**Facts on the heart / by Richard C. Cabot.**


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# FACTS

*on*

# THE HEART

*By*

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ILLUSTRATED

*PHILADELPHIA AND LONDON*

W. B. SAUNDERS COMPANY

1926

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# PREFACE

I cannot too strongly insist that a large part of this book represents not my work but that of others. The necropsies and necropsy records of the 1906 cases on which my conclusions rest, are the work of Drs. James H. Wright and Oscar Richardson. A good many of these necropsies I have witnessed. All of their protocols I have studied in whole or in part. But my task has been merely to collect, arrange, and at times to interpret the results of these pathologists' labors so far as they relate to cardiovascular disease. I have made no attempt to decide for myself any question of gross or microscopic pathology but have gratefully accepted and tried to utilize the decisions of those who did the work.

On the clinical side I have studied and have had abstracted the ward records of 1906 cases decisively diagnosed post-mortem. Starting from the post-mortem diagnoses I have worked back into the clinical records corresponding. A good many of these clinical records depict the sufferings of patients who were under my charge in the wards and in a fair number I wrote the record of the cardiovascular examination and clearly recall the patients. In the great majority of instances however, the patients were cared for by other members of our staff and I am confined to a study and interpretation of other men's work. I am indebted to Drs. Paul D. White, Hugh Cabot, Edward L. Young Jr., William H. Smith, William D. Smith and Maurice Fremont-Smith for some of the printed discussion of cases.

In abstracting the clinical and pathological records many have assisted me. Dr. Harry Taylor, now in China, Dr. J. H. Newburgh of Ann Arbor, Michigan, Dr. Byron C. Darling of New York, Dr. Mary W. Lawson of New London, Conn., Dr. James H. Young, Dr. E. H. Heath and Dr. W. R. Redden of Boston, Mr. John M. Porter of the Harvard Medical School and, above all, my secretaries, Miss Alice G. O'Gorman and Miss Florence Painter, have gathered and arranged material for me.

My own task has been to put together and edit this material, since no one else appeared anxious to undertake so prosaic and time-consuming a task.

I appreciate clearly that the data here gathered together would have been far more valuable in many ways had they represented throughout the observations of one man. But had this been so, one set of preconceptions and leading ideas would have guided and perhaps to some extent marred the observations. There is some advantage as well as a considerable disadvantage in the fact that most of the observations on which I rest were made without any idea of the use to which they might later be put.

To all of those whose names are mentioned above, as well as to the staff of the Massachusetts General Hospital, I acknowledge my gratitude and indebtedness. I trust that this book may to some extent carry out their interests as well as mine.

Especially to Dr. Oscar Richardson I am indebted, not only as one of those by whose daily labors I have profited, but for the time and pains he has taken in re-reading and interpreting pathological records and sometimes in re-examining old specimens to clinch or expunge a diagnosis.

The book differs from all those previously written on heart disease (so far as I know) in basing its conclusion wholly on the study of cases which came in the end to necropsy. The chapter on syphilitic aortitis, for example, was written by abstracting all the necropsies from 1897 to 1919 in which this lesion occurred and then finding what was recorded and remembered about them on the clinical side.

It is true that the pathologist may have overlooked something or mistakenly identified some lesion. But considering the care with which the necropsies were made and the systematic and detailed records that were dictated from them at the time, I believe the percentage of error is small.

If anyone reads and compares the statistical tables, he may be puzzled to see that the total number of cases of a particular disease as given in the early tables differs from the same total given in later tables. This is due to the incompleteness of our records. In relation to age and sex, for instance, we may be able to prepare a table based on complete data from 100% of the necropsies of that disease. Yet in relation to the examination of the heart we may be able to study only 75% of these. Hence the totals of the latter table will be smaller.

Obviously there is very little about treatment in these pages. Most of the little that I know on that subject is to be found in the records or discussions of some of the illustrative cases.

Very few people should even try to read the whole of this book. I should advise most readers to read the opening and the closing chapters, and the summaries at the end of each section, and then to look over as many of the illustrative cases as seem interesting.

RICHARD C. CABOT.

CAMBRIDGE, MASS.,
*February*, 1926.

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# FACTS ON THE HEART

## CHAPTER I

### REVIEW OF 4143 LESIONS IN 1906 CASES OF CARDIAC DISEASE. TABLES OF FREQUENCY

#### I. IN EXAMINING A PATIENT SUPPOSED TO HAVE HEART DISEASE, WHAT ARE THE DIAGNOSTIC EXPECTATIONS WHICH EXPERIENCE JUSTIFIES?

The first point on which this book hopes to shed light is this: when a physician is called to attend a patient who seems to be suffering from cardiovascular disease, what are the important probabilities and the more remote possibilities which he should consider? If we have some basic knowledge about what is rare and what is common, it may give us valuable clues.

The first and in some ways the most important point of all is to know that

Most "Heart Disease" is imaginary.

Those who think or fear that they have heart disease usually turn out on careful examination to be free from it. Nearly 10% of Harvard freshmen in one of the classes examined by Dr. Roger I. Lee and his assistants believed themselves to have a "weak heart" or some more definite heart disease. 14 out of 18 successive patients sent to me by their physicians quite recently for supposed heart disease, had, in my opinion, perfectly sound hearts. The patient with "effort syndrome," complaining of his dyspnea, cardiac pain and palpitation, yet with a normal heart, is not the commonest type. More often it is a patient who has fainted or been dizzy or had a little precordial pain and whose physician has heard a (quite harmless) systolic murmur somewhere in the precordia.

That such cases are more numerous than those with organic heart disease is difficult to prove, though strongly suggested by the experience of anyone who has examined many college students and middle-aged women. But whether or not this pseudo-heart disease, this groundless fear of heart disease, is as common as I believe it to be, it is certainly common enough to make the thought of it an essential

part of our mental furniture whenever we begin the examination of a supposedly cardiovascular case. For the needless sufferings and sacrifices of such patients are often very great. To my knowledge boys have given up their chosen life work, girls their prospect of marriage, business men their favorite projects because of a false diagnosis of heart disease. The fears, disappointments and ailments resulting from such a diagnosis may be enough to render a person's life miserable. And all this one can sweep away by a clear and positive reassurance based on a thorough examination. I know few greater services that a physician can perform, few that give him livelier satisfaction.

**Narrowing the Field.**—Next to this fundamental knowledge of the frequency of supposed but unreal heart disease, one is helped by a *narrowing of the field of possibilities*. That we do not need any longer to consider and search for fatty degeneration of the heart, fatty overgrowth upon the heart, brown atrophy, myocarditis, the senile heart, etc., simplifies our work and makes us more apt to make a correct diagnosis.

**The Commonest Lesions.**—This series of 1906 necropsied cases, (see Table 1) covering all the cardio-vascular material in the 4000 necropsies done at the Massachusetts General Hospital between 1896 and 1919 is enough, I think, to constitute a fair sample and give a fairly accurate picture of heart disease as one may expect to find it in most parts of the United States. Certain districts where syphilis or endemic goiter are especially prevalent will have a higher incidence of syphilitic or thyrotoxic heart disease, but in most parts of the country the proportion of the different types will be found, I think, to be about the same as in New England.*

(a) For example our conclusion that 77% of all heart disease is due to simple hypertrophy and dilatation of the heart (or hypertensive cardiovascular disease) without valve lesions will, I believe, be found to represent approximately the facts in any large series of necropsies made in the United States and Canada.

Further results of this study are:

* Of course no comparisons are of value except on the basis of necropsy statistics, which so far as I know do not exist in this country.

TABLE 1.—SUMMARY OF MATERIAL

A. Total cardiovascular lesions, 4143 in 1906 persons, (out of 4000 necropsied from 1896 to 1919).

B. All cases showing cardiac hypertrophy and dilatation ..... 1209 cases
All cases without hypertrophy and dilatation ..... 697 cases

Total ..... 1906 cases

| | | | |
|---|---|---|---|
| I. Hypertrophy and dilatation.* | with other lesions | 1053 | 1209 cases |
| | alone | 156 | |
| II(a) Arteriosclerosis | with H. & D. | 668 | 1051 cases |
| | without H. & D. | 383 | |
| III(b) Arteriosclerosis with artscl. degen. kidney | with H. & D. | 93 | 142 cases |
| | without H. & D. | 49 | |
| IV. Nephritis,* acute, subacute and chronic | with H. & D. | 275 | 377 cases |
| | without H. & D. | 102 | |
| V. Chronic non-deforming endocarditis | with H. & D. | 148 | 241 cases |
| | without H. & D. | 93 | |
| VI. Acute and subacute endocarditis | with H. & D. | 118 | 180 cases |
| | without H. & D. | 62 | |
| VII. Valve lesions | with H. & D. | 184 | 220 cases with |
| | without H. & D. | 36 | 334 single lesions |
| VIII. Chronic pericarditis | with H. & D. | 89 | 114 cases |
| | without H. & D. | 25 | |
| IX. Acute pericarditis | with H. & D. | 102 | 186 cases |
| | without H. & D. | 84 | |
| X. Myocarditis incl. abscess (15), infarct (26) | with H. & D. | 83 | 99 cases with |
| | without H. & D. | 16 | 130 single lesions |
| XI. Syphilitic aortitis | with H. & D. | 63 | 94 cases with |
| | without H. & D. | 31 | 108 single lesions |
| XII. Pernicious anemia | with H. & D. | 22 | 23 cases |
| | without H. & D. | 1 | |
| XIII. Leukemia | with H. & D. | 9 | 9 cases |
| | without H. & D. | 0 | |
| XIV. Hypoplastic aorta | with H. & D. | 8 | 19 cases |
| | without H. & D. | 11 | |
| XV. Goiter with cardiac results | with H. & D. | 6 | 10 cases |
| | without H. & D. | 4 | |
| XVI. Congenital heart disease | with H. & D. | 6 | 10 cases |
| | | 4 | |

Total no. of lesions ..... 4143

* Nephritis is here included as a local vascular lesion producing in most cases a cardiac effect through the changes in its glomerular capillaries or in its larger vessels.

(b) That rheumatic valvular disease is approximately twice as common as all the varieties of syphilitic aortitis combined, or

(c) five times as common as syphilitic *valvular* disease (aortic regurgitation).

(d) That mitral stenosis, existing alone or combined with other valve lesions, is about three times as common as all other rheumatic valve lesions combined,

(e) That mitral stenosis uncomplicated is about twice as common as any other single valve lesion.

I believe, this picture is what anyone who looks for the facts will find to be true in any part of this country and probably in many parts of the other continents.

## II. THE MATERIAL HERE STUDIED

**The Probable Error in Table 1.**—These figures have been verified and re-verified again and again until they are as nearly correct as I can make them. It is sometimes a matter of judgment, however, whether a slight puckering on a valve shall be considered sufficient to throw it into the group of *deforming* valvular lesions with a genuine obstruction or leak, or whether it shall be placed with the cases of chronic *non-deforming* endocarditis. Valve measurements alone do not settle it. A valve may have considerably less than the average circumference (considering the age, development and musculature of the individual) and yet show no deformity and be in fact normal. In another case a valve may have the normal circumference and yet be so rigid and adherent as to be incompetent. Some balancing of conflicting evidence, then, and some chance for error enter these statistics (and all other statistics) not because of inaccurate counting but because the assignment of each case to a particular pile-to-be-counted involves questions of judgment which may be very difficult. Most of them, however, are simple and so the possible error from this source is not great. For example: most cases of acute endocarditis are easy to recognize at necropsy. It is only in the occasional case that we are uncertain as to the nature of a few minute tags on a valve. In most cases of syphilitic aortitis the lesions can be readily and positively identified by an experienced pathologist. But in a few cases in every hundred he will remain in some doubt as to the nature of the lesions, since the *treponema pallida* is not often to be found within the time that can be devoted to the search for it.

Having myself gone over the evidence on which all these pathological diagnoses were based and having discussed with the pathologist his reasons for the more doubtful divisions, I am confident that the error in any of the figures of this table does not exceed 5%, an error

which will not invalidate any of the conclusions or predictions which are based upon them.

**Comments and Conclusions Drawn from the Whole Material.**—(a) Nearly half of all the individuals necropsied at the Massachusetts General Hospital during approximately a quarter of a century had suffered from some cardio-vascular lesion. Some of these lesions are trifling. For example, some of the cases show only arteriosclerosis (slight or considerable) without any cardiac enlargement or other important lesions. Such cases of slight and *isolated* arteriosclerosis amount to 265 out of our total 1051 cases of arteriosclerosis. So far as we know, these scleroses did no harm to the cardiovascular system and were of no importance to the circulation. Adding to these the 71 cases of nephritis without cardiac hypertrophy, pericarditis or other cardiovascular lesion, 39 cases of chronic non-deforming endocarditis uncomplicated, 10 cases of hypoplastic aorta without cardiac enlargement, and 5 other odds and ends, we reach a total of 390 cases to be subtracted from the gross total of 1906 in order to ascertain the *net total of patients with important cardiovascular lesions;* 1516 *out of* 4000, *or* 38%.

It must be further borne in mind, however, that although the cardiovascular lesion was, I believe, a factor in producing death in all these 1516 persons, it was sometimes a *minor* factor, not the only or the chief one. Some of the 19 cases of acute endocarditis and some of the 12 cases of acute pericarditis without other cardiovascular lesions, (Table 4) were of the *terminal* type, the main underlying cause of the patient's illness and death being a cancer or a diabetes. But the above figures show, somewhat to my surprise, that these restrictions apply to but a few per cent of the cases, so that with less than 5% of error, one can say: *a cardiovascular lesion is an important factor in* 38% *of all deaths.*

(b) In 1846 of the persons here studied, there were 4037 cardiovascular lesions, or more than 2 apiece, on the average. As a matter of fact the 16 lesions in Table 1 occur in 209 different combinations. Only 644 out of the 4037 lesions occurred singly. There were 625 cases with two cardiovascular lesions, one member of the combination being hypertrophy and dilatation in 502 cases and arteriosclerosis in 248.

In 379 cases there were three lesions,* 359 including hypertrophy-and-dilatation as one of the two, and 261 of this 359 including hypertrophy-and-dilatation and arteriosclerosis as two of the three.

* In all the figures of Paragraph (b) a heart with valve lesions counts as one, even when two, three, or four valves were affected.

157 cases had four lesions; all but two of these included hypertrophy-and-dilatation as one member of the combination; 130 of the 155 included hypertrophy-and-dilatation and arteriosclerosis as two of the four in the combination; and 53 of these 130 cases had hypertrophy-and-dilatation, arteriosclerosis, and nephritis as three members of the quartet of lesions.

36 cases had five lesions, of which hypertrophy-and-dilatation was a member of 34; arteriosclerosis and hypertrophy-and-dilatation were members of 28 of these 34; nephritis, arteriosclerosis and hypertrophy-and-dilatation were members of 13 of these 28.

Five cases had six lesions. In four of these the combination of hypertrophy-and-dilatation, arteriosclerotic nephritis (or arteriosclerotic kidney) and chronic endocarditis recurred.

| | |
|---|---|
| Single lesions | 644 |
| Two lesions | 625 |
| Three lesions | 379 |
| Four lesions | 157 |
| Five lesions | 36 |
| Six lesions | 5 |
| | 1846 |

The *chronic infections* (rheumatic and syphilitic) affected 665 persons.

The *acute infections* (endocarditis, pericarditis, myocardial abscess) affected 358 persons.

The *chronic degenerative changes* (arteriosclerosis, hypertrophy and dilatation of the heart, myocarditis) affected 809 persons.

The congenital deformities cut very little figure. There was a hypoplastic aorta in 19 cases; 10 occurring as the only cardiac lesion and 9 with other lesions; the other congenital lesions of the heart occurred *alone* in only four cases (10 cases in all had congenital lesions).* So that the infections, acute and chronic and the degenerative-reparative lesions divide the whole material into three main groups:

| | | |
|---|---|---|
| (1) | †Chronic infection (rheumatism, syphilis) | 665—36% |
| (2) | Acute infection (pericarditis, endocarditis) | 358—19% |
| (3) | Degenerations (hypertrophy and dilatation of the heart, arteriosclerosis, myocarditis) | 809—44.3% |
| (4) | Congenital lesions *occurring alone* (29 in all) | 14—7% |
| | | 1846 100% |

* 29 congenital lesions in all or 1% including 19 of hypoplastic aorta and 5 of harmless valve anomalies.

† I have included here the cases of chronic pericarditis and chronic non-deforming endocarditis.

## III. MANIFEST CARDIOVASCULAR LESIONS

But though all but a few of the lesions here listed doubtless had some effect on the heart's powers, we get another and probably a fairer view of cardiac disease as a whole if we confine ourselves to *manifest lesions*, i.e., those in which there is at necropsy and in life definite evidence of deficient heart power as shown in dropsy of the

TABLE 2.—NO. OF CASES AND PERCENTAGES OF HYPERTROPHY AND DILATATION AND OF CHRONIC PASSIVE CONGESTION IN ALL LESIONS

| | Total cases | H. & D.* | | Single cause | C.P.C.† | | Remarks |
|---|---|---|---|---|---|---|---|
| | | No. | % | | No. | % | |
| Mitral stenosis | 107 | 87 | 84 | 26 | 55 | 53 | |
| Mitral & aortic stenosis | 40 | 37 | 93 | | 25 | 62.5 | |
| Combined valve lesion | 33 | 32 | 97 | | 24 | 71 | |
| Aortic stenosis | 28 | 20 | 71 | | 23 | 82 | |
| Nephritis, chronic | 198 | 180 | 90 | 67 | 107 | 54 | |
| Nephritis, subacute | 66 | 21 | 32 | | | | |
| Nephritis, acute | 113 | 53 | 47 | | | | |
| Nephritis, amyloid | 21 | 3 | 14 | | 1 | | |
| Pericarditis, chronic | 112 | 88 | 78 | 16 | 34 | 30 | (Several with valve or other lesions such as to make C.P.C.) |
| Goitre | 10 | 6 | 60 | 2 | 5 | 50 | |
| Leukemia | 9 | 9 | .... | 7 | 4 | | |
| Pernicious anemia | 23 | 22 | 96 | 12 | 12 | 50 | |
| Endocarditis, acute | 180 | 118 | 66 | 19 | 80 | 56 | (In many, the C.P.C. was accounted for by other lesions.) |
| Pericarditis, acute | 186 | 119 | 64 | 12 | 67 | 36 | (31 accounted for by other lesions, the rest unexplained) |
| Endocarditis chronic non-deforming | 237 | 160 | 67 | 17 | 67 | 28 | |
| Arteriosclerotic kidney | 175 | 93 | 52 | 2 | 6 | .... | |
| Arteriosclerosis | 1051 | 668 | 63 | 248 | | | |
| Myocarditis | 91 | 86 | 94 | 4 | 56 | 63 | (Many had other causes for H. & D. and for C.P.C. present) |
| Syphilitic aortitis | 92 | 57 | 61 | 19 | 26 | 27 | |
| Congenital heart | 10 | 6 | 60. | ... | 1 | 10 | |
| Pure mitral regurgitation | 10 | 7 | 77 | ... | 5 | 50 | |
| Pure aortic regurgitation | 13 | 13 | .... | ... | 11 | 8 | |
| H. & D. with other diseases | | | | | | | |
| (a) With Artscl. | 343 | (67.6%) | | | | | |
| (b) Without Artscl. | 164 | (32.3%) | | | 230 | 45 | |
| Odds and ends | | | | | | | |
| Tb. pericarditis | 2 | 2 | | | | | |
| Pulmonary artscl. | 2 | 2 | | | | | |
| Amyloid degen. kidney | 2 | | | | | | |
| Artscl. degen. kidney | 2 | (without arteriosclerosis elsewhere) | | | | | |
| Hypoplasia of aorta | 19 | 8 | 42 | | | | |

* Hypertrophy-and-dilatation.
† Chronic passive congestion.

serous cavities and in general chronic passive congestion. From Table 2* we can separate out the cases with chronic passive congestion in each group, giving the results shown in Table 3a.

Among the degenerations referred to on page 22. I have bracketed together arteriosclerosis, myocarditis and hypertrophy with dilatation because it seems to me probable that most of them belong to a single group, namely, hypertensive heart disease—with enlarged but finally failing heart, the acute infections being terminal events and the other lesions historical landmarks of no significance in weakening the heart. In some of the cases of extensive myocarditis this may not be true, but the number of these exceptions is small.

TABLE 3a.—CAUSES OF MANIFEST HEART DISEASE WITH CHRONIC PASSIVE CONGESTION

| | | | |
|---|---|---|---|
| Valvular heart disease (including luetic aortic regurgitation) | 169 | | |
| Chronic pericarditis | 34 | | 203 |
| Acute endocarditis (with hypertrophy and dilatation and probably with hypertension) | 80 | | |
| Acute pericarditis (ditto) | 67 | | |
| Chronic non-deforming endocarditis (ditto) | 67 | | |
| Myocarditis (ditto) | 56 | 270 | |
| Hypertrophy and dilatation (with and without nephritis; "hypertensive heart disease") | | 230 | 500 |
| | | | 703* |

* I have omitted here 12 cases of Pernicious Anemia, 4 of Leukemia, and 5 of Thyroid disease, all of which showed passive congestion at necropsy. I am not sure how these should be classified,

TABLE 3b.—PREDOMINATING LESION IN 465 CASES OF MANIFEST HEART DISEASE

| | |
|---|---|
| Cardiac hypertrophy and dilatation | 230 |
| Valvular lesions | 169 |
| Chronic pericarditis | 28 |
| Acute endocarditis | 18 |
| Acute pericarditis | 10 |
| Myocarditis | 7 |
| Goitre | 2 |
| Pernicious anemia | 1 |

* Although for Table 1 we have included all the cases catalogued in the pathological records, in Table 2 and in the subsequent chapters which elaborate the different items of this table we have often analyzed in detail a number smaller than the total as given in Table 1.

Of our 1906 cardiovascular cases, then, only 465, or one-quarter, produced heart failure. The *latency* of many cases of heart disease is apparent in all the types. (See Table 2.) For example, out of 262* cases of valvular disease as found post mortem, only 169—or 63%—had produced heart failure, as shown in chronic passive congestion. Out of 112 cases of chronic pericarditis only 34—or 30%—were manifested in dropsy.

Only in a minority of the cases that I am here referring to did any single lesion, such as rheumatic valvular disease or chronic pericarditis—occur alone. There are 207 different combinations and overlappings in numbers mentioned on p. 22. In view of this difficulty the 465 cases of manifest heart disease just referred to have been divided up according to what seemed to be the dominating lesion when several were present. In the production of death from chronic passive congestion, the following lesions predominated in the proportion expressed by the figures in Table 3a.

Acute endocarditis and acute pericarditis are here put down because they were the only anatomical lesion present after death in the cardiovascular system in association with 18 cases and 10 cases, respectively, dying a congestive death. In these, as in others of the smaller items in this table, there may well have been an earlier hypertension producing the cardiac hypertrophy and leading to the final breakdown.

But dropsy is of course an extreme, sometimes terminal, manifestation of heart disease. Let us take hypertrophy and dilatation as evidence that extra work has in some way been thrown upon the heart. 1209 cases out of 1906, or 63%, showed hypertrophy and dilatation, the causes for which will be discussed in detail later. Here one may merely note that of these 1209 cases of hypertrophy and dilatation only 184 are due to valvular disease and 88 to chronic pericarditis. The vast majority (¾ of all) are cases of "simple cardiac hypertrophy" associated with nephritis and with arteriosclerosis, often combined with acute terminal lesions like acute endocarditis and acute pericarditis, or with historical landmarks like myocarditis and the non-deforming valve scars.

Still, it is hard to get a fair picture of heart disease from these figures, because there are so many combinations and interweavings of the different lesions. When a patient dies with valvular disease, chronic nephritis and chronic pericarditis, with which of these three

* 220 cases of rheumatic valvular disease, plus 42 of syphilitic aortitis with aortic regurgitation.

are we to class it when we come to separate the different types of cardiac trouble? In view of these difficulties, we have separated out the uncomplicated cases in which we can deal with *single lesions*. The results appear in Table 4. Here again simple cardiac hypertrophy, with or without arterio-sclerosis and nephritis, is vastly the commonest lesion. It makes up 469 cases out of 656 or 71% of the whole. Rheumatic valvular disease comes next with 69 cases. These two items together amount to 538—or 82%—of the 654 cases in which these single lesions had produced hypertrophy and dilatation as evidence of interference with the heart's action. Among the other 116 cases serious enough to produce hypertrophy and dilatation chronic pericarditis (16) and syphilitic aortitis (20) are the important items, though they only amount to 2%+ each of the whole. In most of the remaining 78 cases the diagnoses represent terminal or vestigial lesions, the real cause of the trouble being probably the same as that producing the 469 cases of enlarged heart without local lesions to explain it,—i.e., *hypertension, the* great cardiac malady.

TABLE 4.—1145 SINGLE LESIONS (WITH AND WITHOUT H. & D.)

| | Without H. & D. | With H. & D. | Total |
|---|---|---|---|
| Arteriosclerosis | 265 | 248 | 513 |
| Chronic non-deforming endocarditis | 39 | 17 | 56 |
| Acute endocarditis | 35 | 19 | 54 |
| Valve lesions | 17 | 69 | 86 |
| Acute pericarditis | 69 | 12 | 81 |
| Chronic pericarditis | 13 | 16 | 29 |
| Arteriosclerotic degeneration, kidney | 2 | 2 | 4 |
| Luetic aortitis | 19 | 20 | 39 |
| Myocarditis (with abscess and infarct) | 6 | 4 | 10 |
| Hypoplastic aorta | 10 | 5 | 15 |
| Pernicious anemia | 1 | 12 | 13 |
| Leukemia | 0 | 7 | 7 |
| Goitre | 2 | 4 | 6 |
| Nephritis | 11 | 67 | 78 |
| Hypertrophy and dilatation alone | | (154) | 154 |
| | 489 | 656 | 1145 |

## IV. RELATIVE FREQUENCY OF THE DIFFERENT CARDIOVASCULAR LESIONS

Haven Emerson* found that among 4566 patients treated for heart disease in nine New York hospitals† during 1920 or 1921 (twelve months) the diagnoses of the primary condition stood:

* "The Nation's Health," Vol. V, No. 6, June, 1923.

† Bellevue, King's County, Metropolitan, City, Neurological, New York, Presbyterian, St. Luke's, and Mt. Sinai.

| Lesions as diagnosed in New York hospitals | No. of cases | % |
|---|---|---|
| Valvular disease | 1845 or | 40 |
| Myocarditis | 1966 or | 43 |
| Endocarditis | 398 or | 9 |
| Cardiac hypertrophy | 184 | |
| Arrhythmia | 61 | |
| Pericarditis | 46 | 8 |
| Aneurism | 38 | |
| Angina pectoris* | 28 | |
| | 4566 | |

* Not mentioned among "complications," hence this number 28 appears to represent all the cases so diagnosed.

Comparing these with our findings in 465 cases of *manifest heart disease* (i.e., those with chronic passive congestion post-mortem) there were:

| Lesions (Manifest Heart Disease) as found at necropsy | No. of cases | % |
|---|---|---|
| Valvular disease | 169 or | 36 |
| Myocarditis | 7 or | 1 |
| Hypertrophy and dilatation (hypertensive heart disease) | 230 or | 50 |
| Chronic pericarditis | 28 or | 6 |
| Acute endocarditis | 18 or | 4 |
| Acute pericarditis | 10 or | 2 |
| Others | 3 or | 1 |

Taking our cases, manifest and latent (1230), we find the results not very different:

Valvular disease (including 42 syphilitic, 220 rheumatic) ..... 262 or 21%
Myocarditis ..... 89 or 7%
Hypertrophy and dilatation (with chronic nephritis, acute endocarditis, acute pericarditis, chronic pericarditis, chronic non-deforming endocarditis, arteriosclerosis and combinations ..... 879* or 71%

And since myocarditis is not a clinical entity we may say that 77+% of heart disease is non-valvular and 22+% is valvular.

The forty-one aneurisms in our 1906 cases is in marked contrast with Dr. Emerson's thirty-eight aneurisms in 4566 cases, as is also the 186 cases of acute and 114 of chronic pericarditis in our 1906 cases,

* 1209 cases of hypertrophy and dilatation minus Valve disease with H. & D. 184
minus Syphilitic disease with H. & D. 63 } 879.
minus Myocardial disease with H. & D. 83
330

while in the 4566 New York diagnoses of cardiovascular disease, there are only forty-six cases of pericarditis (presumably acute in the vast majority of cases).

Our percentage of acute pericarditis (10%) is ten times as great as theirs, which apparently (like ours) does not include any that were treated only as out-patients and is therefore comparable. Some years ago I found that at the Massachusetts General Hospital we recognized one case in five. In New York they apparently recognize about one in ten. Of aneurisms we recognized twenty-five out of forty-one or 60%. In New York if the same percentage held, our forty-one aneurisms in 1230 cases, (3%) would correspond to 137 aneurisms of which 38 were recognized, or 28%. Myocarditis was recognized six times as often as it was present, valvular disease twice as often.

### V. VARIATIONS OF NECROPSY DIAGNOSES AT DIFFERENT PERIODS

In Table 5 I have separated the post-mortem diagnoses into four groups, taking each thousand necropsies by itself so that we can examine the variations in our recognition of the different lesions during the four periods, 1896–1903, 1903–1907, 1907–1912, and 1912–1919.

The figures show a falling off in the cases of rheumatic valvular disease after 1907. This is not to be accounted for by any known change in the types of patients received at the hospital or coming to necropsy. The same pathologists have applied the same diagnostic criteria to this part of the necropsied material throughout the whole period from 1896 to 1919. Therefore these statistics show that *in this group of cases* there has been a diminution in the amount of fatal rheumatic heart disease during these twenty-three years. So far as I know, this group of cases may be taken as a fair sample, at any rate for the north-eastern part of the United States.

It is notable that the diagnoses of aortic stenosis have *not* decreased in number, while those of mitral stenosis, and of mitral- and aortic stenosis combined *have* fallen from forty-six (in the period 1903–1907) to twenty, or less than half, in the period from 1912–1919.

On the other hand there is no falling off in the amount of pericarditis which is ordinarily included with the valvular stenoses as a "rheumatic" lesion.

The sudden drop in the number of cases of "non-deforming endocarditis" in the last thousand necropsies is I believe of no significance and represents merely a change in the pathologist's views as to the term "endocarditis." Slight thickenings of the

valves, especially in elderly persons, are now not called endocarditis or noted in our anatomical diagnosis.

The apparent increase in fibrous myocarditis is also not significant, and represents probably a slightly altered terminology.

The diagnoses of syphilitic aortitis in the cases belonging to the earlier years were made by re-examining old specimens after the pathologists had come to recognize this lesion. Naturally the cases thus identified are relatively few, so that the increase in the later years means an increase not in the *cases* of syphilitic aortitis but in the diagnoses of this lesion.

TABLE 5.—NECROPSY DIAGNOSES IN EACH 1000 AUTOPSIES TAKEN CONSECUTIVELY FROM 1896 TO 1916

| Disease | 1896 to 1903 | 1903 to 1907 | 1907 to 1912 | 1912 to 1919 | Total |
|---|---|---|---|---|---|
| | Necropsy number 1–1000 | Necropsy number 1001–2000 | Necropsy number 2001–3000 | Necropsy number 3001–4000 | |
| Pure mitral disease (stenosis) | 27 | 33 | 30 | 16 | 107 |
| Mitral and aortic (stenosis) | 11 | 13 | 10 | 4 | 38* |
| Combined valve lesions | 7 | 9 | 8 | 9 | 33 |
| Aortic stenosis | 5 | 3 | 11 | 9 | 28 |
| Acute pericarditis | 43 | 31 | 64 | 48 | 186 |
| Chronic pericarditis | 25 | 26 | 30 | 31 | 112 |
| Acute endocarditis | 48 | 33 | 52 | 47 | 180 |
| Chronic non-deforming endocarditis | 49 | 54 | 100 | 34 | 237 |
| Fibrous myocarditis | 9 | 19 | 33 | 30 | 91 |
| Myocardial abscess | 5 | 3 | 3 | 4 | 15 |
| Myocardial infarct | 3 | 2 | 6 | 8 | 19† |
| Pure aortic regurgitation (rheumatic) | 1 | 5 | 3 | 4 | 13 |
| Pure mitral regurgitation (rheumatic) | 3 | 0 | 2 | 3 | 8 |
| Syphilitic aortitis | 13 | 15 | 28 | 36 | 92 |

* In the text two more cases from another series have been used.

† In the text one more case has been used from another series.

## CHAPTER II

### RHEUMATIC HEART DISEASE. FATAL CHOREA

**The Material Used.**—In all cases I have started from the demonstrated *post-mortem* lesions and worked back to the clinical findings corresponding. I have paid no special attention to the clinical diagnosis in deciding which cases to include under each group, but have based all classifications and statistics on the measurements and descriptions of the necropsy records.

A valve has been considered obstructed or stenosed when its circumference was found to be markedly diminished either by the rigid end results of chronic endocarditis, by soft vegetations, or by the combination of both. When soft vegetations were not in such a position or of such a size as to produce obstruction the case has been studied not as a valve lesion but in the chapter on "Acute and Subacute Endocarditis."

Hypertrophy and dilatation of the heart has been judged and classified (like the valve lesions) wholly on the *post-mortem* data,—the weight of the heart, the thickness of its walls, and the size of its cavities all considered in relation to the size of the individual. A big man has a big heart. The same cardiac dimensions if existing in a small woman or in a child would be considered evidence of hypertrophy.

**Terms.**—1. When the mitral valve alone was found at necropsy to be diseased, or when the disease of other valves was so slight as apparently not to interfere with the opening and closing of the valve, I have called the cases "*pure mitral*" or simply "*mitral.*"

2. When there are important deformities both in the mitral and in the aortic valves I have called the cases "*mitral-and-aortic,*" emphasizing by the two hyphens the fact that I am not here referring to two separate groups of cases but to one set of combined lesions.

The cases grouped as "pure mitral" number 107 and those called "mitral-and-aortic" come to 40.

3. Isolated disease of the aortic valve is called "*pure aortic,*"—28 cases.

4. The cases in which the *mitral-aortic-and-tricuspid* valves were all involved, (23 examples), those with lesions in *mitral-aortic-tri-*

*cuspid-and-pulmonary* valves (2 cases), the combination of *mitral-and tricuspid* disease (6 cases), and of *pulmonary-and-tricuspid* (2 cases) have here been all lumped together under the term "OTHER COMBINATIONS."

In all the cases classed under these four groups—208 in total—*stenosis* was the lesion anatomically demonstrated *post-mortem*. This is all that the necropsy can demonstrate. But from the half-open position in which the diseased valves were rigidly fixed, it seems clear that there must have been some regurgitation as well as some obstruction of the blood stream. A valve fixed half open will not open fully, but neither will it shut.

A *valve lesion*, single or combined, means therefore in the following section, a deformity believed to have caused *stenosis and regurgitation* although it may sometimes be called simply a "lesion," a "disease" or a "stenosis."

The rare cases of *regurgitation* alone, due to rheumatic endocarditis will be discussed separately. They make up only 23 cases in all.

By the terms "rheumatism" and "rheumatic" I mean a disease believed to originate as a general septicemia (point of entrance unknown), which may "settle" in the *joints* ("rheumatic fever") in the *subcutaneous tissues* ("rheumatic nodules") or in the *tonsils* (tonsillitis), may cause *brain* symptoms (Sydenham's chorea) or may be implanted in the *pericardium*, in the *myocardium* or on the *heart valves* without any other localization. Ordinarily when the disease affects the heart it has had some earlier localization (in the joints, tonsils, or brain, sometimes in the subcutaneous tissues also). But it does not seem strictly true to say that the rheumatic joint trouble *causes* the endocarditis which so often follows. Both are presumably caused by the same organism—the same septicemia—which may localize its activities first in the heart and later in the joints, or in the reverse order. In case the heart lesion appeared first (as is not very rarely the case) we should not say that the heart trouble *caused* the joint trouble. It is as incorrect, I think, to say that acute polyarthritis causes endocarditis. Both manifest a single cause.

The proof of a septicemia is not yet to be obtained.

No "arthritis" has been considered "rheumatic" in the analysis of these cases unless it involved many joints, produced marked prostration, ran a short course and left the joints sound in the end. The chronic, stiffening, apparently afebrile lesions often called rheumatism, especially in elderly people, have received no consideration here.

**The Type of Endocarditis Found Post-mortem in 208 Cases.**—I include here under "Rheumatic Valvular Heart Disease" as the subject of this chapter (see Table 6), any valvular obstruction, whether produced by a soft, acute, vegetative process (ten cases in 208), by the chronic deforming end-result of an earlier acute endocarditis, or by the combination of both.*

I have included these acute cases in part because in 53 out of 208 cases, or 25%, the chronic process was overlaid at the time of death by a fresh endocarditis. In some cases (see Table 48) this acute septic process, involving in its effects the myocardium and all the organs of the body, may be assumed to be the immediate cause of death as, in earlier stages of the disease, similar acute exacerbations are believed to produce many of the decompensations formerly explained by mechanical overstrains.†

*Valvular lesions* (such as mitral obstruction) are here conceived to be produced (a) (as is usually the case) by the hardened, shrunken, adherent end-results of an acute endocarditis; (b) by acute soft inflammatory vegetations: (c) by a combination of both.

TABLE 6.—TYPE OF ENDOCARDITIS

| | Mitral | M-A | M-A-T | M-T | Aortic | P-T | 4 valves | Total |
|---|---|---|---|---|---|---|---|---|
| Chronic | 75 | 23 | 17 | 3 | 23 | 2 | 2 | 145 |
| Acute | 8 | 2 | 0 | 0 | 0 | 0 | 0 | 10 |
| Chronic and acute | 24 | 15 | 6 | 3 | 5 | 0 | 0 | 53 |
| | 107 | 40 | 23 | 6 | 28 | 2 | 2 | 208 |

Since there appear to be all possible transitions between the various types, and all sorts of combinations of them, it seems best to include them all under a single heading, "*rheumatic valvular heart disease.*" Very probably there are differences, at the extremes quite marked differences, both in the type of micro-organism and in the clinical course of acute cases as contrasted with chronic. But the

* It is often impossible to decide the chronicity of the so-called verrucose endocarditis. Sometimes the character of the minute masses, such as softness, reddening, and fragility, is apparent, and the process is of course acute. But frequently this is wanting, and they appear as firm, grayish to gray-red, warty granules apparently organized, and in these instances are chronic in nature, but of more recent origin than the usual accompanying, underlying chronic endocarditis. (Note by Dr. Oscar Richardson.)

† See Breaks in compensation from Endocarditis: Charles Hunter Dunn, Jour. Amer. Med. Assoc., Feb. 9, 1907.

border-line cases are so many that I doubt the wisdom of separating them.

## MITRAL STENOSIS, PURE AND COMPLICATED

**Age of Onset.**—In Table 7 I have endeavored to estimate the age at which the cardiac lesion started in this group of necropsied cases. This estimate is made by assuming that the first attack of rheumatism or chorea known to the individual or his parents coincided with the beginning of the endocarditis; or, when no history of rheumatism or chorea could be obtained, by dating the cardiac disease from the onset of cardiac symptoms. Obviously this last measure is misleading since there is in most if not in all cases a latent or symptomless period, often lasting for years. The frequency with which (especially in women) we find mitral stenosis in the course of a routine physical examination undertaken by reason of complaints unconnected with the heart proves the existence of this latent period in many cases, perhaps in all except the acute vegetative types of endocarditis.

Taking the figures of Table 7 as they stand, with all the errors inherent in them, it appears in 42 out of 63 cases, or 67% that the heart disease began before the thirtieth year ("pure-mitral" cases). When the mitral stenosis was complicated by stenosis at other valves the disease began before the thirtieth year in 77%. Taking all the well-studied cases in one group, 73% of them began before thirty.

Compare these figures with the estimates in 239 similar ambulant cases examined by me. There 104 out of 130 well studied "pure-mitral" cases or 80% began apparently before the 30th year. Of the mitral-and-aortic cases 85% began before the 30th year. Although the diagnosis in these cases was not verified by necropsy the histories are probably better than those in the necropsied series. So that I am inclined to believe that the true percentage of mitral cases beginning before 30 is nearer 82% than 73%.

*Sex in Relation to the Age of Onset.*—In males the disease apparently begins earlier than in females if the indication of my rather scanty figures is borne out by larger statistics. Thus in Table 7, 90% of the males with mitral-and-aortic disease and 70% of the "pure mitral" cases began before the thirtieth year, while in women only 60% of the pure mitrals and 71% of the mitral-and-aortic cases began before thirty. Combining all groups we find:

| | |
|---|---|
| Men | 77% before 30 |
| Women | 68% before 30 |

In the ambulant cases examined by me in life but not at necropsy, 88% of the men and only 76% of the women were under the 30th year. This correspondence with the figures in necropsied cases is striking.

TABLE 7.—NECROPSIES. AGE OF ONSET

| Ages | Mitral | | Mitral and Aortic | | Other comb. | | Total | | |
|---|---|---|---|---|---|---|---|---|---|
| | m | f | m | f | m | f | m | f | both |
| 0– 9 | 2 | 4 | 6 | 1 | 1 | 4 | 9 | 9 | 18 |
| 10–19 | 10 | 10 | 9 | 3 | 3 | 4 | 22 | 17 | 39 |
| 20–29 | 10 | 6 | 4 | 2 | 4 | 4 | 18 | 12 | 30 |
| 30–39 | 7 | 9 | 2 | 1 | 3 | 4 | 12 | 14 | 26 |
| 40–49 | 2 | 2 | .. | .. | .. | 1 | 2 | 3 | 5 |
| 50–59 | .. | 1 | .. | .. | 1 | .. | 1 | 1 | 2 |
| | 31 | 32 | 21 | 7 | 12 | 17 | 64 | 56 | 120 |

TABLE 8.—LIVING CASES. AGE OF ONSET IN 239 CASES OF RHEUMATIC HEART DISEASE, EXAMINED IN OUT-PATIENT DEPARTMENT FOR THE YEARS 1919, '20, '21, '22

| Age | Mitral | | Mitral and Aortic | | Total | | |
|---|---|---|---|---|---|---|---|
| | Male | Female | Male | Female | Male | Female | Both |
| 0– 9 | 13 | 15 | 14 | 3 | 27 | 18 | 45 |
| 10–19 | 16 | 27 | 23 | 14 | 39 | 41 | 80 |
| 20–29 | 12 | 21 | 10 | 4 | 22 | 25 | 47 |
| 30–39 | 3 | 15 | 3 | 2 | 6 | 17 | 23 |
| 40–49 | 1 | 4 | 1 | 2 | 2 | 6 | 8 |
| 50–59 | 1 | 1 | 3 | 0 | 4 | 1 | 5 |
| 60–69 | 0 | 1 | 0 | 1 | 0 | 2 | 2 |
| Unknown..... | 6 | 14 | 5 | 4 | 11 | 18 | 29 |
| | 52 | 98 | 59 | 30 | 111 | 128 | 239 |

## DURATION OF LIFE

Out of 36 pure-mitral necropsied cases, 27 (or over ⅔) are estimated as having lived ten years or more since the onset of the disease. Six lived apparently 25–35 years with their stenoses.

The term of life in the mitral-and-aortic cases is strikingly shorter. In more than half the cases, the disease appeared to have killed the patient in one year. The average duration in the 31 cases is three

years as compared with 15 years in the pure mitral cases. Only one lived more than 10 years.

In the other "combined lesions" (with fewer cases involving the aortic valve) the prognosis seems to be better than in the mitral-and-aortic cases, though worse than in the pure-mitral cases. Nine out of 28 lived 10 years or more, only two of 28 succumbed within the first year and the average duration of the disease was 10 years.

Average duration in pure-mitral cases = 15 years
Average duration in mitral-and-aortic = 3 years
Average duration in combined lesions = 10 years

In the living cases the figures are of course not compared. (See Table 10.)

TABLE 9.—ESTIMATED DURATION OF LIFE IN 95 CASES OF RHEUMATIC HEART DISEASE ENDING MOSTLY BY PASSIVE CONGESTION

| Years | Mitral | Mitral and aortic | Other comb. | Total |
|---|---|---|---|---|
| 1 | 1 | 16 | 2 | 18 |
| 2 | 1 | 7 | 3 | 10 |
| 3 | 0 | 1 | 1 | 2 |
| 4 | 1 | | | |
| 5 | 3 | .. | 3 | 5 |
| 5–10 | .. | 5 | .. | 5 |
| 6 | 3 | 0 | 1 | 4 |
| 7 | 0 | 0 | 5 | 5 |
| 8 | 0 | 0 | 1 | 1 |
| 9 | 0 | 0 | 1 | 1 |
| 10 | 2 | 0 | 1 | 3 |
| 11 | 1 | 0 | 0 | 1 |
| 12 | 2 | 0 | 2 | 3 |
| 14 | 3 | 0 | 1 | 3 |
| 15 | 4 | 0 | 0 | 4 |
| 16 | 2 | 0 | 0 | 2 |
| 17 | 0 | 0 | 2 | 2 |
| 18 | 1 | 0 | 0 | 1 |
| 20 | 2 | 0 | 0 | 2 |
| 21 | 1 | 0 | 1 | 2 |
| 22 | 2 | 0 | 1 | 3 |
| 23 | 0 | 1 | 0 | 1 |
| 24 | 1 | 0 | 0 | 0 |
| 25 | 1 | 0 | 0 | 1 |
| 26 | 1 | 0 | 1 | 1 |
| 31 | 1 | 0 | 0 | 1 |
| 34 | 1 | 0 | 0 | 1 |
| 35 | 2 | 0 | 0 | 2 |
| 41 | 0 | 0 | 1 | 1 |
| "Many" | 0 | 1 | 1 | 2 |
| | 36 | 31 | 28 | 95 |

The lesion dated supposedly from a rheumatic attack in seventy-two, and from a choreic attack in six cases. The estimated duration of the lesion was as in Table 9.

This group of cases with a *known* cause appears to occur in younger people than the group in which no cause is clear (see below).

The average age of the thirty-six patients with "pure-mitral" disease is thirty-five years, and the average duration of the lesion 15 years. Most of these cases died from passive congestion ("congestive death").

TABLE 10.—LIVING CASES. ESTIMATED DURATION OF LIFE IN 239 CASES OF RHEUMATIC HEART DISEASE EXAMINED BY ME IN THE OUT-PATIENT DEPARTMENT IN THE YEARS 1919, 1920, 1921, 1922

| Years | Mitral | | Mitral and aortic | | Total |
|---|---|---|---|---|---|
| | M | F | M | F | |
| Less than 1 year | 2 | 8 | 3 | 2 | 15 |
| 1 year | 6 | 12 | 8 | 1 | 26 |
| 2 years | 2 | 7 | 8 | 1 | 18 |
| 3 years | 9 | 7 | 5 | 3 | 24 |
| 4 years | 2 | 8 | 2 | 1 | 13 |
| 5 years | 9 | 11 | 2 | 0 | 22 |
| 6 years | 3 | 3 | 3 | 3 | 12 |
| 7 years | 4 | 4 | 4 | 1 | 13 |
| 8 years | 1 | 3 | 1 | 1 | 6 |
| 9 years | 1 | 5 | 3 | 1 | 0 |
| 10 years | 0 | 2 | 1 | 3 | 6 |
| 11 years | 1 | 3 | 1 | 1 | 6 |
| 12 years | 0 | 1 | 3 | 1 | 5 |
| 13 years | 0 | 0 | 2 | 1 | 3 |
| 14 years | 0 | 1 | 0 | 1 | 2 |
| 15 years | 0 | 1 | 0 | 1 | 2 |
| 16 years | 3 | 3 | 2 | 0 | 8 |
| 17 years | 0 | 0 | 1 | 0 | 1 |
| 18 years | 1 | 1 | 0 | 1 | 3 |
| 19 years | 0 | 1 | 1 | 1 | 3 |
| 20 years | 1 | 2 | 2 | 0 | 5 |
| 25 years | 1 | 0 | 1 | 1 | 3 |
| 29 years | 0 | 1 | 1 | 0 | 2 |
| 34 years | 1 | 1 | 0 | 1 | 3 |
| 38 years | 0 | 1 | 0 | 0 | 1 |
| Many years | 1 | 4 | 0 | 0 | 5 |
| Unknown | 4 | 8 | 5 | 4 | 21 |
| | 52 | 98 | 59 | 30 | 239 |

**Age and Length of Life in Rheumatic Heart Disease.**—1. *Age.* The figures in Table 7 show that rheumatic valvular heart disease (defined as above) occurs at every age, lasts till old age, and is compatible with long life. In the 10% "pure-mitral" cases, 33 or 30% were over fifty years of age at the time of death; while of the 40 mitral-and-aortic cases 14 or 35% had survived the fiftieth year. From the point of view of prognosis in youthful cardiac sufferers these facts are somewhat reassuring, though the majority (in "pure-mitral" cases 70%) die before fifty.

The worst decade for mitral cases is that between thirty and forty, while the danger-point for mitral-and-aortic is fifty to sixty.

Males with "pure-mitral" stenosis and combinations of this with pulmonary and tricuspid disease die usually before forty; only one-fourth survive the fiftieth year. But if they have both mitral and aortic stenosis they are slightly older at death than with mitral alone.

Aortic stenosis is the longest-lived lesion of the rheumatic group. 76% of the cases lived beyond forty, and almost 50% survived the fiftieth year. *It is a lesion of elderly men,* but when women have it (which is rare) they live even longer than men.

2. *Duration of Disease.*—(a) The actual number of years occupied in each case by the disease before death and the causes of death (congestive or non-congestive) are shown in Table 9 and Table 48. The first of these points—the duration of the disease—can be settled only with an approximation to accuracy. We may surmise that the disease began at the date of the first (often the only) attack of acute rheumatism or chorea, a date which can be fixed with approximate accuracy in 86 of our cases. Assuming this to be true we can say that 35, *or* 40%, *of our cases lived ten years or more.*

(b) The table of *ages at the time of death* in necropsied cases (Table 11) represents the only certain data that I have. But it is of some interest to compare with this the ages of 315 cases diagnosed as rheumatic heart disease by me in out-patient practice or in private practice (see Table 12). These ages given at the time the patient was first seen, are to be taken as representing a point *near* the time at which symptoms first appeared. In this group (see Table 12) the decade from ten to nineteen is the most important one. It is striking that only 17 or 5% fall within the first decade of life while 87 or nearly 27% come for advice within the next ten years, and 173 or 55% before the thirtieth year.

TABLE 11.—AGE (AT THE TIME OF DEATH) AND SEX

| | Mitral | | M-A | | M-A-T | | M-T | | Aortic | | P-T | | 4 valves | | Total | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | m | f | m | f | m | f | m | f | m | f | m | f | m | f | m | f | |
| 0– 9 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 2 |
| 10–19 | 3 | 2 | 5 | 3 | 1 | 1 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 12 | 6 | 18 |
| 20–29 | 12 | 9 | 3 | 0 | 2 | 4 | 2 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 19 | 15 | 34 |
| 30–39 | 12 | 12 | 6 | 2 | 3 | 8 | 1 | 1 | 2 | 0 | 1 | 0 | 0 | 1 | 25 | 24 | 49 |
| 40–49 | 8 | 15 | 6 | 1 | 0 | 2 | 0 | 0 | 7 | 0 | 0 | 0 | 0 | 0 | 21 | 18 | 39 |
| 50–59 | 8 | 8 | 4 | 6 | 0 | 1 | 0 | 0 | 5 | 2 | 0 | 0 | 1 | 0 | 18 | 17 | 35 |
| 60–69 | 2 | 11 | 1 | 1 | 1 | 0 | 0 | 1 | 6 | 1 | 0 | 0 | 0 | 0 | 10 | 14 | 24 |
| 70–79 | 0 | 4 | 2 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 3 | 4 | 7 |
| | 45 | 62 | 27 | 13 | 7 | 16 | 3 | 3 | 25 | 3 | 1 | 1 | 1 | 1 | 109 | 99 | 208 |
| Males..... | 45 | | 27 | | 7 | | 3 | | 25 | | 1 | | 1 | | 109 | | |
| Females.... | 62 | | 13 | | 16 | | 3 | | 3 | | 1 | | 1 | | 99 | | |
| | 107 | | 40 | | 23 | | 6 | | 28 | | 2 | | 2 | | 208 | | |

Returning now to the necropsied cases (Table 11) we see that the end of the disease comes most often in the years from thirty to thirty-nine. Its earliest symptoms (if the ante-mortem diagnoses of Table 8 are correct) come twenty years earlier. I refrain from concluding that the average length of the disease is twenty years. The two sets of figures cannot legitimately be combined in this way. Moreover, only in the death-age figures are we certain of the diagnosis.

3. *Sex.*—It has usually been stated that mitral stenosis (pure or combined with other lesions) is commoner in women than in men. In this series of 180 proved mitral cases ("pure" and combined) there were 84 males to 96 females. But as in our necropsies from all sources males make up 65.4% these figures certainly show a considerable excess of females. Women do not come into our wards as often as men do. The difference in the number of autopsied cases may well be accounted for by women's greater unwillingness to trust themselves to a hospital, or by their relatives' greater unwillingness to grant necropsy in the case of a female than in that of a male.*

*The actual number of deaths from heart disease of all sorts is about the same in women and in men. See for example the Statistical Bulletin of the Metropolitan Life Insurance Co., June, 1925 (Vol. VI, No. 6).

TABLE 12.—LIVING PATIENTS

Age and Sex

| | Age at first consultation | | | | | | | | Total |
|---|---|---|---|---|---|---|---|---|---|
| | Mitral private cases | | Mitral | | M + A | | Aortic | | |
| | Males | Females | Males | Females | Males | Females | Males | Females | |
| 0– 9 | 1 | 2 | 4 | 4 | 5 | 1 | 0 | 00 | 17 |
| 10–19 | 6 | 3 | 19 | 27 | 21 | 7 | 3 | 1 | 87 |
| 20–29 | 2 | 14 | 7 | 19 | 17 | 10 | 0 | 0 | 69 |
| 30–39 | 1 | 11 | 11 | 30 | 8 | 7 | 1 | 0 | 68 |
| 40–49 | 7 | 8 | 6 | 10 | 2 | 2 | 2 | 1 | 38 |
| 50–59 | 0 | 3 | 3 | 7 | 4 | 2 | 1 | 0 | 21 |
| 60–69 | 0 | 2 | 1 | 1 | 2 | 1 | 0 | 0 | 7 |
| 70–79 | 0 | (7)* | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| | 17 | 50 | 51 | 99 | 59 | 30 | 7 | 2 | 308 |
| | * Age unknown | | | | | | | | 7 |
| | | | | | | | | | 315 |
| | Sex | | | | | | | | |
| Males... | 17 | | 51 | | 59 | | 7 | | 134 |
| Females. | 50 | | 99 | | 30 | | 2 | | 181 |
| | 67 | | 150 | | 89 | | 9 | | 315 |

Comparing these figures with those obtained in out-patient work and in private practice, we find that (see Table 12) in the living out-patient and private cases there is a considerable excess of females, 181 to 134 males, even though among *all* the patients coming *for all causes* to our Out-Patient Department there are but fifty-eight women to every seventy-one men. Hence this excess of women with mitral stenosis is greater than the figures show.

Taking now the Out-Patient Department figures of heart disease, chorea, and rheumatism in children up to sixteen, we find that 59% of those treated as "rheumatic cardiac disease" in 1921 and 61% of those treated either as rheumatic heart disease, as chorea, or as rheumatism, were girls.

TABLE 13.—SOURCE OF CASES

| | Mitral | M + A | Aortic | Total |
|---|---|---|---|---|
| (a) Out-Patient Department (for years 1919, 1920, 21, 22) | 150 | 89 | 9 | 248 |
| (b) Private practice | 67 | .. | .. | 67 |
| | 217 | 89 | 9 | 315 |

In the "pure-mitral" cases of all ages the females are more than twice as numerous (149 to 68) so long as we confine our attention to living (and therefore doubtful) cases. In the necropsied cases of "pure mitral" disease there were sixty-two females to forty-five males. But as already said this gives too slight an impression of the excess of females, since our necropsied series is not a fair sample of the sick adult population, but contains a disproportionate number of males. Putting together all our facts:

(a) as to necropsied cases in a "selected" material,

(b) as to all living cases examined by me,

(c) as to all out-patient cases of rheumatic heart disease,

(d) as to all out-patient cases of rheumatic heart disease in children,

(e) as to all out-patient cases of chorea,

(f) as to all out-patient cases of acute rheumatism,

we may conclude that the female sex is a strong factor in determining the incidence of mitral disease, whether "pure" or combined.

I have already called attention to the fact that in the mitral-and-aortic lesions the males predominate, while in the pure aortic lesions females are the exception.

**Relation of Sex to Duration of Diseases.**—When we study the relation of sex to the age at which the fatal illness occurred, we see that *males break down earlier than females*. Thus in Table 11 it appears that in females only 40% (24 of 62 cases) of the pure mitral and 59% (22 of 37 cases) of those associated with other valve lesions died before their fortieth year. Combining both groups we find that in only 47% of the 96 (not counting the cases of "pure" aortic stenosis) *female* cases did death occur before forty. In other words more than half of them (53%) get by the fourth decade.

Of the 84 men (not counting aortic stenosis) only 40% (33) lived past forty. 60% (27 of 45 cases) of the "pure mitral" and 76% (39 of 51) of the remaining cases in men died under forty.

We may surmise that this is due to the greater muscular activity called for by the men's occupations, but for reasons stated elsewhere I doubt whether this explanation holds. I cannot explain the women's greater tenacity of life.

## ETIOLOGY IN MITRAL STENOSIS

**The Relation of These Lesions to Rheumatism, Chorea, and Tonsillitis.**—In 92 cases the records on this point are fairly complete. They show that rheumatic arthritis was a factor in 73 or 78%; that chorea was remembered in only 7 cases or 6%, while tonsillitis seemed important in 7 cases or 7%. In the remaining 7 cases none of these evidences of infection was recorded.

The surprising thing to me in these cases is the small number of choreas. Perhaps chorea, even when fatal (see page 312), causes a type of endocarditis not apt to lead to serious deformities of the valves. I find that only in 7% of cases seen clinically did definite mitral stenosis follow chorea in patients without joint symptoms. Thus in 116 living cases of "pure mitral" disease, and in 73 mitral-and-aortic disease there were but eight cases with a history of *chorea without rheumatism*, that is, only 7%, which corresponds closely with 6% in the necropsied cases.

As to the importance of *tonsillitis* in these cases it is difficult to say anything definite, on account of the frequency of this infection in all sorts of people. Our records do not clearly separate the cases in which tonsillitis has been frequent and severe from those in which it was merely remembered to have occurred once or more in the individual's lifetime. Our records show only 9 of 174 autopsied cases and 31 of 170 clinical cases with records of tonsillitis. This is no more than would be found in any series of healthy people.

**Number of Attacks of Rheumatism (or Chorea).**—An attempt was made to ascertain *how many attacks* of rheumatism or chorea had occurred during the lifetime of these patients. The memory of such events is notoriously faulty and the following table is therefore of very little value.

TABLE 14.—NUMBER OF ATTACKS OF RHEUMATISM (OR CHOREA) IN MITRAL DISEASE "PURE" OR OCCURRING WITH OTHER LESIONS

| No. of attacks | Mitral | | Mitral & Aortic | | Other combinations | | Total |
|---|---|---|---|---|---|---|---|
| | Males | Females | Males | Females | Males | Females | |
| 1 | Rheum. 13<br>Chorea 2 | Rheum. 9<br>Chorea 4 | Rheum. 5<br>0 | Rheum. 1<br>0 | Rheum. 11<br>Chorea 1 | Rheum. 7<br>Chorea 3 | Rheum. 46<br>Chorea 10 |
| 2 | Rheum. 2 | Rheum. 4 | Rheum. 7 | Rheum. 1 | Rheum. 2 | Rheum. 1<br>Chorea 1 | Rheum. 17<br>Chorea 1 |
| 3 | Rheum. 2 | Rheum. 2 | Rheum. 1 | Rheum. 1 | 0 | 0 | Rheum. 6 |
| 4 | 0 | 0 | Rheum. 1 | 0 | 0 | 0 | Rheum. 1 |
| 5 | Rheum. 1 | 0 | 0 | 0 | 0 | 0 | Rheum. 1 |
| 6 | 0 | Rheum. 1 | 0 | 0 | 0 | 0 | Rheum. 1 |
| 7 | 0 | 0 | Rheum. 1 | Rheum. 1 | 0 | 0 | Rheum. 2 |

**Previous Infections Other than Rheumatism.**—In Table 15 are listed the infections remembered by the individual, or in the case of children by the parents, as having occurred in the course of his previous life. It seems to me apparent from these figures that no infection other than that manifested in rheumatism and chorea can as yet be definitely correlated as an etiological factor in mitral disease. The histories of any 173 people with the same age and sex distribution would contain, I think, at least this number of pneumonias, scarlet fevers, etc. Doubtless some cases of *scarlet fever complicated by streptococcus infection* may be the precursors of some types of heart disease. But for scarlet fever alone these figures show no connection to endocarditis.

*Pneumonia* in fatal cases shows a fair percentage of *acute* endocarditis, especially when the streptococcus is present (as in the "influenza" pneumonias). But on the whole when we consider the great frequency of pneumonia and the relative rarity of mitral disease at all ages, it is difficult to put down pneumonia as an etiological factor, even a minor one.

Equally groundless in these cases is the theory that syphilis or arteriosclerosis can produce the peculiar lesions of mitral stenosis. Lesions of syphilis recognizable post-mortem were not found in a single case, and the percentage of *known* syphilitic infections was notably below the figures to be found by questioning hospital patients in general. (See R. C. Cabot: Relative Frequency of the Different Diseases Prevalent in Boston and its Vicinity; Shattuck Lecture, Mass. Medical Society, June 13, 1911.) A calcified column ("bone") was found at the root of the mitral valve in three cases and seemed

like a lesion which might be explained by arteriosclerosis. But these calcified masses caused no stenosis. (See page 204.)

TABLE 15.—PREVIOUS INFECTIONS OTHER THAN RHEUMATISM IN 173 CASES OF RHEUMATIC HEART DISEASE

| | Mitral | M + A | Other comb. | Total |
|---|---|---|---|---|
| Scarlet fever | 10 | 4 | 3 | 17 |
| Measles | 10 | 3 | 1 | 14 |
| Pneumonia | 7 | 3 | 6 | 16 |
| Typhoid | 3 | 2 | 2 | 6 |
| Smallpox | 3 | 0 | 0 | 3 |
| Diphtheria | 2 | 3 | 2 | 6 |
| Pertussis | 2 | 3 | 0 | 5 |
| Mumps | 4 | 1 | 0 | 5 |
| Syphilis | 4 | 0 | 0 | 4 |
| Gonorrhea | 3 | 0 | 0 | 3 |
| "Grippe" | 0 | 4 | 0 | 4 |

TABLE 16.—TUBERCULOSIS, ACTIVE OR OBSOLETE

| | Mitral | Mitral and Aortic | Other comb. | Total |
|---|---|---|---|---|
| Pulmonary, active | 0 | 0 | 1 | 1 |
| Adrenal, active | 0 | 1 | 0 | 1 |
| Miliary | 2 | 0 | 0 | 2 |
| Obsolete | 16 | 1 | 9 | 23 |
| Absent | 88 | 38 | 23 | 142 |
| Doubtful | 1 | 0 | 0 | 1 |
| | 107 | 40 | 33 | 180 |

The idea fathered by some French writers that tuberculosis is an etiological factor in rheumatic heart disease finds no support in these figures. Moreover there is but one case in which an *active* phthisis has accompanied or complicated rheumatic heart disease. There were two miliary cases, one affecting the pleura and liver, one of "pseudo-miliary" tuberculosis in the kidneys, and one of active tuberculosis of the right adrenal.

Even obsolete tuberculosis, though carefully searched for in 176 necropsies, was found in but twenty-three cases.

## SYMPTOMS OF MITRAL STENOSIS

TABLE 17.—109 NECROPSIED CASES. TABULATION OF THE EARLIEST CARDIAC SYMPTOM

| Earliest Symptom | Mitral No. of cases | Durations and No. of cases for each | M + A No. of cases | Durations and No. of cases for each | Other lesions No. cases | Durations and No. of cases for each | Total |
|---|---|---|---|---|---|---|---|
| Dyspnea | 34 | 24 yrs. . . . . 1<br>10–15 yrs. . . . . 10<br>1–9 yrs. . . . . 12<br>1–6 mos. . . . . 11 | 13 | 23 yrs. . . . . 1<br>5–10 yrs. . . . . 5<br>2 mos.–2 yrs. . . . . 3<br>2 wks.–2 mos. . . . . 4 | 10 | 17 yrs. . . . . 1<br>15 yrs. . . . . 1<br>6–7 yrs. . . . . 3<br>4–12 mos. . . . . 4<br>? . . . . . 1 | 57 |
| Dyspnea and palpitation | 12 | 1–5 yrs. . . . . 5<br>1–5 mos. . . . . 7 | 6 | 5 mos.–2 yrs. . . . . 6 | 5 | under 1 yr. . . . . 2<br>3 yrs. . . . . 2<br>8 yrs. . . . . 1 | 23 |
| Dyspnea, palpitation, cough | 0 | . . . . . | 1 | 3 yrs. . . . . 1 | 0 | . . . . . | 1 |
| Dyspnea and edema | 2 | 1–4 wks. . . . . 2 | 0 | . . . . . | 0 | . . . . . | 2 |
| Dyspnea, orthopnea, cough | 0 | . . . . . | 1 | 2 weeks. . . . . 1 | 0 | . . . . . | 1 |
| Dyspnea, precordial pain | 1 | 6 days. . . . . 1 | 0 | . . . . . | 0 | . . . . . | 1 |
| Dyspnea, palpitation, edema and cough | 1 | 3 yrs. . . . . 1 | 0 | . . . . . | 0 | . . . . . | 1 |
| Dyspnea and cough | 3 | 2 yrs. . . . . 1<br>2–6 wks. . . . . 2 | 1 | 2 mos. . . . . 1 | 1 | 6 mos. . . . . 1 | 5 |
| Dyspnea and orthopnea | 0 | . . . . . | 1 | 7 mos. . . . . 1 | 2 | 2 mos. . . . . 1<br>6 mos. . . . . 1 | 3 |
| Orthopnea | 1 | 7 wks. . . . . 1 | 1 | 3 mos. . . . . 1 | 0 | . . . . . | 2 |
| Palpitation | 1 | 5 yrs. . . . . 1 | 0 | . . . . . | 1 | 13 yrs. . . . . 1 | 2 |
| Attacks of palpitation and tachycardia | 1 | ? | 0 | . . . . . | 0 | . . . . . | 1 |
| Precordial pain, palpitation | 1 | ? days | 0 | . . . . . | 0 | . . . . . | 1 |
| Palpitation and dizziness | 0 | . . . . . | 1 | ? yrs. . . . . 1 | 0 | . . . . . | 1 |
| Cough | 2 | ? | 1 | 4 wks. . . . . 1 | 0 | . . . . . | 3 |
| Cough and edema | 1 | 3 mos. . . . . 1 | 0 | . . . . . | 0 | . . . . . | 1 |
| Edema | 1 | 2 wks. . . . . 1 | 1 | 1 wk. | 2 | 2 wks. . . . . 1<br>2 mos. . . . . 1 | 4 |
| | | | | | | | 109 cases |

**Earliest Symptoms (Necropsied Cases).**—Table 17 shows as might be expected that *dyspnea*, either alone or in company, was present at the onset of symptoms in fifty-three out of sixty-one cases of "pure-mitral" stenosis in which this point was carefully inquired into. In thirty-four cases, or over half, dyspnea appeared alone, was the earliest symptom, and often persisted for years before any other symptoms were noticed.

In the mitral-and-aortic cases, twenty-three out of twenty-seven had dyspnea alone or in combination as their first symptom, while in the other combined stenoses 18 of 21 began in the same way. *In the total group of* 109 *carefully questioned patients*, 94 *began with dyspnea, alone or accompanied.*

**Earliest Symptoms (Out-Patient Cases).**—In the living cases (Table 18) *dyspnea* was the first complaint in sixty-one out of eighty-five carefully questioned "pure mitral" cases, and in thirty out of forty-seven "mitral-and-aortic" cases, i.e. in about three-quarters of all cases.

*Palpitation* was noticed first in ten out of thirty-three "pure mitral" cases and in ten out of twenty-six "mitral and aortic," i.e. in one-third of the total cases.

*Cough* was the earliest symptom in eleven out of twenty-seven "pure mitral" and five out of twelve "mitral and aortic," i.e. in about one-third.

*Precordial pain* of some sort was the earliest complaint in eight out of twenty-five "pure mitral" and sixteen out of twenty-seven "mitral and aortic." The mitral-and-aortic cases show a higher percent of pain,—over one-half.

*Edema* was the first point noticed in four "mitral" cases out of twenty-one, and five "mitral and aortic" out of fifteen.

TABLE 18.—TABULATION OF THE EARLIEST CARDIAC SYMPTOM

Living Cases

| Symptom | Mitral | M + A | Total |
|---|---|---|---|
| Dyspnea | 61 | 30 | 91 |
| Precordial pain | 8 | 16 | 24 |
| Palpitation | 10 | 10 | 20 |
| Cough | 11 | 5 | 16 |
| Edema | 4 | 5 | 9 |
| | | | 160 cases |

Taking all symptoms noted, whether early or late, their relative frequency in this whole group of living mitral cases. with or without aortic disease, was as follows:

| | |
|---|---|
| Dyspnea | 132 |
| Palpitation | 59 |
| Precordial pain | 52 |
| Edema | 36 |

In contrast with the necropsied cases most of which were studied only at the end of life, these relatively early cases (though their diagnosis is never quite certain) seem to show a much greater frequency of *pain* as a complaint (especially in the "mitral-and-aortic" cases). The relation of pain to dyspnea is as fifty-two to one hundred and thirty-two in the early (living) cases, while it is only as twenty-one to one hundred and twelve in the late (necropsied) cases.

Palpitation and dyspnea were noted together at the onset of twenty-three cases or about one-fourth.

In most respects these tables show no notable differences of symptomatology as between the "pure-mitral" and the mitral-and-aortic cases.

The percentage of patients who noticed edema at *some* time before hospitalization is 12 out of 40 in the mitral-and-aortic series, as compared with 43 out of 107 in the mitral series and 17 out of 33 in the other combinations,—i.e. the mitral and aortic cases showed early edema in 30%, the pure mitral in 40%, and the other combinations in 51%.

**Symptoms in General.**—The figures of Table 19 refer to the patient's complaints before entering the hospital, not to the terminal sufferings. The familiar symptoms of cardiac patients here occur in very much their familiar order. It is perhaps significant that in the mitral-and-aortic cases *dyspnea* is relatively more frequent (75%) than in the mitral (50%). The "combined" stenoses give the highest of all (82%).

The fact that only 55 out of 107 "pure-mitral" patients are known to have had any dyspnea at all before entering the hospital is surprising, indeed almost incredible. But if it represents the truth it goes toward explaining why the diagnosis was altogether missed in about the same percentage of cases. Examining this similarity more closely we find that *in 40 out of 52 of the undiagnosed cases dyspnea was absent.* Under these conditions the cardiac histories may well have been less searchingly taken.

*Palpitation; Early or Never.*—Another notable fact is that palpitation (often, perhaps usually, equivalent to auricular fibrillation) appears either early or not at all. It was noticed in only eighteen "pure mitral" cases *but in 12 of these it was noticed as early as any symptom.* In seven out of eleven mitral-and-aortic cases the same "early or never" tendency appears, while in the remaining "combined stenoses" palpitation came early or never in five of the 9 recorded cases. So that (combining the three groups) we may say: In mitral disease (with or without other valve lesions) palpitation is recorded only 38 times among 180 cases, but in 24 of these 38 or 63%, this palpitation appeared either as the earliest symptom or with the earliest symptoms. [These data do not agree with those of the Out-Patient Department series wherein only one-third of those with palpitation manifested it as a "first complaint."]

These figures as to palpitation may be compared with our rather scanty data on the pulse as recorded during the patients' stay in the hospital. Of the mitral cases 41 out of 67 cases with definitely recorded pulse observations showed arrhythmia. In all but three of these the arrhythmia is so described as to make it highly probable that auricular fibrillation was present. But it is certainly surprising that only 41 out of 67, or approximately 61%, should have manifested this obvious change in any such degree as to get it into our hospital records. Arrhythmia has been, throughout the years covered by this series of cases, one of the points most clearly expected and looked for by physicians and internes in sick patients of all types, especially, but not only, in those believed to have heart disease. It is not usually difficult to recognize the existence of *some* arrhythmia when one feels the pulse as often as is the routine practice of physicians, internes and nurses at the Massachusetts General Hospital.

In the "mitral-and-aortic" cases, 24 had regular and 10 irregular pulses while in the other "combined stenoses" 15 were regular and 12 irregular. Combining the three groups, we have 63 of 127 or almost exactly 50% with irregular pulses. The greater part of these I believe (knowing the conditions of observation, and the characteristics of internes and nurses at the Massachusetts General Hospital) would not have been recorded as "irregular" unless an *absolute* arrhythmia or auricular fibrillation were present. Hence I think we may conclude that 50% of arrhythmias (mostly fibrillations) is not far from the correct figure in hospitalized cases of mitral disease. But we must realize that a large number of these cases were hospitalized not for heart disease but for all sorts of medical and surgical

TABLE 19.—FREQUENCY OF CERTAIN SYMPTOMS IN RHEUMATIC HEART DISEASE
Necropsied Cases

| Symptoms | Mitral | M + A | Comb. lesions | Total |
|---|---|---|---|---|
| Dyspnea | 55 | 30 | 27 | 112 |
| Edema | 43 | 12 | 17 | 72 |
| Cough | 42 | 11 | 16 | 69 |
| Palpitation | 18 | 11 | 9 | 38 |
| Vomiting | 13 | 4 | 4 | 21 |
| Precordial pain or distress | 8 | 2 | 11 | 21 |
| Orthopnea | 7 | 6 | 4 | 17 |
| Abdominal pain | 8 | 0 | 1 | 9 |
| Indigestion | 5 | 1 | 2 | 8 |
| Nausea | 4 | 0 | 0 | 4 |
| Chills | 3 | 1 | 1 | 5 |
| Gas and colic | 2 | 0 | 0 | 2 |
| Headache | 2 | 0 | 0 | 2 |
| Vertigo | 1 | 0 | 1 | 2 |
| Malaise | 1 | 0 | 0 | 1 |
| Pain in legs | 0 | 0 | 1 | 1 |

TABLE 20.—FREQUENCY OF CERTAIN SYMPTOMS IN RHEUMATIC HEART DISEASE
Living Cases

| Symptoms | Mitral | M + A | Total |
|---|---|---|---|
| Dyspnea | 85 | 47 | 132 |
| Palpitation | 33 | 26 | 59 |
| Precordial pain | 25 | 27 | 52 |
| Cough | 27 | 12 | 39 |
| Edema | 21 | 15 | 36 |

conditions, the heart disease appearing at necropsy as a surprise. In a group of cases hospitalized for heart disease itself the figures would be quite different.

This 50% represents to a considerable extent the terminal events. It may be that the discrepancy between the 63 arrhythmias noticed in the hospital and the meagre 38 observed by the patients as "palpitation" represents a late onset of fibrillation. It seems more probable, however, that minor degrees of arrhythmia, including many fibrillations, passed unnoticed by the patients.

Aside from this *early or never* feature in the patient's *awareness of his heart-beats*, the other symptoms listed in Table 19 call for

little comment. The abdominal pain may represent congestion of the liver. The precordial distress only once presented definitely anginoid characteristics. In this case, No. 810, a patient forty-two years of age, without history or physical evidence of syphilis, had for three months intense squeezing and boring pain in the left chest and shoulder, sometimes lasting thirty-six hours at a time. The coronaries, aorta and myocardium showed nothing remarkable at necropsy.

The absence of bowel disturbances is only what experience leads us to expect in all types of cardiac disease. It is nevertheless remarkable that the definite and obvious congestion which necropsy almost always shows in these cases should have no correlation with diarrhea, with obstinate constipation, or with abdominal pain.

If orthopnea really occured but 17 times in 149 patients previous to their hospitalization it is remarkable. I strongly suspect, however, that better history taking would have changed this figure.

## PHYSICAL SIGNS OF MITRAL STENOSIS

**General Physical Examination.**—1. *Nutrition* was good or fair in 65% of the uncomplicated cases. (See Table 21.) When emaciation was present it was usually explainable by complications such as malignant endocarditis, diabetes, abscess of the lung, cancer of the pancreas, gullet, uterus, breast, gall-bladder, etc. The remaining 21% of poorly nourished patients had presumably suffered unusually from loss of sleep, pain, or nausea. Sepsis may well have been a factor also.

TABLE 21.—STATE OF NUTRITION

| | Mitral | M + A | Comb. | Total |
|---|---|---|---|---|
| Obese | 7 | 1 | 1 | 9 |
| Good | 49 | 17 | 19 | 85 |
| Fair | 14 | 4 | 4 | 22 |
| Poor | 23 | 13 | 3 | 39 |
| Emaciated | 5 | 3 | 2 | 10 |
| Not given | 9 | 2 | 4 | 15 |
| | 107 | 40 | 33 | 180 |

2. *Jaundice.*—(Table 35.) Only 6 cases showed icterus. Of these one showed cancer of the pancreas, one gallstones, one cirrhosis

of the liver. Of the remaining 3 cases, one was of the ulcerative type of endocarditis, one had a terminal infection. Thus it appears that in only one case out of 180 are we forced to fall back on hepatic stasis and so on the mitral stenosis itself as an explanation of jaundice.

3. *Cyanosis.*—In slight degree this was noted in 10 out of 180 cases, in marked degree in 62, or 72 in all. On the other hand its definite absence was noted only in 37 out of 180 cases, so that in fact it may be the rule though in our records it was usually not noted.

4. *Leucocytes.*—(Table 40.) We have studied 123 cases. As a rule only one count, made at the patient's entrance to the hospital, is recorded, though in febrile cases counts are usually recorded once a week or more. Out of these 123 cases, 102 were free from non-cardiac complications such as might cause leucocytosis. Among these 102 there were 28 or about one-fourth which showed a leucocytosis of over 15,000.

Correlating these 28 cases with the conditions found post-mortem, we find that leucocytosis, like fever (see p. 67) was generally associated with embolism and thrombosis, with complicating pneumonia, meningitis, acute endocarditis, pericarditis, or general sepsis. Occasionally no cause could be found. Sometimes acute endocarditis is present without leucocytosis (nine of twenty-eight cases in *this* group).

**Cardiac Enlargement.**—The data are collated in the following table:

TABLE 22.—CARDIAC ENLARGEMENT

| Necropsied cases | Mitral | M + A | Comb. lesions | Total |
|---|---|---|---|---|
| Heart not enlarged transversely | 22 | 3 | 6 | 31 |
| Heart slightly enlarged transversely | 13 | 10 | 4 | 27 |
| Heart markedly enlarged transversely | 18 | 10 | 7 | 35 |
| Heart greatly enlarged transversely | 18 | 10 | 5 | 33 |
| Heart enlarged considerably downward | 16 | 0 | 9 | 25 |
| Total | 87 | 33 | 31 | 151 |
| **Living cases** | | | | |
| Heart showing "definite enlargement" | 127 | 81 | .. | 208 |
| Heart showing no "definite enlargement" | 23 | 8 | .. | 31 |
| Total | 150 | 89 | .. | 239 |

"Slightly enlarged" means that the apex was not over 2 cm. beyond its normal position.

"Markedly enlarged" means that the apex was 2 cm. to 4 cm. beyond its normal position.

"Greatly enlarged" means that the apex was not over 4 cm. beyond its normal position.

"Enlarged considerably downward" means that the apex was in the sixth, seventh, or eighth interspace.

*Comment.*— 1. The mitral-and-aortic cases and the "other combinations" fail to show, as might be expected, any greater frequency and degree of enlargement than the "pure mitral" cases. Thus 60+ % (20 of 33) of the mitral-and-aortic cases were "markedly" or "greatly" enlarged, as compared with 60−% (52 of 87) of the mitral cases. But at the other end of the scale, 25% of the mitral cases manifested no cardiac enlargement in life, while only 9% of the mitral-and-aortic cases, and 18% of the "other combinations" failed to show hypertrophy.

2. In both groups the apparent enlargement was mostly transverse. Only 16 of 87 mitral cases, four of forty-two mitral-and-aortic cases, and 9 of the combined lesions showed an apex impulse in the sixth, seventh, or eighth space. It is contrary to the usual teaching that the cases complicated by aortic disease should show no more lowering of the apex impulse than the "pure mitral" cases. In fact there seemed to be actually less downward enlargement (5%) in the "aortic-and-mitral" than in the "pure mitral" cases (18%). But these figures are probably not significant.

Comparing these estimates with the facts found regarding enlargement post-mortem, we find the following. Clinically the heart appeared to be of normal size in 25% of the necropsied *pure mitral cases* and 15% of the living cases. At necropsy there were 16% of normal sized hearts.

In the mitral-and-aortic group, 9% of the necropsied cases appeared to have hearts of normal size, while in the living cases the % appeared to be also 9. At necropsy there were 7% in this group. In the 30 combined lesions 6 (or 20%) seemed to show hearts of normal size, while at necropsy there was but one out of thirty-three that was free from hypertrophy, though considerably dilated.

At the other extreme the comparisons are as follows:

PURE MITRAL

Markedly + (2–4 cm. out) ante-mortem = 20% of 87 cases
500–600 grams post-mortem = 22% of 104 cases

Greatly + (over 4 cm. out) ante-mortem = 20% of 87 cases
600 grams or more post-mortem = 6% of 104 cases

MITRAL + AORTIC

2–4 cm. out in 30% ante-mortem
500–600 grams in 25% post-mortem

Over 4 cm. out in 30% ante-mortem
Over 600 grams in 22% post-mortem

On the whole the agreement is fairly close.

TABLE 23.—WEIGHT OF HEART AT NECROPSY

| | Mitral | M + A | Comb. lesions | Total |
|---|---|---|---|---|
| 200–300 grams | 15 | 1 | 0 | 16 |
| 300–400 grams | 32 | 10 | 6 | 48 |
| 400–500 grams | 27 | 9 | 10 | 46 |
| 500–600 grams | 23 | 10 | 8 | 41 |
| 600–750 grams | 7 | 6 | 4 | 17 |
| Over 750 grams | 0 | 3 | 3 | 6 |
| "Large" | 0 | 1 | 0 | 1 |
| Not recorded | 3 | 0 | 2 | 5 |
| | 107 | 40 | 33 | 180 |

TABLE 24.—HYPERTROPHY AND DILATATION AT NECROPSY

| | Mitral | M + A | Comb. lesions | Total |
|---|---|---|---|---|
| Hypertrophy and dilatation present | 83 | 32 | 29 | 144 |
| Hypertrophy and dilatation absent | 18 | 3 | 0 | 21 |
| Hypertrophy present | 6 | 5 | 3 | 14 |
| Dilatation present | 0 | 0 | 1 | 1 |
| | 107 | 40 | 33 | 180 |

TABLE 25.—DILATATION OF CAVITIES

| | Mitral | M + A | Comb. lesions | Total |
|---|---|---|---|---|
| All cavities greatly dilated | 4 | 2 | 0 | 6 |
| All cavities greatly dilated, right more than left | 2 | 0 | 2 | 4 |
| All cavities dilated | 33 | 18 | 13 | 64 |
| All cavities dilated, right more than left | 3 | 3 | 4 | 10 |
| All cavities dilated, left more than right | 1 | 2 | 1 | 4 |
| All cavities dilated, auricles more than ventricles | 3 | 0 | 3 | 6 |
| All cavities dilated esp. right auricle & left ventricle | 1 | 0 | 0 | 1 |
| All cavities dilated esp. left auricle | 3 | 0 | 0 | 3 |
| All cavities dilated esp. right auricle | 2 | 0 | 1 | 3 |
| All cavities dilated esp. right ventricle | 2 | 0 | 0 | 2 |
| All cavities dilated esp. left ventricle | 1 | 3 | 0 | 4 |
| | 55 | 28 | 24 | 107 |
| Right auricle and ventricle dilated | 5 | 0 | 0 | 5 |
| Right auricle and ventricle and left auricle | 7 | 0 | 0 | 7 |
| Right auricle and ventricle and left ventricle | 1 | 0 | 1 | 2 |
| Right and left auricle dilated | 7 | 1 | 1 | 9 |
| Right and left auricle and right ventricle | 3 | 2 | 1 | 6 |
| Right and left auricle and left ventricle | 1 | 0 | 1 | 2 |
| Right and left ventricle dilated | 2 | 0 | 0 | 2 |
| Right and left ventricle and left auricle | 1 | 0 | 0 | 1 |
| Right ventricle and left auricle | 2 | 0 | 0 | 2 |
| Left auricle and ventricle dilated | 1 | 1 | 0 | 2 |
| Left auricle and ventricle and right auricle | 1 | 0 | 0 | 1 |
| Left auricle and ventricle and right ventricle | 0 | 0 | 1 | 1 |
| Left auricle alone | 2 | 0 | 0 | 2 |
| Right auricle alone | 0 | 0 | 1 | 1 |
| Left ventricle | 1 | 0 | 0 | 1 |
| No dilatation present | 18 | 8 | 3 | 29 |
| | 107 | 40 | 33 | 180 |

1660—(Mitral-aortic-and-tricuspid) extreme dilatation of auricles, right ventricle also dilated, left contracted.

303—(Pulmonary Stenosis) three cavities dilated, especially right auricle.

3945—(M-A-Stenosis) three cavities dilated, especially left ventricle.

*Heart Weights.*—What can we conclude from Table 23?

The obvious and outstanding fact is that mitral stenosis produces less hypertrophy when alone than when it is linked with similar

disease in other valves. There are fifteen small hearts in 107 cases of pure mitral disease, or 14%, and there are forty-seven (including these fifteen) which are not much enlarged, 43%. In the mitral-and-aortic cases only 27% fall in the corresponding group and in "other combinations" only 18%.

At the other end of the scale there are very few "pure mitral" hearts weighing over 600 grams, (6%) while in the mitral-and-aortic cases 25% of the hearts weighed 600 grams or more.

Yet on the whole the differences are less striking than might have been expected. Only at the extremes is there a striking contrast.

Table 24 however makes these differences considerably sharper. If we take into account the relation of heart-weight to the size of the whole body, as is done in deciding whether a heart is hypertrophied or not, it appears that in eighteen of 107 pure mitral cases hypertrophy was absent. This is 16%, or nearly one-sixth of all cases. In the mitral-and-aortic cases the corresponding percentage is but 7%, and in the "other combinations" zero.

*General Dilatation (in 35 to 40%).*—As regards the dilatation of the several heart chambers Table 25 shows on the whole *surprisingly little preponderance of right-sided change.* In seventy cases out of 180 rheumatic valve lesions of all sorts, the dilatation affects all the chambers alike. This is 39%. In the pure mitral cases the percentage of general dilatation is thirty-five—not much less.

*Preponderance of Dilatation* (Table 25).—When there is any difference between the two sides of the heart in respect to the amount of dilatation, the right side is more dilated in 40 cases out of 180, the left side in 21. Taking the pure mitral cases alone, the corresponding figures in 104 cases are twenty-five and twelve.

*Lop-sided Dilatations.—When the pathologist finds no general dilatation but certain chambers picked out by the disease, "pure mitral" is usually the lesion.* Thus there are thirty-four of these lop-sided dilatations in the 107 "pure mitral" cases, only four in forty mitral-and-aortic cases, and six in the thirty-three "other combinations."

The favorite specialization of the dilatation in "pure mitral" cases is in the right auricle and ventricle with the left auricle (ten cases), next in the right and left auricle (7 cases), and next in the right auricle and ventricle (five cases). *In all these groups the left auricle is included thirty-two times,* so that it seems to be the chamber hardest hit. But the right auricle (thirty-one times) is a *very* close second.

When all cavities were dilated the right side was more stretched than the left in ten cases and the left auricle more than the right in only five.

On the whole, then, these observations, accurate and reliable as we have every reason to believe, show that *in the "pure mitral" cases there is very little preponderance of right-sided dilatation*, while in the "combined valve lesions" right sided preponderance was more striking than in the pure mitral cases.

TABLE 26.—CARDIAC MURMURS

| Necropsied cases | Mitral | M + A | Combined lesions | Total |
|---|---|---|---|---|
| No record | 1 | ..... | ..... | 1 |
| No murmurs | 28 or 27% | 12 or 30% | 2 or 6% | 42 or 24% |
| Systolic murmur only | 30 or 28% | 7 or 18% | 7 or 21% | 44 or 24% |
| Presystolic murmur only | 10 or 8% | 1 or 2% | 1 or 2% | 12 or 6% |
| Presystolic + Systolic | 26 or 24% | 3 or 7% | 12 or 36% | 41 or 22% |
| Systolic + Diastolic | 10 or 9% | 10 or 25% | 6 or 18% | 26 or 15% |
| Three murmurs | 2 or 2% | 6 or 15% | 5 or 15% | 13 or 7% |
| Diastolic only | ..... | 1 or 2% | ..... | 1 or ½% |
| Total | 107 | 40 | 33 | 180 |

TABLE 27

| Living cases | Mitral | M + A | Private (mitral) | Total |
|---|---|---|---|---|
| No murmurs | 6 or 4% | 3 or 3% | 5 or 7% | 14 or 4% |
| Systolic murmur only | 10 or 7% | 11 or 12% | 5 or 7% | 26 or 9% |
| Presystolic murmur only | 51 or 34% | 10 or 11% | 25 or 37% | 86 or 28% |
| Diastolic murmur only | 6 or 4% | 17 or 19% | 0 or 0% | 23 or 7% |
| Presystolic & diastolic | 28 or 19% | 35 or 39% | 13 or 19% | 76 or 24% |
| Presystolic & systolic | 27 or 18% | 0 or 0% | 17 or 25% | 44 or 14% |
| Diastolic & systolic | 2 or 1% | 3 or 3% | 1 or 1% | 6 or 2% |
| 3 murmurs | 20 or 13% | 10 or 11% | 1 or 1% | 31 or 10% |
| Total | 150 | 89 | 67 | 306 |

## Comments.

**Cardiac Murmurs.**—1. The 45% of necropsied pure mitral cases in which presystolic or diastolic murmurs were heard at or near the apex corresponds closely with the 48.5% in which the diagnosis of mitral stenosis was made during life.

2. We know that in many cases of mitral stenosis a systolic murmur at the apex is all that we hear *unless* we stir up the heart and bring out the presystolic or diastolic by exertion (or by a drug like amyl nitrite), or listen carefully for a mid-diastolic in the recumbent position. This we should be unlikely to do unless we suspected the presence of the stenosis. In many cases of this series (surgical cases) there was no such suspicion. But such suspicions should be aroused (as apparently they were not in fifty-four of these cases):

(a) By any history of rheumatism or chorea.

(b) By an unusually sharp, slapping or thumping quality of the first heart sound.

(c) By the "explosive" quality of the systolic murmur.

(d) By the absence or feebleness of the second heart sound at the apex and in the second right interspace.

(e) By the doubling of the second sound in the pulmonary area, at the apex, and along the left sternal border (third heart sound).

(f) By the age of the patient (youth).

(g) By the evidence of auricular fibrillation, decompensation, or passive congestion not otherwise explained.

In the necropsied *mitral-and-aortic cases* a presystolic or diastolic murmur was heard in 21 out of forty (or 52%) so that our diagnostic average was higher than in the pure mitral cases. As a rule we heard in this group either a systolic murmur alone (18%) or no murmur at all (30%).

In the "other combinations," though their total is but 33 cases, we did rather better, recognizing a presystolic or diastolic murmur in 71% of them, a systolic only in 21% and no murmur in the remainder. But of the multiplicity of the lesions we had no idea. In the whole group of 180 cases our success in recognizing some mitral lesion appears to be expressed as 50%.

To explain the 50% of failure we may say that:

In the present group of cases there were many elderly and moribund patients without rheumatic or choreic histories, patients entering the hospital for surgical or other non-cardiac complaints.

**Heart Sounds.**—Moreover our records show that the suggestive changes in the heart sounds (see *b*, *d*, and *e* above) often most helpful (in that they lead us to search for a presystolic or diastolic murmur, at rest in the recumbent position or after physical or pharmacal stimulation of the heart) were not often looked for at all in this group of cases. Thus there is no record whatever of the quality of the apical first sound in 74 out of 107 "pure mitral" necropsied

cases, and in 30 out of forty mitral-and-aortic cases. In other words this suggestive sign of mitral stenosis ("sharp first sound") was ignored in ¾ of the examinations.

Doubling of the 2nd sound at the or along the left sternal margin (third heart sound) was apparently not looked for.

TABLE 28.—HEART SOUNDS (NECROPSIED CASES)

| Necropsies | Mitral | M + A | Other comb. | Total |
|---|---|---|---|---|
| First sound sharp | 11 | 5 | 4 | 20 |
| First sound accentuated | 12 | 3 | 7 | 22 |
| First sound loud | 4 | .. | .. | 4 |
| First sound thumping | 1 | .. | .. | 1 |
| First sound rough | 1 | .. | .. | 1 |
| First sound not clear | 1 | .. | .. | 1 |
| First sound valvular | 1 | .. | .. | 1 |
| First sound impure | .. | 1 | .. | 1 |
| First sound not heard | .. | 1 | 4 | 5 |
| First sound snapping | 1 | .. | 1 | 2 |
| First sound negative or no record | 75 | 30 | 17 | 122 |
| | 107 | 40 | 33 | 180 |
| 2d at apex present | 2 | 3 | 1 | 6 |
| 2d at apex absent | 4 | 7 | 5 | 16 |
| 2d at apex diminished | 2 | 0 | 2 | 4 |
| 2d replaced by murmur | 1 | 1 | 0 | 2 |
| 2d not recorded | 98 | 29 | 25 | 152 |
| | 107 | 40 | 33 | 180 |

A comparison with the living cases (though of course their diagnosis is never certain) shows what, with expectant attention, one might have heard in most of the cases coming to necropsy. Thus in 134—or 89% out of 150 pure-mitral cases—examined in life by me, the first sound at the apex was "sharp" or accentuated, and in only fourteen was it normal, diminished or absent.

In the mitral-and-aortic living cases this sign was not so frequently present. 63 out of 89 (70%) showed an accented first sound, while in twenty-one it was normal or absent.

Accentuation of the pulmonic 2nd sound was present in 60 out of 150.

TABLE 29.—HEART SOUNDS (LIVING PATIENTS)

| Heart sounds | Mitral | M + A | Total |
|---|---|---|---|
| First sound sharp or First sound accentuated | 181 | 63 | 244 |
| First sound doubled | 3 | 4 | 7 |
| First sound diminished | 2 | 0 | 2 |
| First sound absent | 5 | 14 | 19 |
| First sound normal | 7 | 7 | 14 |
| First sound not recorded | 19 | 1 | 20 |
| | 217 | 89 | 306 |
| 2d at apex present | 48 | 39 | 87 |
| 2d at apex absent | 50 | 41 | 91 |
| 2d at apex doubled | 49 | 3 | 52 |
| 2d at apex diminished | 4 | 5 | 9 |
| 2d at apex not recorded | 66 | 1 | 67 |
| | 217 | 89 | 306 |
| Aortic 2d doubled | 44 | 1 | 45 |
| Aortic 2d accentuated | 16 | 29 | 45 |
| Aortic 2d diminished | 17 | 8 | 25 |
| Aortic 2d absent | 3 | 34 | 37 |
| Aortic 2d normal | 12 | 17 | 29 |
| Aortic 2d questionable | 11 | 0 | 11 |
| Aortic 2d not recorded | 114 | 0 | 114 |
| | 217 | 89 | 306 |
| Pulmonic 2d accentuated | 95 | 35 | 130 |
| Pulmonic 2d accentuated and doubled | 3 | 2 | 5 |
| Pulmonic 2d doubled | 111 | 1 | 112 |
| Pulmonic 2d diminished | 0 | 5 | 5 |
| Pulmonic 2d absent | 2 | 30 | 32 |
| Pulmonic 2d normal | 6 | 16 | 22 |
| | 217 | 89 | 306 |
| Pulmonic 2d greater than Aortic 2d | 103 | 33 | 136 |
| Aortic 2d greater than P2 | 17 | 21 | 38 |
| Pulmonic 2d equal to A2 | 17 | 12 | 29 |
| Pulmonic 2d and A2 absent | 0 | 23 | 23 |
| Pulmonic 2d not recorded | 80 | 0 | 80 |
| | 217 | 89 | 306 |

Doubling of the 2nd sound to the left of the sternum—or a third heart sound—(I cannot always distinguish these if, indeed, they are different) was present in 83 out of 150 living patients.

Diminution or absence of the 2nd sound at the apex I noted in 36 out of 150 cases.

Our records in the necropsied cases are woefully meagre as regards that very helpful confirmatory sign, *doubling of the second sound (or a third heart sound)* to the left of the sternum. In only *six* of 104 necropsied mitral cases, and four of the 40 mitral-and-aortic cases was any attention paid to this sign. Yet as already stated I believe that this is a much more common and valuable sign than accentuation of the pulmonic sound. It was noted in 55% of the living pure mitral cases.

The same is true of a third helpful sign, the *weakening or obliteration of the apical second sound*. It was noted as weak or absent in only thirteen cases out of 149, and present in five cases. In the remaining 131 cases, 87%, the whole matter was ignored. I believe from clinical experience that this sign grows more and more frequent as the case progresses in severity, i.e. as decompensation increases. In the moderately or entirely compensated pure-mitral cases seen by me in private or out-patient practice, 36 out of 150 or 26% showed this change.

*Accentuation of the pulmonic second sound* is a sign which I have found of little value in the diagnosis of mitral disease or indeed of any other disease.

Of the living cases in only 40%—a small minority—could I find any distinct accentuation. The frequency and value of this sign has certainly been much exaggerated. I regard it as rather rare in ambulant cases.

The *diastolic murmurs* in these mitral cases are sometimes early diastolic but more often mid-diastolic. They are the rule in the moderately advanced and far advanced stages of the lesion, while presystolic (or *late* diastolic) murmurs are what we expect (at any rate after exertion) in the earlier stages of the lesion. Some very early cases show only a mid-diastolic obtained best after rest in the recumbent position.

TABLE 30

## Palpable Thrill

*In* 183 *ambulatory mitral cases* there was a thrill in ninety-three or 50%:

| | Cases | |
|---|---|---|
| Thrill felt best at apex in | 57 | |
| Thrill felt best at apex, extending to aortic | 1 | |
| Thrill felt best at apex, extending to base of heart | 1 | |
| Thrill felt at aortic area alone | 1 | |
| Thrill felt at pulmonic area alone | 1 | |
| No record of place | 32 | 93 |
| Thrill felt during presystolic period | 67 | |
| Thrill felt during systole alone | 6 | |
| Thrill felt during diastole alone | 6 | |
| No record of time | 14 | 93 |

*In* 89 *ambulatory mitral-and-aortic cases* twenty-six showed thrill:

| | Cases | |
|---|---|---|
| Thrill felt in | 24 | |
| Double thrill felt in | 2 | 26 |
| Systolic and presystolic at apex | 1 | |
| Systolic at apex | 1 | |
| Presystolic at apex | 8 | |
| Presystolic at apex, systolic at aortic area | 1 | |
| Diastolic at apex | 2 | |
| Diastolic at pulmonic area | 1 | |
| Diastolic no record of place | 1 | |
| Systolic at aortic | 9 | |
| Systolic no record of place | 2 | |
| | | 26 |

Thus in 119 out of 272 ambulatory cases the diagnosis rested in part on the presence of a purring thrill palpable after exertion or at rest. In a few cases (fifteen) this thrill was not confined to the apex region but could also be felt at the base of the heart. I suppose this variation depends chiefly on how violent the vibrations are.

**The Pulse.**—Out of 107 necropsied "pure mitral" cases our records are of some value in sixty-seven. Of these twenty-six showed regular pulses, most of them small in volume and of low tension* but a good many entirely normal in all respects. *There is no characteristic pulse of mitral disease.*

Forty-one were arrhythmic, and of these thirty-eight are described so that it is probable that *absolute* arrhythmia, and so auricular fibrillation, was present i.e. in about 50% of the well-recorded cases.

* Two are described as of high tension.

The three remaining are called "intermittent" and may have represented other types of arrhythmia.

In three cases the word "Corrigan" is used, and twice a capillary pulse is recorded. These cases are of special interest because we know that no chronic aortic disease, no arteriosclerosis, or hyperthyroidism was present. In one case the pulse is easily explained by the presence on the aortic valve of large fresh vegetations which probably caused regurgitation. In another *chronic pericarditis, that universal confuser of all circulatory signs, was present.* The third case was not accounted for.

Comparing the pulses in the two groups of cases in Table 31, it seems that *arrhythmia is nearly twice as common in the pure-mitral cases as in the mitral-and-aortic.* Why this is so I have no idea. In the mitral-and-aortic cases the regular pulses greatly out-number the irregular by 2 to 1; in the "other combinations" also the regular pulses are slightly but definitely in the majority. It would seem to follow, if these figures are correct, that "pure mitral" disease is more apt to be associated with arrhythmia than any of the more complicated lesions.

The Corrigan Pulse in the mitral-and-aortic series was noticed only ten times or 22%. This figure is of considerable interest. It shows (if the clinical records are correct) that when we are in doubt whether or not an aortic lesion is present beside the recognized mitral lesion, we cannot often count on the Corrigan pulse as an indication of aortic disease. In four-fifths of the cases the associated stenosis at the mitral (and usually at the aortic) valves prevents the pulse from jerking and collapsing.

TABLE 31.—THE PULSE

Necropsies

| Pulse | Mitral | M + A | Other combinations | Total |
|---|---|---|---|---|
| Regular | 26 | 24 | 14 | 64 |
| Irregular | 41 | 10 | 12 | 63 |
| Corrigan | 3 | 10 | 5 | 18 |
| Capillary | 2 | 7 | 1 | 10 |
| High tension | 2 | 4 | 2 | 8 |
| Low tension | 48 | 11 | 9 | 68 |
| Fair tension | 5 | 5 | 2 | 12 |
| Good tension | 10 | 15 | 2 | 27 |

TABLE 32.—LIVING CASES

| Pure-Mitral and Mitral-and-Aortic* | |
|---|---|
| Corrigan | 36 |
| Capillary | 16 |
| Pistol shot | 9 |
| Durozier | 3 |
| Waterhammer | 2 |
| Normal or no record | 181 |

TABLE 33.—CONDITION OF THE ARTERIES

| Necropsied cases | Mitral | M + A |
|---|---|---|
| Palpable | 14 | 9 |
| Thickened, brachials tortuous | 3 | |
| Palpable and sclerosed | 3 | |
| Not palpable | 11 | 9 |
| Bounding, not palpable | 1 | |
| No record | 75 | 22 |
| | 107 | 40 |

**Blood Pressure.**—Nothing definite or characteristic can be made out. It is noteworthy, however, that the presence of mitral stenosis does not prevent very high pressures in cases complicated by chronic nephritis. Thus in Case 3283 (complicated by glomerulonephritis

TABLE 34.—BLOOD PRESSURE
Living Cases

| | |
|---|---|
| *Mitral* (59 measured). | |
| Highest diastolic pressures: 155, 120 (2), 115, 110 (2), 105 (3), 100 | 10 cases |
| Highest systolic pressures: 210 (2), 195, 185, 180 (2) | 6 cases |
| Highest pulse pressures: 105, 90. | |
| *Mitral-and-aortic* (45 cases measured). | |
| Systolic blood pressure 100–120 | 14 cases |
| Systolic blood pressure 120–130 | 13 cases |
| Systolic blood pressure 130–140 | 9 cases |
| Systolic blood pressure 140–150 | 2 cases |
| Systolic blood pressure 150–160 | 6 cases |
| Brachial 120/30, popliteal 190/20 | 1 case |

in a patient of twenty-three) pressures of 210/130, 220/130, 280/110, and 210/140 were recorded during the twelve days of his life in the hospital. The mitral orifice measured eight cm. and showed a well marked stenosing deformity.

In another mitral stenotic patient dying at sixty-four of a cerebral hemorrhage with complicating pneumonia, the systolic pressure reached 175. In thirty other valve lesions there were only six records of systolic blood pressure ranging from 105 to 180, with three records of diastolic pressure, 50, 65, and 80.

The living patients illustrate the same point.

**Hoarseness and Aphonia.**—Only one of the cases here analyzed necropsy 3422 showed during life either of these symptoms, though Dr. Paul D. White* has shown them to be not infrequent clinically in mitral cases which did not happen to come to necropsy. Probably poor observation accounts for this discrepancy.

TABLE 35.—POST-MORTEM EVIDENCES OF PASSIVE CONGESTION

| + Present o Absent | 107 cases Mitral | | 40 cases M + A | | 33 cases Other comb. | | 180 cases Total | |
|---|---|---|---|---|---|---|---|---|
| | + | o | + | o | + | o | + | o |
| Cyanosis | 39 | 68 | 12 | 28 | 21 | 12 | 72 | 108 |
| Jaundice | 4 | 103 | 1 | 39 | 1 | 32 | 6 | 174 |
| Anasarca | 22 | 85 | 4 | 36 | 1 | 32 | 27 | 153 |
| Hydropericardium | 27 | 80 | | | | | | |
| Hydrothorax* | 45 | 62 | 19 | 21 | 15 | 18 | 79 | 101 |
| Ascites | 35 | 72 | 17 | 23 | 13 | 20 | 65 | 115 |

| | Mitral | M + A | Other Comb. | Total |
|---|---|---|---|---|
| * Hydrothorax, right | 8 | 3 | 3 | 14 |
| Hydrothorax, left | 6 | 2 | 2 | 10 |
| Hydrothorax, double | 30 | 14 | 8 | 52 |
| Hydrothorax, place not stated | .. | .. | 3 | 3 |
| | | | | 79 cases. |

* Paralysis of the left recurrent laryngeal nerve associated with mitral stenosis, Paul D. White, M.D. and Joseph Garland, M.D., Archives of Int. Med., Sept., 1920, Vol. 26, p. 343.

**Evidences of Passive Congestion at Necropsy.**—*Anasarca* was recorded twenty-two times in the 107 pure mitral cases and four times in the 40 mitral-and-aortic cases.

*Hydropericardium* was recorded twenty-seven times in the 107 mitral cases. In the other cases we have no record.

*Hydrothorax* forty-five times in the 107 mitral cases and 19 times in the 40 mitral and aortic. Thus the two series show no significant difference in the % occurrence of this symptom. *Aortic disease when added to mitral disease neither increases nor diminishes the amount of terminal pulmonary congestion produced by mitral disease alone.*

What seems most surprising in these figures is that 56% *of the cases show no hydrothorax at all!*

In the positive cases the fluid was confined, or largely confined to the right chest in fourteen cases and to the left in ten cases—a discrepancy favoring the right side less than clinical observation would lead us to suppose.

In nineteen out of forty cases the hydrothorax was not recognized in life. Part of this error may be due to a reluctance to disturb dying patients in order to establish a fact which is of little or no use to prognosis or treatment. Twice hydrothorax was diagnosed in life but not found at necropsy. Four times we found it in life only on the right side while the pathologist found it on both sides.

*Ascites* was also rather surprisingly infrequent in both mitral and mitral-and-aortic cases. It was present in thirty-five of 107 mitral cases and 17 of 40 mitral-and-aortic cases,—practically identical fractions, each representing about one-third of the group. In the "combined lesions" it was twice as frequent (20 of 33).

In two-thirds of all cases the ascites was recognized in life. In the remainder we missed it. In four cases we "found" ascites when it was not there.

The *spleen* was firm and elastic at necropsy in 68 cases, soft in 33 out of a total of 101 cases in which a definite observation is recorded. Thus two-thirds show passive congestion and one-third suggest infection. In life the spleen was felt but five times in 144 cases. In one of these five, infarction was found at necropsy.

These cases seem to show that *as a rule death does not occur in mitral disease by means of decompensation and passive congestion.*

When "pure mitral" disease is itself the cause of death, decompensation is usually present. But mitral disease is often present not as a cause of death but as an historical landmark, harmless so far as we know, to the individual. In our series this was the case

in 60 cases out of 107— or nearly three-fifths—an astonishing result. (Further analysis of the non-congestive deaths is given on page 77).

**How Explain the Cases without Dropsy.**—Sixty of 107—or 3/5 of the pure mitral cases died without any hydrothorax and with little or no dropsy elsewhere. In most cases this fact is explained by the fact that compensation was good to the last, the patients dying of embolism and thrombosis (ten), pneumonia (five), sepsis (five), acute endocarditis (four), general peritonitis (four), cancer (four), apoplexy (three), meningitis (two), leukemia, uremia, and hemorrhage, each one. In four cases death was unexplained.

In the 40 Mitral-and-Aortic cases, 21 or over half showed no hydrothorax; among the "combined lesions," 18 of 33 were also free of hydrothorax.

TABLE 36.—CONDITION OF THE LIVER IN LIFE

| | 107 cases mitral | 40 M + A | 33 comb. lesions | 180 total |
|---|---|---|---|---|
| Liver enlarged (felt below ribs) | 26 | 14 | 19 | 59 |
| Tenderness also in | (21) | (9) | (8) | |
| Not demonstrably enlarged | 48 | 17 | 10 | 75 |
| Not recorded | 33 | 9 | 4 | 46 |

TABLE 37.—CONDITION OF THE SPLEEN AFTER DEATH

| | 107 mitral | 40 M + A | 33 comb. lesions | 180 total |
|---|---|---|---|---|
| Spleen firm, "rubbery" | 54 | 19 | 16 | 89 |
| Spleen soft | 22 | 11 | 1 | 34 |
| Spleen normal | 0 | 1 | 4 | 5 |
| Spleen enlarged | 1 | 0 | 5 | 6 |
| Spleen not recorded | 30 | 9 | 7 | 46 |

**Condition of the Liver.**—The figures in Table 36 seem to show that during life hepatic enlargement is obvious in slightly under one-half of the pure mitral cases, over one-half of the mitral-and-aortic cases and nearly 2/3 of the "other combinations."

The decompensating power of multiple valve lesions is very slightly greater than that of mitral stenosis alone. This is only what we should expect and bears out what is shown in the post-mortem records of passive congestion (Table 35). There we see that *hydrothorax* is produced by pure mitral disease in 42% of cases, by mitral and

aortic disease in 47.5%, and by the "other combinations" in 45%. *Ascites* occurred in 33% of pure mitral cases, in 42.5% of mitral-and-aortic cases, in 40% of "other combinations." The records of *hydropericardium* are incomplete.

**The Spleen.**—The necropsy record of a firm and "rubbery" spleen represents passive congestion. Enlargement is mentioned in only six cases and is relatively slight, especially in length and breadth, although the organ is often plumper than normal. Its lack of longitudinal enlargement accounts for the fact that in heart disease we can so rarely feel its tip during life unless an infarct is lodged there.

The soft spleens are usually associated with acute endocarditis or other septic complications.

**Urinalysis.**—The cases showing definite glomerulonephritis and those associated with renal infarcts are collated in Tables 42 and 45 which show that about ⅕ of all cases of mitral disease ("pure" or complicated) are associated with nephritis of some type. In 40 uncomplicated cases eight showed normal urine and thirty-two showed albumin and casts. In only four of these thirty-two was the albumin or the casts in unusual amount, i.e., more than the traces of albumin and the few hyalin and granular casts to be expected as a result of passive congestion. These four cases showed the following:

TABLE 38.—URINE IN PASSIVE CONGESTION DUE TO MITRAL DISEASE

| Case | Pathological state | Albumin | Casts |
|---|---|---|---|
| 1 | Simple passive congestion | Large trace | Many |
| 2 | Simple passive congestion | 1 to 2% | Many |
| 3 | Meningitis as complication | Trace | Many |
| 4 | General peritonitis as complication | Slight trace | Many |

*Comments.*—Considering these cases in connection with those complicated by definite nephritis, it is obvious that urinary examination does not enable us to distinguish nephritis from the conditions accompanying (a) passive congestion of the kidney, (b) acute infectious diseases with cloudy swelling of the kidney. This conclusion I proved many years ago* but until recent years it was been unfashionable in medical circles to admit it.

* Cabot, R. C.: The Diagnosis of Renal Functions, N. Y. Med. Journ., May 12, 1906.

**Fever.**—Fever was present (continued or remittent) during most or all of the hospital stay of seventy-six out of 180 patients (see Table 39). The probable explanation in 55 cases was as follows:

| | | |
|---|---|---|
| 1. | Complicating pneumonia | 21 |
| 2. | Acute endocarditis | 10 (or possibly 11) |
| 3. | Meningitis | 4 |
| 4. | General sepsis | 12 |
| 5. | Pericarditis | 3 |
| 6. | General peritonitis, arthritis, leukemia, uremia, abortion each one | 5 |
| | | 55 |

*Thrombus—Fever.*—In addition to these there remains an interesting group of thirteen cases in which nothing was found post-mortem to explain fever except *thrombi, emboli, fresh infarcts* and their results. In six of these the thrombi were confined to one or both auricular appendages (the left in five). This association of thrombi with fever may not be a causal one. Certainly thrombi are sometimes associated with bacteria. In other cases the disintegration of a clot might cause a protein fever. On the whole it seems not improbable that ball thrombi or other clots in the auricles are part of a process which not infrequently causes fever.

TABLE 39.—TEMPERATURE

| Temperature | Mitral | M + A | Other comb. | Total |
|---|---|---|---|---|
| Subnormal | 2 | 1 | 2 | 5 |
| Normal (under 99) | 18 | 8 | 7 | 33 |
| 99–100 | 11 | 2 | 3 | 16 |
| Up to 101 | 14 | 5 | 3 | 22 |
| Up to 102 | 12 | 2 | 2 | 16 |
| Up to 103 | 1 | .. | .. | 1 |
| Up to 104 | 13 | 4 | 4 | 21 |
| Degree not recorded | 36 | 18 | 12 | 66 |
| | 107 | 40 | 33 | 180 |

Ordinarily when an unexplained fever arises in a case of valvular heart disease we begin to speculate on the possibility of acute endocarditis, and regard this possibility as strong if embolic phenomena subsequently appear. But from this series of cases it appears that unexplained fever in cardiac cases is associated *as* often post-mortem

with intracardiac clots or visceral infarcts as it is with fresh valvular endocarditis.

**Leucocytosis.**—Leucocytosis usually accompanied these fevers of all types. There was nothing of special interest about it in these cases except that in eight out of twenty-one "acute" cases of endocarditis leucocytosis was *absent*.

TABLE 40.—LEUCOCYTE COUNT

| Leucocytes | Mitral | M + A | Other comb. | Total |
|---|---|---|---|---|
| 0– 5,000 | .. | 2 | 2 | 4 |
| 6,000– 7,000 | 7 | 1 | 2 | 10 |
| 8,000– 9,000 | 10 | 2 | 4 | 16 |
| 10,000–11,000 | 9 | 2 | 1 | 11 |
| 12,000–13,000 | 12 | 3 | 4 | 19 |
| 14,000–15,000 | 16 | 2 | 3 | 21 |
| 16,000–17,000 | 6 | 3 | 1 | 9 |
| 18,000–19,000 | .. | 2 | .. | 2 |
| 20,000–21,000 | 9 | 3 | .. | 12 |
| 22,000–30,000 | 11 | .. | 2 | 13 |
| 31,000–50,000 | 3 | 1 | 7 | 11 |
| Slight leucocytosis | .. | .. | 1 | 1 |
| Moderate leucocytosis | .. | 1 | .. | 1 |
| Marked leucocytosis | .. | 2 | 1 | 3 |
| Total | 83 | 24 | 28 | 135 |

TABLE 41

| | Mitral | M + A | Other comb. | Total |
|---|---|---|---|---|
| Total cases | 83 | 24 | 28 | 135 |
| Of these there was leucocytosis in | 66 | 19 | 20 | 105 or 80% |
| No cause for leucocytosis outside the heart in | 57 | 19 | 13 | 89 or 85% |

*Comments.*—1. In terminal stages, from two-thirds to four-fifths of the cases in each group showed fever.

2. In one-fifth of the cases this fever might be explained by the acute endocarditis found post-mortem. Other common causes are pneumonia and general sepsis.

3. In the remainder thrombosis occurred thirteen times, leaving two cases with fever quite unexplained. Thrombosis thus appears to be associated with most of the "unexplained" fevers of mitral disease.

4. Nearly four-fifths of all cases showed a leucocytosis, particularly marked in the "combined lesions." The cause of this leucocytosis was usually within the heart (clot or endocarditis).

**Infarction of the Kidneys, Lungs, and Spleen.**—Whatever the precise cause of infarcts, however they are related to embolism, to thrombosis, or to simple passive congestion, the data of these necropsies regarding them may well be presented here (Table 42).

TABLE 42.—INFARCTS

| Infarcts in the | Mitral | M + A | Other comb. | Total |
|---|---|---|---|---|
| Kidney | 39 | 14 | 8 | 61 |
| Lungs | 33 | 7 | 11 | 51 |
| Spleen | 23 | 12 | 7 | 42 |
| Liver | .. | 1 | .. | 1 |
| Gut | .. | .. | 1 | 1 |
| Total no. of cases with infarctions in these organs. | 57 (of 107) | 19 (of 40) | 20 (of 33) | 96 (of 180) |

From these figures it is clear that:

(1) In 57 out of 107 cases infarctions were present, ordinarily of the lungs, kidneys, spleen or liver. 43 of this 57 were in uncompensated cases with general dropsy and congestion. Only 14 occurred in well compensated, non-congestive cases.

(2) Infarcts occur in 19 out of 40 or about ½ the "mitral-and-aortic cases and in 20 of 33 or 43% of the "other combinations."

(3) Infarcts are slightly commoner in the kidneys than elsewhere, perhaps because they are easier to see in this organ than in the lungs.

### *The Diagnosis of Infarctions*

*Pulmonary infarction* one may recognize clinically in about one-third of the cases, those namely in which it leads to an hemoptysis not otherwise explained. This was the case in nine out of the thirty-three well-studied "pure mitral" cases. In one of the cases there was also sharp pain in the right side of the chest at a point corresponding

with the position of the infarct. In the other twenty-four the infarcts were not and probably could not have been recognized.

*Infarct of the kidney* was known to be associated in one case out of thirty-nine with a sharp pain in the right hypochondrium, which led to a guess that infarction had occurred. The urinary findings in the cases showing infarction post-mortem were in no way characteristic or suggestive. Four cases showed only the "slightest possible trace" of albumin, four others a "slight trace" with casts, one a "large trace" alone, and one a large trace with casts, but this latter case had also a general peritonitis. It is entirely reasonable that the urinary findings should not be characteristic, for the infarcts found post-mortem were of all ages. Only a fresh infarct could be expected to produce hematuria and that for a brief period alone.

*Splenic infarct* was suspected in two cases out of the 42 in which it was found post-mortem. In one of these there occurred on three occasions a sharp sudden pain in the splenic region without any other assignable cause. In the presence of decompensated valvular disease such pain might naturally suggest infarct.

TABLE 43.—EMBOLIC AND THROMBOTIC LESIONS AT NECROPSY IN 180 CASES

| Embolism and Thrombosis in | Mitral | M + A | Other comb. | Total |
|---|---|---|---|---|
| 1. *Heart.* | | | | |
| Left auricular appendage | 15 | 4 | 2 | 21 |
| Both appendages | 5 | 0 | 0 | 5 |
| Ball thrombus, left auricle | 4 | 0 | 0 | 4 |
| Other thrombi, left auricle | 2 | 1 | 0 | 3 |
| Right auricular appendage | 2 | 0 | 3 | 5 |
| Place not stated | .. | .. | 3 | 3 |
| Left ventricle | 0 | 0 | 0 | 0 |
| Total cardiac thrombi | 28 | 5 | 8 | 41 |
| 2. *Pulmonary vessels* | 9 | 1 | 0 | 9 |
| 3. *Peripheral vessels* | 7 | 2 | 0 | 9 |
| 4. *Cerebral vessels* | 1 | 2 | 1 | 4 |
| 5. *Kidney vessels* | 1 | 1 | 0 | 2 |
| 6. *Spleen vessels* | 1 | 1 | 0 | 2 |
| 7. *Liver vessels* | 0 | 1 | 0 | 1 |
| 8. *Intestinal vessels* | 1 | 0 | 0 | 1 |
| 9. *Innominate vein* | 1 | | | |
| Total lesions | 49 | 13 | 9 | 69 |

In a third case, a spleen which turned out to contain an infarct was palpable during life, but no suspicion of the infarct was aroused, nor should it have been suspected, since infarcts often do not enlarge the spleen and other causes of splenic enlargement could not here be excluded. But it has been my experience with necropsied cases not included in this series that *when a spleen has been observed during life to grow notably larger within a few days, the diagnosis post-mortem is always infarct.* Leukemic and "infectious" spleens grow more slowly.

*Comment.*—The well known tendency for thrombi to form in the left auricle when its outlet is blocked by mitral stenosis and its current is slowed by auricular fibrillation, receives abundant illustration in these necropsies. *In the presence of an advanced case of mitral stenosis with fibrillation,* we must realize that *the chances of intracardiac thrombosis are one in four* and that in over 90% of these cases this thrombus will be in the left auricle. The danger of detaching this clot when we stir up the previously passive and fibrillating auricle with quinidine has been recently emphasized by Paul D. White and others. Cerebral embolism and death have been thus produced.

Next to the heart, the lungs, the brain and the peripheral vessels are oftenest involved.

**Clinical Evidences of Embolism or Thrombosis.**—1. *Cerebral* (13 cases).* In eleven of the 104 "pure mitral" cases, and in two of the "mitral and aortic" cases there were signs and symptoms pointing to a focal brain lesion.

In ten of these 13 cases a sudden hemiplegia was the central fact. This affected the right side of the body in six cases, the left in two cases, while in one case the right arm and the left side of the face are recorded as paralyzed. In the tenth case the side is not stated in the record. Four cases showed signs of "apoplexy" or cerebral hemorrhage, but without hemiplegia.

These cases do not bring out the clinically familiar fact that the embolic hemiplegias of mitral stenosis give a relatively good prognosis when compared with hemiplegias associated with hypertension and arteriosclerosis. But neither do they contradict it.

2. *Pulmonary.*—The recognizable cases have already been referred to with pulmonary infarct.

3. *Peripheral.*—(See Table 50.)

* This does not contradict Table 43 where only 4 cases are listed as showing cerebral lesions at necropsy. Permission to examine the brain is often, indeed usually, refused in our cases.

**Auricular Thrombi and Arrhythmia.**—In 23 cases out of thirty-seven, clots in the left or right auricles were associated with an extreme irregularity of the pulse so described (in most cases) as to warrant the inference that auricular fibrillation was present. In three cases similar clots occurred apparently without any fibrillation during the period of observation. Fibrillation without clots occurred in only four cases. In two other cases the records were not clear.

From these figures it appears that intracardiac clots are usually preceded by fibrillation.

TABLE 44.—INFECTIOUS PULMONARY COMPLICATIONS FOUND AT NECROPSY

| Pulmonary lesion | Mitral | M + A | Other comb. | Total |
|---|---|---|---|---|
| Lobar pneumonia | 5 | 2 | 3 | 10 |
| Bronchopneumonia | 7 | 0 | 0 | 7 |
| Organizing pneumonia | 1 | 1 | 1 | 3 |
| Resolving pneumonia | 0 | 1 | 0 | 1 |
| Pulmonary abscess | 3 | 0 | 1 | 4 |
| Empyema | 1 | 1 | 0 | 2 |
| | 17 | 5 | 5 | 27 |

*Comment.*—1. Twenty-seven in 180 cases, or about one-seventh of the whole "rheumatic" group showed a definite pulmonary disease other than passive congestion, edema, hydrothorax, or "hypostatic pneumonia." These twenty-seven cases were all of pneumonia or its results.

2. There were 4 pulmonary abscesses 3 of which occurred in cases showing a partially organized pneumonia. Abscess and organization were doubtless aspects of the same process.

3. From the frequency of these complicating pneumonias we may suppose, therefore, that the pulmonary congestion due to mitral disease favors inflammation of the lung though it certainly does not favor tuberculosis.

**The Occurrence of Nephritis and Other Renal Complications in Mitral Disease.**—Nephritis, acute or chronic, was found post-mortem in 18 of 107 mitral cases (17%), in 12 out of 40 mitral-and-aortic cases (30%), and in 4 of the 33 "other combinations." Whether there is any significance in the fact that nephritis was twice as common in the mitral-and-aortic as in the simple mitral cases I cannot say. The outstanding fact is that *late in the course of mitral disease we may expect the presence of glomerulonephritis in one-fifth of all*

*cases* (35 in 180). Doubtless a similar nephritis occurs and heals in the earlier stages of many more cases.

Only six of these 35 cases of glomerulonephritis were suspected in life. Here suspicion was aroused usually by the condition of the urine, sometimes by high blood pressure, edema, or uremic symptoms in addition to the urinary abnormalities. In the remaining 29 cases the urine showed nothing suspicious.

Of the 16 cases of *acute* glomerulonephritis proved post-mortem, one showed a normal urine; one showed a slight trace of albumin and many casts; one showed a trace of albumin, many casts, blood and pus. In the rest there was nothing pointing to nephritis.

Of 7 cases of subacute glomerulonephritis, four were recognized by the presence of albumin in considerable quantities with many casts and other clinical manifestations.

One case of *chronic* glomerulonephritis showed only a slight trace of albumin and many casts.

TABLE 45.—NEPHRITIS WITH VALVE LESIONS

| | Mitral | M + A | Combined lesions | Total | |
|---|---|---|---|---|---|
| Acute glomerulo nephritis | 10 | 5 | 2 | 17 | |
| Subacute glomerulo nephritis | 0 | 5 | 2 | 7 | |
| Acute and chronic nephritis | 0 | 1 | 0 | 1 | 35 or 25% |
| Chronic nephritis | 8 | 1 | 0 | 9 | |
| Amyloid degeneration | .. | 1 | .. | 1 | |
| No nephritis | 89 | 23 | 21 | 133 | |
| No record | .. | 4 | 8 | 12 | |
| | 107 | 40 | 33 | 180 | |

**Anginoid Attacks.**—In necropsies 600, 810, 1098 and 3040 there were attacks simulating angina. One of these has already been analyzed (see p. 49). Another occurring in connection with abortion in a woman of twenty-three, lasted six days as "pain" over the heart,—apparently the first attack and leading immediately to death. No cause was discoverable post-mortem. I do not consider this angina pectoris.

Another case occurred in a man of twenty-seven with a well-marked history of syphilis (hard chancre, headache, alopecia, sore throat, treatment by mercury pills). He had had substernal pain on exertion for two years. Necropsy showed lesions probably to be interpreted as syphilitic aortitis. The mouths of the coronary art-

eries were not occluded and the aorta and coronaries were otherwise negative. This is the only case in our 180 showing the association of a syphilitic and a rheumatic lesion in the same heart, exemplifying once more the queer fact of an association between syphilis and angina pectoris even when the aorta is not diseased.

TABLE 46.—CASES SHOWING POSITIVE (POST-MORTEM) CULTURES

| | Organism | Source | Type of endocarditis | Complications |
|---|---|---|---|---|
| Mitral | Streptococcus 17 | Blood 14<br>Spleen 1<br>Liver 1<br>Liver and Spleen 1 | Acute 3<br>Chronic 10<br>Acute & Chronic 4 | General Peritonitis 2<br>Local Peritonitis 1<br>Erysipelas 1<br>Cancer of the liver 1 |
| | Pneumococcus 4 | Blood 3<br>Liver 1 | Chronic 4 | Lobar Pneumonia 3 |
| | Pseudo Pneumococcus 1 | Blood 1 | Acute & Chronic 1 | Lobar Pneumonia 1 |
| | B. muc. capsul. / B. Coli 1 | Blood 1 | Chronic 1 | Bronchitis and Ulcerative Colitis 1 |
| | B. Coli 1 | Blood 1 | Chronic 1 | Local Peritonitis 1 |
| | Diplococcus 1 | Blood 1 | Acute & Chronic 1 | |
| M + A | Streptococcus 7 | Not Stated | Chronic 1<br>Acute & Chronic 6 | Appendicitis 1<br>Malignant Endoc. 1<br>Nephritis 1<br>General Peritonitis 1 |
| | Pneumococcus 5 | Not Stated | Chronic 3<br>Acute & Chronic 2 | Pneumonia, lobar 2<br>Pneumonia, focal 1<br>Pneumonia, organizing 1 |
| | Staphylococcus 2 | Not Stated | Acute & Chronic 2 | |
| Comb. lesions | Streptococcus 3 | Blood 3 | Acute & Chronic 2<br>Chronic 1 | Pneumonia & Bronchiectasis 1<br>Erysipelas 1<br>Meningitis 1 |
| | Pneumococcus 1 | Blood 1 | Acute & Chronic 1 | Pneumonia 1 |
| | Pseudo pneumococcus 1 | Blood 1 | Acute 1 | Pneumonia 1 |

| | | | | |
|---|---|---|---|---|
| Total | Streptococcus | 27 | Positive cultures | 44 |
| | Pneumococcus | 10 | Negative cultures | 99 |
| | Staphylococcus | 2 | No record | 37 |
| | Pseudo-Pneumococcus | 2 | | 180 |
| | B. muc. Capsulatus & B. Coli | 1 | | |
| | Diplococcus | 1 | | |
| | B. Coli | 1 | | |
| | | 44 | | |

**Blood Cultures.**—Very few attempts were made during the life of these 180 patients to isolate any organism from the circulating blood, and only one urinary culture is recorded, No. 3566: staphylococci. Out of 11 cases tested in life the blood in eight was negative, 3 positive,—streptococcus in all cases.

*Post-mortem* cultures were made in 143 cases. Of these forty-four were positive (24%) and ninety-nine negative. In the forty-four positive cases one showed only bacillus coli which was very possibly due to a complicating local peritonitis; one showed staphylococcus albus (presumably contamination); two showed staphylococcus aureus; *twenty-seven cases showed a streptococcus; ten cases a pneumococcus;* two a pseudopneumococcus, one bacillus mucosus capsulatus, one a few diplococci. The pseudopneumococci and eight of the ten positive pneumococcus cultures came from cases complicated by pneumonia.

In the twenty-seven streptococcus cases acute endocarditis was present post-mortem fifteen times. Two of these were of the ulcerating type; the rest showed minute vegetations only. In one of the others, showing only chronic endocarditis, there was a terminal erysipelas; in two others general peritonitis had supervened, upon hysterectomy and upon appendectomy respectively. General peritonitis was also present in connection with two of the cases of acute endocarditis with positive streptococcus cultures.

From these cultures I can draw no conclusions of significance. Streptococcus cultures post-mortem occur only in 15% of our necropsied cases. Nevertheless in non-cardiac cases of this chronic exhaustive type the percentage is not far different.

The only point of interest is the absence of the influenza bacillus and of the gonococcus, organisms not infrequently mentioned in the literature of endocarditis and both of them organisms which our pathologists have been especially interested to search for.

The two staphylococcus cases showed curiously enough no local abscesses at necropsy. One showed endocarditis of the acute polypous (as well as chronic) type; in the other the lesions were purely chronic.

**Has the Size of the Mitral Orifice Any Relation to Its Clinical Manifestations?**—Nothing of significance could be learned on this topic from the study of the present series. Some of the cases with extreme narrowing seemed to be rather more subject to repeated attacks of decompensation closely following one another. For instance:

Necropsy 2748: mitral 1 cm. 2 decompensations in 1 year: no acute endocarditis.
Necropsy 2226: mitral 2.5 cm. 2 decompensations in 1 year: no acute endocarditis.
Necropsy 2091: mitral 1.5 cm. 2 decompensations in 1 year: no acute endocarditis.
Necropsy 1806: mitral 1. cm. 2 decompensations in 1 year: no acute endocarditis.

But in contrast:

Necropsy 2960: mitral 1.2 cm. Nothing characteristic in course.
Necropsy 2635: mitral 1.5 cm. Nothing characteristic in course.
Necropsy 1675: mitral 1.8 cm. Nothing characteristic in course.
Necropsy 973: mitral 1.5 cm. Nothing characteristic in course. Died of pneumonia.

On the whole it does not appear that the size of the stenosed mitral orifice is in itself a recognizable factor in determining what or how much the individual shall suffer (see Table 47).

TABLE 47.—DIMENSION OF THE MITRAL VALVE

| Size of orifice | Mitral | M + A | Other comb. | Total |
|---|---|---|---|---|
| 9. cm | 5 | 0 | 0 | 5 |
| 8.5 cm | 6 | 5 | 0 | 11 |
| 8. cm | 9 | 8 | 2 | 19 |
| 7.5 cm | 7 | 4 | 3 | 14 |
| 7. cm | 15 | 3 | 3 | 21 |
| 6.5 cm | 4 | 2 | 1 | 7 |
| 6. cm | 7 | 1 | 2 | 10 |
| 5.5 cm | 1 | 0 | 2 | 3 |
| 5. cm | 8 | 1 | 1 | 10 |
| 4.5 cm | 5 | 0 | 0 | 5 |
| 4. cm | 5 | 0 | 1 | 6 |
| 3.5 cm | 3 | 1 | 3 | 7 |
| 3. cm | 2 | 1 | 2 | 5 |
| 2.5 cm | 2 | 0 | 1 | 3 |
| 2. cm | 0 | 0 | 1 | 1 |
| 1.5 cm | 5 | 2 | 1 | 8 |
| 1. cm | 2 | 0 | 0 | 2 |
| "Fish-mouth" | 0 | 1 | 0 | 1 |
| "Buttonhole" | 1 | 0 | 0 | 1 |
| "Very small" | 2 | 0 | 0 | 2 |
| "Not very small" | 0 | 0 | 1 | 1 |
| Admits forefinger tip | 10 | 3 | 0 | 13 |
| Admits middlefinger tip | 2 | 0 | 0 | 2 |
| Admits littlefinger tip | 0 | 0 | 5 | 5 |
| Admits closed blades of scissors | 1 | 0 | 0 | 1 |
| Admits two finger tips | 0 | 1 | 0 | 1 |
| Admits three finger tips with difficulty | 1 | 0 | 0 | 1 |
| Not recorded | 1 | 4 | 3 | 8 |
| Over 9 cm | 3 | 3 | 1 | 7 |
| | 107 | 40 | 33 | 180 |

*Comment.*—55% of the mitral cases, 28% of the mitral-and-aortic cases, and 55% of the "other combinations" showed a mitral ring measuring 7 cm. or less instead of ten cm.,—the average for adults. It would seem that the "pure mitral" cases tend to a greater degree of narrowing than the "mitral and aortic" cases. Possibly this may help to account for the fact that passive congestion is as often marked at necropsy in the "pure mitral" cases as it is in the "mitral-and-aortic." A very narrow mitral perhaps does as much harm as a fairly narrow mitral *plus* some aortic stenosis.

## MODE OF DEATH

TABLE 48.—MODE OF TERMINATION IN 106 CASES OF MITRAL STENOSIS

| | |
|---|---|
| Passive congestion | 49 |
| Non-cardiac disease (cancer, nephritis, etc.) | 33 |
| Embolism | 12 |
| Acute sepsis | 10 |
| Sudden death, unexplained | 2 |
| | 106 |

1. *Deaths from Chronic Passive Congestion.*—Only forty-nine out of our 106 cases of "pure" mitral disease died a "congestive death." In these forty-nine the symptoms and signs before death and the findings after death were predominantly those of passive congestion, though in thirteen cases there was *also* a slight acute endocarditis,—believed to be of the terminal type and not the main cause of death.

Of these forty-nine cases with "congestive death," thirty had a typical rheumatic or choreic history, and nineteen did not. These nineteen cases were on the average older than the thirty rheumatics, averaging forty-five years and having complained of dyspnea and other cardiac symptoms for seven and a half years. In three of these (fifty, sixty-three, and sixty-five years of age) the symptoms had been noticed "for years."

2. *Deaths from Other Diseases with Latent Mitral Stenosis.*—There were thirty-three cases of this type—nearly one-third of the whole. The main causes of death are shown in the following table.

TABLE 49.—CAUSES OF DEATH IN LATENT CASES OF MITRAL DISEASE

| | |
|---|---|
| Infections (Pneumonias 5, General Peritonitis 3) | 14 |
| Neoplasms | 6 |
| Cerebral Hemorrhage | 4 |
| Post-operative Exhaustion | 5 |
| Nephritis | 2 |
| Cirrhosis | 1 |
| Chronic arthritis with bed-sore | 1 |
| | 33 |

Passive congestion was absent at necropsy in these cases.

3. *Embolic Deaths.*—In twelve patients embolism was the only or the chief cause of death. The site of the emboli was as follows:

TABLE 50.—SITE OF EMBOLI CAUSING DEATH IN MITRAL STENOSIS

| | |
|---|---|
| Peripheral (one or both legs) | 5 |
| Peripheral and cerebral | 1 |
| Cerebral | 1 |
| Mesenteric | 1 |
| Pulmonary artery | 2 |
| Pulmonary and renal arteries | 1 |
| Abdominal aorta, iliac and left renal | 1 |
| | 12 |

4. *Acute Septic Deaths.*—An accompanying acute endocarditis, with the general septicemia resulting, seemed the chief cause of death in ten cases. These have also been counted among the cases of Acute Endocarditis in Chapter VII but are included here because the mitral valve was definitely stenosed.

5. *Sudden Death Unexplained.*—Two patients died suddenly and without any notable symptoms or signs until the coma abruptly appeared. These cases are of sufficient rarity and interest to deserve description in detail.

*Necropsy* 1621 was performed on the body of a white American woman of thirty-five who had had chorea in childhood but no rheumatism. She was brought to the hospital in collapse March 1, 1906, and died on March 5th. There was a vague history of similar attacks in 1903 and 1905, but as she had had many epileptiform attacks (the nature of which is not clear) we cannot be sure that the seizures of 1903 and 1905 were due to the cardiac disease. She had not been known to be ill and had been found in coma by her brother who brought her to the hospital. Though she was but thirty-five there was well-marked arteriosclerosis of the pulmonary artery and of the aorta. There was a mitral stenosis (orifice three cm.) and a moderate hydropericardium but no other dropsical effusions.

*Necropsy* 1981 concerned a man of forty who was brought in unconscious and died the same day. He had worked till the day before entrance, and had not been known to be ill.

Necropsy showed a mitral orifice of 4.5 cm., a non-deforming chronic endocarditis of one aortic cusp, chronic passive congestion with hydropericardium, and hydrothorax. There was also a general arteriosclerosis and some unexplained cicatrices in the liver—not definitely syphilitic.

TABLE 51.—ERRORS IN DIAGNOSIS

| | Mitral | M + A | Other lesions |
|---|---|---|---|
| Recognized ante-mortem | 52 | 13 | See below p. 160 |
| Unrecognized ante-mortem | 54 | 27 | p. 160 |
| Doubtful | 1 | | p. 160 |

## DIAGNOSTIC ERRORS

I have disregarded the clinical diagnoses written on the records because in many instances these diagnoses have shown strong internal evidence of having been copied from the necropsy record. I have considered the cases as rightly diagnosed during life whenever a presystolic or diastolic murmur was recorded or heard at or near the cardiac apex and not otherwise explained. When systolic murmurs only were heard, or no murmur at all, I have considered the case as not diagnosed in life, because *in the absence of other signs pointing to mitral stenosis* I know no way in which a systolic murmur alone can be held to justify such a diagnosis.

**Reasons for Failure to Recognize Pure Mitral Stenosis.**—(1) *The absence of a history of cardiac symptoms and the obvious presence of some other disease.* This was the case in 35 of 54 of our failures. Under these conditions cardiac examination is often superficially made, especially in surgical wards where 35 of these patients were treated.

(2) A very short period of observation and a moribund patient. This condition obtained in 6 cases.

(3) Failure to recognize any murmur but a systolic at the apex: 18 cases.

(4) No murmur whatever recognized: 21 cases.

(5) Lack of realization of the fact that there are certain reasons for *expecting* mitral stenosis and so for searching for evidence of it with particular attention. Such reasons apparently ignored in this series are:

(a) The presence of *arterial embolism* or thrombosis (hemiplegia, peripheral gangrene, *in relatively young patients* (e.g. embolic gangrene of right foot at 29, cerebral embolism with hemiplegia at 35, gangrene of both feet at 44).

(b) The presence of *decompensation with a rheumatic or choreic* history and without hypertension or syphilis. Knowing that under these conditions mitral stenosis (with or without aortic disease) is present in almost 80% of all cases, one would look for it harder and more successfully than is the case in this series.

(c) *Suggestive cardiac signs other than a murmur*, e.g. in Necropsy 1926 the heart was greatly enlarged (7th interspace, 20 cm. from midsternal line), the apex 1st sound was short and sharp, the pulmonic 2d accentuated, and the aortic 2d barely audible. These signs in a woman of thirty-five, who had had decompensation almost without intermission for a year and who had no hypertension, no nephritis, and no evidence of syphilis, should make us assume, even if no murmur is present, that mitral stenosis is present. (We might well expect also adherent pericardium or tricuspid stenosis, neither of which was in fact present here.)

(d) *An apical systolic murmur alone with rheumatic or choreic history, arrhythmia or decompensation in a person under* 45 should always make us suspect that repeated and careful search with the patient in various positions at rest or after exercise will discover a diastolic or presystolic murmur not audible at all times. In decompensated cases we cannot rightly stimulate by exertion that increased cardiac vigor which often "brings out" diastolic or presystolic murmurs in addition to the obvious systolic which is almost invariably present. But a whiff of amyl nitrate will serve the same purpose without harming the patient.

(e) *With signs of acute (ulcerative, malignant) endocarditis* one should remember that in 56% of such cases discoverable in life a chronic deforming endocarditis is also present and therefore to be searched for or at least suspected.

### MITRAL STENOSIS. ILLUSTRATIVE CASES

1. Latent.
2. Latent with cirrhotic liver.
3. With *recurrens* paralysis.
4. With acute endocarditis.
5. With embolic gangrene of legs.

#### Necropsy 4292

An American dentist of thirty-eight entered January 14, 1922. His mother died of valvular heart disease. One uncle died insane.

He had the minor diseases of childhood. At ten years of age he was in bed for two months with "rheumatic fever," all the joints

being swollen, red, hot and very painful on motion, and at eighteen he was in bed for a month with a second attack. Since then he had at various times had one or two joints painful for a day or two. At twelve he had tonsillitis. In general his health had been very good. Occasionally he had had some epigastric distress and belching of gas after meals when he was very tired and worried. He urinated once at night, chiefly because he awoke in the night and went from habit. At twenty-six he had gonorrhea for a month. He denied syphilis. His best and usual weight was 137 pounds, his present weight 135.

Before the present illness he was a heavy smoker. He denied the use of alcohol.

Three and a half years before admission after an especially hard day's work he was awakened suddenly in the night with severe palpitation, orthopnea, coughing and wheezing with about a half a cupful of bloody frothy sputum. The attack lasted most of the night but gradually passed off by morning, as did his rapid heart action. Six months later he had a similar attack followed by a little cough which lasted a week. From this time his symptoms had gradually increased. He began to tire easily. Any unusual exertion caused a feeling of epigastric distress with some dyspnea, which had been steadily increasing. From time to time he had mild attacks like the ones described. Two years before admission he had a third very bad attack similar to the first but lasting longer. At this time he was particularly tired and nervous, having been worried a great deal about his condition and his business. He continued to work hard however for the next six months. Then his gradually increasing distress and dyspnea on exertion became very severe. His heart action was also quite irregular, and had continued to be so. He stayed two months in a hospital with much improvement. Five days after leaving it he was forced to return for another month. He gradually got back to his work and did well for six months. Then his old symptoms of fatigue, palpitation, dyspnea, nervousness and sleeplessness began to come back. Six months before admission his ankles became swollen. He was having considerable cough with frequent hemoptysis, never more than an ounce. He stayed in the hospital again for two months with much improvement. Three months before admission he went back to work and did well for a short time before his symptoms began to return. Three weeks before admission both knees and ankles became swollen, red, hot and very painful on motion. This was soon followed by some pain in his

elbows and wrists. A week later he developed a red purpuric rash on both legs. His dyspnea became much worse. He had been in bed most of the time since, although he had improved. For a year and a half he had taken digitalis from time to time with good effect. For a week he had not taken any.

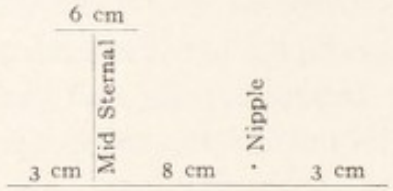



FIG. 1.—Cardiac measurements by percussion.

Physical examination. Well nourished. Weight 132 pounds. Skin dry. Faint macular rash on the chest. Slight acne on the back. The tonsils showed crypts on the left. Lungs negative. Heart. Apex impulse not seen or felt. Measurements as shown in

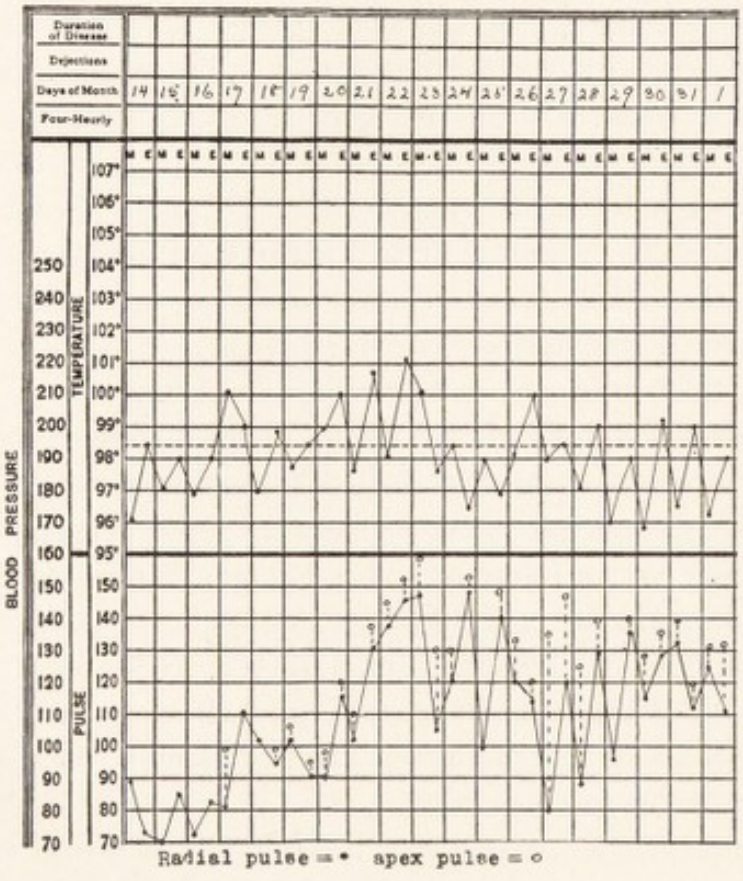



FIG. 2.

the diagram. Action absolutely irregular, not rapid. Sounds of good quality. $P_2$ equal to $A_2$. Systolic murmur heard during the longer systoles. Pulses low volume and tension. Arteries normal. Blood pressure 120/90–95/70. Abdomen and genitals negative

except for a reducible left inguinal hernia 2.5 cm. in diameter and a very slight right inguinal hernia. Rectal examination. A few external tabs. Extremities negative. Pupils equal, regular, reacted poorly to light and distance. Reflexes normal.

Temperature and pulse as shown in the chart. Respirations 19–35 until January 30, then 37–50. Urine $\bar{3}$ 16–52 in 24 hours. Sp. gr. 1013–1034. Acid at all four examinations. A slight trace to a very slight trace of albumin at the last two, leucocytes at three. No sugar. Renal function 50%. Blood: Hgb. 75%–80%. Leucocytes 8600–52,000. Polynuclears 73%. Reds and platelets normal. Wassermann negative. Electrocardiograms, January 15, auricular fibrillation, coarse. Rate 85. Flat T wave. On ten days, Janunary 17–28, auricular flutter. See table. X-ray. See Fig. 3. Chest 25.5 cm. transverse inside diameter.

| | Auricular rate | Ventricular rate | Block |
|---|---|---|---|
| Jan. 17 | 265 | 130 | Varies; usually 2:1 |
| Jan. 18 | 255 | 130 | |
| Jan. 19 | 255 | 130 | |
| Jan. 20 | 285 | 120 | Varies. |
| Jan. 21 | 325 | 162 | 2:1 |
| Jan. 22 | 320 | 160 | 2:1 |
| Jan. 24 | 340 | 170 | 2:1 |
| Jan. 25 | 330 | 165 | 2:1 Sometimes 3:1 |
| Jan. 27 | 320 | 160 | 2:1 |
| Jan. 28 | 280 | 140 | 2:1 |

Dr. Paul D. White noted January 25, "Under quinidine sulphate auricular flutter was induced. When the quinidine was stopped and digitalis given to saturation the rate of the flutter circus movement increased from 255 to 340, but neither normal rhythm nor auricular fibrillation has ensued. Coincident with the progress of the flutter there has been an acute infection (fever, leucocytosis) responsible for at least a part of his symptoms of discomfort. The infection, probably respiratory, apparently subsiding now." The night of January 27 the flutter showed signs of breaking. Every five or ten beats there occurred several ventricular escapes. January 29 the heart was still in flutter, but the ventricular rate was slower. He was very restless and nervous. January 31 the heart action was still regular and rapid. There were râles at the bases, more on the left.

The patient was cyanotic and looked very ill. He grew rapidly worse. The chest was filled with râles with fluid at the bases. February 2 the patient died.

Dr. White adds, "The action of quinidine was unfavorable in this case."

*Clinical Diagnosis.*—Chronic valvular heart disease (mitral stenosis).

Myocardial insufficiency.

Auricular flutter.

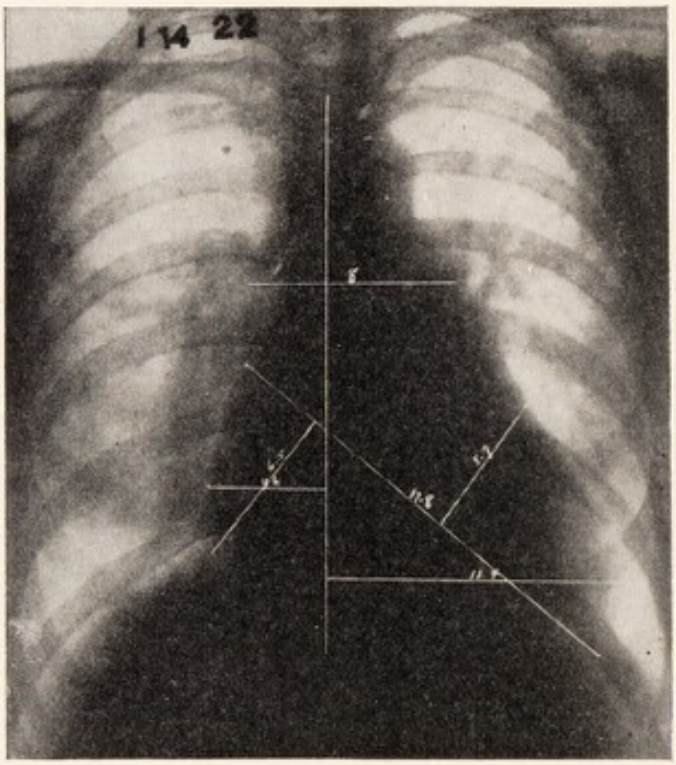



FIG. 3.—Necropsy 4292. Mitral stenosis. Mural thrombi of the auricular appendices. Embolic thrombosis of branches of the pulmonary artery. X-ray shows general enlargement of the heart shadow, the greatest prominence in the region of the auricles, particularly the left. (Roentgenological Department, Massachusetts General Hospital.)

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the mitral valve with stenosis.

Acute endocarditis?

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Infarcts.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral valve (stenosis).

Hypertrophy and dilatation of the heart.

Mural thrombi of the auricular appendices.

Embolic thrombosis of branches of the pulmonary artery.
Infarcts of the lungs.
Chronic passive congestion, general.
Acute serofibrinous pericarditis.
Acute serofibrinous pleuritis, left.
Chronic pleuritis, right.

DR. OSCAR RICHARDSON: The head was not examined. The face was dusky and the lips purplish. The skin elsewhere was smooth and pale. There were no signs of purpura at this time. The feet and ankles were slightly swollen and pitted slightly. The subcutaneous tissues were a little wet. The stomach and intestines showed chronic passive congestion. The mesenteric and retroperitoneal glands were negative. The liver at the time of necropsy was at the costal border.

The pleural cavities: on the right 200 c.c. of thin brownish fluid, clear; on the left 1500 c.c. of thin cloudy fluid and fibrin; and the lower lobe of the lung on this side was coated with dirty reddish fibrinous material. The trachea, bronchi and bronchial glands were negative. The lungs showed chronic passive congestion, many infarcts, and in places the branches of the pulmonary artery leading to the areas of infarction showed occluding plugs.

The pericardium contained about 200 c.c. of thin cloudy fluid with fibrin. The visceral and parietal surfaces were reddened and coated with fibrin. The heart weighed 545 grams, considerably enlarged. The myocardium was of good consistence, pale brown-red. There was no evidence of myocarditis. The following measurements are interesting because they picture mitral stenosis. The left ventricular wall 10 mm., a little flabby; the right 6 mm., good consistence, the columnae carneae a little flat on the left, thickened on the right. The cavities on the left side: the ventricle was full sized and showed nothing remarkable; the left auricle was full sized and the wall was thickened. In the left auricular appendix a short distance from the tip, a mural thrombus,—not uncommon in mitral stenosis. The right ventricle and right auricle showed considerable dilatation, and in the right auricular appendix a few mural thrombi, bits of which had passed over into the branches of the pulmonary artery, appearing as the plugs mentioned. The aortic valve showed a little fibrous thickening, nothing very definite, which faded out into the marked thickening of the mitral valve. It is impossible definitely to say whether there had ever been any infection on that valve or whether it was a compensatory thickening. The only thing that makes one

think there may have been infection was that below the cusps on the endocardium there was a small fibrous patch, which is abnormal; and that possibly may have been a little patch of endocarditis *x* years ago. It is of no great importance now, and of course had no relation to the hypertrophy and dilatation of the heart. The mitral valve was in the form of a flattened ovoid mass; and in the center of this mass was the crescentic orifice of the valve, three and a half cm. long, with the margins practically fused for one centimeter and closely approximated eleswhere. Their surfaces were very irregular, somewhat coral-like in places, and altogether the valve showed marked deformity and stenosis, with thickening and shortening of the chordae tendineae.

The coronaries showed a little scattered sclerosis. The aorta and branches were out of the picture. The liver showed nutmeg markings, that is chronic passive congestion. The gall-bladder, bile-ducts, spleen, adrenals and kidneys were negative except that they showed more or less congestion. The genito-urinary organs were negative.

**Necropsy 4605**

An American railway clerk of twenty-six entered January 3, 1920, complaining of dyspnea and weakness. His mother died of cancer of the stomach. As a child the patient's health was not good. He easily took infections, and had pertussis, scarlet fever, mumps, diphtheria, measles, and rare earache. At twelve he was laid up in a hospital three months with "nervous prostration." From the age of seventeen to twenty-two he had malaria for several weeks every summer. He had rare sore throats. A year before admission he had influenza lasting ten days. With this he had bilious attacks and vertigo. He occasionally urinated once at night. Several years ago he weighed 125 pounds, his best weight. His usual and present weight was 122. Until six months before admission he sometimes drank large amounts of alcohol.

At seventeen, during a severe cold, he had with rather sudden onset an attack of "congestion." He was put to bed and had dyspnea, orthopnea, slight fever, persistent deep-seated cough with blood streaked sputum, and frequent attacks of vomiting without nausea, with small amounts of blood in the vomitus. He noted for the first time palpitation and precordial pain, at times severe, preventing sleep. For over a week he was confined to bed. Within three weeks he was allowed to go back to his work. Except for general moderate asthenia, dyspnea and palpitation on exertion there had been practically no change in his condition until two years before admission. Then

he had a sudden chill in the afternoon. The next morning a moderately severe dull pain in both lower quadrants gradually developed. He was taken to a hospital. The next day moderate fever developed. The pain although continuous for two days was not severe enough to prevent sleep. He was in a hospital a week, during the last four days of which he was moderately jaundiced. The fever lasted only four days and was never over 101°. Ten weeks before admission he had a fleeting chill. The next day he observed swelling of the precordia and sudden onset of dyspnea when walking slowly on a level. He returned to work, but was dyspneic whenever not sitting absolutely quiet. The precordial swelling increased. During the next five weeks he had palpitation, tachycardia, dyspnea on walking even a few steps, orthopnea, enlarged liver and swollen abdomen. The ankles

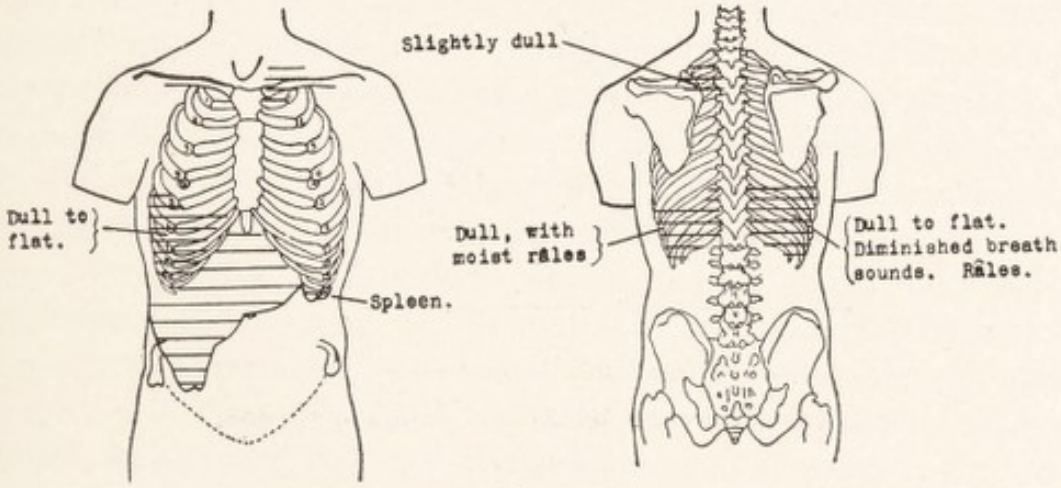



FIG. 4.

and lower legs were edematous. After a week of complete rest in bed with medication the orthopnea, dyspnea and palpitation disappeared and the swelling of the abdomen, ankles and precordia markedly diminished. During this period he had moderate cough. After working for two weeks the symptoms gradually increased. An increasing heavy sense of discomfort in the abdomen, although painless, tended to keep him awake at night.

Examination showed a fairly well developed and nourished man with bright red lips and cheeks. The sclerae, throat and tonsils were slightly injected. The jugulars were much distended. There was slight general adenopathy. There was bulging of the precordia. The lungs signs were as shown in Fig. 4. The diaphragm excursion was slight. There was diffuse cardiac impulse over the whole precordia and felt in the midaxillary line. The sounds were absolutely irregular in quality. There was a pulse deficit of 52. The first

sound at the apex was loud and snapping. The pulmonic second sound was accentuated. A soft blowing systolic murmur was heard over the precordia, loudest at the apex. There was a short middiastolic murmur, loudest at the apex. The pulses were of small volume and tension. The beats varied in force. The blood pressure was 150/85 to 110/80. The abdomen was slightly distended, with shifting dullness in the flanks. The liver dullness extended from the fourth rib to 10 cm. below the costal margin. The edge was felt 15 cm. below, slightly tender. The spleen was just felt. There was moderate edema of the ankles. The pupils were dilated. The right was greater than the left. The knee-jerks were sluggish.

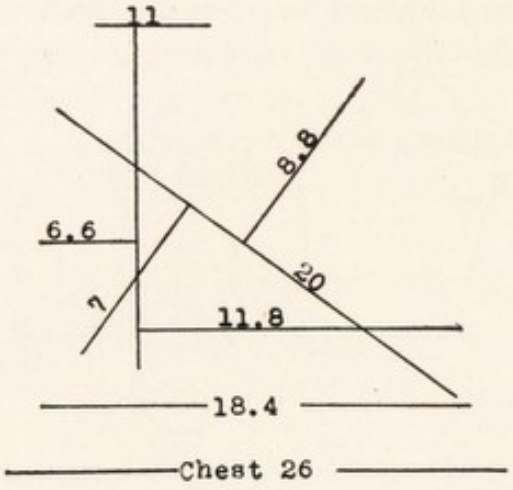



FIG. 5.

The temperature was 96.1° to 99.9°, the pulse 28 to 84 (radial), the respiration 12 to 26. The output of urine was 16 to 90 ounces, the specific gravity 1.018 to 1.030. The urine was cloudy at three of five examinations, alkaline at one, showed a slight trace to the very slightest trace of albumin at two, rare leucocytes at three. The renal function was 60%. The hemoglobin was 70%. The leucocytes were 6400 to 15,800, the polynuclears 70%. A Wassermann was negative. X-ray January 7 showed the lower part of the right chest dull, the costophrenic angle obliterated, perhaps because of an extensive high diaphragm on this side, perhaps because of a little fluid at the right base. Both lung roots were considerably thickened and the lung markings prominent. There were some calcified glands. The heart shadow was very much enlarged. There was marked prominence in the region of the left auricle. January 21 the shape of the heart was much the same as at the previous examination. There was marked increase across the extreme upper part of the heart shadow. Although

there was general enlargement, the *prominence was most marked* in *the region of the left auricle.* Electrocardiogram January 3 showed auricular fibrillation, rapid ventricular rate, 120, right ventricular preponderance. January 5 there was auricular fibrillation, ventricular rate about 55, right ventricular preponderance, bigeminal pulse due to ectopic ventricular contractions (probably the result of digitalis).

Under a single dose of digitalis the pulse dropped markedly and the irregularity and the deficit were less marked. "The prognosis is absolutely bad,—probably a matter of months at most." By January 10 there was no friction over the liver. The ascites was much less. January 17 he was still digitalized on the initial dose of one gram. January 29 he was discharged partially relieved.

December 14, 1923, nearly 4 years later he was again seen unconscious and *in extremis.* His sister said that under treatment in the Cardiac Clinic of the Out-Patient Department of this hospital he had been in "good health" apparently, with no complaints. His last visit to the Clinic was in August, four months before admission. Since his father's death the previous spring he had been somewhat more introspective and worried about himself than previously. The morning of admission he felt well and cheerful. He arrived at his place of business without in any way over-exerting himself, and sat down at his desk. A little while later his head was seen to drop forward, he stiffened out, and then collapsed in his chair. He had been unconscious from 7 a.m. to 1 p.m. A physician had given stimulants, etc., without effect.

Examination showed him cyanotic, with pinkish frothy sputum pouring out of the mouth and nose. There were bubbling râles throughout both chests. The cardiac dullness was increased. The pulse was not felt at the wrist. There was no edema. There is no record of the chart, urine or blood. The pulse was slightly irregular at first, later very irregular. The blood pressure was 100/-, gradually dropping.

Caffein, adrenalin and atropin were injected intramuscularly, digifolin intravenously and adrenalin, caffein and atropin intracardiacally with only slight temporary effect. Venesection was without effect, as the blood was so thick and black it barely dripped. The patient stopped breathing, and in spite of cardiac massage and artificial respiration died half an hour after admission.

*Clinical Diagnosis (from Hospital Record).*—Acute pulmonary edema.

Mitral stenosis.

*Dr. Richard C. Cabot's Diagnosis.*—Acute pulmonary edema. Chronic endocarditis of the mitral valve. Stenosis.

Ball thrombus?

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral valve. Stenosis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Slight edema piae.

Obsolete tuberculosis of a mesenteric lymphatic gland.

Slight chronic pleuritis, left.

Dr. Richardson: There was a small amount of thin pale fluid in the peritoneal cavity. The esophagus and gastro-intestinal tract showed some areas of ecchymosis and reddening, the reddening of course that of passive congestion. One of the mesenteric glands showed marked calcareous degeneration,—obsolete tuberculosis.

The liver was two and a half cm. below the costal border. The diaphragm on each side was at the sixth rib. In each pleural cavity there were a few c.c. of thin fluid slightly blood stained. There were no adhesions on the right; on the left a few in the region of the upper lobe. The trachea and bronchi contained much pinkish froth and frothy fluid. As far as that goes it would indicate, of course, edema of the lungs. The lungs showed passive congestion and also much edema.

The heart weighed 560 grams. That is considerably enlarged. The myocardium was pale brown-red, with a right ventricle wall of five to eight mm., indicating of course at once some obstruction on the left side or some obstruction in the pulmonary valve. The pulmonary valve was negative. The tricuspid valve was increased in circumference, but otherwise was negative. There was no hypertrophy of the left ventricle wall. The cavity of the left ventricle was full sized. The left auricle was considerably dilated and the wall somewhat thickened. The right cavities showed much dilatation. The auricular appendices were free. The foramen ovale was closed.

The mitral valve showed much deforming fibrosis with areas of fibrocalcareous change. This deforming process formed a flat collar about the orifice of the mitral and reduced the opening to a button-hole-like cervice one and a half cm. by five mm. There was some thickening and fusion of the chordae tendineae,—all told, a marked

mitral stenosis. In places on the endocardium of the left auricle, above the mitral valve, there were here and there small areas of fibrous roughening, and in one or two places these were covered with thin smooth calcareous shells, the end result, of course, of endocarditis extending to the auricular surface. The aortic valve measured 6.5 cm. and was negative. The coronary arteries were free and negative. Altogether this was a typical picture of mitral stenosis, with hypertrophy of the right side of the heart, some dilatation on the left, more especially in the left auricle, and marked hypertrophy and dilatation on the right. The aorta and great branches, the pulmonary artery and veins, the venae cavae, the portal vein and radicles were frankly negative.

The liver weighed 1325 grams (normally 1200–2400) and showed chronic passive congestion. It was 2½ cm. below the costal border.

The spleen weighed 354 grams (normally 80–180), was moderately enlarged and showed chronic passive congestion, that is, dark brownish-red elastic tissue. There are three accessory spleens. The kidneys were rather small but showed chronic passive congestion.

Dr. Cabot: Dr. Richardson, do you realize that this man went to his job feeling perfectly well on the morning that he was brought here, that he was brought here at one o'clock, and died in half an hour,—went from good health to death in four and a half hours,—and that nothing that you have read in any way explains that—does it?

Dr. Richardson: Of course if one says edema of the lungs, you ask me what that is due to; and then if I say the heart muscle gave out, we are still in the same place. There was no medication in this case, was there?

Dr. Cabot: I do not believe that anything given him killed him. He was under our care in the Out-Patient Department when last heard from before the day of death.

Dr. Richardson: I mention that on account of a case that stands out very distinctly in my memory, a medico-legal case in which the patient was being treated at the Out-Patient Department. At her death, which was rather sudden, the case was made medico-legal. I think I found as many as half a dozen bottles of all kinds of drugs for heart disease which she had accumulated and kept under the pillow, eating them as she saw fit. That is a remote possibility of course, but it could happen. It was a perfectly clear-cut case so far as the anatomy went.

Dr. Cabot: The case seems then merely to give one more illustration of how little we know about the immediate cause of death. It

does not seem as if in medical literature our complete ignorance on that matter has been stressed as much as it should be. We know pretty well why he lived for four years; we do not know at all why he died at the end of four years. I do not know any more striking example of how little we know in these cases.

DR. RICHARDSON: Associated with this comparatively sudden exit did he do any lifting or anything unusual?

DR. CABOT: No, it is particularly stated that he did not.

AN INTERNE: He rode seven miles in an open car coming here after this thing had started, and was very much worse after he got here. He was riding against the wind.

DR. CABOT: But he collapsed at his desk before that.

DR. RICHARDSON: These patients do that, don't they—I do not mean collapse to death necessarily, but collapse?

DR. CABOT: No, I do not think they do when in good compensation, as he was.

DR. MEANS: Did the microscopic examination of the heart muscle show anything definite that might throw any light on this?

DR. RICHARDSON: No. The myocardium macroscopically and microscopically was negative as regards chronic interstitial myocarditis. There was of course the marked hypertrophy and dilatation of the right heart, and the mitral orifice or crevice measured 1½ cm. by 5 mm., as compared with the usual measurement of 10 cm. in circumference.

### Necropsy 4525

A Lithuanian rubber factory operative of thirty-three was referred November 27, 1922, from the Out-Patient Department, where he came complaining of swelling of the face and legs. Examination in the Industrial Clinic showed pallor, edema of the eyelids, pyorrhea, black dots on the gums, and weak hand grip, both sides. His past history was negative except for occasional headaches and nosebleeds. He smoked twenty cigarettes a day and occasionally took alcohol.

Seven weeks before admission he began to have cough with a little white frothy sputum, followed by a pain in the stomach. November 20 his legs and ankles began to swell. Since this time he had had frequent urination by day and two or three times at night, and dyspnea, occasional vomiting, and a cough which disturbed his sleep. The urine had been cloudy and his mouth had felt dry in the morning. He felt very well.

Examination showed a well nourished man with puffy face and eyelids and some orthopnea. There were many acne scars on the

back of the neck and head. The mucous membranes were pale. There were a few bean-sized cervical and some small axillary glands. The lungs were clear in front. The lungs showed râles in both backs. The apex impulse of the heart was seen and felt in the fifth space 14 cm. to the left. The left border of dullness was 14 cm. to the left of the midsternum, the right border 4 cm. to the right, the supracardiac dullness 7 cm. There was a presystolic thrill. The pulmonic second sound was markedly accentuated. A rumbling diastolic murmur followed the first sound, which was largely replaced by a loud systolic transmitted to the axilla. Both murmurs were best heard over the apical region. The blood pressure was 160/85 to 140/95. The abdomen was slightly prominent, with edema of the wall and shifting dullness in the flanks. There was massive edema of the feet and legs. The fingers and toes showed marked clubbing. There were many purpuric spots over both lower legs. The knee-jerks were unequal, the pupils normal.

The temperature was 97° to 100.6°, the pulse 62 to 110, the respiration 20 to 31. The output of urine was 32 to 62 ounces, with one drop to 16 ounces January 3. The specific gravity was 1.008 to 1.020. The urine was cloudy at nine of twelve examinations, alkaline at four, and showed a very slight trace to a very large trace of albumin at nine, red blood cells at all but one, 50 to 150 per high power field at all but three, leucocytes at seven, an occasional granular cast at the first three, hyaline casts at the last. The renal function was 30 to 38%. The hemoglobin was 60 to 70%, the leucocytes 4400 to 7000, the polynuclears 72 to 48.5%, the reds 3,392,000 to 4,976,000. December 22 there was 9.5% atypical mononuclears, some vacuolated cells, lymphocytes of all sizes and shapes, many not typical, platelets apparently diminished, red cells slightly achromic, a moderate number of small oval cells and some true microcytes. A blood culture December 16 showed Gram-positive diplococci; others December 19 and 22 were negative. The non-protein nitrogen was 50.7 mgm. to 47.1 mgm. A Wassermann was negative. Examination of the fundi December 22 showed the retinal vessels tortuous, the nerve heads somewhat blurred; possibly small congenital hemorrhage of the left nerve head. A throat consultant found the tonsils small, not infected. The nose showed a tendency to atrophic retinitis. There was no pus. The sinuses were clear. No focus of infection was found. X-ray November 29 showed the sinuses normal in density and outline. The right frontal sinus was somewhat larger than the left. The teeth were negative.

December 22 there were no changes as far as could be made out in the feet. The fingers however showed distinct increase in the size of the tips of the terminal phalanges. (See illustration.)

By December 2 the edema was going. There were still a good many red cells and a trace of albumin in the urine with a little pus. No foci of infection were discoverable. His industrial history was gone into twice without proving much as to the possibility of lead posioning. His work had nothing to do with mixing. His house was piped with lead pipes. His gums showed a blue line, but with the



FIG. 6.—The tips of the terminal phalanges show distinct increase.

magnifying glass it had not the characteristics of a lead line. Of the classical symptoms he had had only constipation. Reëxamination of an over-stained blood smear showed no stippling. The platelets were increased. December 17 there were a few petechiae on the feet. December 23 the visiting physician found the spleen three inches below the costal margin. The red blood count tended gradually downward. There were occasional new pin-point purpuric spots on the feet. *The spleen enlarged slowly but steadily.*

January 20 he was discharged unrelieved.

After his discharge he felt much better for two months. March 12, 1923, records of the Out-Patient Department showed edema of the extremities, purpuric spots, râles in the lungs, heart condition

about the same, blood pressure 140/98, hgb. 60%, reds 3,880,000, smoky urine. Orders, digitalis gr. iss. About March 20 he began having dyspnea on slight exertion and general weakness. Soon after this he began having cough, at first unproductive, later with white sputum. April 6 he began to have edema of the lower extremities and Aprial 8 edema of the eyelids. April 6 he noticed also numerous spots on the legs. Since his discharge he had slept with two pillows. He had urinated twice at night until April 4, then four or five times. He had stayed at home, and his greatest exertion had consisted in daily walks.

April 10, 1923, he was readmitted to the wards. Examination showed frequent cough, pyorrhea, shotty cervical and left axillary glands, and engorged veins in the supraclavicular space on the right. The chest expansion was limited. The right chest was full of coarse

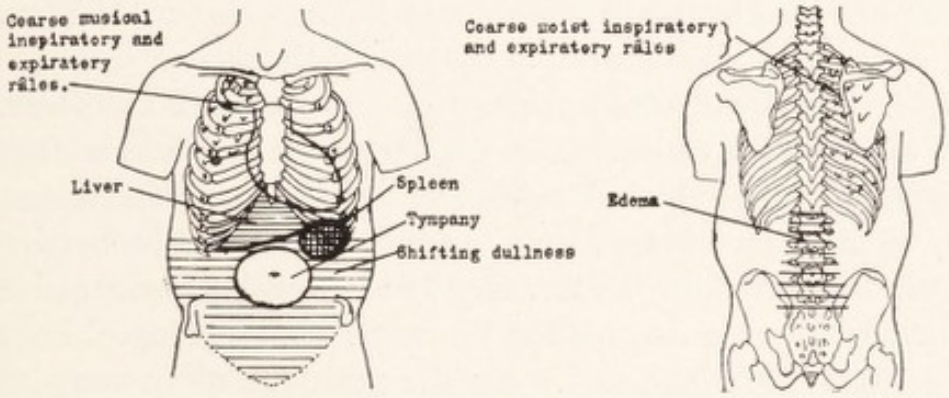



FIG. 7.

musical inspiratory and expiratory râles. There were a few coarse râles at the left posterior apex. The heart was enlarged downward and to the left. The impulse was heaving. At the apex, 14 cm. to the left in the sixth space, was a short faint systolic murmur. A long rough harsh diastolic was heard over the lower precordium. At the base the pulmonic second sound was accentuated and greater than the aortic second, with a blowing systolic and diastolic murmur. There was a questionable thrill at the apex. The blood pressure was 120/85 to 150/90. The abdomen showed a large area of shifting dullness. (Fig. 7.) The liver was four finger-breadths below the costal margin, the spleen one or two finger-breadths below. There was marked edema of the sacrum and ankles. The finger tips were clubbed, with an inflammatory zone around the nails. There were red thickened areas the size of a dollar on the palms.

The temperature was 94.6° to 102.7° (rectal), the pulse 53 to 127, the respiration 20 to 34. The output of urine was 32 to 80 ounces,

the specific gravity 1.010 to 1.014. The urine was reddish and cloudy at one of eight examinations, showed a slight trace to a large trace of albumin, leucocytes, many red blood cells, and rare to many granular casts at all, cellular at four. The renal function was 10 to 15% (four tests). The hemoglobin was 60%, the leucocytes 9700 to 25,000, the polynuclears 64%, the reds 3,800,000 to 3,960,000, with some variation in size and occasional oblong cells. The platelets were descreased. Blood cultures April 21, 23 and 24 were negative. The non-protein nitrogen was 58.2 mgm. April 13, 64.5 mgm. April 18, 91 mgm. April 24, 110 mgm. May 2, 163.5 mgm. May 8. A Wassermann was negative.

It was difficult to restrain the patients fluid intake. By April 17 he was losing a little puffiness about his face. The temperature was a little more steady and the pulse slightly lower. The administration of mercurochrome April 23 was followed by a chill, and the patient felt very uncomfortable the rest of the day.

April 25 a hematoma appeared on the inside of the cheek as a result of biting the cheek. Two days later the area was infected and a crusted gray area showed. A smear showed many organisms of all sorts, many spirochete forms, bacilli, cocci, streptococci, some fusiform bacilli. April 28 Dr. C. Morton Smith noted considerable numbers of the organisms of the Vincent's angina group. The cheek was painted with diarsenol. By the 30th the ulcer was a sizable slough.

The patient became increasingly edematous and stuporous. The leucocyte count and the non-protein nitrogen rose. May 8 the patient quietly died.

*Clinical Diagnosis (from Hospital Record).*—Rheumatic heart disease.

Mitral stenosis.

Subacute bacterial endocarditis.

Acute nephritis.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the mitral valve.

Mitral stenosis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Infarcts of the spleen and kidneys.

Terminal infection?

*Anatomical Diagnosis.*—Chronic and subacute endocarditis of the mitral valve.

Hypertrophy and dilatation of the heart.
Chronic passive congestion, general.
Hydrothorax, right.
Ascites.
Slight hydropericardium.
Anasarca.
Infarcts of the spleen and kidneys.
Chronic pleuritis, left.
Obsolete tuberculosis of a bronchial gland.
Chronic peritonitis.
Wet brain.

DR. RICHARDSON: This man had pop-eyes.

A HOUSE OFFICER: That should have been noted in the record. There was an appearance of exophthalmos, but not very marked.

DR. CABOT: That rather goes to support those who believe there is nephritis in the case. That goes with nephritis.

DR. RICHARDSON: The pia showed marked edema, infiltration with thin pale fluid, and there was considerable in the brain tissue. The vessels of Willis, the sinuses, the pineal and pituitary glands were negative. That leaves nothing but a wet brain.

The fingers and toes showed well marked clubbing.

Much frothy fluid ran from the mouth.

The feet and ankles were swollen and pitted on pressure, and there was a little edema of the thighs. Scattered over the legs and feet and on the skin of the trunk were those purplish areas spoken of in the clinical picture.

The peritoneal cavity contained at least 200 c.c. of clear straw-colored fluid. The serosa of the intestine showed scattered over it here and there small hemorrhagic areas.

The gastro-intestinal tract on section showed chronic passive congestion.

The anterior margin of the right lobe of the liver was twelve and a half cm. below the costal border. The diaphragm on the right was at the fifth rib, on the left at the sixth interspace. That is down at least a rib's width on each side.

The right pleural cavity contained about 1000 c.c. of fluid. On the left there was no fluid, but the cavity was obliterated by old adhesions. These adhesions were juicy, but there was no free fluid.

There was much brownish-red frothy fluid in the trachea and bronchi. The bronchial glands were slightly enlarged, brownish-red,

juicy, congested; one of them was transformed into fibrocalcareous material, obsolete tuberculosis.

DR. CABOT: That is all an old process?

DR. RICHARDSON: Absolutely. The difference between the right and the left lung is slightly important. We have much fluid on the right, none on the left. But the left lung was much wetter than the right one. The left of course was bound up in the adhesions.

The pericardium showed 100 c.c. of thin clear fluid,—no pericarditis. The heart weighed 685 grams. For this man a heart of 350 grams would have been a fair size. In the text books they give tables of heart weights, but every man's heart must be considered in comparison with the makeup and muscular development of the man. The myocardium was of fair consistence and brown-red. I could not make out any areas of myocarditis, meaning by myocarditis the replacement of muscle tissue by fibrous tissue. The thicknesses were four to five mm. on the right, ten on the left,—perhaps a little thin on the left, and full thickness on the right. The columnae carneae were well marked. So that all told it was a muscular heart.

There was much dilatation on the left, moderate dilatation on the right. The valve circumferences were as follows: the mitral 13½ cm. —usually ten; but in spite of that the mitral was the seat of chronic endocarditis and some subacute endocarditis. That appeared on the valve in this fashion. The curtain itself showed some diffuse fibrous thickening rather generally and in two places was definitely thickened. Rising from that was a fringe of small masses which at the bases were perhaps a little firm, but as we went towards the tip grew spongy and soft. So that we have some old endocarditis and on top of it this subacute endocarditis.

DR. CABOT: There is rather surprisingly little chronic endocarditis.

DR. RICHARDSON: At one end of that valve there was quite a large patch extending up on the endocardium of the left auricle, occupying an area at least three or four cm. across.

DR. CABOT: Is that the subacute process?

DR. RICHARDSON: Yes, on its surface; but at the base it was a little fibrous. Again, under the mitral curtain it curved around on the under surface of the valve.

The aortic, tricuspid and pulmonary valves were out of the picture except for some increase of the circumference of the tricuspid.

Here we have a rather curious condition,—a valve which measured laid open 13½ cm. and yet had masses of vegetations on it which when the valve was shut one would think would decrease its

circumference, a paradoxical condition. When those masses were together I should say there was a little decrease. There was an increase in the circumference when the valve was laid open, but when that was brought together there probably would be a slight decrease in the circumference. The blood would have a smaller space to go through.

The coronaries were free and negative. That gives us a chronic and subacute endocarditis with hypertrophy and dilatation; and I could find nothing else as the basis for that hypertrophy and dilatation.

The aorta and great branches, the pulmonary artery, veins and vena cava were negative. In other words, the circulatory apparatus, except that it had been the means of conveying from this mitral valve smaller and larger bits to the spleen and kidneys, was out of the picture. It did convey to them the small bits of vegetation and it did set up in them infarctions.

The liver, which was a little large, showed beginning congestion. The spleen weighed 465 grams and was the seat of infarcts. The adrenals were negative.

The kidneys were rather large, weighing 375 grams. They showed the infarcts, and macroscopically I could make out no definite nephritis. Of course there was passive congestion. There was nothing the matter with the kidney vessels, no arteriosclerotic changes in the renal branches; that is, none of the fibrosis which we see in the section surfaces and which is one of the landmarks of arteriosclerotic nephritis. The only other thing in the kidneys was that in each kidney there were infarcts. Some of these infarcts seemed to be old, others more recent. That is in harmony with the picture on the valve,—some of it old, organized, some of it softer and more in the subacute stage.

Culture from the heart blood showed no growth. This case from the anatomical side looked like one of those where the organism is of the *viridans* group; and in those cases it is not unusual to get a culture and also not unusual to find none. Once in a while we get them at necropsy, and in one case I have seen the blood stream was negative and I found the organsim in an infarct of the spleen, which was soft and broken down. But it all depends on whether the fish are running in the stream and whether we know the hour when to fish. At intervals the stream seems to clear up and the organisms all go into their lairs. They come out again, and if we are there with the material we can get them.

In the region of the gall-bladder there were a few old adhesions.

DR. CABOT: Was there any change in his fingers between the first entry and the second?

A HOUSE OFFICER: Yes, sir. There was apparently a very rapid development. There were no hemorrhages in the finger tips, although they were present in other parts of the body. The clubbing even at the firsty entry was very marked.

DR. CABOT: I think this is a very interesting case. Perhaps the greatest interest is that he did not have nephritis.

A PHYSICIAN: Will you review why you barred that?

DR. CABOT: In the first place, he never had a high blood pressure.

A PHYSICIAN: What do you call a high blood pressure—one hundred and sixty?

DR. CABOT: I depend a good deal more on the diastolic than on the systolic. He must have a diastolic of over one hundred and a systolic of more than this—I won't try to say how much more—before I call it elevated.

It is only at the end that he had any high non-protein nitrogen, only at the end that he got anything approaching fixation of gravity, only at the end that his renal function by the red test was low at all. He never had any uremia, or anything to approach a uremic state. The clubbed fingers which he had from the start do not come from nephritis. They come only when we have something other than nephritis; he had that something from the beginning. He never, even to the very end, had a low enough renal function by the red test to make us sure of chronic nephritis. He never got below ten.

A PHYSICIAN: Were the emboli septic emboli?

DR. CABOT: I suppose so. We have to say they were bacterial emboli, but they were of low virulence. They never produced any sepsis around the nails.

It seems to me very interesting to know that we can get a high rising non-protein nitrogen from renal infarcts. I was banking a good deal on the fact that I am confident that when a spleen grows under observation it is infarcted. If we have splenic infarct, then renal too. If infarcts are blocking the kidney that will account for the loss of kidney function even though there is no nephritis.

The condition of the mitral valve seems very interesting too, because on the whole we have to say he *did* have mitral stenosis even though the circumference was increased, because the masses on the valve narrowed the opening.

**Necropsy 4289**

An American elevator man of forty-four was referred to the wards from the Out-Patient Department January 25, 1921, where he had come complaining of dyspnea of three weeks' duration. He had "rheumatism" at twenty-two in the soles and small joints of the feet; no fever; at twenty-five or thirty "soft chancre" for a week. He denied syphilis. At thirty he had a bubo, which "broke" and healed in a week. He drank much beer until prohibition in 1920. He rarely urinated at night. His best weight was 145 pounds, his usual weight 135.

A year before admission he noticed palpitation on exertion, brought on he believed by the strong winter winds. For three months this persisted. One evening he had hemoptysis five or six times, though he had no cough or edema. After this he was better, though the palpitation on exertion persisted in less degree. Five weeks ago the strong winter winds brought the dyspnea back. Dull precordial pain always followed the dyspnea, at first for short periods, later for longer. A week and a half later he got his feet wet, "caught cold," and began to have attacks of coughing with dyspnea once a night, the attacks increasing in length until they had lasted all through the last three nights, with orthopnea and "asthmatic" coughing and white sputum, lately as much as a cupful, mostly expectorated in the evening and early night. Three weeks ago he began to have fairly sharp steady pain in the precordia and left lower chest in the posterior axillary line when he opened the elevator door. This seemed to be getting worse.

Examination showed a fairly well nourished man weighing 120½ pounds, with flushed cheeks. (Cyanosis of the nose and lips is mentioned in the Out-Patient examination.) There were some carious teeth and pyorrhea. The nasal septum deviated slightly to the right. There was mucus in the nasopharynx. The lung signs were as shown in Fig. 10. The apex impulse of the heart was seen and felt in the fifth space 8.5 cm. to the left. The measurements by percussion and by X-ray are shown in Figs. 8 and 9. The action was absolutely irregular. The sounds were of poor quality, the pulmonic second sound greater than the aortic second. There was a presystolic roll at the apex and a systolic murmur over the precordia. No thrill was felt. The pulses were normal, the artery walls palpable. The systolic blood pressure was 135, the diastolic 75. Electrocardiogram showed auricular fibrillation, ectopic ventricular contractions, rate 100, only moderately irregular. The liver dullness

began at the fifth space. The edge was felt 4 cm. below the costal margin. The genitals, extremities, pupils and reflexes were normal.*

January 25 and 26 the temperature was 100.4° to 98°, the pulse 100 to 83, the respiration 32 to 20; afterwards the temperature was not elevated (occasionally 97° or a little below), the respirations were not remarkable, the radial pulse was 92 to 50; deficit 2 to 15




FIG. 8.—Measurements by percussion.

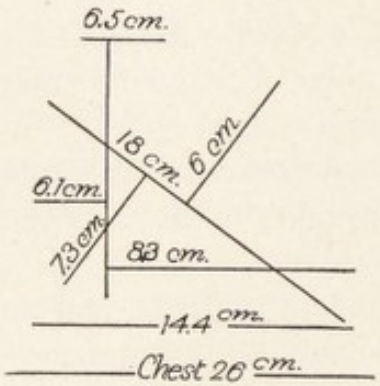



FIG. 9.—Measurements by X-ray.

The amount of urine was 15 to 80 ounces, the specific gravity 1.034 to 1.016. The urine was cloudy and alkaline at four of five examinations; no albumin or sugar. The renal function was 50%. The blood was normal. Two Wassermanns were negative. Fluoroscopic examination showed the findings seen in Fig. 11, and also showed the diaphragm very low, and forced respiratory excursion limited on both sides.

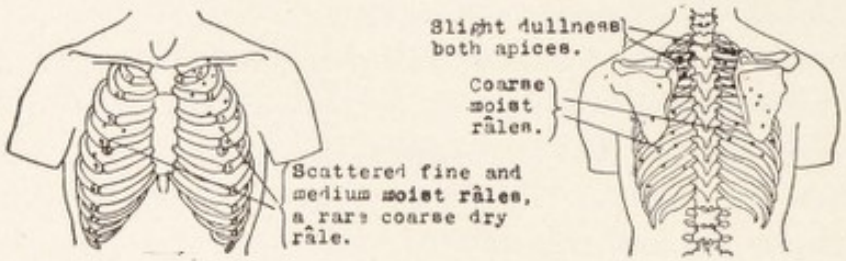



FIG. 10.

The orders were as follows. January 25 soft solids, codein gr. ¼ by mouth at 7:30 p.m., repeat s.o.s. tonight; January 26 saturated

* (From the Out-Patient record.) Heart. Apex impulse heaving, slightly outside the nipple line. Some enlargement to right and left. $\frac{4\frac{1}{2}}{11}$ First sound at the apex very sharp, preceded by a crescendo murmur. A questionable thrill at the apex. Abdomen negative throughout. In the left inguinal region was a scar, probably of old bubo. Otherwise genitals negative. Extremities cyanosed. No edema.

solution KI ten drops t.i.d., codein gr. ¼ by mouth s.o.s.; February 7 fluid extract of convallaria 2 c.c. t.i.d.; February 10 convallaria 5 c.c. t.i.d.; February 11 convallaria 10 c.c. t.i.d.; February 12 convallaria 15 c.c. t.i.d.; February 13 discontinue convallaria; February 14 digitalis leaves gr. iii. t.i.d.

January 30 it was noted that the pulse was coming down and the heart's action was more regular. There were now a few extrasystoles. February 3 the deficit was quite small. Convallaria was

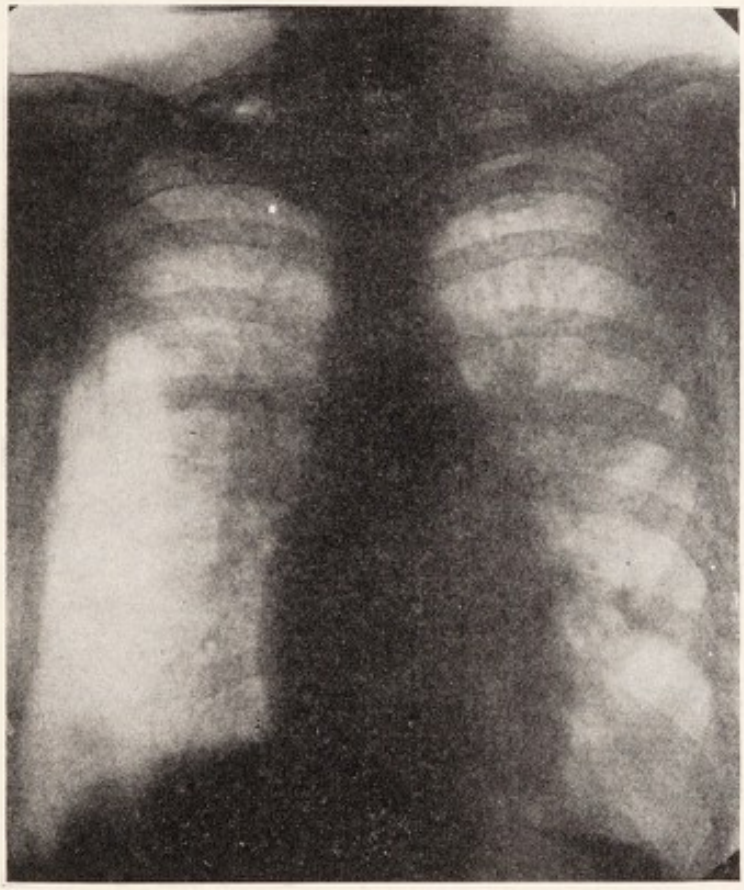


FIG. 11.—The heart shadow is distinctly enlarged, but almost entirely in the region of the auricles. There is marked prominence of the heart shadow in the region of the left auricle. The appearance is characteristic of mitral stenosis with dilatation of the auricles. The hilus shadows and lung markings are increased on both sides. The right upper chest is less radiable than the left. Extending across the right lung field from the hilus to the chest wall is a dense linear shadow suggesting thickened interlobar septum. The costophrenic angles are clear.

found to have practically no effect. February 14 he was put upon digitalis. February 15 there was improvement in the condition. February 19 he was discharged, with instructions to take a small dose of digitalis.

March 5, 1921, he returned to the Out-Patient Department complaining of fatigue and dyspnea on very little exertion, frequent swelling of the feet, and early morning cough. His appetite and sleep were fair. He was taking no digitalis.

Examination showed a pulse deficit of 27 (apex 106, radial 79). The heart showed absolute irregularity; a low rumbling diastolic murmur at the apex with a blowing systolic just inside (tricuspid area); the pulmonic second sound accentuated; the first sound at the apex not much accentuated. The lungs were filled with sonorous and crackling râles, groans and wheezes, most marked at the right base behind. There was cyanosis of the lips, nose, cheeks and ears.

He was ordered digitalis leaves 0.1 gram t.i.d. for three days, then once a day until March 10, when he was told to return to the Cardiac Clinic.

March 10 he felt quite well, though he was still short of breath on exertion. He had taken the digitalis as ordered. His weight was found to be 130½ pounds. Examination showed the apex pulse to be 58, the radial 56. He had the typical mitral facies. The diastolic murmur was very marked, typical of mitral stenosis. He was ordered to take a grain and a half of digitalis once or twice a day and report again in four weeks.

April 7 he felt pretty well. He had taken one pill daily except for three days when he took none. He was hoarse, and had been so at times for two months. On examination he weighed 133 pounds. The diastolic murmur was very marked. The left border was 10 cm. from midsternum,—a straight left border suggestive of left auricular enlargement. He was ordered a grain and a half of digitalis once a day; to report again in two months.

June 9 examination showed his weight to be 132 pounds. He felt fairly well. Electrocardiogram showed auricular fibrillation, right ventricular preponderance, a rate of 90. The murmur was as before. He was hoarse. He was ordered a grain and a half of digitalis once or twice a day; to report again in two months. He was referred to the Throat Department with a question as to laryngeal paralysis.

August 4 he was feeling very well. The hoarseness continued. He had been taking digitalis regularly twice a day for the first three weeks, then once a day. He was working regularly. On examination the apex rate was 72, the radial 64. There was a mitral diastolic murmur at the apex as before, and absolute arrythmia. He was told to continue the digitalis as before and to report again in two months. He now went to the Throat Room, where the only finding was diseased tonsils which it was thought should be removed if the condition would warrant operation.

October 27 he reported that he had kept on with the digitalis until it gave out the week before. He was getting very short of breath again.

Examination showed his weight to be 132½ pounds. The systolic blood pressure was 130, the diastolic 80. The apex pulse was 82, the radial 82. There was absolute arrythmia and a typical mitral diastolic murmur. He was told to take digitalis as before and to report again in three months.

*Discussion by Dr. Paul D. White.*—It is obvious from the history and physical examination that this patient had rheumatic heart disease with mitral stenosis, auricular fibrillation, and a tendency to failure of the congestive type. Although it is possible that there has been a syphilitic history in the past there is no reason to believe that it is in any way a factor in the causation of his present heart condition. He has a typical mitral facies. The report of the physical examination made in the ward January 25, 1921, states that the action of the heart was absolutely irregular, sounds of poor quality, presystolic roll at the apex and a systolic murmur over the recordia. A somewhat similar examination made in the Out-Patient Department just prior to admission to the ward I feel is quite inaccurate. My opinion is based on the findings made in the Cardiac Clinic in March, where the absolute irregularity was still found, and a low rumbling diastolic murmur at the apex without a presystolic accentuation. This low rumbling diastolic murmur beginning at a short interval after the second sound and heard best at the apex (in the reclining position with a bell stethoscope) is typical of mitral stenosis when auricular fibrillation is present. One does not find the presystolic murmur of mitral stenosis in the presence of auricular fibrillation, except rarely when the heart is beating very fast. The much more important murmur is the mid-diastolic rumble which was the only murmur in his case in diastole. His mitral stenosis must have been of years' duration. His auricular fibrillation quite probably started at the time he first noticed palpitation a year ago.

One rarely finds auricular fibrillation in rheumatic heart disease without mitral involvement, and very frequently the two conditions, mitral stenosis and auricular fibrillation, go together. It is for this reason that I insist on the absolute need of recognizing the mid-diastolic murmur when these two conditions are present together. As a matter of fact even when the rhythm is normal the mid-diastolic murmur is still present if there is much mitral stenosis, but there is in addition a presystolic accentuation which used to be regarded as the

only important murmur in mitral stenosis. I am surprised to find that the records even at the present time are still full of such inaccuracy as is found here in both the record made in the ward and the first examination in the Out-Patient Department. It is high time that the diastolic rumble of mitral stenosis in auricular fibrillation be described as such and not falsely interpreted as a presystolic roll.

Electrocardiogram confirmed the diagnosis of auricular fibrillation, and also showed evidence of preponderance of the right ventricle and occasional ectopic contractions. One of the ward notes, January 30, states that there were a few extra systoles. It takes a clever man to diagnose extra-systoles in the presence of auricular fibrillation unless they are coming in a bigeminal fashion. Therefore I should feel doubtful about this statement.

Now as to treatment. In the ward he was put to bed and given soft solids and codein. This sounds like common sense. Another order appears for potassium iodide. Just why this is given I do not know unless for its action as an expectorant, but we have no proof that it would help the condition of fibrillation or rheumatic heart disease. From February 7 he received fluid extract of convallaria, and continued to receive it for six days in steadily increasing doses. At this particular time investigation of the digitalis-like action of convallaria and apocynum was being carried on in the wards. They are said to belong to the digitalis group, but it was felt that they had not been properly classified. The dose of convallaria in the pharmacopeia is stated to be ½ of 1 c.c. in fluid extract. Having already determined that larger doses were necessary to produce any effect at all in cases of auricular fibrillation we gave two c.c. three times a day at the start of the therapy in this case. Three days later this was increased to 5 c.c. three times a day, one day later to 10 c.c. three times a day, and still a day later 15 c.c. three times a day. About twenty-four hours after this, since convallaria was having little or no effect and since it was necessary to have a digitalis action in this case, the convallaria was stopped and digitalis given at the dose of three grains three times a day in the standardized leaf. This makes two-tenths of a gram three times a day. Quite rapidly there was improvement from the digitalis, such as was hoped for, with the reduction of apex rate and clearing up of some of the symptoms of failure. The convallaria even in doses thirty times the dose recommended in the pharmacopeia had failed to produce the desired effect.

When the patient was discharged he was given instructions to take small doses of digitalis. I find no note indicating how much he

was told to take. However, it was obvious that he took too little, for when he returned to the Out-Patient Department March 5 he was feeling badly. He had some edema, cyanosis, his lungs showed râles, he was very dyspneic, his apex rate was 106 and his radial 79, making a deficit of 27. He was told to take $\frac{1}{10}$ of a gram of digitalis three times a day. He returned March 10 feeling very much better. His apex rate had dropped to 58, his radial rate was 56, a deficit of two. This observation is a very important one. It shows the need of giving a proper amount of digitalis, and it shows the excellent control obtained by studying the apex rate and the pulse deficit. He was told to continue on $\frac{1}{10}$ of a gram of digitalis leaf once or twice a day as needed. This he has done most of the time. Since the first observation was made in the Cardiac Clinic he has been quite well. He had been at work and has stated occasionally that he felt very well. Investigation of his hoarseness revealed nothing, although it is quite likely that there is some pressure on his left recurrent laryngeal nerve from the markedly enlarged left auricle, which in turn through the pulmonary artery presses against the aorta and constricts the nerve.

The moral from this experience is as follows: If digitalis is needed, as it is in auricular fibrillation, it should be given in proper doses. The patient should be quickly saturated with the drug, and the saturation would be complete in within 48 to 72 hours, the dose depending on the weight of the individual. Here we found that $\frac{6}{10}$ of a gram a day quickly saturated this patient in our first observations in the Cardiac Clinic. From $\frac{1}{10}$ to $\frac{2}{10}$ of a gram is excreted daily by the average patient. Enough additional digitalis should be given daily, therefore, to make up for this amount excreted. By the maintenance of saturation the apex rate is kept down within normal bounds,—it usually should be between 55 and 75,—the heart contracts strongly and has plenty of rest. For this particular purpose digitalis is practically a specific, and it is in fibrillation of course that it acts so dramatically.

Another important lesson is that drugs of the type of convallaria, —this should also include apocynum and squill,—may well be omitted. Digitalis does much more in benefiting the heart than any of these drugs. Any other reasons that we have been able to observe for which they might be given are absent. The dosage is inaccurate, and they are not primarily diuretics. I see no reason why they should be given any more. Cactus is a drug which has been included by some in their treatment of heart disease. It has been given in

enormous quantities in experiment without effect. It is my feeling that this drug should be omitted from cardiac therapy, at any rate from treatment of heart disease with failure.

With regard to digitalis I feel that it is given far too often and when it is given it is often given wrongly. Digitalis may be recommended for any one of the following three conditions, first, auricular fibrillation, second, auricular flutter, and third, heart failure, no matter what the rhythm. As a prophylactic of heart failure or auricular fibrillation in an acute infectious disease such as typhoid or pneumonia without cardiac damage I feel that it has no place. In fact it is possible to produce fibrillation by the drug alone. Digitalis is an irritant to the heart and a drug for which we should have great respect, using it only when we have a clear indication for its use. This also applies to the preparation of patients for surgical operation, or for emergency use in so-called surgical shock, where again it has no place. (My experience is entirely like Dr. White's R.C.C.)

This patient has just been admitted to the ward (Nov. 8) specifically for the treatment of auricular fibrillation. This treatment consists in the use of quinidine sulphate. Quinidine sulphate has a drug action which is one of the most dramatic in the whole field of functional therapy. It abolishes auricular fibrillation or auricular flutter in at least two-thirds of all the cases in which it is tried. It does not of course cure heart disease, but in many cases it restores normal rhythm and relieves the patient of disturbing palpitation and of the likelihood of failure. It is not a drug, however, to be given indiscriminately. At the present time investigations of its use are being carried on all over this country and elsewhere. It is my present feeling that it is one of the most valuable steps in advance in our cardiac treatments to be made since Withering introduced digitalis over one hundred years ago. Its action is well described by Sir Thomas Lewis in the British Medical Journal for October 1, 1921. Lewis had previously shown that auricular fibrillation and auricular flutter are due to a curious circus movement of a contraction wave in the auricle. This movement continues until broken up in some way. Ordinarily it persists throughout life in the human being. Increase of the refractory period of the auricular muscle is needed to break up this circus movement. Quinidine does this. The drug itself was investigated and found to have a specific action in these abnormal rhythms by Frey in Berlin during the great war. Lewis's investigations of the mechanism of the circus movement were going on independently. When these two very important pieces of work

were coördinated a marked advance in the treatment of heart disease was effected.

At the present time out of thirty-five cases of auricular fibrillation and auricular flutter at the Massachusetts General Hospital we have restored normal rhythm, at least temporarily, in twenty-three.

*Diagnosis.*—Rheumatic heart disease with mitral stenosis.

Auricular fibrillation.

Failure of the congestive type.

*History, Continued.*—After his discharge from the hospital he did not go back to work for three weeks. He had been taking two pills daily. He had no precordial pain, but became dyspneic on climbing two or three flights of stairs. He still slept propped up with pillows. At times he awoke at night and coughed up a mouthful of white frothy sputum. He had rare cough during the day. He had been able to work comfortably.

Examination was as before except for the points noted. His cheeks were flushed. The lungs showed slight dullness at the right apex and coarse moist râles at both bases. The apex impulse of the heart was seen and felt in the fifth space 10 cm. to the left. The action was irregular, slow. The pulmonic second sound was accentuated, fibrillating. There was a long rumbling mid-diastolic murmur. The left border of percussion dullness was 10.5 to the left of midsternum, 2.5 cm. outside the midclavicular line, the right border 4.5 to the right, the supracardiac dullness 4.5 cm. Rectal examination showed a slightly enlarged prostate. A Wassermann was negative. Electrocardiogram November 8 showed auricular fibrillation, rate 75, upright T wave; November 9, auricular fibrillation, rate 70, diphasic T wave.

This patient, the thirty-sixth of our quinidine series, received a total of 13.8 grams of quinidine sulphate in five and a half days without restoration to normal rhythm and also without any toxic effects whatever. The dose varied from 0.2 gram twice the first day to 0.08 gram five times on the fifth day. He must be recorded as one of our quinidine failures. The duration of his arrhythmia may be one of the reasons for its continuance even under quinidine therapy. He was discharged from the ward November 13, 1921, and will continue to be followed in the Cardiac Clinic of the Out-Patient Department, being kept constantly on digitalis.

The records of the Out-Patient Department Cardiac Clinic show that January 25, 1922, the patient reported shortness of breath for the past three weeks, hemoptysis five or six times one evening.

When seen in the wards January 26 he was in respiratory distress and found it difficult to talk. January 23 at the beginning of a cold snap he began to have unusual dyspnea with palpitation and coughed up considerable white sputum. He continued at work, but went home with some difficulty. January 25 he had dyspnea and palpitation on the slightest exertion and was unable to go to work. His legs were swelled to the knees.

Examination showed no dullness in the lungs. There were many moist râles below the angles of both scapulae and from the third to both sixth ribs in front. The apex impulse of the heart was seen and felt in the fifth space 8 cm. to the left. The left border of percussion dullness was 9.5 cm. to the left of midsternum, the right border 4 cm. to the right, the supracardiac dullness 5 cm. The action was irregular, probably slow fibrillation, but there was a slight tendency to bigeminy. The first sound was loud and snapping, the pulmonic second sound accentuated. A presystolic murmur was heard over the precordium, and a faint systolic murmur, difficult to make out because of râles and grunting respiration. A thrill was palpable, maximum in the third space. The pulses were of low volume and tension. The blood pressure was 125/60. There was very marked edema of the ankles.

The temperature was 100.6°, rising steadily to 104°, the pulse 70 to 101, the respirations 19 to 50. The amount of urine is not recorded. The specific gravity was 1.019. The urine was alkaline and there was a trace of albumin. The hemoglobin was 80%. The leucocytes were 8400 to 14,800, the polynuclears 88%. A Wassermann was negative. Electrocardiogram showed auricular fibrillation, rate 100, ectopic ventricular contractions, flat T wave, small complexes.

The morning of January 27 slight dullness was found at the right apex and around the spine of the right scapula with groaning râles throughout the right lung and moist râles at both bases. The dyspnea seemed out of proportion to the other symptoms. That evening there was dullness at both bases with rise in temperature and respiration. That night the patient died.

*Clinical Diagnosis (from Hospital Record).*—Chronic valvular heart disease.

Mitral stenosis.

Auricular fibrillation.

Bronchopneumonia.

Pulmonary edema.

*Dr. Paul D. White's Diagnosis.*—Rheumatic heart disease.

Mitral stenosis.

Cardiac enlargement.

Bronchopneumonia.

Chronic passive congestion.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral valve (stenosis).

Thrombosis of left auricular appendix.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Hydrothorax, right.

Slight anasarca.

Chronic pleuritis.

DR. RICHARDSON: We were not permitted to examine the head. In the peritoneal cavity there was about fifty c.c. of thin pale clear fluid. The mucosa of the gastro-intestinal tract was reddened, velvety, juicy, showing chronic passive congestion. The mesenteric and retroperitoneal glands were negative.

The right pleural cavity contained 500 c.c. of thin pale clear fluid. On the left side the cavity was obliterated by old adhesions, but the adhesions were wet. The lungs showed a typical picture of chronic passive congestion, with a few small areas of questionable bronchopneumonia, but the macroscopical examination shows only chronic passive congestion in these places.

The pericardium contained twenty-five c.c. of thin pale clear fluid; negative. The heart weighed 545 grams,—considerably enlarged. If he had had a heart weighing 350 it would have been a good sized heart for him. The myocardium was pale brown-red and a little flabby, toneless. The wall of the left ventricle was eleven mm. thick; it was a little thin if anything. The right was four to six mm., which of course is about twice as thick as usual, indicating some obstruction in the region of the mitral valve or something in the pulmonary valve. We can dismiss the pulmonary valve at once because it was negative, and it is a very unusual place to have lesions. The columnae carneae are part of the musculature, and it is well to note them. On the left they were negative; on the right side they were thick. So that we found marked hypertrophy of the right side of the heart, the wall generally thickened. The cavity of the left ventricle was negative as usual in mitral stenosis where the lesion is pure mitral. The left auricle was dilated, the wall slightly thickened, and in it there was a very large frank adhering thrombus. The right auricu-

lar appendix was free. The cavities on the right side were dilated and distended with blood clot. The valve measurements were: the aortic seven, the tricuspid 14½, the pulmonary 9; these valves were out of the picture. The mitral showed marked stenosis. In the situation of the valve was a flat ovoid fibrocalcareous mass in the center of which was a very small crescentic opening. The chordae tendineae showed marked shortening and thickening,—a typical picture of mitral stenosis with a mural thrombus in the left auricular appendix.

The coronaries were free and capacious and showed a few scattered small fibrous plaques. The pulmonary artery and veins, the aorta and great branches, and the venae cavae were negative. The liver, pancreas, spleen, adrenals, kidneys, showed chronic passive congestion.

In the gall-bladder were 16 yellowish-brown crystalline stones. The bile ducts and the duct of Wirsung were free and negative.

### Necropsy 4046

An American leather-worker of forty-six entered May 14, 1908. He had scarlet fever, measles, mumps and pertussis in childhood, "rheumatism" (swelling and tenderness in many joints) at fourteen and again at forty-two, confining him to bed four weeks each time, at twenty-eight a sore on the penis followed by a bubo, at thirty-one gonorrhea. For years he had had frequent attacks of "bronchitis" every winter, with wheezing respiration, usually worse at night, and cough often with a cupful of sputum at night. At forty-two he had pneumonia. He formerly drank several glasses of beer and whiskey daily and had an occasional spree. For two years he had taken very little alcohol.

A year before admission he had a slight attack of dyspnea, cough and palpitation similar to the present one lasting seven to ten days. Two and a half months ago he began to have attacks of dyspnea, palpitation and cough with mucoid sputum and on two occasions a mouthful of blood. He was in bed two weeks. After this he felt perfectly well and intended to go to work again. May 9 he had a recurrence of the symptoms. In both attacks his legs swelled in the evenings and often his eyes and face were puffy in the mornings. His hands had occasionally been swollen. Ever since the onset he had had more or less constant gnawing pain in the lower midabdomen radiating to the right hypochondrium, not related to eating, increased by exercise. For three months his bowels had not moved without

catharsis, and when straining at stool "the bowel sometimes turned wrong side out." He had had two or three attacks of dizziness since the onset lasting only a few seconds.

Examination showed a well nourished man with many old faded and pigmented macules and papules on the skin of the trunk and right arm. There was a small wen on the back of the neck. The mucosae were of fair color, a little pale. The throat was generally reddened. There were enlarged glands in both axillae and in the left groin below Poupart's. The costal borders flared. The apex impulse of the heart was diffuse over the lower precordia and in the left epigastrium, seen and felt in the sixth space 12 cm. from midline, 1½ cm. outside the nipple line, corresponding to the left border of dullness. The right border was 4 cm. to the right. No supracardiac dullness was made out. There was very slight systolic retraction in the region of the apex. No definite thrill was felt. There was absolute arrythmia. The first sound was loud but not flapping. The pulmonic second sound was greater than the aortic second. The second sound was accented at the apex. A blowing systolic murmur was heard at the apex transmitted to the axilla, a faint diastolic murmur in the third and fourth spaces along the left sternal margin. The pulses were normal, the artery walls tortuous and beaded. The blood pressure was 165/95 to 140/80. Harsh breathing was heard throughout the lungs, with many groans and wheezes, and fine râles in both axillae. There was slight tenderness in the right upper quadrant. The liver dullness extended from the fifth space to 3 cm. below the costal margin, where an indefinite edge was felt. The right pupil was larger than the left. Both were slightly irregular but reacted to light and distance. The reflexes were normal.

The temperature was 97° to 99°, usually 98° or below. The pulse was 112 to 80, the respiration 30 to 25 to May 16. Both were afterwards normal. The urine and blood were normal. A Wassermann was negative. The sputum showed moderate numbers of bacteria, mostly pneumococci; no tubercle or influenza bacilli. The X-ray is shown in Fig. 12.

The patient gained rapidly. Action was taken by the Social Service to find him lighter work. At his discharge, May 27, the heart was slightly irregular, the sounds much the same as at entrance.

In June, 1910, a polygram taken in the Out-Patient Department showed auricular fibrillation. Examination showed a systolic and a short diastolic at the apex. He was taking digifolin gr. iss as needed. The pulse at the apex was 70, at the wrist 66. He was ordered to

continue digifolin. A diastolic murmur was heard first in February of the previous year. In July X-ray showed no evidence of gall-stones. The following January he felt badly and was slightly cyanotic. Examination in the Out-Patient Department showed râles at both bases, the liver two fingers down; no edema. The heart rate was 45; no deficit except for occasional premature beats. The first sound was replaced by a murmur.

January 30, 1917, he returned to the wards reporting several severe attacks since the previous admission, the first three weeks after discharge following very strenuous work. He rested at home until Aug-



FIG. 12.—Necropsy 4046. Plate I. Mitral stenosis. X-ray six years before death. (Not taken at seven feet.) Heart shadow enlarged. Great vessels not abnormal. Dense mass at both lung roots, probably glands. Lung markings extending out from lung roots thickened and mottled. Angle between heart and diaphragm indistinct.

gust, 1908, then took light work. A year before readmission and again in the summer of 1916 he had attacks each lasting four or five days, with bronchial trouble, dyspnea, palpitation and distressing cough. Between the attacks he had been in good condition. Two weeks before admission he was laid up by an attack more severe than the former ones. He felt comfortable while staying quiet, but on light exertion became very dyspneic and had palpitation and severe cough.

Examination was as before except for the points noted. He was fairly well nourished. The mucous membranes were slightly cyanotic. There was barrel chest. The lungs showed sibilant

râles in front, moist crackles at the base. The nipple line was 10½ cm. to the left of midsternum, the apex impulse 10 cm., the border of dullness 13½ cm. to the left. The right border was 4½ cm. to the right. The supracardiac dullness was 6 cm. There was absolute irregularity. The first sound at the apex was replaced by a loud blowing systolic murmur transmitted to the axilla. There was a softer blowing diastolic at the apex. The pulmonic second sound was much accentuated. The pulses were absolutely irregular, of fair volume and tension. All the apex beats came through. The systolic blood pressure was 130 to 140. The liver dullness extended from the sixth rib to 2 cm. below the costal margin. The edge was not felt. The pupils were equal, regular.

The temperature was 95.7° to 98.8°, the pulse 38 to 70, the respirations 17 to 32. The amount of urine was 90 to 10 ounces, cloudy, specific gravity 1.010, findings negative. The hemoglobin was 80%, the leucocytes 8000 to 13,000, the polynuclears 64%, eosinophils 10%. A Wassermannn was negative. The renal function was 40%. The blood nitrogen was 30 mgm. per 100 c.c.

The radial pulse continued to stay about 40 for the first four days, it was supposed because of digitalis treatment before entrance. There was no edema except slight moisture in the bases of the lungs. By February 7 he was feeling very well. The pulse rate was between 50 and 60. The dull pain in the right upper quadrant which he had previously, had cleared up. February 8 he was discharged relieved, with advice to rest several weeks and to return to the Out-Patient Department once a week.

July 2, 1918, he came to the Out-Patient Department. He was better, slightly cyanotic. There was probably fibrillation, but slow. He was working. October 23 he reported influenza three weeks earlier. November 10 electrocardiogram showed auricular fibrillation with inversion of T wave. The heart was working very well. February 6, 1919, the heart measurements were as shown in Fig. 13. February 20 he had had no digitalis for four weeks. There was edema, dyspnea, ascites, hydrothorax. The apex pulse was 120, the radial 90.

January 12, 1920, he entered for the third time. He had done no steady work for three years. Since his stay in the hospital his heart had been decompensated most of the time, with an occasional period of a few days to a week when he felt well. During the attacks of decompensation he had edema of the feet and lower legs. He had dyspnea on slight exertion, palpitation, and a variable

amount of cough. His bowels were constipated. He urinated at night and had occasional burning micturition. He was troubled by gas and sour stomach. There was almost constant gnawing but not severe pain in the right upper quadrant varying with severity of heart symptoms. He now used three or four pillows. He slept poorly and had nightmares.

On examination his skin was dusky, with generalized punctate papules. His mucosae were slightly pale. There was very marked pyorrhea. The left chest was dull to flat throughout the front, with musical râles and prolonged expiration. There was dullness with occasional crackling râles at the left apex behind. There was flatness from the midscapula to the base with crepitant râles, bronchophony, and distant bronchial breathing. The apex impulse of the heart was in the sixth space 8.5 cm. to the left. The left border of dullness was 11.5 cm. to the left, the right border 3.5 cm. to the right, the supracardiac dullness 7 cm. The sounds were irregular, rapid, of good quality. The heart was in fibrillation. The pulmonic second sound was accentuated. There was a soft systolic murmur at the apex transmitted to the axilla. The pulses were normal, the artery walls palpable. The systolic blood pressure was 150, the diastolic 90. There was slight tenderness in the right upper quadrant. The liver dullness extended from the fourth rib to 4 cm. below the costal margin. The edge was felt. There was dullness over Traube's space. The fingers were clubbed. There was moderate edema of both legs and feet. The pupils were normal.

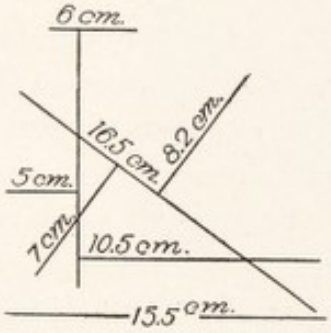



FIG. 13.—Heart measurements by X-ray February 6, 1919, Out-Patient Department.

The temperature was 96.1° to 102°, the pulse 58 to 115, with tow drops to 43–49, and a rise to 120 the day of death. The respirations were 14 to 40. The amount of urine was 15 to 105 ounces. A Schlayer test January 19 showed the specific gravity 1.008, 1.008, 1.010, 1.010, 1.014, 1.010; the slightest possible trace to a very slight trace of albumin in all but one of six specimens; total chlorides 1.085–2.214 gm.; four specimens cloudy, two alkaline. In six other examinations the specific gravity was 1.026–1.037; there was a slight trace to a very large trace of albumin at all, cloudiness at one, bile at one, hyalin casts at five, leucocytes at five, red blood corpuscles at three. The renal function was 20 to 30%. The hemoglobin was 70%, the leucocytes 7400 to 8400 the polynuclears 64%. The

reds showed slight achromia, some anisocytosis and poikilocytosis. The non-protein nitrogen was 45.7 mgm. The creatinin was 1 mgm. per 100 c.c. of blood. A Wassermann was negative. A chest tap gave 1000 c.c. of clear blood-tinged fluid which showed 600 cells, specific gravity 1.004, a negative culture, total chlorides 6.2 gm. X-ray showed the heart shadow very much enlarged to the right and the left. The outline of the various chambers was obliterated. The diaphragm could not be made out on the left side.

The chest tap gave some relief. The patient slept very restlessly, and on some days looked very ashy. Diuretin gr. xv t.i.d. for five days was followed by good diuresis, but the urinary output soon fell again. He continued to have pulse deficit. Without diuretin the chest began to fill up again with fluid. He responded poorly to digitalis. He was drowsy most of the time. January 30, after an interval of ten days, the diuretin was begun again. February 13 the temperature rose to 102°, and each of the following days reached that point. The edema increased markedly. February 15 he died.

*Clinical Diagnosis (from Hospital Record).*—Chronic endocarditis, mitral valve.

Myocardial insufficiency.

Arteriosclerosis.

Bronchopneumonia.

Auricular fibrillation.

Possible adherent pericardium.

*Dr. William H. Smith's Diagnosis.*—Mitral stenosis and regurgitation.

Possible adhesive pericarditis.

Arteriosclerosis.

Bronchiectasis.

Chronic passive congestion of liver and kidney.

Probable terminal infection.

*Anatomical Diagnosis.*—Chronic endocarditis of mitral valve, stenosis.

Thrombus in left auricle.

Slight chronic endocarditis of the aortic valve.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Hydropericardium.

Hydrothorax.

Ascites.

Anasarca.

Infarcts of the lungs.

Chronic pleuritis.

DR. RICHARDSON: This is a typical case of mitral stenosis. The heart weighed 660 grams, was markedly enlarged, the right ventricle wall seven mm., the left ventricle wall eight mm.,—that is, the right ventricle wall markedly thickened, the left wall rather thin. There was much dilatation of the left auricle and great dilatation of the right ventricle and auricle. The mitral valve was the seat of a deforming mass, reducing its curcumference to a crescent-shaped opening about one and a half cm. in length, which is shown in the picture.

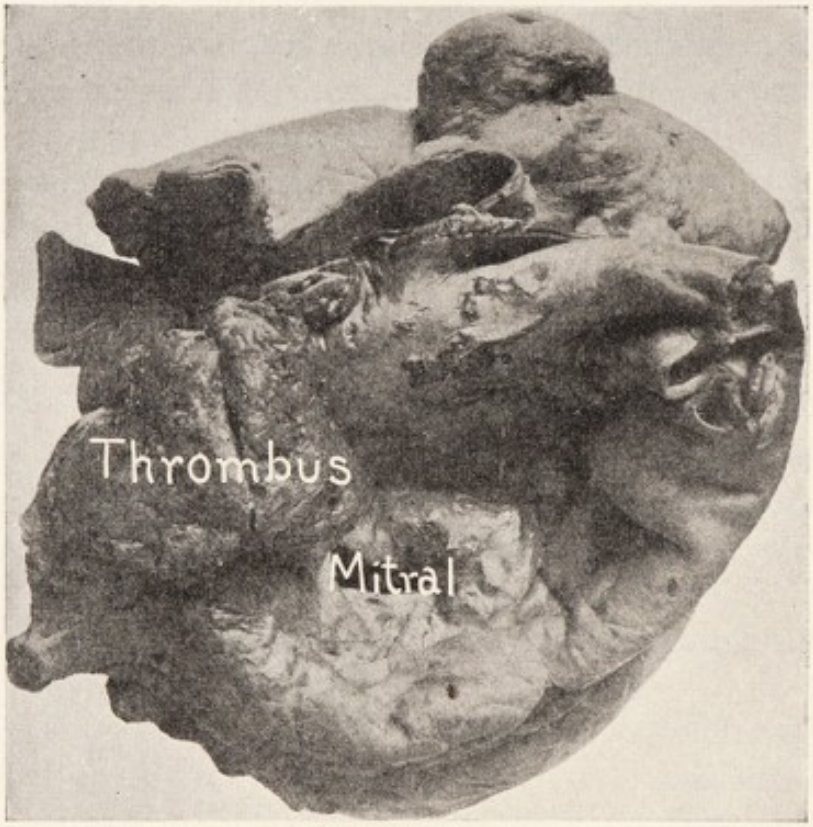



FIG. 14.—Mitral stenosis. Left auricle viewed from above. Large thrombus in the left auricular appendage.

In the left auricular appendage there was a large adhering thrombus. (See Fig. 14.) The aortic valve showed a few small areas of chronic endocarditis, otherwise was negative. The other valves were negative.

The kidneys showed chronic passive congestion.

The liver was large, 2300 grams, and was about a hand below the costal margin. It showed typical nutmeg markings. The spleen showed chronic passive congestion.

The gastro-intestinal tract showed a reddened, velvety, juicy mucosa, that which accompanies chronic passive congestion.

There was no growth from the heart blood.

The lungs showed chronic passive congestion, and there was a slight amount of chronic pleuritis.

The anatomical diagnosis here is in logical sequence. It is a typical case.

**Necropsy 3768**

A Swedish housewife of thirty-one entered August 16. The history was obtained with difficulty from her husband. He did not know whether she had had rheumatic fever, tonsillitis, etc. She had a fever many years ago in which she lost her hair. She often had headaches, and had had some trouble with her vision; at times she had seen double. She had sore throat two or three times a winter. Three years before admission she had some urgency and frequency. During the past three years she had lost a good deal of weight. For two years she had had intermittent slight cough with a small amount of white sputum. She had had night sweats. Her bowels were constipated. She had had eructations of gas and pyrosis. She had been married four years, and had never been pregnant.

Two years before admission dyspnea, weakness, and dizziness gradually developed. Three months before admission she was in a hospital for ten weeks. After six weeks more in a convalescent home she was discharged four weeks ago feeling very well. August 6 she had pain in the left hypochondrium, increased by straightening up and taking deep breath, accompanied by cough, and keeping her awake. August 8 she began to cough up white sputum. August 10 the pain left the side and developed in the right flank and hypochondrium, becoming so severe that she screamed when touched. Next day she was better. August 13 she began to vomit everything she took. Her physician said he had given her too strong medicine. The night before admission the vomiting ceased. She could not lie on her left side, and had to be lifted to change position. For three days she had had constant severe pain on one or both sides, and had seemed to grow weaker. The day of admission for the first time she coughed up a little bloody sputum.

Examination showed a fairly well developed and poorly nourished woman, cyanotic and very dyspneic. The mucous membranes were pale. The teeth were poor. There was some pyorrhea. The throat and tonsils were reddened. The apex impulse of the heart was in the fifth space. There was no enlargement to percussion. The aortic second sound was greater than the pulmonic second. There was a presystolic roll followed by a loud and sudden first sound.

A slight thrill was felt. The artery walls were not palpable. The blood pressure was 75/60. The lung signs were as shown in Fig. 15. The pupils were regular. The left was greater than the right. The right reacted slightly; the left was stiff and dilated. The rest of the examination was not made because of the patient's poor condition.

The temperature was normal, the pulse 100 to 110, the respiration 48 to 30. The urine is not recorded. The hemoglobin was 80%, the leucocytes 27,000 the polynuclears 89%. A Wassermann and a stool examination were negative.

The patient became very cyanotic and dyspneic. Coarse râles developed throughout the chest. The night of entrance she had a profuse cold sweat. August 17 she died.

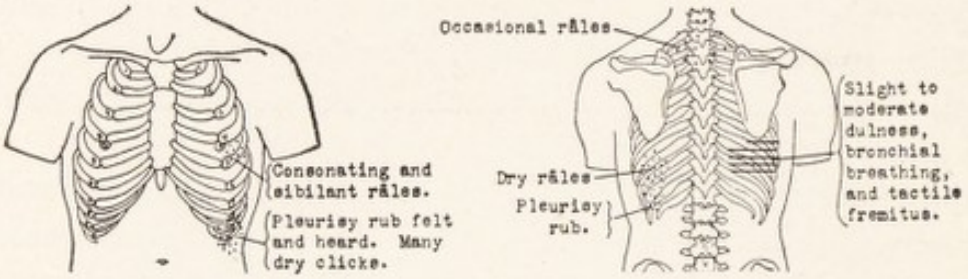



FIG. 15.

*Clinical Diagnosis (from Hospital Record).*—Mitral stenosis.
Myocardial weakness.
Dry pleurisy.
Pulmonary tuberculosis? (Miliary?)
*Dr. William H. Smith's Diagnosis.*—Mitral stenosis.
Emboli in the lungs.
Auricular thrombosis.
Acute endocarditis?
*Anatomical Diagnosis.*—Chronic endocarditis. (Mitral stenosis.)
Acute endocarditis.
Ball thrombi in the auricles.
Hypertrophy and dilatation of the heart.
Infarcts of the lungs, spleen, and kidneys.
Ecchymoses on the epicardium and peritoneum.
Acute pleuritis.
Chronic passive congestion.
Ascites.
Slight chronic pleuritis, right.

DR. RICHARDSON: The circumference of the mitral valve was only 3 cm. There were ball thrombi in each auricle, a combination which we have never found before.

With a lesion in the mitral valve, stenosis, we should expect to find hypertrophy of the right ventricular wall. That is precisely what we did find. The heart weighed 337 grams, showing a hypertrophy of 100 grams for a woman of her size. The aortic, tricuspid, and pulmonary valves were negative. The wall of the left ventricle was 8 mm. thick (normally 12 mm.); being in front of the obstruction it had no work to do. The wall of the right ventricle measured 5 mm.; having work to do, it hypertrophied. The coronary arteries were negative. The heart muscle was good, and there was a good blood supply. In typical cases of mitral stenosis the changes come back of the obstruction; the left auricle, the right ventricle, the right auricle, show marked dilatation. The left ventricle is out of the question altogether; in a way it does not have so much to do, as the blood gets through the valve in much smaller amounts than it does normally.

There were several small ball thrombi in each auricle. The infarcts went on the right to the lungs, on the left to the spleen and kidneys.

The culture was negative. I speak of that because it is the clinching fact in these cases of infection. If we get three or four things that mean infection, but do not get the other one, in all probability it means infection. In this case we had acute endocarditis, acute pleuritis, and on the epicardium small areas of ecchymosis. From the microscopical examination we add to that as evidence of the presence of infection the acute degeneration of the epithelium of the renal tubules. Though we did not get a growth in the blood stream, the case stands as though there was an acute infection present.

The lungs showed infarcts. Minute dissection of the organs was not done, however, for we sent them to a medical college for teaching purposes. The chronic passive congestion, of course, was the end result of the obstruction at the mitral valve.

We usually find ball thrombi in the left auricle where behind a mitral stenosis the stasis in the blood stream is great, especially in the auricular appendix and where the blood supply to the auricular wall may be poor. The thrombus becomes established on the wall of the appendix, gradually increases in size, and pushes out into the auricular cavity. As the thrombus increases in size its pedicle of attachment to the auricular wall becomes more and more attenuated,

until finally the thrombotic mass is freed from its attachment and becomes what we call a ball thrombus. If this floats down over and closes a buttonhole mitral then sudden death occurs. In this case particles from the left auricular thrombus may have gone up into the cerebral vessels and caused the disturbances mentioned in the clinical record. This was a typical case showing thrombi in the auricles and what they can do.

### Necropsy 1410

An Irish housewife of twenty-seven entered May 26. She gave a history of measles and pneumonia as a child, pleurisy at twenty-one. Five weeks before admission her left shoulder and knee became painful, without redness or swelling. After two days the condition extended to the right elbow, then to the ankles. Occasionally the pain returned to the left shoulder. The day before admission there was slight pain in the right wrist. At entrance both knees were painful.

Examination showed a well nourished woman with pale skin and mucous membranes. The tongue showed a heavy white coat. The throat was slightly reddened. There was no enlargement of the heart to percussion. The action was regular. A presystolic thrill was felt at the apex. A loud presystolic murmur was heard at the mitral area, but loudest at the apex, transmitted toward the axilla and ending in a sharp first sound. The second sound at the apex was scarcely audible. The pulmonic second sound was greater than the aortic. The pulses were of fair tension, otherwise normal. Over the left back there was slight dullness on percussion, with slightly diminished vocal and tactile fremitus. The abdomen was normal. Both knees were somewhat puffy and painful on motion. The left pupil was greater than the right. Both reacted normally. The abdominal reflexes and the knee-jerks were not obtained.

The temperature was 101.1° to 98.1° the pulse 115 to 80, the respiration 19 to 29. The output of urine was 11 to 55 ounces, the specific gravity 1.014 to 1.026. There was a very slight trace of albumin to none, rare hyaline casts at four of five examinations, granular casts at the last, very rare blood cells at two. The hemoglobin was 85%. There were 10,300 leucocytes.

May 28 the temperature had dropped to nearly normal and the patient was practically free from pain. By the thirty-first there was only occasional slight pain or stiffness. She continued alternately very comfortable and troubled with pain, chiefly in the knees. The

heart condition remained unchanged. After June 20 the temperature was 100° to 101° every afternoon. June 29 numerous moist râles were heard in both lungs, with a few dry crackling râles just below the left clavicle. The breathing was of the bronchovesicular type. Ten grains of potassium iodid was given three times a day. She looked pale and weak, but had no complaints, and except for the fever would have been up. The night of June 30 she vomited half a cupful of greenish material three times. The pulse was strong, and she felt better after each attack. Three-quarters of an hour after the last one she died.

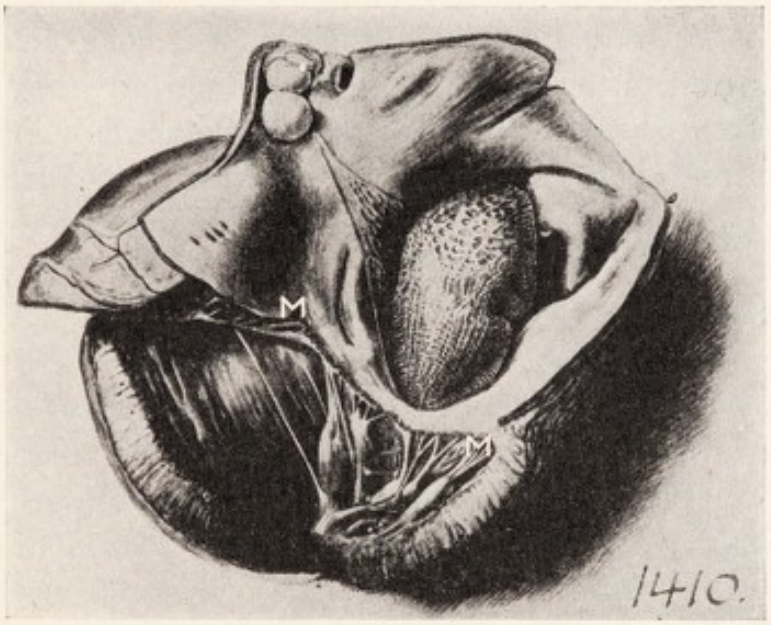



FIG. 16.—Necropsy 1410. Ball thrombi in left auricle. Fine cockscomb vegetations on edge of stenosed mitral valve. The large thrombus occludes the mitral orifice. (From a drawing by H. F. Aitken. Dr. Oscar Richardson.)

*Clinical Diagnosis (from Hospital Record).*—Acute articular rheumatism.

Mitral stenosis.

*Dr. William H. Smith's Diagnosis.*—Chronic endocarditis.

Acute endocarditis?

Acute or subacute glomerulo-nephritis?

*Anatomical Diagnosis.*—Chronic and acute endocarditis of the mitral and tricuspid valves. Mitral stenosis.

Thrombi in the left auricular appendix.

Ball thrombus in the left auricle.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Hemorrhagic infarctions of the lungs.

Thrombosis of a branch of the pulmonary artery in the lower lobe of the right lung.

Ecchymoses in the visceral pericardium.

Infarcts of the skin.

Anasarca.

DR. STEELE: The heart weighed 430 grams. The mitral valve was stenosed, its circumference being 3½ cm. (normally 10 cm.). Resting over this valve and occluding it was a ball thrombus 6½ × 5½ × 1½ cm., which occupied a great part of the left auricle. The mitral and tricuspid valves showed granulations such as are found in streptococcic endocarditis. There was also thrombosis of a branch of the right pulmonary artery, and hemorrhagic infarcts in several places in the lungs.

There was no macroscopic evidence of glomerulo-nephritis.

No culture from the heart was made, owing to the incision restriction, but a culture made from the liver showed the presence of a streptococcus.

DR. RICHARDSON: Thrombi are usually found in the left auricle and are commonly associated with mitral stenosis. In this case the stasis of the blood stream in the left auricle due to the stenosis, associated with the character of the blood and the streptococcus infection, are the sources of the thrombus formation. These ball thrombi, judging from the cases we have seen, arise in all probability in the following manner. The conditions favorable to the erection of thrombi are, of course, more favorable in the conical sac-like portion of the auricle known as the auricular appendix, and thrombi in the auricle are usually found in the region of the appendix. It was so in this case.

The picture of the heart of this patient shows remarkably well this evolution of the ball thrombus. The so-called wave markings on the thrombus are well brought out, and the cox-comb-like granulations (M, M) on the free margin of the stenosed mitral valve are well indicated.

### Necropsy 2960

Un unmarried Irish housemaid of thirty-five entered October 20. Her mother died at thirty-five of "dropsy." The patient had scarlet fever and measles in childhood. At twenty-nine she had "rheumatism" for four or five weeks affecting many joints. Since this attack she had had twinges in damp weather. Her catamenia were often irregular, with much backache. She had one child living and well. The perineum was repaired seven years ago. She had had

slight whitish vaginal discharge and attacks of headache and dragging pain for six years. Since the attack of rheumatism she had noticed steadily but very gradually increasing dyspnea on exertion. For the past year this had bothered her a good deal while at work. For three weeks she had felt tired all the time, and the dyspnea had markedly increased. She had also had much distension and belching of gas after meals. For two weeks she had had some urinary frequency, with burning. A week ago she began to vomit after meals. Her stomach retained only liquid food. She had little nausea, but much stomach heaviness. For five days she had had slight soft swelling of the ankles and occasional pains in the ankles and knees. She had also had frequent chilly sensations and dull frontal headache. She had slept poorly because of hacking cough and smothering sensations in the lower chest followed by attacks of dyspnea. She had been orthopneic. She had done a little work every day. Her bowels had been very constipated for years.

Examination showed a pale, cyanotic and dyspneic woman five feet and a half inches in height, weighing 104½ pounds. The apex impulse of the heart was in the fifth space 4 cm. outside the nipple line. The right border was 4 cm. to the right of midsternum. The action was tumultuous. The sounds were of poor quality. At times a rolling presystolic was clearly heard, ending in a sharp first sound and followed by a systolic murmur. There was a loud systolic in the second right space. The jugular seemed to fill, though slowly, from below. An occasional presystolic thrill was felt. The pulmonic second sound was weaker than the aortic second. The blood pressure 115/85–125/85. The lungs showed flatness and diminished fremitus in the right lower chest below the angle of the scapula. Just at the angle there was egophony and bronchial breathing. The abdomen was full, with shifting dullness in the flanks. The liver dullness extended from the sixth rib to a hand's breadth below the costal margin, where a tender edge was felt. There was moderate soft edema of the legs. The pupils and knee-jerks were normal.

The temperature was 98.2° to 95.1°, (usually 96°–97°.) The pulse was 60 to 90. The respirations were normal. The output of urine was occasionally 15 ounces or less. The specific gravity was 1.025 to 1.020. There was a very slight trace of albumin and a moderate number of granular casts at all of four examinations. The renal function was 10%. The hemoglobin was 80%. There was 15,000 to 23,000 leucocytes, 79% polynuclears. The reds were normal. A blood culture October 20 showed no growth. The fundi were normal.

Until the 27th vomiting was the most prominent symptom. Digipuratum, digitalis, salts and elaterium were all vomited. Still she passed a fair amount of urine. Hot air baths gave marked relief. The jugulars were distended. There was marked lessening of the ascites. November 8 caffein sodium benzoate gr. v 4 i.d. was given and well retained, but no diuresis followed. November 13 she began to spit blood. November 11 diuretin suppositories gr. xv 4 i.d. were given. The vomiting became more severe, and theocin gr. iii every four hours in capsules was substituted. November 16 Seidlitz powders were given one every hour during the day. The patient failed rapidly and died the next morning.

*Clinical Diagnosis (from Hospital Record).*—Double mitral lesion.

Myocardial failure.

*Dr. Richard C. Cabot's Diagnosis.*—Acute and chronic endocarditis of the mitral valve, stenosis, with probably a stretching of the tricuspid.

Predominant hypertrophy of the right ventricle.

Very possibly a thrombus in the left auricular appendage.

Hydrothorax, right.

Passive congestion of all the organs.

Probably no nephritis; if any, acute or subacute.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral valve, stenosis.

Thrombosis of the left auricular appendix.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Hydrothorax, left.

Ascites.

Edema of the lower extremities.

Obsolete tuberculosis of tracheal lymphatic glands.

Chronic pleuritis.

Chronic interstitial pneumonitis of the right lung, bony plates in the pleura.

Chronic perihepatitis.

Chronic pelvic peritonitis.

Slight arteriosclerotic degeneration of the kidneys.

The heart weighed 337 grams. The left ventricle wall measured 8 mm.—very thin; the right 4 or 5 mm.,—thick. The mitral valve was of the button-hole type, and measured 12 mm. Normally it should be 10 cm. The tricuspid showed a slight amount of fibrosis, but measured 13.5 cm., normal size.

The liver weighed 925 grams,—small and nutmeg.

The kidneys showed scarcely any change, certainly no cause for uremia.

The stomach showed nothing so far as could be made out. The autopsy was, however, done 346 hours after death.

### Necropsy 3746

An Irish-American housewife of forty-eight entered June 12. Since childhood she had had occasional slight attacks of tonsillitis. At thirty-six she began to have rheumatism. At forty-one she came to the Out-Patient Department of this hospital for relief of pain in the ankles and the left arm, giving a history of swollen ankles for the past two years, dyspnea on exertion, and very profuse flowing. Her catamenia had always been irregular, with some dysmenorrhea. For the past four or five years she had had flowing most of the time. She urinated three or four times by day and six or seven time at night. Her best weight was 165 pounds, her usual weight 145 pounds. She thought she had lost a good deal in the last two or three years.

Seven years before admission she began to get out of breath on slight exertion. Her ankles swelled during the day but went down during the night. At the Out-Patient Department a harsh systolic murmur was heard all over the precordia and in the axilla. The specific gravity of the urine was 1.006. There was the slightest possible trace of albumin and a few granular casts. She was advised to enter the hospital, but did not. Since this time her ankles had continued to swell and the dyspnea had increased. She had become less able to work. Three weeks ago she was obliged to go to bed, mainly on account of dyspnea. About the same time she noticed that her abdomen was getting larger. She developed a cough, raising large amounts of thick yellow sputum. Her dyspnea had increased to orthopnea. She often waked at night with an oppressed feeling and had to sit up and gasp.

Examination showed a fairly well developed and nourished woman with yellow skin and sclerae and pale mucosae. The apex impulse of the heart was in the fifth space 10 cm. to the left of the midsternum. The left border of dullness was 13 cm. to the left; the right border was not found. The supracardiac dullness was 6.5 cm. There was absolute irregularity of action. The sounds were of poor quality. The first sound at the base was replaced by a blowing systolic murmur. There was a soft systolic at the aortic area. The pulmonic second sound was accentuated. The pulses were of poor volume and ten-

sion. The blood pressure is not recorded. The artery walls were palpable. The right breast was edematous. The right lung was dull to flat from the midscapular region to the base posteriorly and from the third to the seventh rib anteriorly, with diminished breath, voice and fremitus, and with moist râles above. The left lung showed moist râles to the level of the sixth rib in front and to the inferior angle of the scapula behind. The abdomen was distended, the edema extending to the back. The umbilicus was level. The liver dullness was not made out. The extremities showed much edema of the feet and legs, some of the hands and arms. Rectal and pelvic examinations were not done. The pupils and reflexes were normal.

The temperature and respirations were not remarkable. The pulse was 80 to 112. The amount and specific gravity of the urine are not recorded. There was the slightest possible trace of albumin, bile, diacetic acid, hyalin and granular casts, and leucocytes. The hemoglobin was 40%. (?), the leucocytes 14,400, the polynuclears 87%. The reds were 2,046,000, with slight variation in size and shape, much achromia and some polychromatophilia; no blasts or stippling. The plates were somewhat reduced. A Wassermann was negative. The urea nitrogen* was 24 mgm. per 100 c. c. of blood. An abdominal tap gave 8 ounces of straw-colored fluid, the specific gravity 1.010, 170 cells, all lymphocytes.

The patient became comatose six hours after entrance. The day after entrance the visiting physician was able to hear no second sound. There was marked edema of the walls of the chest and the abdomen and brawny edema of the back, thighs, and legs. He found no evidence of malignancy on rectal examination, and felt no definite tumor anywhere. The uterus was not felt. The cervix was not nodular. Two lumbar punctures gave a negative fluid under great pressure. Slight improvement followed the tap. June 14 the patient died.

*Clinical Diagnosis (from Hospital Record).*—Cardio-renal disease.
Severe anemia. Primary?
Cerebral edema.
Chronic nephritis.

*Dr. William H. Smith's Diagnosis.*—Mitral disease.
Anemia, primary? secondary?

*Anatomical Diagnosis.*—Chronic interstitial hepatitis with slight fatty metamorphosis.

*The normal urea nitrogen is 12 to 15 mgm. per 100 c.c. of blood.

Chronic endocarditis of the mitral valve.
Submucous myoma of the uterus.
Anemia.
Hyperplasia of the bone marrow of the right femur.
Arteriosclerotic degeneration of the kidneys.
Hypertrophy and dilatation of the heart.
Chronic passive congestion, general.
Hydrothorax.
Anasarca.
Cholelithiasis.
Chronic pleuritis, right.

DR. RICHARDSON: There was a slight amount of fibrous sclerosis in the aorta and the great branches, but nothing unusual.

The liver weighed 930 grams and was small. The organ showed some thickening of the capsule, which was smooth and glassy. In some few places this capsule rounded over small masses of liver substance, giving in those regions a slightly granular aspect to the surface. In other places slender prolongations of the capsule extended into the liver substance. The liver tissue itself was rather homogeneous, and, although not typical of an extreme case of cirrhosis, showed enough to reduce the weight to 930 grams. There were some fatty changes in the remaining liver cells. The interstitial increase was mainly along the lines of the portal vessels, appearing as a network of grayish lines.

In the gall-bladder there were two stones which seemed to have produced no result.

The spleen weighed 420 grams (normally 80–180). The hypertrophy was probably due to chronic passive congestion.

The kidneys weighed 300 grams (normally 200–400) and showed a slight amount of arteriosclerotic nephritis. That means that in areas scattered here and there over the tissue there were collections of fibrous tissue and some sclerosis of the arteries.

The heart weighed 570 grams, a weight which, with the lesions I have mentioned, seems to be excessive. The right ventricle was 4–5 mm., the left ventricle 11 mm. The cavities were considerably dilated on the right side; not so much on the left. All this goes with the fact that the only change in any of the valves was in the mitral, which was somewhat shortened, and presented a thickened irregular fibrous ridge along the free margin, dotted over in places with small fibrocalcareous areas. The chordae tendineae reaching to those areas were also shortened and thickened. We call that chronic

endocarditis. The other valves were negative. The enlargment of the heart is to be attributed partly to the mitral valve, partly to the arteriosclerosis of the aorta and the great vessels, partly to the arteriosclerotic degeneration of the kidneys, and partly to the anemia, plus, of course, the wear and tear of life, which is always a factor. The right side was more hypertrophied than the left. The myocardium showed in places what were probably a few areas of fatty degeneration, but other than that was negative.

The bone marrow of the right femur looked, grossly, like the bone marrow in pernicious anemia. It was of a rather dark red color and cut out easily, but was a little mushier than it is in typical cases of pernicious anemia. Still, if I were compelled to write the diagnosis from the gross appearance I should not hesitate to say that it was primary anemia. The microscopical examination, however, did not quite bear out that opinion. It was not sufficiently typical to enable us to write definitely the diagnosis of pernicious anemia. It was a grave anemia, however.

If the anemia was not primary in this case, but secondary, what is the explanation of its orgin? Within the uterine cavity there was a polyp about three inches long, the surface of which was coated with blood. Whether over a long period of time that polyp could bleed enough to produce an anemia as grave as this is the question. I should say that it could. The polyps may be of any size or shape. On microscopical examination you will find that there is a network of large thin-walled vessels running over the surface of the polyp. For some reason, either because of a change in the pressure or something else, these thin-walled capillary vessels break, with the result that there is a bleeding mass, the blood dripping from various points. It is not uncommon to find in these cases a history of long bleeding. That seems to have been the case here.

The anemia was a factor, I think, in her dyspnea.

A PHYSICIAN: How large was the uterus?

DR. RICHARDSON: It was considerably enlarged, enough to demonstrate that when there is a mass in the uterine cavity the organ does enough work in trying to get rid of it to produce some hypertrophy.

If we did not have the polyp in the uterine cavity it would be harder to prove that the case was not one of pernicious anemia.

### Necropsy 3422

A housewife of forty entered January 5 for the relief of cough. Her father, one brother, and one sister died of consumption. The

patient cared for all three during their illnesses. She had "inflammatory rheumatism" at seventeen and off and on for the next ten years, keeping her in bed four times for six weeks. She had had chronic cough with yellowish sputum since she was seventeen. Her bowels were always constipated. She had never been pregnant. Her best weight was 135 pounds, her present weight about 100.

For the past fifteen years she had tired very easily and had had dyspnea on slight exertion. Nine months ago she found she could not do as much work as usual. Her legs and ankles swelled. She went to a hospital, from which the following report was sent. "The patient was admitted May 20 last. The heart was somewhat enlarged. The apex impulse was diffuse, best felt in the fifth space, anterior axillary line. There was a possible presystolic thrill. The heart action was irregular in force and rhythm. The first sound at the apex was very sharp and accentuated and preceded by a rumbling murmur. A systolic murmur was heard all over the precordia and transmitted into the axilla. On entrance there was considerable cyanosis and much edema of feet and ankles. She was discharged June 30 feeling better than for years." Since her discharge she had been in bed more or less. Weakness and fatigue had been the characteristics of her illness. Recently her cough had troubled her more. During the past five days she had become hoarse, and now spoke in a hoarse whisper.

Examination showed a poorly developed, fairly well nourished woman with slightly cyanotic lips and mucosae and slight exophthalmos. The apex impulse of the heart was seen and felt in the sixth space. The supracardiac dullness in the first space was 11.5 cm., in the second space 9 cm. The other borders are not recorded, but the visiting physician notes, "Heart enlarged and displaced to the left." The action was absolutely irregular. The pulmonic second sound was greatly increased. At the apex was a sharp first sound. A systolic blowing and a diastolic roll were heard at the apex, where a thrill was felt. The blood pressure was 110/60. The lungs were hyperresonant. The left chest was more resonant than the right, and moved more. The breathing was much diminished on the right. At the right apex in front there was dullness below dull tympany. There was no coin sound and no amphoric phenomena of any sort. The left chest showed increased compensatory (resonance). In the right lung the breath sounds were almost absent, high-pitched, with an occasional musical râle. (See Fig. 17.) On the left there was compensatory breathing. The abdomen was distended, dull

in the flanks. The liver dullness extended from the fifth rib to 5 cm. below the costal margin; tender. The feet showed slight edema.

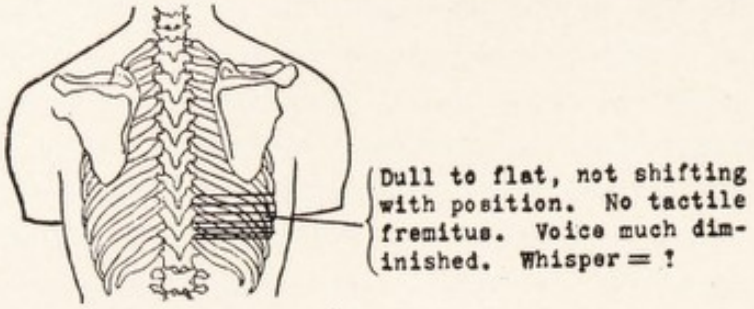



FIG. 17.

The right pupil was greater than the left. Both were dilated. They reacted to light. The other reflexes were normal.

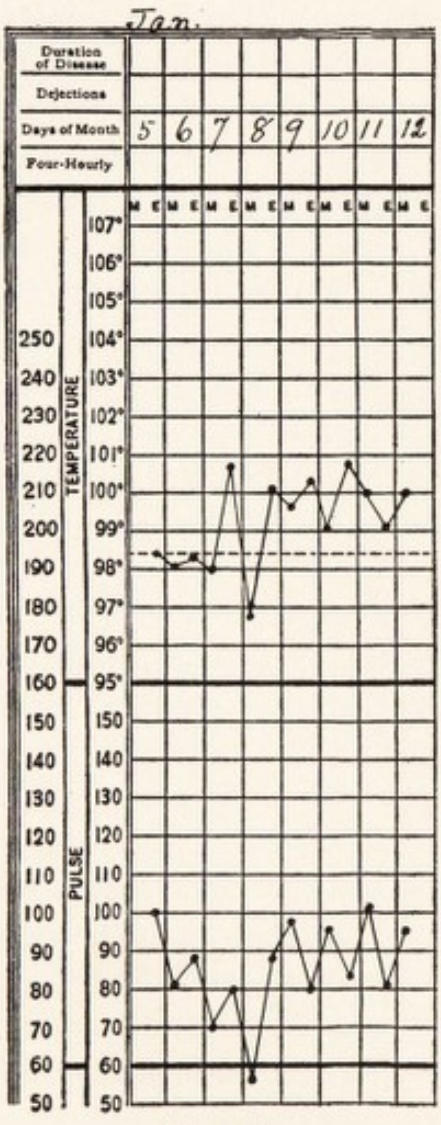



FIG. 18.

The temperature and pulse are shown in Fig. 18. The respiration was 50 at entrance, afterwards usually 30. The output of urine was normal, the specific gravity 1.030–1.032. The sediment showed some pus and granular casts at one of two examinations. The hemoglobin was 60%. The leucocytes were 28,000 at entrance, 5400 January 11, the polynuclears 88%, the reds 4,048,000, with moderate achromia. A Wassermann was negative. The sputum showed tubercle bacilli. A radial tracing and electrocardiogram at entrance showed auricular fibrillation. Fluoroscopic examination showed right hydropneumothorax with extreme displacement of the heart to the left.

The patient was put upon magnesium sulphate $\bar{3}$ss in the morning, ammonium chloride gr. v t.i.d. p.c., and sodium veronal gr. v. January 6 the right chest was tapped and 50 ounces of thick pea-soup-like fluid withdrawn. The specific gravity is not recorded. The smear snowed pus cells and small mononuclears; no organisms. A culture showed pneumococci. A guinea pig inoculated

with the fluid showed a negative necropsy February 11. The patient was very much more comfortable after the tap. The heart, however, did not come back at all. The fluid did not reaccumulate during the next three days, and the general condition and heart rate and quality remained about the same. There was a very definite coin sound over the right chest. January 9 a throat consultant reported complete paralysis of the left recurrent laryngeal nerve.

January 11, just as she was in preparation for another tap, she grew more dyspneic and cyanotic. The heart became rapid and more irregular, and a mass about the size of a grape-fruit appeared in the abdomen in the region of the umbilicus, firm and definite in outline, not fluctuating. She grew rapidly worse, and died January 12, asphyxiated by her sputum.

*Clinical Diagnosis (from Hospital Record).*—Pyopneumothorax, right.

Acute cardiac dilatation.

Mitral stenosis and insufficiency.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the mitral valve; stenosis and regurgitation.

Ball thrombus in the auricular appendage.

A piece in the mesenteric artery.

Tuberculosis of the lungs.

Probably pyopneumothorax.

Probably compression atalectasis.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral valve; mitral stenosis.

Pyopneumothorax, right. (2000 c.c. of frank pus.)

Left laryngeal paralysis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Chronic passive congestion of the liver.

Hydropericardium.

Ascites.

Anasarca.

Arteriosclerosis of the aorta and its great branches, moderate.

Arteriosclerosis of the pulmonary artery and its branches, moderate.

Cholelithiasis.

Old infarcts of one kidney.

Chronic pelvic peritonitis.

Chronic appendicitis.

Chronic pleuritis.

Heart: *The myocardium was negative.* The left ventricle wall was 11 mm., the right ventricle 8 mm., (greatly thickened). The cavities were enlarged. The mitral valve was of the button-hole type, 16 × 5 mm. The chordae tendineae were fused with the valve all the way down to the papillary muscles. The valves were otherwise negatve. There were no fresh vegetations. There was a clot in the left auricular appendage, presumably an ante-mortem clot.

Lungs: The pus was walled off in the pleural cavity on the right for about the lower two-thirds. That portion of the lung was collapsed against the spinal column in compression atalectasis. At one point the pleura showed an area suggesting that a portion of the lung had been adherent, and had pulled off, establishing communication between the bronchi and the pleural cavity; hence pyopneumothorax. The other third of this lung, isolated in its own compartment, was not remarkable. The apex and the bronchial lymphatic glands were negative. The left lung was voluminous, and showed chronic passive congestion. Otherwise it was negative. No record as to the cause of *recurrens* paralysis. There was no tuberculosis anywhere and no abdominal tumor!

**Necropsy 2646**

An American housewife of twenty-nine entered June 27, 1910. Her father died of trouble with the heart and liver after suffering with acute rheumatism. She had had scarlet fever, kidney trouble, chickenpox and mumps. At fourteen she was ill a year with rheumatic fever and was in bed six months. She had one miscarriage at twenty-two. About once each winter she had a cold. She had occasional attacks of nausea and vomiting. She urinated three or four times at night. She had frequent frontal headache. A few months before admission she weighed 200 pounds, her best weight. She now weighs 159.

In the summer of 1909 she began to have dyspnea and backache and to tire easily. During the winter she had a cold, but worked in a laundry until a month before entering the hospital, when she began to feel worse.

Examination showed a well nourished woman breathing somewhat rapidly. The skin was moist, hot and loose, the muscles flabby, the mucous membranes slightly cyanotic. Her only tooth was a carious stump. The tongue showed a dry brown-gray coat. The throat was reddened, the pharynx dry. The left side of the chest over the precordia was more prominent than the right. There was diffuse tumultuous heaving over this area. The apex impulse of the

heart was seen and felt in the fourth, fifth and sixth spaces, with the point of maximum intensity at the fifth space. The left border of dullness was 16 cm. from midsternum in the left fifth space, anterior axillary line, the right border 4.5 cm. to the right in the fourth space. There was no increase in the substernal dullness. The action was irregular, rapid. There was a short powerless impulse at the apex with a presystolic leading up to a first sound which was practically replaced by a harsh blowing systolic murmur heard over the entire precordia, loudest just inside the apex and transmitted to the axilla and back. The pulmonic second sound was greater than the aortic second, snapping, accentuated and reduplicated. The pulses were equal, irregular in rhythm, rapid, of low volume and tension. Not all beats reached the wrist. The artery walls were not palpable. The systolic blood pressure was 105. The lungs showed fair expansion, good resonance throughout except in the left axilla below the fourth rib, where there was dullness to flatness. Posteriorly there was good resonance except at the left base below the angle of the scapula. The breath sounds were rather harsh. There were a few moist râles at both bases, more marked on the left. The abdomen was full and held somewhat rigid. It was tympanitic throughout, with slight general tenderness, more marked in the region of the sigmoid and the liver. The liver dullness extended from the fifth rib to two finger-breadths below the costal margin, where the edge was felt, tender. The fingers and toes were slightly clubbed. The pupils were normal, the knee-jerks very sluggish.

The temperature was 98° to 102.8° until July 20, when there was a terminal drop from 97.9° to 96°. The pulse was 69 to 120, the respiration 24 to 46. The amount of urine was normal, the specific gravity 1.009 to 1.016. The urine was cloudy at four of five examinations, showed a slight trace to the slightest possible trace of albumin at three, hyalin and granular casts with cells attached at two, with blood attached at one, a few red blood cells at another. The hemoglobin was 95% to 85%, the leucocytes 36,000–12,000–39,600, the polynuclears 90%–70%–91%. The reds showed polychromatophilia at one of three examinations. No Wassermann is recorded.

At entrance the patient was very uncomfortable, breathing rapidly, and had considerable pain in the right arm and leg. Aspirin gave relief but was followed by marked delirium and some vomiting. After this she had a feeling of weakness in the entire right half of the body. From entrance she had had a feeling of numbness on this side. After digipuratum had been given for four days the apex and wrist

beats became synchronous and the pulse rate was much slower and steadier. There was a definite presystolic thrill and murmur, a loud first sound and a systolic murmur at the apex. No murmurs at the base were made out. The lungs showed a few râles at the bases. There was considerable edema over the sacrum and a small amount of shifting dullness in the abdomen. There was very little edema of the legs. A small bed sore over the coccyx healed very slowly.

From July 7 the patient lost ground. Her memory was very poor. She looked very pale and weak. She had very little dyspnea and no palpitation. The temperature was very irregular. She had three sudden attacks of sharp pain in the region of the spleen lasting half an hour to two hours and keeping her awake. She seemed to be in a kind of stupor most of the time, and fell asleep while people were talking with her, but could easily be roused and then seemed perfectly rational. A blood culture showed a slight growth of staphylococcus aureus.

By the 17th she was much worse. There was a steady aching in the splenic region with attacks of sharp pain. July 21 a blood culture showed a growth of atypical streptococci in chains and clumps, weakly Gram-staining. For two days the pulse could not be felt in the left hand, and for several hours before death none in the right. The heart became permanently enlarged. The apex impulse, very diffuse and flapping, could easily be felt as far as the midaxillary line, the right border two inches from midsternum, and the lower border in the sixth and seventh spaces. The action was very irregular and rapid, and so tumultuous that the thrill could not be timed. Still a fairly definite presystolic murmur and a tremendous systolic murmur were heard at the apex. No murmurs were made out in the aortic area. The left lung showed dullness at the base with diminished breath sounds. This lung was full of moist râles. The right lung was clear. The patient lay on her left side. There was only slight edema, and no shifting dullness, but the abdomen was considerably distended so that it was impossible to palpate the liver or spleen. Finally she had almost constant attacks of pain in the region of the spleen, with considerable tenderness there. July 23 she died.

*Clinical Diagnosis (from Hospital Record).*—Acute ulcerative mitral endocarditis.

Aortic disease?

Chronic mitral endocarditis.

Perisplenitis.

Chronic passive congestion.

Multiple infarcts.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the mitral valve.

Mitral stenosis and regurgitation.

Acute endocarditis.

Thrombosis and embolism.

Infarcts of kidney, spleen, brain (?)

Chronic passive congestion.

Hypertrophy and dilatation of the heart.

Streptococcus septicemia.

*Anatomical Diagnosis.*—1. PRIMARY FATAL LESIONS.

Fibrous endocarditis of the mitral and aortic valves.

Verrucose endocarditis of the aortic valve.

Polypous endocarditis of the mitral valve.

Streptococcus septicemia.

2. SECONDARY OR TERMINAL LESIONS

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Infarcts of spleen and kidneys.

3. HISTORICAL LANDMARKS

Urethral caruncle.

DR. RICHARDSON: We were not permitted to examine the head. There is nothing in the record about anasarca or edema.

DR. CABOT: There was very little in life.

DR. RICHARDSON: There was a good amount of subcutaneous fat. The peritoneal cavity contained no fluid. Scattered over the peritoneum were small purplish-red ecchymotic areas, and there were a few on the pericardium.

DR. CABOT: But there were none of those areas on the skin?

DR. RICHARDSON: None.

The liver was two fingerbreadths below the costal border. The diaphragm was at the third interspace on the right and the sixth on the left. For some reason the diaphragm was perhaps a little high on the right and it was low on the left, indicating of course, so far as it goes, the presence of fluid or something abnormal in the left cavity.

The right pleural cavity contained no fluid. In the left there was a small amount of thin pale fluid. There were no adhesions, which is rather unusual at almost any age.

The glands were out of the picture.

The lungs showed no areas of infarction or consolidation. The tissues were a little leathery, brownish-red, yielding a brownish-red fluid showing the so-called heart cells, that is the large cells filled with

pigment,—chronic passive congestion of the lungs, moderate in amount.

The pericardium was out of the picture except for the spots mentioned. The heart weighed 470 grams. In a woman of twenty-nine, if the heart had weighed 230 or 240 grams, it would have been all right for her. So there is considerable hypertrophy. The right ventricle wall measured four mm., the left twelve,—both fairly thick. The left auricle wall measured two to four mm. That is rather unusual. A thick left auricular wall should mean that it was working against something. The columnae carneae were flattened in the left ventricle, fairly marked on the right. The cavities, more especially on the left side, were enlarged. That would indicate something on the aortic valve. The mitral valve circumference was 12 cm., the aortic 7 cm., the tricuspid 12 cm. The mitral and the tricuspid in this heart were of the same dimensions; that is a little large for the mitral, and the tricuspid was about what it might be if this heart was normal. The mitral and the aortic valve showed some diffuse fibrous thickening,—a certain amount of chronic endocarditis.

Along this thickened mitral valve there was a mass of grayish to yellowish soft vegetations. It was very large and irregular, presenting, tit-like projections and extended down along the chordae tendineae. At one point it extended for a short distance on to the body of the mitral valve. A short distance above this, on the inner surface of the auricle, there was another flattened mass of these vegetations.

The aortic cusps showed the fibrosis mentioned and running along them some verrucose endocarditis,—that is, chronic and acute. So that these two valves showed chronic and acute endocarditis, the acute being more massive on the mitral valve and in minute amount on the aortic but still present on both valves. The tricuspid and pulmonary valves and the coronary arteries were out of the picture. The aorta and great branches, the pulmonary artery, veins and vena cava were negative.

The liver weighed 2413 grams, a large liver, but all it showed was chronic passive congestion.

The gall-bladder showed no stones, the bile ducts were free the pancreas negative, the duct free.

The spleen weighed 417 grams, considerably hypertrophied, and showed definite infarcts. There was a question raised which makes of value a statement as to whether the infarcts in this spleen were soft.

They were soft. The largest one measured 3½ cm. across. That is quite large.

The kidneys were a little large, the cortex rather wide, but other than for some chronic passive congestion and a few infarcts they were negative.

The uterus and adnexa were negative.

The gastro-intestinal tract showed no definite lesions of any sort except a little congestion.

Culture from the heart blood at the time of necropsy showed a moderate growth of micrococci growing in long chains and clumps with many involution forms after three days,—the *streptococcus viridans*.

The vegetations on the mitral were so massive that we used the term polypous to describe them.

DR. CABOT: The only thing we got wrong was that I thought there was a mitral stenosis due to a chronic lesion. There was a mitral stenosis due to these fresh vegetations. I do not know how we are ever going to tell the difference between a mitral blocked by a chronic permanent lesion and a mitral blocked by acute vegetations like this.

It is obvious that there is something in this case which has produced hypertrophy and dilatation of the heart. We are not accustomed to thinking that acute endocarditis produces cardiac hypertrophy and dilatation. Is it possible that this disease has been going on over a year? She had very little chronic endocarditis, not enough, I should think, to produce hypertrophy and dilatation. It seems to me we have to say that this is a case which has been going on for a year, that these vegetations have been going on for months and months; they have obstructed the circulation and so produced this hypertrophy. It is an idea which has not yet taken its place in our ideas about the heart that a purely soft process can last so long and make so much obstruction of the valve that hypertrophy and dilatation occur as in this case. I do not know what else to say as to the cause of the big heart. Did that problem present itself to you, Dr. Richardson, why that heart was so big?

DR. RICHARDSON: Yes, it did. It makes me think of the hearts in chorea. In those cases we find changes on the valves, sometimes on all three valves. It is a fibrosis not so very marked, but still there is some hypertrophy of the heart and more than one would think the chronic endocarditis accounted for. It seemed to throw it back into the physiological pathology rather than the anatomical.

DR. CABOT: Otherwise I do not see that we have need to change anything of the reasoning we made here. We notice that the nycturia, if it was a real thing, was present without kidney disease.

DR. RICHARDSON: The anatomy here is interesting when we note the thickness of the auricular wall which is accounted for by the stenosis produced by the mass of vegetations.

DR. CABOT: There is a little fibrous process, Dr. Richardson says, on the aorta and on the mitral,—not enough to do anything in the way of stenosis or regurgitation, which has to be accounted for by the soft vegetations. There is nothing in the aorta itself except these vegetations.

DR. RICHARDSON: How does the pulse work in these chorea cases —is it high?

DR. CABOT: I do not think it is any different from the pulse in any other failing heart. It is not high over long periods ordinarily.

**Necropsy 861**

A married Canadian laundress of sixty entered March 10. She gave a history of scarlet fever and smallpox in childhood; no other serious illnesses until the present one. She had had to do hard work to support her husband and three children for twenty years and felt worn out. For five weeks she had felt miserable.

Two weeks and a half before admission she had sudden onset of cramps in her feet, and both legs felt numb to the knees. There was pain below the knees. The right leg was better in a few hours, but the left had continued numb and painful. A little over two weeks ago she began to be dyspneic and noticed that her heart beat fast and irregularly. She had had cough recently, especially at night.

Examination showed a fairly well nourished woman. The mucous membranes were pale. There was a systolic murmur in the axilla. The pulse was weak, irregular and intermittent. The lungs showed numerous squeaks and grunts. The abdomen was negative. The left leg below the knee was enlarged, firm, and painful on pressure. The vessels in the calf were blue and cord-like. The foot was blue-black to above the ankle. The femoral pulse was felt in the groin and above the popliteal space. The right foot was negative.

Before operation the temperature was 97.2° to 100°. The pulse was 150 at entrance, afterwards 90 to 110. The respirations were 27 to 31. The urine and blood are not recorded.

The patient was given one-fortieth of a grain of strychnia every four hours and tincture of digitalis ten minims three times a day with

good result. The pulse became much more steady and better in quality. The toes became dry and black, and an area of redness appeared below the knee. She complained of pain in the precordia and the right thigh and leg. March 16 the right foot was slightly cold. An icebag over the heart gave some relief. March 17 she felt better, had less pain, and the pulse was better, though still rather weak and very irregular.

March 18 the left leg was amputated at the junction of the lower and middle thirds of the thigh. Pathological examination showed the foot and leg entirely black and gangrenous. The cutaneous and deep veins were found thrombosed throughout, and there was an embolus at the bifurcation of the popliteal artery, above which the artery was also thrombosed. The patient was in poor condition at the end of the operation. The next day the pulse was irregular. The night of March 19 she was delirious. She complained of severe pain in the right leg. Next day the right foot and leg were mottled and purple to the knee. No pulse was felt in the tibials or the popliteal.

March 22 the right leg was amputated at the junction of the lower and middle thirds. There was very little bleeding. The femoral vessels were plugged by thrombi. Pathological examination showed an area of commencing gangrene on the outer side of the foot. The entire leg as high as the knee was slightly yellowish. Dissection showed the veins everywhere thrombosed, and in the popliteal artery at its bifurcation a grayish, more or less discolored thrombus. Below, the arteries contained but little blood, while above they were filled with clots, and several thrombi were adherent to the walls. The patient was in better general condition after this operation than after the first.

March 30 she had an attack of pain and discomfort in the epigastrium and tingling and numbness in the right arm. The arm and hand became cold and white. The pulse was present in the subclavian artery but not in the brachial. April 1 there was a suggestion of pulse in the right wrist, and the hand was warm. On the fourth there was a feeble right radial. The wounds were clean and solid. She felt very well. April 16 there was a flicker of pulse in the right brachial and the radial.

April 17 she had right hemiplegia. April 18 she died.

*Clinical Diagnosis (from Hospital Record).*—Embolic gangrene of leg.

*Dr. Maurice Fremont-Smith's Diagnosis.*—Embolism and thrombosis.

Chronic and endocarditis (mitral stenosis?).

Hypertrophy and dilatation of the heart.

*Anatomical Diagnosis.*—Chronic endocarditis (mitral stenosis).

Hypertrophy and dilatation of the left auricle.

Streptococcus septicemia.

Thrombosis of the left auricular appendix.

Embolic thrombosis of the right axillary artery.

Hemorrhages into basal ganglia on the left side.

Multiple infarcts of the spleen and kidneys.

Double amputation at lower third of the thighs for embolic gangrene.

Obliterating thrombosis of the left common iliac and external iliac veins.

Infarcts in the left lung.

Ulcer of the duodenum.

Hemorrhage into the small intestine.

Malformation of the left common iliac vein.

Chronic perihepatitis.

Cholelithiasis.

DR. FREMONT-SMITH: Was that a hemorrhage into the basal ganglia? It was not a hemorrhagic infarct?

DR. RICHARDSON: I should think from the description that those were of the nature of infarcts, because the cerebral vessels were not remarkable.

A PHYSICIAN: Was there any evidence of bacterial endocarditis on that mitral?

DR. FREMONT-SMITH: The temperature never went above 100°.

DR. RICHARDSON: No, there was not.

DR. CABOT: Was this mitral stenosis pathologically like any other mitral stenosis?

DR. RICHARDSON: Yes.

DR. CABOT: I am interested in this case because it is a type of mitral stenosis that does not get adequate mention in the literature. We have quite a group of cases of mitral stenosis in our necropsies in which the first thing that anybody knows is gangrene. The very first event is not congestion or anything suggesting the heart at all. It is peripheral gangrene.

DR. BOCK: There is another group of which we had three examples in one year, of extreme mitral stenosis in patients between twenty

and thirty years of age with no cardiac symptoms until the sudden onset of pulmonary edema followed by death.

DR. CABOT: Would you call that passive congestion, only very sudden?

DR. BOCK: I should hesitate to call an acute pulmonary edema, which comes on very suddenly, passive congestion.

DR. CABOT: How many years ago were those cases?

DR. BOCK: One or two. There were three cases. I think we had two and Dr. Magrath the medical examiner had one. I saw all three. Dr. Magrath said that that was an event that he had seen before occurring in uncomplicated mitral stenosis in young people.

## POINTS ON MITRAL DISEASE

1. As to etiology our knowledge may be summed up by saying the rheumatic infection (including arthritis, chorea, and probably some cases of tonsillitis), is the only one which has any known relation to chronic valvular disease involving the mitral valve. The cases of acute or subacute streptococcic endocarditis probably represent a different type of disease which, however, may be superimposed upon a rheumatic valvular scar.

2. In these 180 cases there was no evidence either of the gonococcus or of the influenza bacillus as an etiological factor.

3. There is a definite predominance of females over males among the cases of pure mitral disease. When the other valves are also involved there is no such predominance.

4. Mitral disease is compatible with long life, and if the patient gets beyond the second decade his valvular lesion is usually not the cause of his death. Intercurrent infections, trauma or surgical operations are more likely to end life, the mitral lesion remaining latent.

5. Over half the patients with mitral disease die a non-congestive death, that is, half do not die of heart disease itself. In the cases of *pure* mitral trouble only two-fifths die a congestive death.

6. Even when the mitral disease is complicated by stenosis at the aortic or one of the other valves, dropsy supervenes before death in only half of the cases.

7. The size of the slit left in the stenosed mitral curtains has no relation to the clinical signs during life. Patients with only a small slit left in the mitral aperture may have no more serious symptoms or signs of congestion than those with a much larger orifice.

8. Chronic passive congestion is no commoner in the cases of mitral disease complicated by aortic disease than in the cases of mitral disease alone.

9. Fever and leucocytosis when they occur, usually turn out to be due to causes within the heart itself, that is, to acute endocarditis or to intracardiac thrombosis.

10. Thrombosis in the auricular appendages, especially the left, is to be expected in a quarter of the decompensating and fibrillating mitral cases.

11. Glomerulonephritis acute or chronic is to be expected before the end of life in one-fifth of the cases of mitral disease.

12. Arrhythmia due to auricular fibrillation is twice as common in pure mitral disease as in the mitral cases with aortic stenosis as well.

13. When mitral disease occurs alone, uncomplicated by stenosis on any other valve, the reduction in the size of the orifice reaches a greater degree than when aortic disease complicates the picture.

14. Over half the cases of mitral disease are not recognized during life in hospital patients. This is presumably due to the considerable number of such cases occurring in surgical wards or dying of pneumonia and other infections without having presented any cardiac complaints during life.

15. The importance of suspecting and especially investigating the possibility of mitral disease, even when no presystolic or diastolic murmur is detected, should be remembered in the following conditions:

(a) In cases of cerebral or peripheral embolism occurring in relatively young persons and presumably not due to arteriosclerosis.

(b) Arrhythmia and decompensating heart disease without evidence of syphilis or hypertensive cardiovascular trouble, and especially if there is a rheumatic history.

(c) Evidence of an acute endocarditis (since a chronic mitral endocarditis usually underlies this lesion).

(d) The presence of a sharp first sound and a dull or feeble second sound at the apex; doubling of the second sound (or a third heart sound) along the left sternal margin; absolute arrhythmia without known cause in a young person.

16. Pure mitral stenosis without any other valve lesion is more than twice as common as the combination of mitral stenosis with valve lesions (mitral-and-aortic, for instance), and considerably more common than all the other combinations put together.

17. Mitral regurgitation is one of the rarest of valve lesions, and cannot with any certainty be recognized in life.

## Multiple Valve Lesions

Although most of the tables in the preceding pages include the valve lesions other than "pure" mitral stenosis and show the statistical difference between this lesion and the more complicated stenoses, it seems well to recapitulate here the main features of the commoner combined lesions separated by themselves.

## Mitral and Aortic Stenosis

*Sex.*—Males predominate 27 to 13 in the necropsied cases and 59 to 30 in the living cases. This is in sharp contrast with the series of pure mitral cases,—females 62, males 45 in the necropsies and 149 to 68 in the living patients. Why this is so I have no idea, but it is one of the items going to make up *the widespread association between diseases of the aortic valve and the male sex.*

*Age.*—The age incidence at the first attack of rheumatism or chorea, at the time of first seeking medical advice, and at the time of death, is about the same in the mitral-and-aortic cases and in the pure mitral cases, yet (as already noted) the term of life in mitral-and-aortic cases from first complaint to death is (in those necropsied) strikingly shorter than in the pure mitral cases or in the other "combined lesions" (see Table 17, p. 44).

Thus the "pure" mitral case's average duration is 15 years.

The mitral-and-aortic case's average duration is 3 years.

The "combined lesions" case's average duration is 10 years.

The significance of these figures however is somewhat diminished by comparison with the non-necropsied cases (Table 10, p. 36). Here of course we have no such certainty as to the diagnosis as we have in the necropsied cases. But on the face of the figures the Mitral-and-Aortic cases live longer than the pure Mitral cases.

*Multiple Attacks of Rheumatism or Chorea.*—Here there is a distinct difference between the mitral-and-aortic cases, thirteen out of seventeen of which had multiple attacks and the pure mitral cases, only twelve out of forty of which had had multiple attacks.

## SYMPTOMATOLOGY

*Dyspnea* is relatively more frequent (73%)as an early complaint in mitral-and-aortic cases than in pure mitral cases (50%). The same thing is true of other "cardiac symptoms." This is another

way of stating the fact that in relatively few of the mitral-and-aortic cases is the cardiac lesion latent. The patient is less apt than is the "pure" mitral patient to die of some infection, operation, or trauma before compensation has failed.

In the clinical picture of "living cases" (not necropsied) precordial pain of some sort was an *early* feature in sixteen out of twenty-seven mitral-and-aortic cases, and of only eight out of twenty-five "pure" mitral cases. In the *later* course of the living cases pain was also a feature more often in mitral-and-aortic cases than in pure mitral cases.

*Arrhythmia and palpitation* (-fibrillation?) were less commonly noticed in the mitral-and-aortic cases (ten out of thirty-four) than in the pure mitral cases (thirty-eight out of sixty-seven).

*Nutrition* was poor in sixteen of forty mitral-and-aortic cases, (40%) as contrasted with twenty-eight out of 107 pure mitral cases (26%).

In the *Physical Examination* of cases which ultimately came to necropsy decided enlargement of the heart was not found any more often in mitral-and-aortic cases (twenty out of thirty-three) than in the pure mitral cases (fifty-two out of eighty-seven). This is somewhat surprising. But the figures are practically the same in the non-necropsied cases,—which showed definite enlargement in 127 to 150 pure mitral (84%) and in 81 of 89 mitral-and-aortic (91%).

Transverse enlargement was *not* found more common clinically in the pure mitral cases than in the mitral-and-aortic. It was the rule in both groups.

**Heart Weights at Necropsy.**—Hypertrophy was present in thirty-seven of forty cases, distinctly more than in the "pure mitral" series, 89 of 107. The degree of hypertrophy is also greater in the mitral-and-aortic series, nineteen of thirty-nine over 500 grams (50%) as against thirty out of 107 in the pure mitral series (28%). This shows how unreliable are our clinical estimates of cardiac enlargement as reported in the last section.

**Dilatation of Cavities.**—Preponderant dilatation of the right side of the heart was present at necropsy in twenty-five pure mitral cases out of 107 (or 23%), and in five of forty mitral-and-aortic cases (12%).

Preponderant dilatation of the left ventricle was present in five of forty mitral-and-aortic and in three of 107 "pure mitral" cases.

Preponderant dilatation of the left auricle was present in twenty-seven of 107 pure mitral cases, and in only three of forty mitral-and-aortic cases. Obviously the strain of the double lesion is more

equally distributed while that of the single mitral lesion falls on the right side of the heart and on the left auricle.

## DIAGNOSIS

The heart was considered enlarged in life in twenty-nine cases of forty. An apical presystolic or diastolic murmur was heard in 21 out of 40 or 52%, so that in ½ the cases we had *some* reason to make a diagnosis of mitral stenosis, especially when a clear rheumatic history was present without evidence of syphilis and in a young person.

In five cases no murmur was heard, and in fourteen a systolic murmur only. In eight cases (20%) a sharp or accented first sound was noted at the apex, but only three of these were associated with presystolic or diastolic murmurs. In the mitral-and-aortic "living patients" sixty-three out of eighty-nine (70%) showed a sharp first sound at the apex.

In seven cases the second sound was absent at the apex.

Doubling of the second sound along the left sternal margin was noticed only four times in forty cases (probably from inattention). Accentuation of the pulmonic second was recorded eighteen times in forty cases.

Arrhythmia (presumably fibrillation) was noted only ten times. The rheumatic history was clear in eighteen cases only.

So far I have been assembling data relative to the diagnosis of the *mitral* stenoses found post-mortem, and I conclude that on the evidence *this* diagnosis was recognized in eighteen out of forty cases.

How about the aortic lesion? A diastolic murmur loudest along the sternal margin or in the second right interspace was heard in *sixteen* cases. This was associated with cardiac enlargement in fourteen, and a collapsing ("Corrigan") pulse and other arterial phenomena in ten cases. *This justifies a diagnosis of aortic regurgitation in sixteen cases out of* 40.

*Direct evidence of aortic stenosis* was seldom sufficient for diagnosis. A basal systolic thrill was recorded in only four cases. Plateau pulse was never recorded. A diminished or absent aortic second sound is noted in six cases, but not one of these is associated with any of the four systolic thrills just mentioned and only four times was the weak aortic second sound associated with a diastolic basal murmur. It is of no considerable value therefore.

Any diagnosis of aortic stenosis in the forty cases of this series which showed it at necropsy had to be based on the assumption that

in rheumatic lesions (such as eighteen of these cases appeared from their history to be) aortic disease almost always produces stenosis as well as regurgitation. With direct evidence of aortic regurgitation in a rheumatic case it is safe to assert that stenosis is present in about ⅘ of the cases (see "pure" rheumatic regurgitant lesions p. 252).

**Diagnostic Summary.**—Of forty cases, the aortic and the mitral stenoses found at necropsy were both recognized in life in eight cases or 20%. The mitral lesion was recognized in ten cases and the aortic alone, in eight, while neither was detected in fourteen.

Mitral and Aortic Stenosis

| | |
|---|---|
| Both lesions recognized in | 8 cases |
| Mitral alone recognized in | 10 cases |
| Aortic alone recognized in | 8 cases |
| Neither recognized in | 14 cases |
| | 40 |

*Passive Congestion.*—Aortic disease when superadded on a mitral lesion produced neither more nor less congestion and dropsy than mitral disease alone.

*Fever and Leucocytosis.*—Fever was present in twenty-two of forty cases (cf. "*pure*" *mitral*, 51 of 107), i.e., slightly more often in the mitral and-aortic series. Leucocytosis was found in nineteen of forty (cf. "*pure*" *mitral*, 66 of 107).

The percentage of infarcts was the same in mitral-and-aortic as in "pure" mitral disease.

*Thrombi in the Auricles.*—Auricular thrombi are recorded in five of forty cases (one in eight) whereas in "pure" mitral disease there were twenty-eight of 107 cases (one in four). Apparently these *auricular thrombi are only one-half as common in mitral-and-aortic disease as in "pure" mitral.*

*Infectious pulmonary complications* are apparently less common in mitral-and-aortic disease (five out of forty) than in pure mitral cases (seventeen in 107).

*Nephritis* is commoner (thirteen of forty cases) than in *Pure mitral* (18 of 107).

*Positive Cultures* at necropsy are also more common (fourteen of forty cases) than in "*pure*" *mitral* (25 of 107). But these differences between the two groups are probably not significant (see Tables 45 and 46).

As to the degree of narrowing of the mitral it appears from Table 47 that the "pure mitral" cases tend to a greater degree of stenosis than the mitral-and-aortic cases.

### MODE OF DEATH IN MITRAL AND AORTIC STENOSIS

21 of 40 cases died from passive congestion.
4 of 40 cases died from passive congestion with sepsis.
8 of 40 cases died from sepsis alone.
7 of 40 cases died from non-cardiac causes.
0 of 40 cases died from embolism.

Of the eight septic cases five were overshadowed by the effects of an acute endocarditis with septicemia, three were cases of surgical sepsis, two of them with general peritonitis.

Of the four cases combining passive congestion and sepsis, three were cases of active septic acute endocarditis engrafted on a chronic process, which had been severe enough to produce marked passive congestion. The remaining case was complicated by pulmonary abscesses due to stenosis of a bronchus from pressure of a gland (tuberculous?) in an Italian boy of fourteen.

Among these seven deaths classed as due to non-cardiac diseases, four were due to pneumonia, one to apoplexy, and two were post-operative deaths.

Comparing these with the "pure mitral" cases we get the following results:

TABLE 52

| Manner of death | Mitral 107 cases | Mitral and Aortic 40 cases |
|---|---|---|
| Congestive deaths | 49 or 47% | 21 or 52% |
| Acute septic deaths | 10 or 9% | 8 or 20% |
| Non-cardiac causes | 33 or 31% | 7 or 17% |
| Embolic deaths | 12 or 11% | 0 |
| Sudden death without known cause | 2 | 0 |
| Congestive and septic death | ..... | 4 |

### SUMMARY OF THE CONTRASTS BETWEEN THE 107 "PURE MITRAL" CASES AND THE 40 "MITRAL-AND-AORTIC CASES

1. *Etiology.*—Mitral-and-aortic disease is common in males, lasts only three years (instead of fifteen) on the average from the first symptom to death, and is much more apt to give a history of multiple attacks of rheumatism (or chorea.)

2. *Symptoms.*—Mitral-and-aortic begins more often with dyspnea, i.e. is less often a latent lesion in a patient dying of non-cardiac disease, is more apt to have precordial pain among its symptoms, and less apt to show palpitation and arrhythmia.

3. *Physical Examination.*—Nutrition is more often poor.

We should expect a record of greater cardiac enlargement than in "pure" mitral cases. But this is not so; neither is there any difference in the shape of the cardiac dullness in the two groups.

4. *Necropsies.*—Hypertrophy of the heart is commoner but is less preponderant on the right side. Passive congestion is no more or less common than in "pure" mitral disease.

Auricular thrombi are but half as common. Nephritis and post-mortem cultures are commoner.

The mode of death—as is to be expected from the relative rarity of intracardiac thrombi—is less often embolic. Indeed *there were no embolic deaths in our forty cases.* Congestive and septic deaths are commoner and those from intercurrent disease or trauma much rarer.

5. *Diagnosis.*—*Some* lesion was recognized in twenty-six of forty cases, but *both* lesions in only eight. The stenotic part of the aortic lesion was hardly ever recognized by physical examination, but might have been inferred with 80% of accuracy from the diagnosis of aortic regurgitation which was made in sixteen out of forty cases.

### ILLUSTRATIVE CASES. MITRAL STENOSIS AND AORTIC STENOSIS

#### Necropsy 4401

An American factory operative of fifty entered September 7, 1922, complaining of dyspnea and weakness. One sister died of tumor of the stomach. The patient's health had always been good except for the present illness. He had influenza in 1918 and questionable influenza in 1921. He and the rest of his family always had pale yellow skin.

At twenty-one he was ill in bed for a week or two with acute rheumatic fever. Since that time he had continually done hard work and had been troubled only a little by dyspnea. At twenty-eight years he was passed for life insurance. His second wife, to whom he had been married nine years, said his heart had been irregular as long as she had known him. Two years ago he noticed that his ankles were slightly swollen for two months. The winter before admission the dyspnea increased both on exertion and at night. A

physician gave digitalis, which slowed his heart but increased the dyspnea and orthopnea. Under some other medicine the condition improved considerably, though the dyspnea and orthopnea were worse than the previous year. A month before admission these symptoms returned, more intense than ever before, and the medicine gave less relief than formerly. During the past few weeks there had been occasional attacks of palpitation during which he had to stop work and spend a few minutes lying flat. Two weeks before admission all the symptoms began to grow progressively worse. He had more dyspnea on the slightest exertion. Orthopnea frequently awakened him at night. He had slight swelling of the ankles. His appetite generally was poor, and he grew weak.

Examination showed a fairly well developed, poorly nourished, orthopneic man, pale, cyanotic and in considerable distress. The tonsils were small and buried. There were a few very small sub-

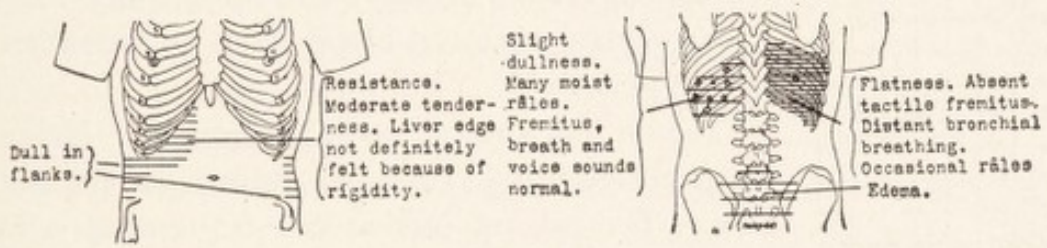



FIG. 19.

maxillary glands. The apex impulse of the heart was seen and felt in the sixth space 12 cm. to the left in the anterior axillary line; also seen in the fourth and fifth spaces, diffuse and heaving, The borders of percussion dullness were 13 cm. to the left, 5.5. cm. outside the nipple line, 4 cm. to the right; the supracardiac dullness 5 cm. At the apex was a loud blowing systolic murmur transmitted to the axilla and back, obscuring the first sound. The second sound was not made out. A low-pitched rumbling diastolic murmur was heard over a small area at the apex. Over the whole base a well marked systolic thrill was felt. There was a loud rough systolic murmur at the base filling all of systole. The second sound was not heard. There was an early diastolic murmur, loudest to the left of the sternum, of different character from that at the apex. There was absolute irregularity of rhythm and force. The action was not very rapid (95). The pulses were absolutely irregular, of poor quality, plateau type. The highest systolic blood pressure was 115, the usual 105, the diastolic 85. The lung signs were as shown in Fig. 19. The abdomen was slightly distended, tympanitic except in the flanks.

(See diagram.) There was moderate edema over the sacrum and marked edema of the feet and ankles extending halfway to the knees. The pupils reacted sluggishly to light. The reflexes were normal.

The temperature and pulse were as shown in Fig. 20. The respirations were 9 to 29. The amount of urine is not recorded except 31 ounces September 8. The specific gravity was 1.026 to 1.028. The urine was cloudy at one of two examinations and showed a slight trace of albumin and leucocytes at both, fine granular and hyalin casts at the first. The hemoglobin was 70%. There were 8000 to 11,000 leucocytes, 70% polynuclears, 4,800,000 reds, slight achromia. The platelets were normal. A Wassermann was negative. A chest tap September 8 in the right posterior axillary line and below the tip of the scapula gave 20 ounces of beet-colored clear fluid which coagulated quickly; specific gravity 1.012, 23 leucocytes, 2410 red blood cells, no organisms; culture negative.

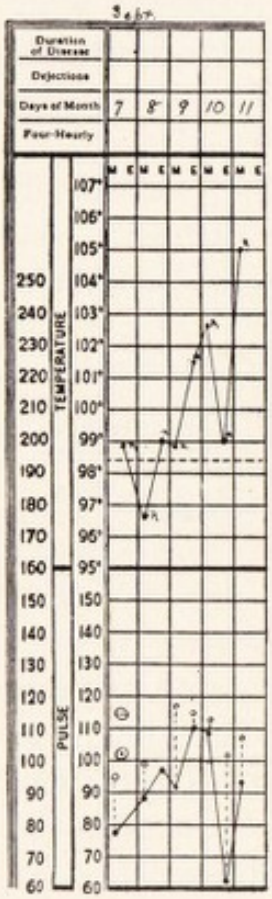



FIG. 20.

The patient showed no improvement. He had constant nocturnal orthopnea, failure and irrationality. The edema increased. September 9 the lungs were full of wet râles, with fluid at both bases. September 11 he died.

*Clinical Diagnosis (from Hospital Record).*—Rheumatic heart disease with mitral stenosis and regurgitation.

Aortic stenosis with regurgitation.

Auricular fibrillation.

Congestive failure.

Chronic passive congestion of lungs and liver.

Hydrothorax.

Bronchopneumonia.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the aortic and mitral valves.

Aortic stenosis and regurgitation.

Mitral stenosis and regurgitation.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Hydrothorax.

Ascites.

Pneumonia?

*Anatomical Diagnosis.*—Chronic endocarditis of the aortic and mitral valves (stenosis).

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Hydrothorax.

Edema of the legs and feet.

Meckel's diverticulum.

Obsolete tuberculosis of the mesenteric glands.

DR. RICHARDSON: Clinically do you find a large heart with aortic stenosis if it is a real stenosis?

DR. CABOT: Yes, certainly.

DR. RICHARDSON: We were not permitted to examine the head. The legs and feet were slightly swollen pitted on pressure. The subcutaneous tissues were moist but not wet. There were a few c.c. of thin pale fluid in the peritoneal cavity. The esophagus, stomach, and intestine showed well marked chronic passive congestion,—reddened, velvety, juicy mucosa oozing thin bloody fluid.

Two of the mesenteric glands were transformed into stony fibrocalcareous material, obsolete tuberculosis.

The right pleural cavity was half full of clear fluid; on the left there was 200 c.c.—hydrothorax, more marked on the right, and some compression atelectasis. The lungs showed frank chronic passive congestion, but I could not find any pneumonia. That does not mean to say absolutely there is no pneumonia in those lungs. It means that I could not find any definite pneumonia. If we took the lung from the apex to the base and cut up every part of it there might be found in it some bronchopneumonia. I was unable to find anything from the culture in the heart blood. So that so far as our evidence goes there was nothing in the way of terminal infection. The trachea and bronchi showed congestion of the mucosa.

The pericardium was negative. The heart weighed 685 grams, markedly enlarged, with a thick myocardium of good consistence, pale brown-red. The left auricular wall showed some thickening, measuring 15 mm., the right three to four mm. That is a thick heart, the kind of heart we expect with aortic stenosis. The left cavities showed considerable dilatation, the right slight dilatation. The cavities were all distended with currant-jelly-like blood clot,—apparently death in diastole. There was a small adherent thrombotic mass in the left auricular appendix. The mitral valve measured seven cm., the orifice of the aortic valve was represented by a slit-like opening 1½ cm. by one cm.—a fairly well marked stenosis in the mitral and

a classical picture of stenosis of the aortic. Of course that connotes a fibrocalcareous fusion of the cusps of the aortic valve and marked fibrocalcareous thickening and deformity of the mitral. There was no evidence on either of these valves of acute endocarditis. The tricuspid valve showed increase of circumference but was otherwise negative. The pulmonary valve was negative.

The coronaries were free, capacious and showed only a slight amount of fibrous sclerosis. There was also a slight amount of fibrous sclerosis in the first portion of the aorta and elsewhere.

The liver showed the typical nutmeg markings of chronic passive congestion. The gall-bladder, bile ducts, pancreas were out of the picture. The spleen did not weigh so much, 115 grams, but was chunky, thick, elastic and dark brown-red,—chronic passive congestion. The adrenals were negative. The kidneys showed chronic passive congestion, otherwise frankly out of the picture. The microscopical examination of the kidneys corroborates what I have said about them. In this case there was a Meckel's diverticulum, but it was perfectly free and negative.

DR. CABOT: I think the fever must be laid to the thrombosis.

### Necropsy 3827

An American clerk of forty-eight entered April 1. His maternal grandparents died of tuberculosis, one brother of epilepsy. In childhood he had scarlet and typhoid fevers, and strained his back by carrying too heavy weight, "with resulting prominence of the shoulder blades and lack of chest development." He had urinated once at night and had had dull frontal headaches once in two or three weeks "all his life," especially brought on by overwork. At thirteen he had what he believed to be epidemic cerebrospinal meningitis, though no lumbar puncture was done. He made a complete recovery. For thirty years he had suffered from gas eructations beginning two hours after meals and lasting one or two hours, somewhat relieved by hot water. At sixteen, twenty-five, and thirty he had attacks of "muscular rheumatism,"—painful, tender but not swollen joints, especially the elbows and knees, and muscles of the calves and arms, lasting about two months. Four years ago he was ill in bed with "grippe" for five weeks, and ever since this had had attacks of dyspnea on exertion with pain in both sides of the chest in front and palpitation. He had to sleep on two pillows. He urinated six times a day and once or twice at night. Three years before admission he had epithelioma of the left cheek, curetted at a hospital. Twelve years

before admission he weighed 155 pounds, his best weight. His usual weight was 148. In the past year it had fallen to 140.

His illness was the third of a series of similar attacks, the first of which followed his "grippe" four years ago. The second kept him from work for two months the year before admission. March 29 he had a third attack of dizziness lasting half a day, followed by weakness and dyspnea on even very slight exertion. He had since this required three pillows at night.

Examination showed a poorly developed and nourished man in distress with dizziness and faintness when sitting up. The mucous membranes were slightly cyanotic. The anterior-posterior diameter of the chest was greater than the lateral diameter. There was slight bulging over the precordia. The apex impulse of the heart was in the fifth space 9 cm. from midsternum and 3 cm. inside the nipple line, corresponding with the left border of dullness. The right border was 6.5 cm. to the right, the supracardiac dullness 9 cm. The pulmonic second sound was accentuated. The sounds were of poor quality. At the apex they were obliterated by loud murmurs, systolic and diastolic in time. The systolic was heard over the precordia. The pulses were thready and of poor volume and tension. The artery walls were palpable. The blood pressure was 85/50. The lungs were normal. The abdomen was retracted. There was tenderness over the liver. The liver dullness extended from the fourth rib to 8.5 cm. below the costal margin in the midclavicular line, 9 cm. below in the parasternal, and 5 cm. below the xiphoid. The liver pulsated. The edge was felt. The genitals and extremities showed nothing of importance. The pupils were irregular, but equal and reacted. The knee-jerks were not obtained. There was a suggestion of a bilateral Babinski; no Kernig.

The temperature was 100° by mouth to 105.4° by rectum, the pulse 91 to 135, the respiration 18 to 32. The amount of urine is not recorded. The specific gravity was 1.025 to 1.010. There was the slightest possible trace to a trace of albumin at both of two examinations, a question of diacetic acid at the first, many leucocytes at the second. The hemoglobin was 80%. There were 21,600 to 26,000 leucocytes. Blood cultures showed staphylococcus albus. The blood urea nitrogen was 24 mgm. per 100 c.c. of blood. A Wassermann was negative. The stools were negative. A lumbar puncture April 2 gave 15 c.c. of clear fluid under 180 mm. pressure, showing 244 cells per cu. mm., 42% polynuclears, 58% mononuclears. Alcohol was slightly positive, Nonne questionable, Wassermann negative.

There was a small fibrin clot in one-half hour in a stained specimen of which no organisms could be demonstrated. A culture showed staphylococcus albus. A lumbar puncture April 3 gave 30 c.c. of cloudy fluid under 140 mm. pressure, showing 840 cells, 59% polynuclears, 41% lymphocytes. Another puncture the same day gave 30 c.c. of cloudy fluid under 200 mm. pressure, cell count 1100, 64% polynuclears, 36% lymphocytes. A clot formed in one hour. Cultures on agar and blood serum showed staphylococcus albus.

The patient was put upon Karell diet,* with fluids limited to 1000 c.c. April 2 his mind was fogged and there was definite Brudzinski. April 3 he was still irrational. 45 c.c. of anti-meningococcus serum was injected, with definite rigidity and struggle on the part of the patient. There was active reflex on the left, and positive Babinski, but no clonus; on the right no reflex and no Babinski. None of the fluids obtained at lumbar puncture showed tubercle bacilli. April 4 the patient suddenly died.

*Clinical Diagnosis (from Hospital Record).*—Acute endocarditis.

Mitral insufficiency and stenosis.

Pulmonary embolism.

Acute meningitis.

Poliomyelitis?

General septicemia.

*Dr. William H. Smith's Diagnosis.*—Extensive chronic endocarditis of the mitral valve.

Possibly staphylococcus albus septicemia.

Possibly fresh vegetative endocarditis.

Possibly staphylococcus albus meningitis.

*Anatomical Diagnosis.*—Chronic and acute endocarditis of the mitral and aortic valves. Stenosis.

Chronic and acute pericarditis.

Septicemia, staphyloccus, pale variety.

Hypertrophy and dilatation of the heart.

Infarcts of the spleen and kidneys.

Abscesses of the kidneys.

Septic arteritis and thrombosis of the hepatic artery with infarcts of the liver.

Phlegmonous enteritis.

Soft hyperplastic spleen.

* 200 c.c. of milk at 8 a.m., 12 m., 4 p.m., 8 p.m.; total 800 c.c. Or ʒ iv at 6–8–10 a.m., 12 m., 2–4–6–8 p.m.

Partial agenesis of liver with cyst.
Cholelithiasis.
Slight chronic pleuritis.
Chronic interstitial orchitis.
(Brain not examined.)

DR. RICHARDSON: A staphylococcus of the pale variety was recovered from the blood and the spleen. Scattered over the skin in many places were minute to small purpuric spots, accounted for, of course, by the staphylococcus septicemia.

The heart weighed 560 grams (normally 200–300), and showed considerable hypertrophy and dilatation. The left ventricle wall

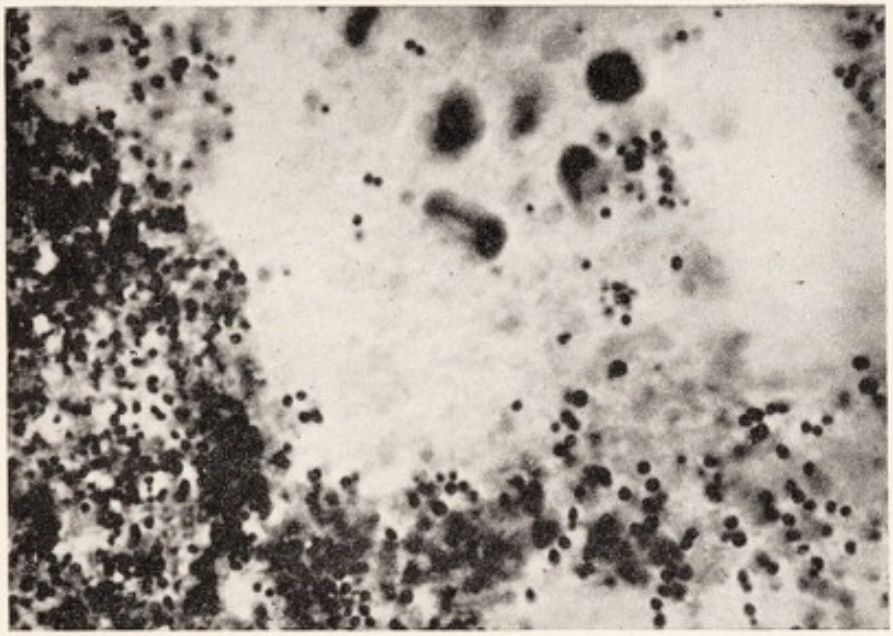


FIG. 21.—Staphylococcus albus septicemia, mitral and aortic stenosis with acute endocarditis. Section of heart valve showing staphylococcus albus. (Photomicrograph by Lewis S. Brown. ×1500. Dr. William H. Smith.)

was beefy,—good muscle. The cavities were dilated markedly on the left and considerably on the right. Both layers of the pericardium, visceral and parietal, were closely bound together by fibrous membranous adhesions, but at one point there was an area about three fingers' breadth on the postero-lateral aspect of the left ventricle where the adhesions were soft, reddish, and mushy. In other words, there was an area of acute pericarditis within the chronic.

The aortic valve orifice would not admit a lead pencil. The cusps were fused into a fibrocalcareous, irregular-surfaced mass, the so-called fish-mouth valve. The surfaces of this irregular mass, both superior and inferior, were coated with a felt-like layer of granular thrombotic material. The mitral valve showed much fibrous thickening and deformity, with a large fibrocalcareous area, the central

portion of which was depressed and the surface roughened, with at one point a coating of thrombotic material. The tricuspid and pulmonary valves were negative. The aorta and its great branches were negative. There was no arteriosclerosis.

The lungs showed some edema; otherwise they were negative.

The spleen was enlarged, soft, pulpy, and showed several infarcts.

There was cholelithiasis; about fifty small gall-stones were recovered. The bile ducts and the gall-bladder were negative.

The kidneys weighed 330 grams (normally 200–400). On removing the capsules the surfaces showed scattered over them minute purplish spots like those on the skin. In other places there were areas that looked like infarcts, the central portions of which were a little softer than usual. They were margined in instances by red borders.

We have in this case, therefore, an old endocarditis established some time ago, with stenosis of the aortic and mitral valves. To this, and within recent times, he has added a staphylococcus infection which finds expression in the acute endocarditis, and, although I did not examine the head, *in all probability a meningitis* due presumably to septic infarcts. The infarctions in the case were really septic; that is, portions of the septic vegetations on the valves containing staphylococci were carried by the arteries to other organs. The spleen and kidneys were the organs in which the infarcts arose because they have the so-called end arteries.

Microscopical examination of the kidneys and of the tissues generally showed a very characteristic picture, corroborative of what we have been saying. In the arteries in the kidneys and in the spleen we found collections of staphylococci. We found the staphylococci also in the liver, and if we had had permission to examine the brain I think we should have found them there.

In the intestine there were purplish areas the central portions of some of which on the mucosal side contained small yellowish-purple areas; that is, septic infarcts in the intestine. In cases of sepsis we may have intestinal disturbance associated with diarrhea, and in some of them we find definite lesions, as we did in this case. The anatomical picture as a whole was as though you had taken a hypodermic syringe containing staphylococci and injected them into the blood in the left ventricle; then, carried by the blood stream, the organisms were distributed throughout the body. It was an experiment conducted by Nature. Unusual as the anatomical conditions were, they formed a good basis for the clinical picture.

There was some question about the conditions in the liver. At one point there was an area of thickening of the capsule, and atrophy of the liver tissue underneath. Below that area of atrophy the liver showed areas not unlike septic infarcts, although infarcts very rarely occur in the liver. A branch of the hepatic artery leading to the region of these areas showed occlusion by thrombotic material. The microscopical examination of the liver tissue showed septic thrombosis of this branch, and also showed that the areas were areas of infarction.

Septicemias due to infection with the pale variety of the staphylococcus are quite rare.

DR. SMITH: The previous typhoid history is of interest as an etiological factor for his gall-stones. It would be of interest to know whether typhoid bacilli were still present in the gall-bladder, suggesting the possibility of his being a typhoid carrier.

### OTHER COMBINED LESIONS

| | |
|---|---|
| Mitral-and-Aortic-and Tricuspid Stenosis | 22 cases |
| Mitral-and-Tricuspid Stenosis | 7 cases |
| Pulmonary-and-Tricuspid Stenosis | 2 cases |
| Mitral-Aortic-Tricuspid-and-Pulmonary Stenosis | 2 cases |
| Total | 33 cases |

Emphasizing some points already alluded to in the chapter on mitral disease we see:

**1. Etiology.**—That in most cases (thirty out of thirty-three) there has been recorded only a single attack of rheumatism. In the "pure mitral" cases we have multiple attacks recorded in nearly one-third, and of the mitral-and-aortic cases two-thirds have had multiple rheumatic attacks.

**2. Symptomatology.**—The mode of death is congestive in two-thirds of the cases as against one-half the cases in the simpler lesions (see Table 52), but it is striking that the cases with *mitral-and-tricuspid lesions do not show passive congestion before or after death.* Only one out of seven cases died a congestive death or showed chronic passive congestion at necropsy.

**3. Necropsies.**—The evidences of passive congestion are, as one would expect, more often severe in these multiple lesions than in the simple cases. Dyspnea and cyanosis in life, cardiac hypertrophy, ascites, enlarged liver, and infarctions are all more often present in the "combined lesions," except as above specified.

**4. The Diagnosis of the More Complicated Valve Lesions.**—Besides the 107 "pure mitral" cases and the 40 mitral-and-aortic,

there were in our series twenty-two cases of *Mitral-aortic-and-tricuspid stenosis*. Out of this group we recognized the mitral lesion alone or in combination in eighteen. We recognized the aortic lesion alone or in combination in nine. We raised the question of a tricuspid lesion in one. We missed all three lesions in four.

In the seven cases showing post-mortem *stenosis of the mitral and of the tricuspid valves*, we recognized the mitral lesion in five, the tricuspid lesion in none at all. In two cases we missed both the mitral and the tricuspid.

We had two cases involving *stenosis of the pulmonary and tricuspid valves*, in one of which we recognized the pulmonary stenosis alone. In the other we missed both lesions.

In two cases necropsy showed *stenosis of all four valves*. In one of these we recognized only the mitral lesion, in the other only the mitral and aortic lesion. We missed the pulmonary and tricuspid lesions in both cases.

Looking back over the whole thirty-three cases of this complicated group one sees that, *though the tricuspid valve was involved* in *every one* of them *we only suspected this disease in one case out of the thirty-three and did not even consider it in the others*. The *pulmonary valve lesion we recognized once*. So that it appears that we can often recognize a mitral lesion especially if it has obvious congestive symptoms to direct our attention to the heart. We can recognize a complicating aortic lesion (when present) in about half the cases. With uncomplicated or pure aortic stenosis we do rather better. *Tricuspid and pulmonary lesions we practically do not recognize at all.*

### ILLUSTRATIVE CASES. VARIOUS COMBINED VALVE LESIONS

#### Necropsy 3549

A Canadian-American housewife of twenty-five entered January 11, 1916, for relief of dyspnea. She had one convulsion as a child. She had "always" been dyspneic, and had never been strong. She had poor appetite, gas, and constipation accompanied by headache over the eyes. She had scarlet fever at nine, chorea for three years beginning at twelve, pneumonia at eighteen, bronchopneumonia at nineteen, bronchitis several times, and many sore throats. Her catamenia had always been irregular.

For four years she had had leucorrhea and precordial pain and urinated once or twice at night. For three years she had coughed and had white frothy sputum. She had been married seventeen months; had never been pregnant. For sixteen months she had had

pain and discharge from the right ear. For a year she had been orthopneic. For four months she had had edema, for three months a good deal of nausea, for three weeks dysuria. A week before admission she coughed up much blood. She lived near a gas plant, and thought she breathed much gas. She slept poorly ten or eleven hours a night. Her intelligence, memory, and speech were primitive. Her best weight was 120 pounds four years before admission, her present weight 112.

For four years she had had much dyspnea, for which she was treated in a hospital twice for a month with relief, and again for a third time. Two years before admission she developed a severe cough and was sent to a tuberculosis hospital for observation. No evidence of tuberculosis was found. Since her marriage she had been much worse. Two weeks before admission she had "grippe." Since that attack she had been very uncomfortable, with aggravated cough, dyspnea, and edema of the ankles.

Examination showed a fairly well-developed and nourished woman 5 feet 6¼ inches in height, (weight January 22, 93 pounds). The mucous membranes were slightly cyanotic, the teeth poor, the tonsils large. The apex impulse of the heart was in the sixth space 12 cm. to the left of midsternum, two inches outside the nipple line. The right border of dullness was 4.5 cm. to the right, the supracardiac dullness 5.5. cm. There was a presystolic thrill. The pulmonic second sound was accentuated and double, the aortic second faint. There was a sharp first sound preceded by a rasping presystolic murmur and followed by a high-pitched systolic. No second sound was heard at the apex. The pulses were of fair volume. The lungs showed many moist râles in the axillae and below the angles of the scapulae, on the right nearly as high as midscapula. There was tenderness over the liver. The liver dullness extended from the fifth rib to eight cm. below the costal margin, where a tender edge was felt. Vaginal examination was not done. The legs showed soft pitting edema from the ankles to the sacrum. The pupils were normal, as were the knee-jerks on reënforcement.

The temperature was 95° to 110° until January 28, then 97.3° to 102° until February 7, then 96° to 99.5°, with one drop to 94.8° February 11. The pulse was 71 to 146, the respiration 16 to 48. The blood pressure was 140/100 to 115/80. The amount of urine was normal except February 5 and 9, when it was 10 ounces. The specific gravity was 1.001 to 1.020. The urine showed a slight trace of albumin at five or six examinations, a few red blood corpuscles at

one, a large amount of pus and blood at one. The renal function January 11 was 45%, January 18 30%, January 31 40%. A urine culture showed a few doubtful bacilli. The hemoglobin was 85%. There were 7800 to 48,000 leucocytes, 83% polynuclears, the reds were normal. A Wassermann was negative. The stools gave a negative guaiac at two examinations. The sputa showed many pus cells and influenza bacilli; no tubercle bacilli at four examinations. Two specimens settled in three layers. A dental consultant in reply

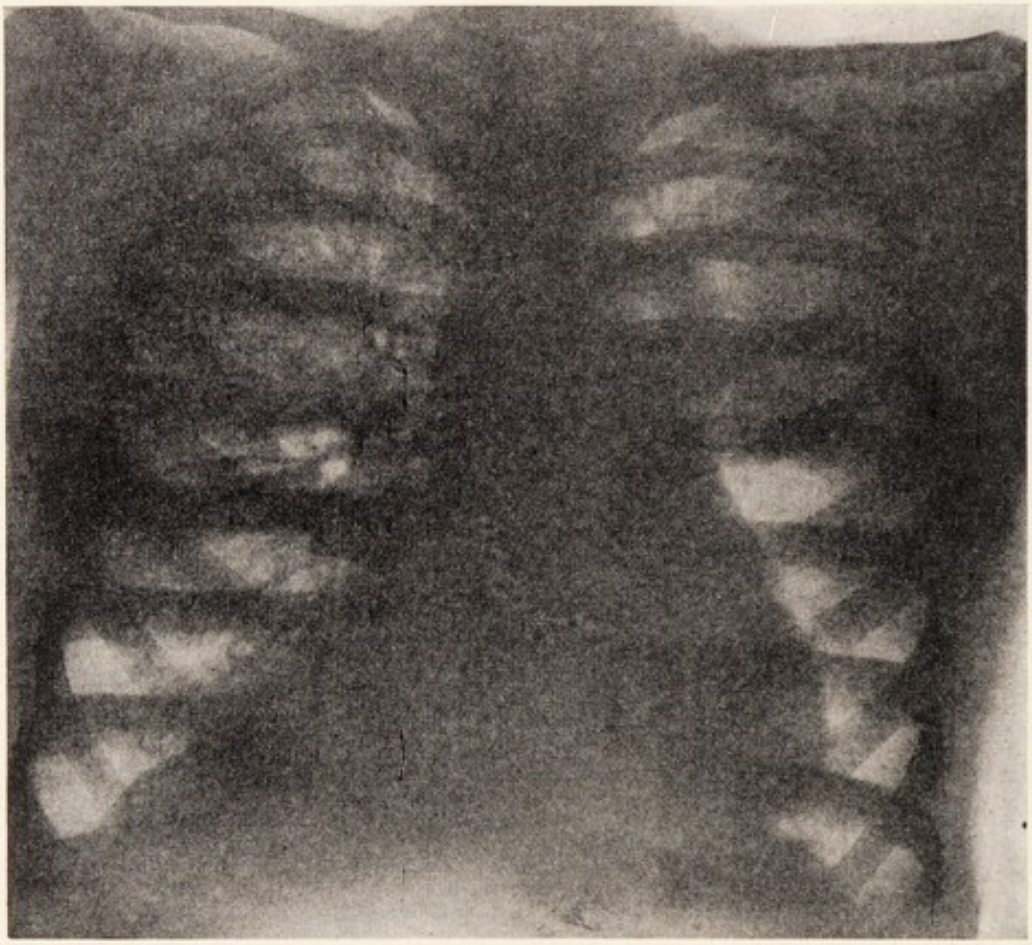


FIG. 22.—Heart symmetrically enlarged, not displaced. Seen from behind. Bronchiectasis with focal pneumonia and gangrene, especially right lung. Several areas suggest cavities. (Roentgenological Department, Massachusetts General Hospital.)

to the question, "Is there alveolar abscess?" reported, "Extraction advised." A throat consultant reported, "Larynx negative as to cause of cough."

January 29 all the back teeth and roots were extracted under ether. The patient was much worse from this time. She had moderate fever. February 2 the pulse was elevated and the murmur rough. For the next week she was very uncomfortable, coughing all night and raising much sputum, occasionally blood-tinged. Evidently she had had much morphia, as it took three-quarters of a

grain to affect her. Her lungs were full of râles, especially the left, where the râles at the apex were very sticky and there was slightly bronchial respiration and some dullness. February 8 she was cyanotic, and seemed worse. X-ray (Fig. 22) showed an extensive pathological process in both lungs, especially the right, more marked in the upper lobes, extending downward and obscuring the cardio-hepatic angle on that side. On the left it extended down to the fourth rib anteriorly. The maximum density was at the roots, extending outward and upward. Enlarged and calcified glands were seen at the roots. There were several areas suggesting cavities, the largest the size of a quarter-dollar, at the lower border of the fourth right rib, a smaller one under the third right, less distinct ones in the left upper lobe. A dense line extended across the right chest horizontally from the lower fourth costosternal angle to the upper border of the seventh rib in the axillary line, near or at the interlobar cleft. The heart was somewhat enlarged symmetrically, not displaced. The diaphragm was low, somewhat irregular, with limited excursion, the angles clear.

The patient's condition became very distressing. She coughed continually, raising reddish-brown purulent sputum, had epigastric pain and occasional vomiting, grew more and more cyanotic and dyspneic, and February 16 died.

*Clinical Diagnosis.*—Chronic nephritis.

Influenza.

Cardiac decompensation.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the mitral valve with stenosis.

Possibly some chronic adhesive pericarditis.

Chronic pneumonitis with pulmonary abscesses or bronchiectasis.

Possibly old interlobar empyema.

Passive congestion of all the organs.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral, aortic, and tricuspid valves.

Hypertrophy and dilatation of the heart.

Septicemia, streptococcus.

Bronchiectasis with focal pneumonia and gangrene.

Infarcts of the superior lobe of the left lung.

Ulcer of the stomach.

Anasarca.

Slight ascites.

Vaginitis papillomatosa.

Old infarct in one kidney.

Obsolete tuberculosis of bronchial lymphatic glands.

Chronic pleuritis.

The heart was considerably enlarged, weighing 404 grams. The right ventricle wall measured 5 mm., the left ventricle wall 10 mm. The auricular walls were thickened. The right side of the heart contained a large amount of currant-jelly-like blood clot. The right auricle was greatly dilated, the right ventricle moderately dilated. The mitral orifice admitted the tip of the little finger. The curtain was the seat of marked fibrous and fibrocalcareous change with great deformity of the valve and decrease in its circumference. The chordae tendineae were shortened, thickened, and fused to the curtain. The aortic valve circumference was 5.5 cm. The cusps showed a moderate amount of diffuse fibrous thickening which was slightly calcareous in places and rendered the walls rather rigid. The thickened walls in places measured from one to two mm. in cross section. The tricuspid valve measured 5 cm. The curtain was the seat of much diffuse fibrous thickening with deformity of the valve and decrease in its circumference. The pulmonary valve measured 7 cm. and was negative.

**Necropsy 4084**

An American girl of twelve entered May 22. She gave an unsatisfactory history which it was expected would be supplemented by relatives. It was not, however. She was apparently a perfectly normal child until a year before admission. She remembered measles, chickenpox and pertussis in infancy (*sic*). She had always been subject to sore throats, but apparently had not had tonsillitis. A year before admission she had an attack of "rheumatism" and was ill in bed for several weeks with swollen, red, and painful joints, particularly the ankles and wrists. Since this time she had had dyspnea on slight exertion, had been unable to attend school or play with other children, and had had slight cough with a small amount of thin yellowish sputum.

Two weeks before admission she began to have fever and marked dyspnea, and since that time had been in bed under treatment by a physician. She had had considerable pain over the epicardium and frequent frontal headaches lasting a few hours each. She had marked cough with considerable watery sputum.

Examination showed a fairly well developed and nourished girl with pale skin and mucous membrane. The throat was slightly injected. The submaxillary glands were swollen and tender. The

chest expansion was less on the left. These was small Harrison's groove. The left border of the sternum was more prominent than the right and somewhat raised. The lung signs were as shown in Fig. 23. The apex impulse of the heart was diffuse in the sixth space 14 cm. to the left. There was a heaving precordial impulse with a wave moving from the apex region toward the sternum. The measurements of percussion dullness show the borders as 14 cm. to the left, 4.5 cm. to the right, the substernal dullness 4.5 cm. The action was rapid. The sounds were of rather poor quality, the pulmonic second sound accentuated. The first sound at the apex was replaced by a loud blowing systolic murmur which took up the whole of systole, followed by a shorter diastolic murmur. There was a rough diastolic murmur over the aortic area and along the left sternal border. The pulses were equal; they tended to be Corrigan in type. A systolic thrill was felt over the precordia. The blood pressure was 106/35. There was definite hyperesthesia. The abdomen showed

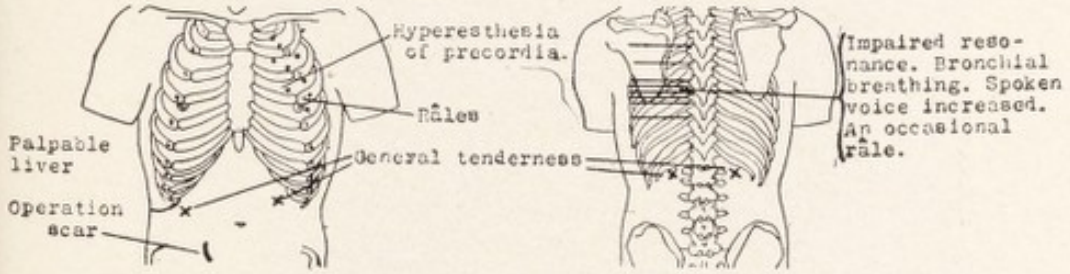



FIG. 23.

general tenderness in both flanks. The liver dullness extended from the fourth rib; the edge was just palpable and tender. There was slight costovertebral tenderness. The genitals were not examined. The fingers showed definite clubbing, and the toes suggested it. The pupils were normal, the knee-jerks equal but diminished. There was no clonus or Babinski.

The temperature was 97°, rising with daily swings of one or two degrees to 101.2°. The pulse was 120 to 140, with one drop to 105 May 25. The respirations were 23 to 47. The output of urine was 16 to 9 ounces, the specific gravity 1.020 to 1.028. The urine was cloudy at all of three examinations and showed the slightest possible trace of albumin and the slightest possible trace of sugar at two, diacetic acid at two, leucocytes at all. The hemoglobin as 80 %, the leucocytes 10,800 to 15,400, the polynuclears 69%, the platelets increased. A Wassermann was negative and a blood culture negative. The stools gave a positive guaiac at both of two examinations. A throat culture showed streptococci, no diphtheria bacilli.

May 26 the signs in the lungs were still more definite. A friction rub was heard in the left back. May 28 the patient died.

*Clinical Diagnosis (from Hospital Record).*—Acute endocarditis.

Myocarditis.

Aortic and mitral disease.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic and acute endocarditis of the aortic valve and possibly the mitral.

Hypertrophy and dilatation of the heart.

Pneumonia, left lung.

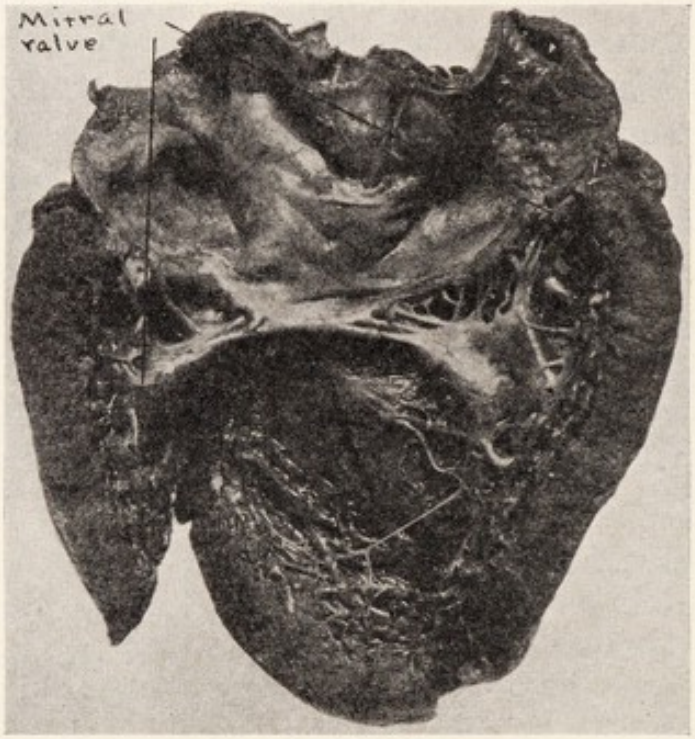



FIG. 24.—Necropsy 4084. Heart in stenosis of the mitral, aortic and tricuspid valves, showing the mitral valve, 7.5 cm. (Photograph by Lewis S. Brown. Dr. Oscar Richardson.)

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral, aortic, and tricuspid valves, stenosis.

Hypertrophy and dilatation of the heart.

Hemorrhagic areas, epicardium and peritoneum.

Chronic passive congestion, general.

Infarct of the right lung.

Focal pneumonia of superior lobe of right lung.

Tuberculous ulcer of small intestine.

Chronic tuberculosis, mesenteric glands.

Status lymphaticus.

Scar of old appendectomy.

DR. CABOT: There was no acute endocarditis?

DR. RICHARDSON: No. In the first place the hypertrophy and dilatation are apparent. The valves show the kind of chronic endocarditis associated with chorea. I do not know that she had it, but this is the type. In the mitral valve there is decrease of circumference, not very much, 7½ cm. But the curtain generally shows fibrous thickening continued to the margin, where it presents as a rounded fibrous ridge with markedly thickened and shortened chordae tendineae. (See Fig. 24.) Dr. Cabot's diagnosis as to the aortic valve is borne out, for the margin of the cusps are thickened and rounded in the way the free margin of the mitral is affected, and

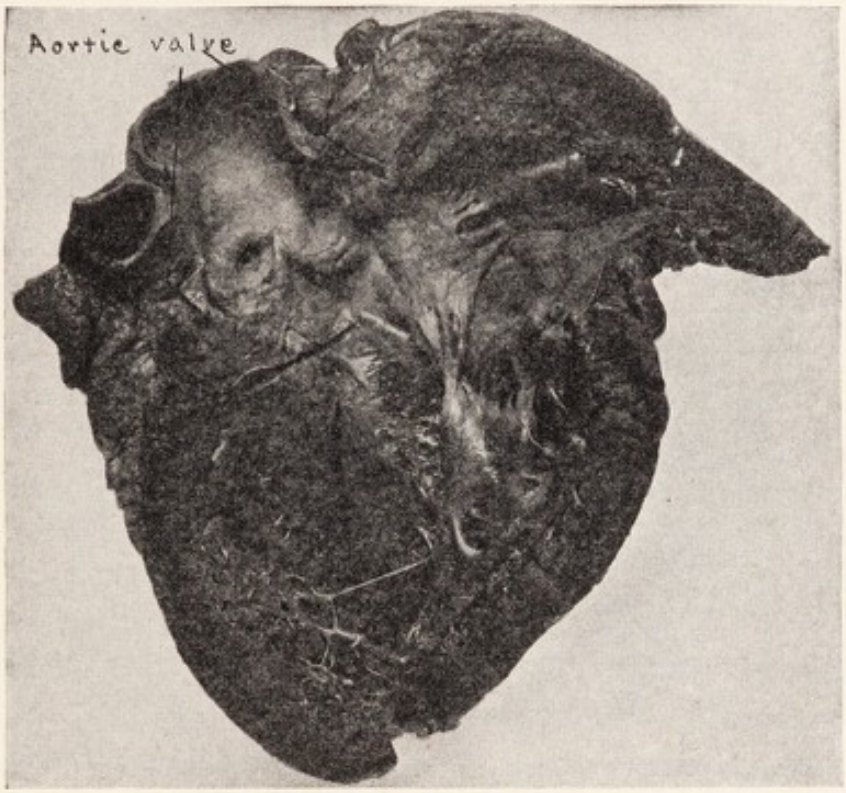



FIG. 25.—Necropsy 4084. The same, showing the aortic valve. (Photograph by Lewis S. Brown. Dr. Oscar Richardson.)

that without making much reduction of the circumference of the aortic valve does nevertheless decrease the width of the cusp and allow some regurgitation. (See Fig. 25.) The process in the tricuspid is slighter than on the other valves. (See Fig. 26.) The pulmonary valve is negative, and gives the standard for a good cusp.

Another interesting point is that extending down from one of the aortic cusps is an oval area of fibrosis which of course *x* years ago was a patch of acute endocarditis. A cross section shows that it extends slightly into the underlying myocardium.

The record should call attention to the fact that petechiae are scattered through the skin and other places. They were present in this case, and they were well marked in the peritoneum too.

The chronic passive congestion is the brown-red, salmon-colored tissue, somewhat leathery, yielding a brownish-red fluid containing the so-called heart cells. There was nothing in the auricular appendices. There was an infarct of the right lung. These occur where there is considerable chronic passive congestion.

The focus of pneumonia was in the upper part of the right lung, and was rather an easy thing to miss, but I happened to be struck by the resistance in feeling of it and therefore examined. With the microscope it was found to be a focus of pneumonia in the superior lobe of the right lung.




FIG. 26.—Necropsy 4084. The same, showing the tricuspid valve. (Photograph by Lewis S. Brown. Dr. Oscar Richardson.)

The thymus gland weighed eleven to twelve grams, and the lymph apparatus generally showed more or less hyperplasia. These conditions we find at times associated with tuberculosis.

In this case there was only a single ulcer along the whole length of the intestine, but microscopically it showed typical tuberculous tissue. In the mesenteric glands there was chronic tuberculosis.

Above the diaphragm there was no tuberculosis of any sort. Below we had an ulcer and the tuberculosis of the mesenteric glands, and of course it is a case with strong evidence in favor of the entrance of the tubercle bacillus by the gastro-intestinal tract.

A PHYSICIAN: This girl had a fever. What do you think it was due to?

DR. RICHARDSON: Fever for how long?

DR. CABOT: Two weeks before she came in and six days here.

DR. RICHARDSON: She had tuberculous ulcer, tuberculosis of the mesenteric glands, and some time within that period, of course, that area of pneumonia.

A PHYSICIAN: What caused the leucocytosis?

DR. CABOT: I suppose it was leucocytosis due to pneumonia. I do not know whether the pneumonia could have been there all this time. In the heart there was no source of fever. The pneumonia was not in the place we suspected. It was at the right top. In the left base it was merely congestion.

A PHYSICIAN: Is there any means of knowing whether the ulcer was bovine tuberculosis?

DR. RICHARDSON: We did not secure the bacillus, but it probably was the bovine type.

### Necropsy 4415

An unoccupied American girl of twenty entered August 1, 1922, complaining of a skin eruption and pain in the cardiac region. Her mother died of Bright's disease. The patient had the minor diseases of childhood and a light case of influenza in 1918. For three weeks she had had some blood stained sputum. Two weeks before admission for a few days she had a yellowish vaginal discharge. She had a tendency to "nervousness." In 1921 she weighed 125 pounds, her best weight; her present weight was 104 or less.

Six years before admission she had a severe sore throat diagnosed as tonsillitis. She was in bed for a week. She returned to school only to be forced to go back to bed by a sudden attack of pain in the arms and legs. She had lameness of the extremities and knife-like shooting pain over the precordia, aggravated by deep breathing. Two months after the initial attack tonsillectomy was done. She was in bed off and on for the next two years for periods of from one to seven weeks. She was very short of breath and orthopneic all this time, and was troubled a great deal by gas. She believed she had some fever during the whole period. After this she felt quite well until January, 1922, danced and walked a normal amount, and had no discomfort of any kind except for a somewhat more than usual amount of gas. In January, 1922, she noticed a small red spot on the left cheek, soon followed by one on the right and two areas on each side of the jaw. These gradually increased until after a month her whole face was exceedingly red. This gradually spread in the course of four months to the neck, chest, arms and hands, excepting the palms.

The lesions on the neck were decidedly painful and cracked. During this time she noticed a small red spot the size of a pea on her great toe.

In May, 1922, she was operated upon at a neighboring hospital for acute appendicitis. The day after the operation she developed pneumonia and was in the hospital for twelve days. During this time the arms cleared except for a few pigmented areas which had persisted.

After a fair convalescence she felt pretty well until six weeks before admission. Then she began to have "a good day and a poor one," and on the poor ones noticed that she had "catching" pains in the side and occasionally in the back. This continued for three weeks. Then she found she could not sleep one night, and the next day was very somnolent, had a heavy oppressive feeling in the chest, and could get her breath only with difficulty, though she did not have to sit up. She had a temperature of 103°. After placing an electric pad on her chest she noticed the return of the skin condition which was present at admission. She had an occasional attack of dizziness and a great deal of gas, nausea and vomiting. She had eaten very little, and had been very dyspneic. She said she had no cough, though the examiner noticed occasional cough. For six weeks she had been in bed, the first three weeks about every other day, the last three weeks constantly. For twenty-one days she had had a temperature of 103° and 104°. The precordial pain had been very sharp and severe. It was somewhat relieved by an icebag and localized in an area about the size of three silver dollars.

Examination showed a well nourished girl with a sharp marginated gyrate eruption on the skin of the lower face and neck and upper chest. More recent parts showed red flaking edges and paler centers. The older portions showed thick crusting and the oldest portions a deep dirty brownish pigmentation. The process in the mucosa and roof of the mouth was red and raw. On the uvula there was a gyrate patch with a whitish border and red center. The apex impulse of the heart and the midclavicle are not recorded. The measurements of percussion dullness were, left border 11.5 cm., right border 2.5 cm., supracardiac dullness 5 cm. At the apex was a presystolic and systolic murmur, along the left sternal border a loud rough diastolic. The aortic second sound was nearly absent. The pulmonic second sound was sharp. There was collapsing pulse and pistol shot in the groins. The blood pressure was 140/0. The lungs showed a few fine râles at both bases posteriorly. The liver edge as 2 cm. down, slightly tender. The spleen was easily palpable 4 cm. below the costal margin. There was a scar in the right lower quadrant.

The temperature was 103.4° to 97.5,° with a terminal rise to 107.1°. The pulse was 155 to 60, the respirations 19 to 71. The output of urine was 8 to 40 ounces, the specific gravity 1.006 to 1.020. The urine was cloudy at two of seven examinations, showed the slightest possible trace of albumin at all, leucocytes at six, red blood cells at four, granular casts at six, hyaline at two. The renal function was 50%. The hemoglobin was 55% to 75%, the leucocytes 24,000 to 4,000, the polynuclears 61% to 75%, the reds 3,050,000 to 4,280,000, slight achromia at one examination, none at another. Three blood cultures were negative; a fourth showed questionable Gram-positive bacilli in one flask. A Wassermann was negative. The stools were negative to guaiac. The sputum was brownish, mucopurulent.

August 6 a swelling appeared on the right arm in the region of the deltoid and behind it, apparently not tender. August 9 she was nauseated and vomited most of what she took by mouth. The heart rate was slower and definitely irregular, without any special rhythm. There seemed to be slow fibrillation. Electrocardiogram August 9 showed auricular fibrillation, rate 70, inverted $T_2$ wave, left axis deviation. Digitalis was stopped. After this she had no nausea and felt much better. By August 12 the temperature was almost normal, the pulse slower. The spleen was barely palpable. The liver was not felt. The râles persisted, but to a smaller extent. August 14 she had return of the precordial pain. It grew more and more severe during the next few days. She required morphia for any rest at all, and at times was forced to sit up and gasp. August 19 the pain began to diminish, and by the 24th was much less. The temperature was also running lower. A swollen tender gland developed beneath the left jaw, disappearing in four days. She felt much better.

September 1 there was dullness to a little above the angle of the right scapula with diminished breath sounds and a few moist râles. The next day moderate edema of the feet was noticed. September 3 this became marked, and she complained of feeling tired. Beginning September 5 she complained of increasing pain in the epigastrium and beneath the lower sternum and of increasing shortness of breath. The edema of the feet was slightly less. The temperature was again rising. Her general complaint was that she had "gas on the heart" and felt the "sleep-start" of the cardiac patient. September 19 the edema of the legs and back began to increase markedly. She felt generally miserable, with pain and much dyspnea.

September 29 a chest tap in the seventh right space, posterior axillary line, gave 600 c.c. of cloudy straw-colored fluid when the patient began to cough. The specific gravity of the fluid was 1.016; leucocytes 3400, rare red cells, polynuclears 87%, lymphocytes 3%, epithelials 10%; no organisms; no clot; culture negative.

The night of October 9 her temperature suddenly went up to 104°, the pulse to 170, and the respirations to 72. Her hands and feet became cold, and she seemed to be moribund. The next morning, however, she was better. The chest showed coarse râles below the angle of the left scapula, dullness and bronchial breathing below the right. The respiration rose to 71, falling the next day to 36–44.

October 11 the right chest was again tapped in the posterior axillary line, the eighth interspace, and 250 c.c. of cloudy straw-colored fluid with-drawn; specific gravity 1.010, 3200 cells, 88% lymphocytes, 22% polynuclears, a few red blood cells, no organisms. October 12 the temperature rose to 107.1°. The pulse was of good quality. That day she died.

*Clinical Diagnosis (from Hospital Record).*—Malignant endocarditis.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic and acute endocarditis of the aortic and mitral valves with stenosis.

Pneumonia with acute purulent pleurisy.

Nephritis?

Chronic adhesive pericarditis?

Hypertrophy and dilatation of the heart.

Probably infarcts of spleen, liver, kidneys.

Chronic passive congestion, general.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral, aortic and tricuspid valves.

Acute endocarditis of mitral valve.

Chronic adhesive pericarditis.

Subacute glomerulonephritis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Edema of feet and ankles.

Slight hydrothorax.

Focal pneumonia, left lung.

Fatty metamorphosis of the liver.

Localized chronic peritonitis.

Obsolete tuberculosis of the mesenteric glands.

Scar of old operation wound.

Slight chronic pleuritis.

DR. RICHARDSON: We were not permitted to examine the head. There was an old operation scar in the right lower quadrant and the appendix was absent. The peritoneal cavity was moist. The gastro-intestinal tract was out of the picture except for some reddening of the mucosa, the beginning of chronic passive congestion. The mesenteric glands showed a little obsolete tuberculosis. There was none anywhere else.

The right pleural cavity contained 200 c.c. of thin pale clear fluid; there was about 50 c.c. in the other cavity. There were scattered old fibrous adhesions. The trachea, the bronchi and the bronchial glands were negative; the bronchial glands were slightly enlarged but with no evidence of tuberculosis.

DR. CABOT: Was there evidence of any recent pleurisy?

DR. RICHARDSON: Not at all, except for what comes later. The right lung showed chronic passive congestion. The left was similar to the right except that in the upper part of the lower lobe near the root of the lung there was a frank area of focal pneumonia, and over that an area of acute pleuritis.

The pericardial cavity was obliterated by adhesions binding the two layers together—chronic pericarditis. The heart weighed 500 grams. This is a very considerable enlargement for a woman of twenty. The myocardium was thick on the right, fairly thick on the left,—four mm. on the right, thirteen on the left,—a general thickness, rather a beefy myocardium. There was a little dilatation on the left, moderate on the right. The mitral valve measured 5½ cm., the aortic 6 cm., the tricuspid 9 cm., the pulmonary 7½ cm., indicating well marked stenosis of the mitral. From the measurement we cannot say anything about the aortic, but there was some change in the aortic valve nevertheless. There was stenosis of the tricuspid, because that valve is normally somewhere about twelve cm. The pulmonary valve is very rarely diseased and was not in this case. The mitral curtain showed diffuse deforming fibrosis extending along the free margin of the valve and showing in places fibrocalcareous degeneration—a pretty fair stenosis of the mitral, and chronic enough so that there was fibrocalcareous material in the lesion. The circumference of the aortic cusps was 6 cm. It might be all right; but what happened was that fibrosis had deformed the margins of the cusps with consequent diminution of their width, producing some regurgitation. The tricuspid showed some fibrosis along the margin of the valve, some shortening and thickening of the chordae tendineae.

The decrease in circumference was sufficient so that we have to say stenosis of the tricuspid. Further complicating the picture, on the roughened surfaces of the fibrocalcareous degeneration of the aortic valve was a soft wad of acute endocarditis. A clear-cut picture of chronic endocarditis and acute, with chronic pericarditis. Of course the result of that was the hypertrophy and dilatation of the heart and the chronic passive congestion.

It often happens here when examining the organs, as happens often in the clinic, that chronic passive congestion disguises any lesion that may be in the kidneys. These kidneys weighed 415 grams, were bluish-red, plump, rather wide cortices, and the section surfaces were bluish-red. That answers well enough macroscopically for chronic passive congestion, but the kidneys nevertheless were the seat of subacute glomerulonephritis. The pelves, ureters, bladder, uterus and adnexa were out of the picture.

The liver showed fatty metamorphosis.

All together this is a clear-cut picture of endocarditis acute years ago and now chronic and acute, both.

DR. CABOT: The spleen was not enlarged?

DR. RICHARDSON: It was slightly enlarged. Another thing—with all the story not a single infarct was found.

### Necropsy 4674

An Irish-American schoolgirl of nineteen came to the Emergency Ward April 27. An unsatisfactory history was obtained from her mother. It was negative except for tonsillectomy and adenoidectomy at four years. When the girl was six years old her father died by trauma. After fainting once the child began to feel weak and listless. A year later she had an attack of red, painful and swollen joints keeping her in bed three weeks. After this she had attacks of joint pain at least once a year, and also had "growing pains." At thirteen she had a particularly severe attack of rheumatic pain, and following it had dyspnea. For the past six winters she had been in bed a great deal, chiefly because of the joints. Two years before admission she was treated by a doctor who said that she should not be out of bed. It was impossible to learn how many periods of dyspnea she had had. For the past ten days she had been vomiting, and for the past week had been very ill indeed, vomiting, unable to sleep or eat, and requiring constant fanning.

Examination showed a young girl with dyspnea and orthopnea yet surprisingly comfortable. The mucous membranes were cyanotic,

the lips red, as if painted. The skin was pale and moist with diffuse acne over the chest and back. The subcutaneous tissue was thin. The muscles were soft. The respiration was accelerated. There was high diaphragm on both sides. The breath sounds were markedly high-pitched at inspiration, otherwise the lungs were normal. The heart was markedly enlarged to the right and left. The apex impulse was in the sixth space 10 cm. from midsternum and 3 cm. outside the midclavicular line. The right border of dullness was 4 cm. from midsternum. The supracardiac dullness was 5 cm. There was precordial heave. A thrill was felt which was difficult to time. Action absolutely irregular. Sounds of good quality. Pulse deficit. One sound, probably the first, markedly accentuated. Murmurs difficult to time. There were no signs of pericarditis or effusion and no compression signs posteriorly. The blood pressure was 110/60. The liver was enlarged almost to the umbilicus and slightly tender. There was slight sacral and pretibial pitting. The temperature was 96 to 101.8°, the pulse 84 to 141 to 40, the respiration 30 to 12. The urine and blood are not recorded.

The patient weighed 100 pounds. One hour after the second dose of digifolin it was found that the heart rate had gradually come down from 140 to 96 at the apex and was irregular. Two hours after the third dose, i.e. after 13⅛ grains had been given intravenously, the apex rate had fallen to 66. After the fall to 96 she felt much better and had good diuresis. After the drop to 66 the improvement was more marked. By the 29th she felt fairly well. She had been vomiting all the morning. At noon the apex rate seemed fairly regular. It was still about 65. There was a slight odor of acetone in the breath. Rectal glucose was started. During the afternoon the pulse was markedly irregular for short periods. By six o'clock the whole condition was changed for the worse, though she felt about the same. She was still vomiting. During the evening the heart was extremely irregular in rate and rhythm over short periods during which it was impossible to enumerate the separate contractions; as nearly as could be made out the rate went up to 90 or 100. At other times it was 65 to 70. At 9 p.m. she had not had any digitalis for twelve hours. The attacks became more frequent and she was more restless during them. When the rate was slow she was markedly apathetic and moribund. She grew rapidly worse, and died rather suddenly during an attack of marked fibrillation.

*Clinical Diagnosis (from Hospital Record).*—Rheumatic heart disease.

Mitral stenosis and regurgitation.

Auricular fibrillation.

Congestive failure.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the aortic and mitral valves with stenosis of each.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral, aortic and tricuspid valves, stenosis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Slight hydrothorax.

Slight hydropericardium.

Slight ascites.

Slight anasarca.

Chronic pleuritis.

Dr. Richardson: The lips were purple, the skin generally pale. In the peritoneal cavity there was about 500 c.c. of thin pale fluid,—slight ascites. The mucosa of the stomach and intestines was velvety, reddened, juicy, and saturated with thin bloody fluid,—chronic passive congestion.

The anterior margin of the liver was 8 cm. below the costal border—i.e., much below. The liver weighed 1010 grams,—not large, although it was so far below the costal border. Yet the tissue showed passive congestion.

Dr. Cabot: That is an interesting point. I never saw that before.

Dr. Richardson: The diaphragm was at the sixth rib on the right, the fifth rib on the left.

The right pleural cavity contained 200 c.c. of thin pale fluid, the left 50 c.c.,—beginning hydrothorax. There is a great variation in the amounts of fluid found in the pleural cavities. Sometimes there is a great deal in the right side and none in the left, and sometimes very little in each; to find very little in one and considerable in the other is not unusual at all. There were old pleural adhesions posteriorly in the region of the upper half of the right lung. On the left there were old adhesions posteriorly. The lung tissue showed well marked chronic passive congestion.

The pericardium contained 100 c.c. of thin clear pale fluid; slight hydropericardium.

The heart weighed 550 grams. That is marked enlargement. The right ventricle wall was thickened, 5 mm. That suggests a

lesion of the mitral valve. The left ventricle wall was 11 mm. There may be something the matter with the aortic, but it will not be very marked. The columnae carneae were well marked. Cavities: Left, much dilatation; right, considerable dilatation. That dilatation suggests changes in probably two valves at least.

DR. CABOT: Was there anything about the left auricle, particularly?

DR. RICHARDSON: Dilatation.

The auricular appendices were negative. The circumference of the mitral valve was about 7½ cm.,—considerable stenosis. The usual circumference would be 10 cm. The mitral curtain showed a fibrous irregular band of thickening extending along the free margin, with some thickening and fusion of the chordae tendineae and considerable diffuse fibrosis and deformity of the valve. A fibrous patch extended from the valve curtain up along the endocardium of the left auricle. That is not an unusual picture in these so-called rheumatic hearts. The acute process is a spreading one, and sometimes extends down along the endocardium below the valve and frequently up into the auricle. In the region of the aortic valve it may spread to the endocardium below the cusps, and sometimes it extends up along the aortic wall, a kind of aortitis, but not luetic. It may confuse one a little as to whether it is syphilitic aortitis or not. The tricuspid valve measured 10 cm., stenosis, and showed considerable fibrosis with contraction and deformity of the curtain and thickening, and shortening of the chordae tendineae. The aortic was 5½ cm. It showed some diffuse fibrous thickening, and the free margins of the cusps were a little rigid and fibrotic; slight stenosis. The pulmonary valve was negative. Of all the valves the pulmonary is the least likely to be diseased; for some unknown reason it best maintains its integrity. The coronaries were free and negative.

The liver showed chronic passive congestion.

The spleen weighed 120 grams,—not increased in weight, but with elastic congested tissue.

DR. CABOT: The kidneys were all right?

DR. RICHARDSON: Chronic passive congestion.

DR. CABOT: No cause for vomiting found except digitalis.

### Necropsy 1249

An Irish housemaid of forty-eight entered August 21. She gave a history of measles, mumps and scarlet fever in childhood. Otherwise she had always been strong and well except for dyspepsia

of several years' standing. For the past two years her catamenia had been very irregular and scanty. The day before admission, three hours after eating cabbage, she was seized with severe abdominal cramps, nausea, vomiting and diarrhea. The symptoms continued all through the day of admission, and she had vomited and retched continually. The vomitus was dark colored and watery. She could not sleep. The cramps were relieved by pressure.

Examination showed a slightly obese woman with markedly irregular heart action and pulse. The heart was not enlarged and showed no other abnormalities. The base of the right lung in front and behind showed slight dullness, many coarse moist râles, with diminished tactile and vocal fremitus and breathing. At the left apex behind there was slight dullness with marked bronchial breathing. The abdomen was much distended, the navel flushed. The muscles were held rigid. There was slight fullness in the epigastrium and slight general tenderness. To the right of the umbilicus was a soft tumor about an inch in diameter under the skin. About three inches below this was a small hard subcutaneous nodule, and in the epigastrium a similar one. The temperature was normal, the pulse 128 to 131, the respirations 23 to 36. The urine showed a very slight trace of albumin; otherwise it was not remarkable. The hemoglobin was 70%, the leucocytes 27,400. The vomitus was dark brown. Microscopical examination showed it to be made up of fibrin and normal red blood corpuscles.

The night of admission the patient did not seem in poor condition. The pain was easily relieved by a hot water bag. After midnight she vomited a few times, castor oil and some food and bile. In the morning she vomited black material which proved to be mostly blood. Her abdomen was much distended and her face ashen. She continued to vomit quarts of black material. Operation was done that morning. Considerable blood tinged fluid escaped when the peritoneum was opened. A portion of the small intestine about twenty-four inches in length seemed to be gangrenous and distended, apparently at the beginning of the ileum. The demarcation line was distinct. The mesentery was thrombosed and friable. The patient died on the operating table.

DR. RICHARDSON: There is a note here upon the operation. Apparently Dr. Scudder must have told me himself, because I have recorded, "The surgeon, Dr. Scudder, states that at the time of operation there was a twist in the jejunum in the region of the involved portion."

DR. YOUNG: A volvulus.

DR. RICHARDSON: That is what that would describe.

*Clinical Diagnosis.*—Acute intestinal obstruction.

Mesenteric thrombosis.

*Anatomical Diagnosis.*—Fibrous endocarditis of the mitral, aortic and tricuspid valves, stenosis.

Obturating thrombus of the superior mesenteric vein.

Hemorrhagic infarction of a portion of the jejunum.

Hypertrophy and dilatation of the heart.

Fibrous adhesions between the ileum and the uterus.

Chronic perihepatitis and perisplenitis.

Chronic hyperplasia of the spleen.

Chronic pleuritis.

Operation wound.

DR. RICHARDSON: A thin brownish-red fluid exuded from the operation wound. The peritoneal cavity contained a moderate amount of thin brownish-red fluid. We have to account for that of course. It came from the region of a strip of infarcted intestine. There were adhesions between the liver and the diaphragm and between the spleen and the diaphragm. These adhesions extended in places to the stomach. The appendix was negative.

The first portion of the jejunum for a distance of about 70 cm. was dark purplish red, the mesentery thickened, purplish. The upper and lower margins of the infarcted strip were rather sharply marked off, the lower line of demarcation being less sharp than the upper. The wall of the jejunum beyond the portion mentioned was slightly reddened, but this soon faded out.

The large intestine was negative. At a point thirty cm. above the ileocecal valve there was a band of adhesions extending from the wall of the ileum to the posterior wall of the uterus. All told there was considerable chronic peritonitis.

There was some chronic pleuritis.

The heart weighed 440 grams,—moderate hypertrophy. The mitral, aortic, and tricuspid valves showed a moderate amount of chronic fibrous endocarditis. The mitral circumference was 7 cm., the aortic 6, the tricuspid 10.5. The coronaries were free and negative. The aorta was negative.

The spleen was quite large, 1030 grams, but all it showed was some increase in the interstitial connective tissue. There was chronic perihepatitis and perisplenitis.

In the region of the jejunum infarction the walls yielded a purplish red fluid, the mucosa was swollen, dark purplish, and the intestine contained considerable thin purplish bloody fluid. The splenic and the inferior mesenteric veins were negative. In the superior mesenteric vein there was an obturating thrombus.

DR. YOUNG: Have you any idea where this thrombus came from?

DR. RICHARDSON: That is the reason why Dr. Scudder's note is recorded. There was no definite source in the body, but if there was a twist there, that is a perfectly good source.



FIG. 27.—Infarction of the jejunum. (Photograph by Lewis S. Brown and Dr. Oscar Richardson.)

DR. CABOT: Could the cardiac lesion by slowing the circulation be regarded as a cause of thrombosis in a vein as far off as this?

DR. RICHARDSON: There does not seem to be quite enough stasis.

DR. CABOT: There was no passive congestion anywhere else? The heart was doing its job?

DR. RICHARDSON: Fairly well apparently.

DR. YOUNG: Isn't it true that there is generally some cardiac lesion in most of these cases of mesenteric thrombus?

DR. RICHARDSON: I cannot say definitely. I do not remember. This thrombus was in the superior mesenteric vein.

It happened that Mr. Brown took a very successful picture of this case which is worth looking at and is much better than anything one can say (Figs. 27 and 28).

A PHYSICIAN: Is there anything to indicate the cause of the chronic peritonitis?

DR. RICHARDSON: Nothing definite.

A PHYSICIAN: The tubes and uterus were normal?



FIG. 28.—Infarction of the jejunum. (Photograph by Lewis S. Brown and Dr. Oscar Richardson.)

DR. RICHARDSON: Yes.

DR. CABOT: How do you account for the blood in the stomach?

DR. YOUNG: It was acute congestion, wasn't it?

DR. RICHARDSON: At the time of necropsy the stomach contained much undigested food material but was otherwise negative. There was bloody fluid in the first portion of the small intestine.

### Necropsy 3566

An Irish housemaid of thirty-two entered February 1 for the relief of pain and distress in the left chest. Her father died of heart disease. She had always been healthy. She normally urinated twice at night. The summer before admission she weighed 150

pounds, her best weight. She now weighed 113. She drank five quarts of water and a quart of milk a day.

For two months she had had night sweats. A month before admission she had moderate epigastric pain, relieved after a week of treatment for dyspepsia. After a few days she had sharp shooting pains in the arms, legs, and feet. A few days later her legs began to swell and her appetite to grow poor. January 29 she began to have severe continuous pain in the cardiac region, not affected by eating or change of position. A previously slight dyspnea became worse. She had palpitation on strong exertion.

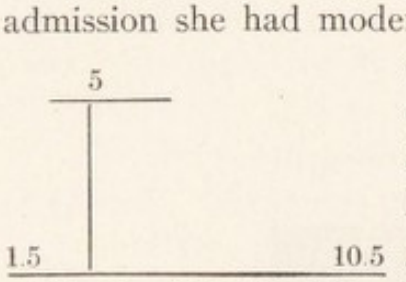



FIG. 29.—Dimensions by percussion.

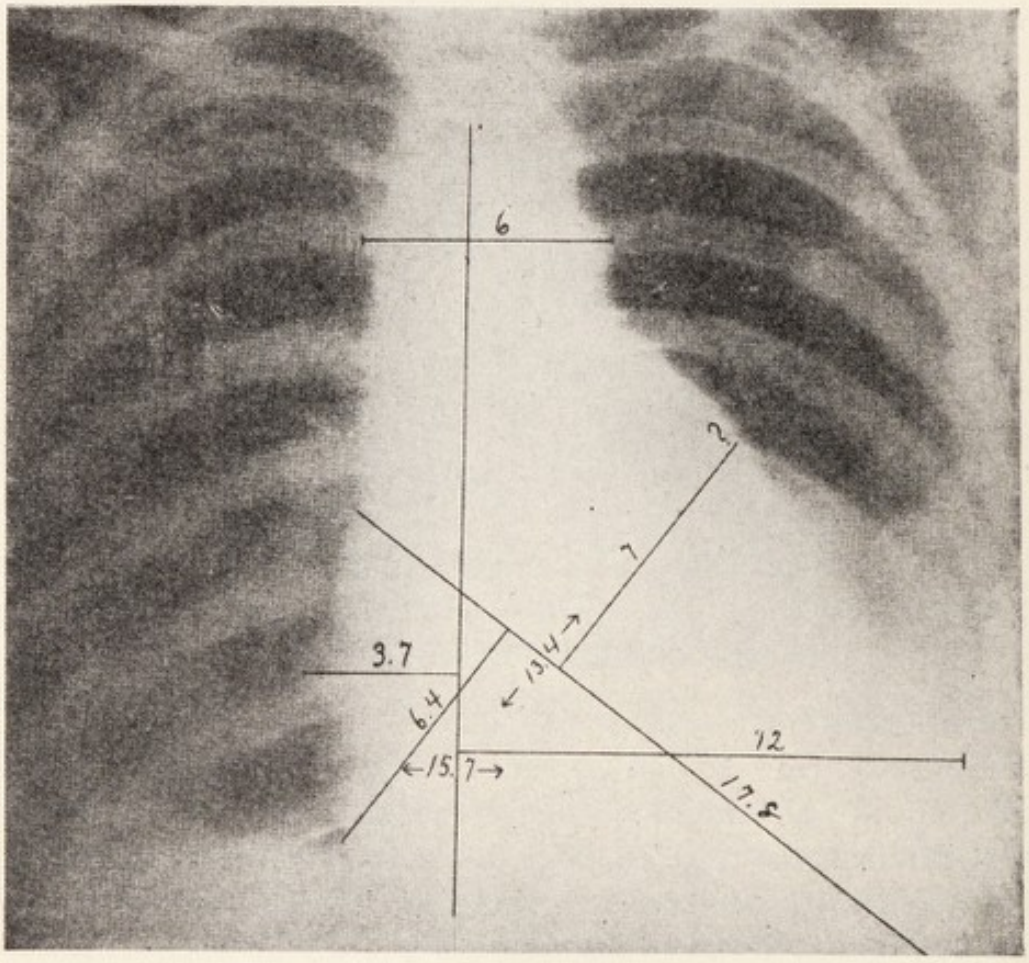



FIG. 30.—Seven-foot plate of hypertrophied and dilated heart with mitral, aortic, and tricuspid stenosis.

Examination showed a well nourished woman. The apex impulse of the heart was in the fifth space. The dimensions by percussion are shown in Fig. 29, the measurements by X-ray in Fig. 30. There was a slight thrill over the apex, whether systolic or diastolic is not

recorded; possibly also over the second right intercostal space. The action was regular, rapid. The sounds were of good quality. The pulmonic second sound was accentuated. A blowing systolic mur-

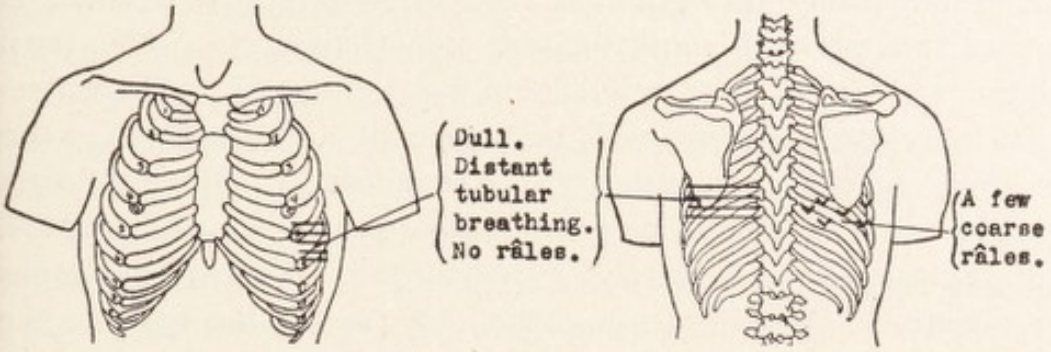



FIG. 31.

mur and a soft diastolic were heard over the aorta area, transmitted to the neck; also at the apex, the systolic transmitted to the axilla. The pulses were of fair volume, low tension, Corrigan in char-

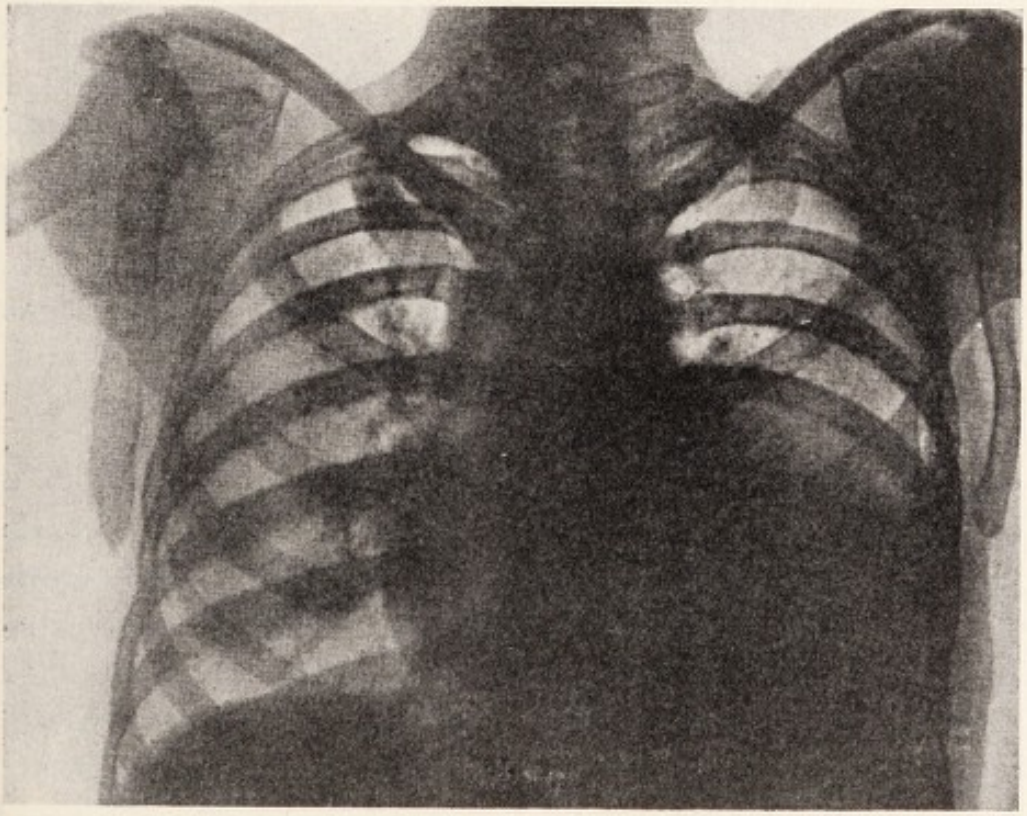


FIG. 32.—Same case showing apparently effusion at left base. Enlargement of the heart, especially in the region of the pulmonary artery and the left auricle. Pathological process at the left base, involving the pleura.

acter. The lung signs are shown in Fig. 31. The abdomen was negative. There was slight edema of the ankles. The pupils and reflexes were normal.

The temperature was 97.5° to 103.5°, the pulse 80 to 120, the respiration 20 to 38. The amount of urine was normal, the specific gravity 1.010 to 1.024. There was a very little albumin at all of eight examinations, rare granular casts at five, cellular at one. A urine culture showed staphylococci; no streptococci. The renal function was 55%. The hemoglobin was 75 to 60%. There were 13,200 to 34,700 leucocytes, 81% polynuclears, 3,954,000 to 3,112,000 reds, slight achromia, irregularity in size and shape. Blood cultures February 2 and 24 showed no growth, February 15 showed doubtful bacilli, February 7 and March 15 streptococci. Two Wassermanns were negative. A stool was negative. X-rays of the roots of the teeth showed a pus pocket and absorption. The chest showed opacity at the left base as high as the fourth rib in front. The diaphragm could not be made out on this side. (See Fig. 32.) The heart was a little displaced to the right.

February 6 the patient suddenly lost power over her left arm could not speak clearly, had a left facial paresis and loss of the pharyngeal reflex. The uvula was pulled to the left. The tongue could not be protruded. The speech grew somewhat clearer; otherwise the condition did not improve. February 11 there was musical murmur in the aortic area. She grew increasingly pale. March 14 she began to complain bitterly of "feeling sick all over," with pain especially in the abdomen. That night she quietly died.

*Clinical Diagnosis (from Hospital Record).*—Mitral stenosis and regurgitation.

Ulcerative endocarditis.

Septicemia, streptococcus.

Cerebral embolus.

Mesenteric infarcts?

*Dr. Richard C. Cabot's Diagnosis.*—Streptococcus endocarditis, acute; also chronic, endocarditis with deformity of the aortic valve, stenosis and regurgitation.

Perhaps some lesion at the mitral valve.

Septicemia, streptococcus.

Serofibrinous pleuritis.

Embolic hemiplegia.

*Anatomical Diagnosis.*—Chronic and acute endocarditis of the mitral and aortic valves.

Chronic endocarditis of the tricuspid valve.

Hypertrophy and dilatation of the heart. (Weight 375 gm. Right ventricle 3–4 mm. Left 10 mm. in thickness.)

Chronic passive congestion, general.
Purulent infarct of the spleen. (Weight 600 gm.)
Infarcts of the kidneys.
Acute glomerulo-nephritis.
Slight fatty metamorphosis of the liver.
Chronic pleuritis, bilateral.
Focus of obsolete tuberculosis, upper lobe, right lung.
Obsolete tuberculosis of a mesenteric lymphatic gland.
The head was not examined.

The circumference of the mitral valve was 8.5 cm. (normally 10), of the aortic valve 7 cm. (normally 7), of the tricuspid valve 9 cm. (normally 11), of the pulmonary valve 8.5 cm. (normally 7).

The aortic and mitral orifices were so much obstructed by masses of acute vegetative endocarditis that it was hard to make a definite statement as to permanent deformities.

The absence of fluid in the left chest post mortem is explainable by the difference in date between the X-ray and the necropsy. Possibly the shadow was due in part to the big spleen.

A streptococcus (probably the s. viridans) was cultivated from the spleen. Yet curiously enough cultures from the heart's blood were sterile.

It is notable though not at all unprecedented that the renal function remained good despite the existence of an acute glomerulonephritis wholly unsuspected during life.

### Necropsy 3596

A widowed American waitress of thirty-four entered April 22 for relief of palpitation. One sister died at thirty of hemorrhage in the brain. The patient's husband died of tuberculosis, one child of tuberculous meningitis six months later.

She had children's diseases and St. Vitus' dance in childhood. She had one miscarriage. At twenty-five she was told she had heart trouble and was advised to rest in the country. She felt perfectly well until the present illness, except for occasional slight dyspnea on exertion for a year. She drank a bottle of porter every night before going to bed. She urinated three or four times by day, two or three times at night. Her catamenia was regular except for four months, a year before admission, when it occurred every two weeks. Her best weight was 121 pounds, her weight a year ago 118. She worked as a waitress, not hard.

A month before admission she became nervous and was unable to sleep. She worried about family affairs, which came to a crisis a week before admission. Six days ago she felt weak, but worked until eleven at night. While walking home she suddenly felt a sense of constriction around the throat, "electric pains" in the arms, and rapid and violent thumping of the heart. Half an hour later she began to vomit, and continued to do so all night and the next day. The palpitation had been less and the pains had disappeared. She had developed a dry cough.

Examination showed a well nourished woman with slightly prominent eyes. The tonsils were ragged. There was possible slight

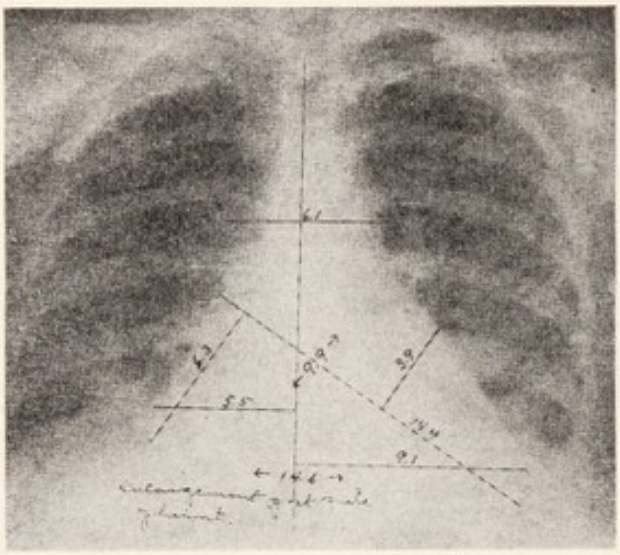


FIG. 33.—Seven-foot plate of hypertrophied and dilated heart in stenosis of the mitral, aortic, and tricuspid, with chronic adhesive pericarditis and auricular flutter. Enlargement of the right side of the heart.

fullness of the thyroid. The apex impulse of the heart was in the fifth space 10 cm. to the left of midsternum. The right border of dullness was 5 cm. to the right. X-ray showed the measurements as in Fig. 33. There was a possible thrill at the apex. The action was regular, very rapid. Electrocardiogram however showed auricular flutter. The first sound at the apex was very loud. The pulmonic second sound was accentuated. There was a harsh systolic murmur at the apex transmitted upward and to the left and a possible diastolic to the right of the apex. The liver dullness extended from the fifth space to two cm. below the costal margin. The edge was felt 5 cm. below the costal margin. The lungs, extremities, pupils and reflexes were normal. There was no tremor.

The temperature, pulse, blood pressure and apex beat are shown in Fig. 34. The respirations were 22 to 44. The amount of urine

was 10 to 35 ounces, the specific gravity 1.018 to 1.008. There was the slightest possible trace of albumin at one of three examinations. Occasional red blood corpuscles were seen once. The renal function was 20%. The hemoglobin was 75%. There were 13,400 to 9600 leucocytes, 73% polynuclears. The reds showed moderate varia-

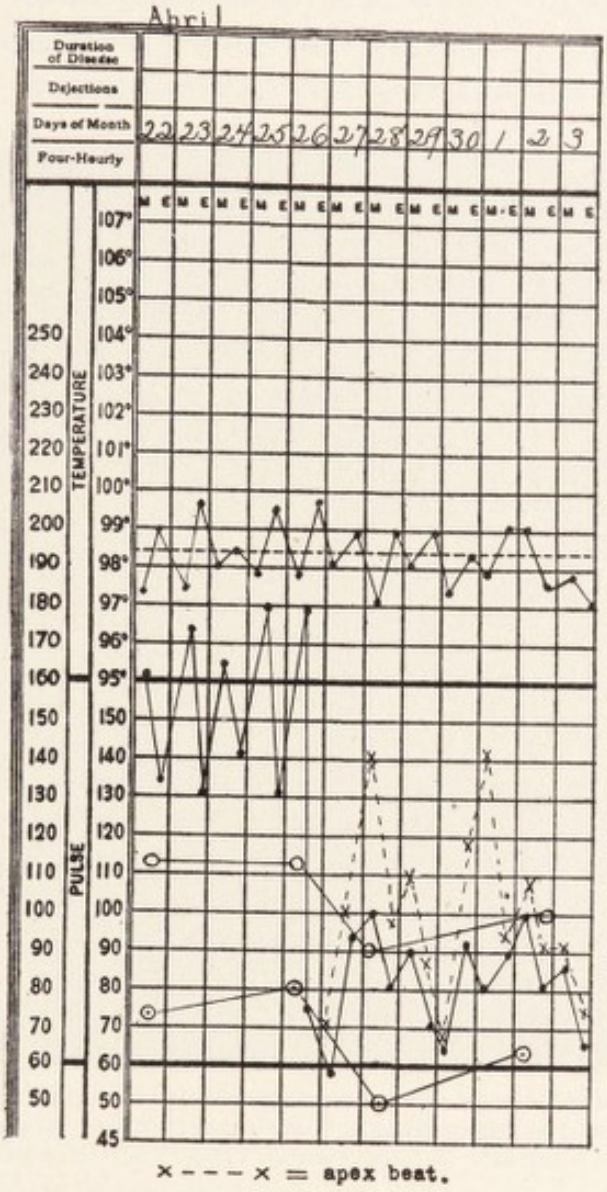



FIG. 34.

tion in size and shape, many polychromatophilic cells, rare stippling. A Wassermann was negative. The stools were negative to guaiac at three examinations. The sputum showed no tubercle bacilli at two tests. X-ray (Fig. 35) showed the right chest less radiant than the left, especially at the apex; considerable thickening of the lung roots, especially on the right and running toward the apex; glands at both roots, some of them calcified; the heart enlarged to the right and left.

April 26 the patient was nauseated, vomited, and her pulse dropped 105 points (180 to 75) and became irregular. Electrocardiogram showed auricular fibrillation. Digifolin was omitted. April 29 the visiting physician heard a loud, very harsh systolic murmur at the aortic area, and wrote, "I believe a diastolic is present different from the one at the apex. This I believe originates at the aortic valve." May 1 digifolin was given again. By May 3 she was responding well to it. The pulse was still irregular. At midnight she was given an

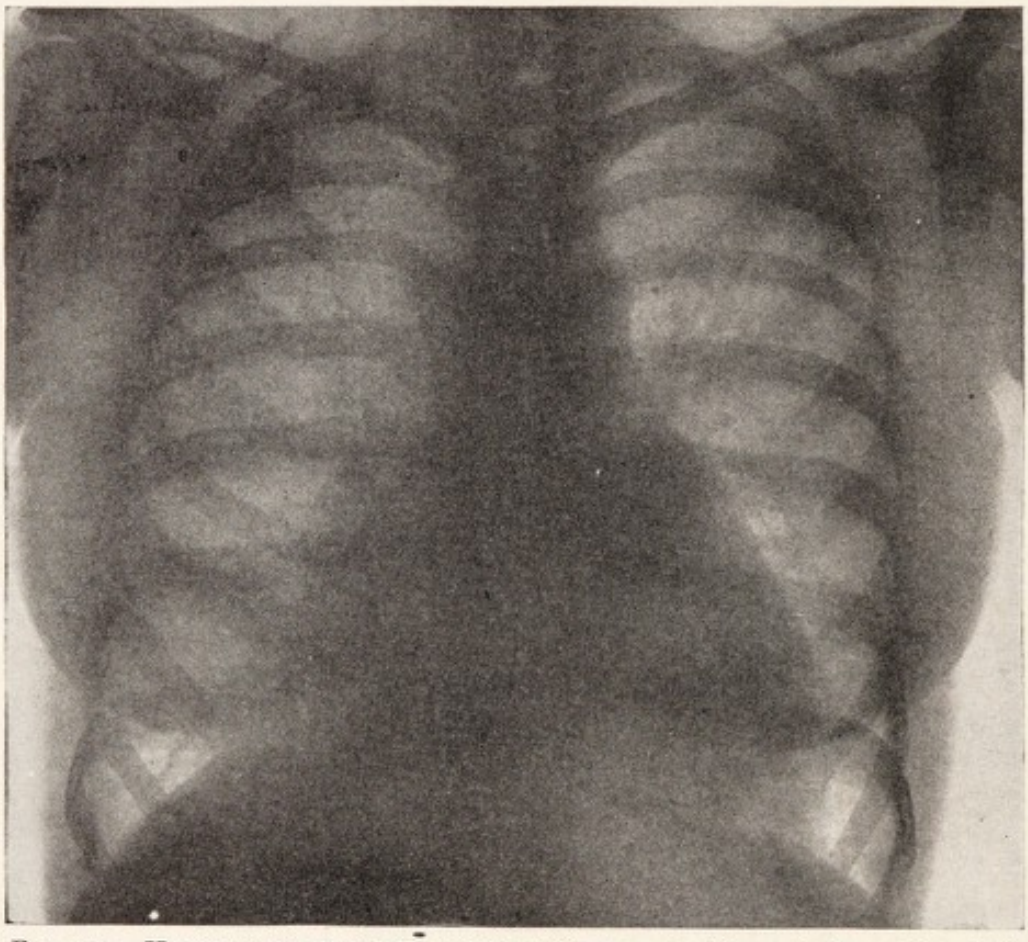


FIG. 35.—Hypertrophied and dilated heart in stenosis of the mitral, aortic and tricuspid, with chronic adhesive pericarditis and auricular flutter. Heart enlarged to right and left. Findings not definite on tuberculosis; rather more like passive congestion.

enema, and felt very well. She sat up to have a sheet changed, and was very comfortable and laughing when she suddenly fell over in a convulsion, more marked on the left side, and died.

*Clinical Diagnosis (from Hospital Record).*—Cardiac flutter.

Auricular fibrillation?

Mitral stenosis?

Cerebral embolism and infarct of the lung?

*Dr. Richard C. Cabot's Diagnosis.*—Goiter heart (to be shown post-mortem by hypertrophy and dilatation).

Possibly a lesion at the aortic, stenosis and regurgitation, though on the whole I vote against it.

Healed tuberculosis of the right lung.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral, aortic and tricuspid valves, (stenosis).

Chronic adhesive pericarditis.

Thrombus in the left auricular appendix.

Thrombus in the right auricular appendix?

Hypertrophy and dilatation of the heart.

Pulmonary embolism.

Chronic passive congestion, general.

Hydrothorax, right.

Slight hydropericardium.

Slight ascites.

Slight arteriosclerosis of the aorta.

Slight chronic pleuritis, left.

Small portion of *persistent thymus* gland.

Slight chronic salpingitis.

DR. RICHARDSON: The heart weighed 466 grams,—considerably enlarged. (Normal weight 200 to 300 grams.) The myocardium of the right ventricle was thick, of the left about normal. The cavities were considerably dilated. The circumference of the mitral valve was 6 cm. (normal 10). The aortic ring was 5 cm. (normal 7).

Death was from pulmonary embolism.

The thyroid was not obviously enlarged. This, however, is perfectly compatible with thyrotoxicosis. Moreover, persistent thymus (which we found) goes very often with thyrotoxicosis.

DR. CABOT: I believe this patient had two things, chronic endocarditis and thyrotoxicosis. A pulse of 180 without arrhythmia is very rarely seen except in thyrotoxicosis.

A PHYSICIAN: She had had heart trouble nine years. Had she had thyrotoxicosis nine years?

DR. CABOT: No. She had had the valve lesion nine years.

### Necropsy 2556

An English housewife of twenty-eight was referred February 28 from the Out-Patient Department, where urine examination showed many red blood corpuscles. Her father died of "shock" at fifty-eight. At twenty-one she had "rheumatism." At twenty-seven she was ill in bed a week with "inflammation of the stomach."

For three years she had had dyspnea and palpitation. For two years she had had almost continuous sharp pain a little to the left of the navel, increased by exertion, especially by walking, which caused nausea and vomiting. A hot drink usually started or aggravated the pain. It was the same day or night. For two weeks it had been so severe that she had vomited two or three times a day. She had pain between her shoulders most of the time. Her appetite and sleep were poor. She had a heavy feeling when she lay down. Her best weight was 95 pounds, her weight two years before admission 90 pounds, her weight February 27, 87 pounds.

Examination showed a fairly well developed and nourished woman with slightly pale skin and mucosae. The apex impulse of the heart was felt in the third space 15½ cm. from midsternum, 4 cm. outside the nipple line. The right border was 3½ cm. from midsternum. A soft blowing systolic murmur was heard all over the precordia. The pulmonic second sound was greatly accentuated, very loud and snapping. The pulses were normal. The blood pressure is not recorded. The lungs were negative. The abdomen was held a little rigid. The point of greatest tenderness was an inch to the left of the umbilicus and a little below it, shading off from this point in all directions. The right pupil was greater than the left. Their reactions and the other reflexes were normal.

The temperature was 97.4° to 101.8°, the pulse 81 to 139, the respirations 24 to 33. The urine was normal in amount, dark green and alkaline at one of seven examinations (bile present), cloudy at two. The specific gravity was 1.017 to 1.026. There was the slightest possible trace to a slight trace of albumin at all examinations, rare hyalin casts at three, red blood corpuscles at one, rare leucocytes at two. Culture from a catheter specimen showed one colony of staphylococcus albus. The hemoglobin was 85% the leucocyte count 13,000 to 36,000. A blood culture was negative. Skin tuberculin test negative. The stool showed a slight amount of admixed blood. Cystoscopy showed a normal bladder, no obvious bleeding from either kidney, the functional activity of both kidneys equal and normal (indigo-carmine). "If she has noticeable hematuria I should like to see her during an attack."

March 3 the visiting physician heard a presystolic murmur before some beats and found at least half the right kidney palpable but not obviously enlarged or tender. By X-ray it appeared enlarged, but the plate was not conclusive. March 6 the double murmur was clear. March 8 the patient rapidly grew blue, the pulse

became rapid and poor, the respiration labored. Early the next morning she suddenly died.

*Bacteriological Report.*—Three guinea pigs inoculated with catheter specimens of urine, two from the right kidney, one from the left, were all negative at necropsy.

*Clinical Diagnosis (from Hospital Record).*—Malignant endocarditis.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the mitral valve, with stenosis and regurgitation.

Acute endocarditis of the mitral valve.

Infarcts in the kidneys and spleen?

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral valve. Stenosis.

Chronic and verrucose endocarditis of the tricuspid valve. Stenosis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Hemorrhagic infarcts of the lower lobe of the right lung.

Slight chronic pleuritis, left.

Focus of obsolete tuberculosis, upper lobe of left lung.

DR. RICHARDSON: This was a case of disease of the heart valves. The kidneys were negative. Nothing was found to give any anatomical basis for the pain mentioned below the diaphragm. But the incision was restricted to the thoracic wall, and the organs were examined *in situ*.

DR. CABOT: Here I think the pathologist is at a loss. He did not find the thing that was there.

A PHYSICIAN: How do you account for the cardiac impulse in the third space?

DR. RICHARDSON: The hypertrophy of the heart in this case was on the right side. It was behind an obstructed mitral, and the other lesion was on the tricuspid; that is, all the lesions were on the right side. The work was consequently thrown on the myocardium of the right side of the heart. In mitral stenosis we do not get hypertrophy and dilatation of the left side of the heart. An added load in this case was the lesion on the tricuspid valve. The valve measured about 7 cm., about one-half its normal circumference. In addition to the chronic endocarditis it showed also some verrucose endocarditis which possibly was acute. The mitral would not admit the tip of the little finger. That threw the whole load on the right side of the heart and gave a wide heart. I did not notice at the

time that the apex was as high as the third interspace. It shows that in making necropsies many points that seem insignificant are of value. Of course I did not know at the time that they called it in the third interspace, and there was nothing to attract my attention to it. I do not think it was as high as that.

A PHYSICIAN: Was a blood culture taken?

DR. RICHARDSON: Yes. It was negative. Septicemia is not always positive by culture.

A PHYSICIAN: Would that be the same reflex pain that we get in a child from pneumonia giving symptoms in the abdomen?

DR. CABOT: I do not think so. It persisted two years. We have to leave as a mystery that pain which we worked so hard over. That does not surprise me. We have many cases in which the post-mortem does not supply us with the information we seek.

A PHYSICIAN: Was that the patient's word for the pain, or was it the observation of the man who had charge of the case?

DR. CABOT: I should judge it was the patient's word.

A PHYSICIAN: There is quite a difference sometimes.

DR. CABOT: You are right. But they did notice tenderness in the place the patient complained of. It certainly sounded as if there had been something there.

A PHYSICIAN: She had a pleurisy.

DR. CABOT: Yes, but it is just a chronic adhesion which we find in about half of all our cases, without pain.

### Necropsy 3982

An American housewife of forty-eight entered August 18, 1919. A grandmother died of gastric carcinoma. The patient considered that she had always been strong and well. From the age of ten to fifteen she had chorea. She had the minor diseases of childhood, scarlet fever, and diphtheria. For twenty-five years she had had hemorrhoids, which in 1902 began an intermittent bleeding, stopping in 1909. At thirty-four she had bronchopneumonia. For ten years she had had occasional sharp grinding precordial pain, better of late, referred to the abdomen or axilla, once in 1907 to the shoulder, when it nearly took her breath away. Her bowels had always been more or less constipated. She passed the menopause at forty-five. That year she weighed 155 pounds, her best weight. Her usual weight was 135, her present weight 125. She thought she had lost 25 or 30 pounds during the past year.

For eight months she had had intermittent smarting, burning and itching of the rectum several times a day, not growing worse, but severe enough to interfere with sleep. There had never been severe pain. Since January she had taken cathartics which had made her bowel movements loose. She thought she had lost flesh, color, and strength. At entrance she was very weak.

Examination showed a fairly well nourished woman with slightly cyanotic skin and mucous membranes. The sclerae were slightly injected. There was right structural scoliosis with slight left lumbar compensating scoliosis and much deformity of the thorax,—prominence of the left chest, the right back, and the left thorax in the midaxillary line. Expansion of the right chest was greater than of the left. There was slight dullness and bronchovesicular breathing at the apex of the left lung. The apex impulse of the heart is not recorded. The borders of percussion dullness were 8.5 cm. to the right, 4.5 cm. to the left, the supracardiac dullness 5.5. The action was irregular. The pulmonic second sound was markedly accentuated. There was a presystolic roll and an apical thrill. The systolic blood pressure was 120, the diastolic 70. The abdomen was somewhat protuberant. The liver edge was 4 cm. below the costal margin. The pelvic examination showed good perineal support. On bearing down the patient began to urinate. She said there was no incontinence. The cervix was bilaterally torn. It was bound down to the posterior cul-de-sac. The fundus was apparently also bound down posteriorly. Rectal examination showed a hard, irregular mass, not tender, completely encircling the rectum, more prominent on the left, extending to the internal sphincter. The fingers showed questionable slight clubbing. The right pupil was greater than the left; otherwise both were normal. The reflexes were hyperactive.

Before operation the chart was not remarkable, the amount of urine not recorded, the specific gravity 1.014 to 1.022; the blood is not recorded. A Wassermann was negative. The stools showed blood and pus and a positive guaiac.

The patient complained of pain in the anus. August 27 operation was done. After the peritoneum was opened palpation revealed an irregular mass in the neighborhood of the left anterior brim of the pelvis. The liver was studded with carcinomatous nodules. The sigmoid was brought into the wound and the mesentery separated from it widely enough to permit the passage of a glass rod over which the sigmoid was sutured to the peritoneum and the fascia.

The loop was not opened. The pathological report on small pieces from the rectum was "carcinoma."

The patient made a good recovery from anesthesia. August 29 the wound was opened and found in good condition. The chart was flat. August 31 she complained of pain in the right side, and a short time later had a rather severe chill. The pulse was 136, the respirations 36, the temperature 99°. Later she complained of pains all over. There was some tenderness in the right flank. The temperature continued to range from 99° to 101.9°, the pulse from 84 to 113. September 3 she was feeling much better, and the wound was in good condition. September 9 she had been vomiting. Medical consultants advised stopping digitalis. The vomiting ceased, the heart action was much better, and by September 12 the chart was flat, yet for no discoverable cause the patient lost ground. September 15 her heart action was very poor. That day she died.

*Clinical Diagnosis (from Hospital Record).*—Carcinoma of the rectum and metastases.

Mitral stenosis.

Phlebitis.

Colostomy.

*Dr. Hugh Cabot's Diagnosis.*—Cancer of the rectum.

Acute peritonitis? or

Acute endocarditis?

*Anatomical Diagnosis.*—Carcinoma of the rectum with metastases in the retroperitoneal and bronchial lymph nodes and the liver.

Thrombosis of the inferior cava and iliac veins.

Embolic thrombosis of branches of the pulmonary artery.

Infarcts of the lungs.

Chronic endocarditis of the mitral and tricuspid valves; stenosis.

Hypertrophy and dilatation of the heart.

Edema of the ankles and feet.

Hydropericardium.

Slight chronic passive congestion, general.

Operation wound, colostomy.

Scoliosis.

The heart weighed 335 grams. The mitral valve measured 4.5 cm., the aortic 7, the tricuspid 7, the pulmonary 8.

### Necropsy 1533

An electrician of twenty-seven came to the Accident Room November 25. His mother died of a paralytic stroke. Except for an

attack of typhoid fever seven years before admission and for rheumatic fever confining him to bed for eight months he had been well. He drank two or three glasses of beer daily and an occasional glass of whiskey. He smoked thirty cigars, six boxes of cigarettes and forty cents worth of plug tobacco weekly. His present illness began four days before admission with sharp pain in the side, chill, and cough, all of which had persisted, with considerable fever. His appetite and sleep were poor.

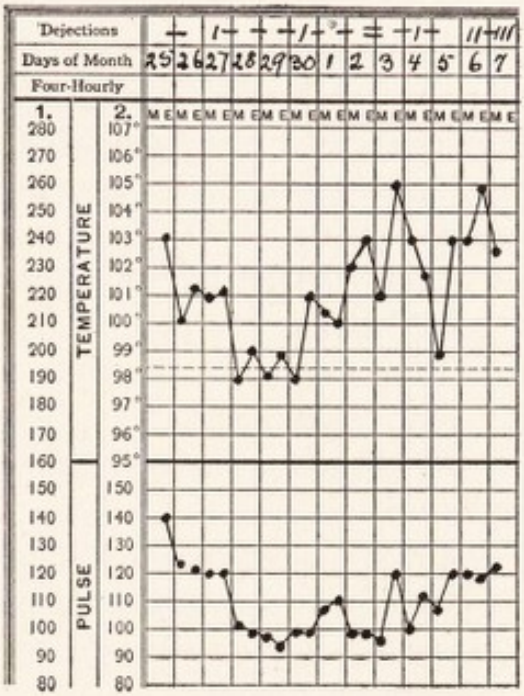



FIG. 36.

Examination showed a well nourished man with pallor of the skin and mucous membranes. The apex impulse of the heart and the left border of dullness were in the fifth space just outside the nipple line, 4½ inches from midsternum. There was no enlargement to the right. The sounds and action were normal. The first sound at the apex was markedly accentuated, heard over the whole precordia. A presystolic and a systolic murmur were heard at the mitral area, loudest at the apex, transmitted to the axilla. A soft diastolic murmur was heard at the aortic area. The pulmonic second sound was greater than the aortic. The pulses and arteries were normal. The right upper chest in front and back showed dullness, increased vocal and tactile fremitus and whispered voice; the front showed also bronchial breathing. At the right base behind were many fine moist râles. The abdomen showed slight general tenderness. There was a nodule in the right epididymis. The knee-jerks were not obtained. The other reflexes were normal. The temperature and pulse were as shown in Fig. 36. The respirations were 28 to 45. The urinary output was 65 to 85 ounces until the day before death, then it fell to normal. The specific gravity was 1.020 to 1.032. There was a trace to a slight trace of albumin at two of three examinations and a few to rare red blood corpuscles at two. The hemoglobin was 100%, the leucocytes 11,000 to 20,000.

November 29 the temperature had dropped to normal. Coarse moist râles were heard over the right back and at the upper right front. December 2 a diastolic murmur was heard best in the third

left interspace, but also in the second right interspace. Fine moist râles were heard all over the base of the right lung behind and at the side. The patient complained of pain in the right shoulder joint and of sharp pain in the splenic region. December 7 the diastolic murmur had become very loud and could be heard all over the precordia. The heart sounds were very loud and snapping. The pulse at the wrist was of rather poor volume and tension. The patient had failed rapidly during the past two days. That night the breathing was somewhat harsh at the left apex and in the axilla. Vocal and tactile fremitus were somewhat increased. The evening of December 8 he suddenly took a turn for the worse, sank rapidly, and in three hours died.

*Clinical Diagnosis.*—Croupous pneumonia.

Arthritis.

Mitral and aortic disease.

Acute endocarditis?

*Dr. Maurice Fremont-Smith's Diagnosis.*—Chronic and acute endocarditis.

Multiple infarcts to the lungs, kidneys and spleen.

Possibly bronchial pneumonia.

*Anatomical Diagnosis.*—Pneumococcus septicemia.

Lobar pneumonia.

Organizing pneumonia.

Chronic and acute endocarditis of the mitral, aortic, tricuspid and pulmonary valves.

Hypertrophy and dilatation of the heart.

Infarcts of the spleen and kidneys.

Dr. Richardson: We were not permitted to examine the head.

The lungs were voluminous. The pleura here and there was coated with fibrinous exudate. Of course there was some passive congestion in the lungs, and in addition in the upper half of each lung areas of pneumonia which in portions resembled a pneumonia in the stage of resolution and in places an organizing pneumonia,—that is, an older combined with a more recent pneumonia.

The heart weighed 493 grams,—considerably hypertrophied—and all of the valves, the mitral, aortic, tricuspid and pulmonary, showed chronic endocarditis in the form of fibrous deforming thickening with if anything decrease in their circumferences,* and on this basis what is called here polypous endocarditis, that is, smaller and larger soft

* The mitral valve measured 8 cm., the aortic 7 cm., the tricuspid 8½ cm., the pulmonary 7 cm.

frank masses of vegetations. That is an acute endocarditis on top of an old. The organism is the pneumococcus all through.

A pneumococcus septicemia, and in the vegetations on the heart valves many pneumococci.

The kidneys were not remarkable except for infarcts. There was a hypertrophied spleen, in places soft, and with infarcts here and there.

I could not make out any infarcts of the lungs, but of course with a source for emboli as there was on the tricuspid valve there might have been some small ones, but none were found.

DR. FREMONT-SMITH: I think we have to assume that the pneumonia was primary, don't we?

DR. RICHARDSON: Yes.

DR. CABOT: That is, you believe that the heart trouble came from the pneumonia?

DR. RICHARDSON: Yes, the acute trouble.

DR. FREMONT-SMITH: Do we ever get a primary pneumococcus septicemia without a pneumonia?

DR. RICHARDSON: Yes.

DR. G. C. CANER: Wouldn't you expect a more sudden onset if it were? There is a long history of smoking up to his admission.

DR. CABOT: Yes. That is a good point. People with acute endocarditis do not smoke that way. Isn't it true, on the other hand, that these vegetations are bigger than those you ordinarily see in a heart accompanying pneumonia? Aren't the vegetations accompanying pneumonia generally little ones?

DR. RICHARDSON: I don't think one can make a definite statement in regard to that.

DR. FREMONT-SMITH: Is pulmonary infarct a common symptom in subacute bacterial endocarditis? There is no reason why it should not occur, but does it happen?

DR. CABOT: I do not remember it. I do not believe it is common.

### Necropsy 2213

An American housewife of thirty-five entered September 21. She gave a history of measles in childhood, two miscarriages, malaria at thirty, rheumatism at thirty-two,—soreness of the knees and ankles without redness and swelling. She was in the habit of taking whiskey about once a week. She passed the menopause eight months before admission. In April, five months before admission,

she was ill in bed six weeks with an attack like the present one. After being up and about for two months she was laid up again for six weeks. After this she was fairly comfortable until two weeks before admission, when her feet began to swell, more at night, and she began to have cough with very little sputum until the past few days. The sputum was thick and yellow, once blood-tinged. She had much dyspnea and palpitation and could not sleep at night because of nervousness.

Examination showed a well nourished woman lying flat without discomfort with very cyanotic and mottled skin, cyanotic lips, and frequent dry cough. The apex impulse of the heart was seen and felt in the fifth space in the anterior axillary line, 14 cm. to the left of the midsternum, 6½ cm. outside the nipple line, corresponding to the left border of dullness. The right border of dullness was 5½ cm. to the right. The heart was fibrillating, the action rapid and very irregular in force and rhythm. A systolic murmur was heard all over the precordia and in the axilla, where it was musical. A presystolic murmur was heard at the apex, loudest just inside the nipple line, where a presystolic thrill was palpable. The pulmonic second sound was slightly accentuated. The pulses were irregular, only about a third of the beats reaching the wrist. The volume was variable. The artery walls were normal. The systolic blood pressure was 130. The lungs showed slight dullness below the lower angles of the scapulae. The abdomen was rather prominent, with diastasis of the recti and lineae albicantes. The liver dullness extended from the sixth rib to 10 cm. below the costal margin, where the edge was felt on a line with the umbilicus. The liver pulsated. There was moderate edema of the legs and slight edema of the thighs. The pupils and reflexes were normal. The temperature was 96.1° at admission, afterwards normal, the pulse 60 to 81, the respirations 20 to 34. The urinary output was 5 to 15 ounces, the specific gravity 1.022. At the single examination there was a very slight trace of albumin and the sediment showed rare red cells. The blood was normal.

September 23 ascites was demonstrated which was not found at entrance. The left border of cardiac dullness was two cm. nearer the median line than at admission. The next day one thousandth of a gram of strophanthin was given intravenously. The heart action was much stronger and steadier for a few hours. The next morning the heart showed alternating strong and weak beats. Only the forcible beat could be felt at the wrist. One-

thousandth of a gram of strophanthin was given intravenously again with transient good effect similar to that of the day before. September 26 one thousandth of a gram was given again. Ten minutes after the injection the patient was in good condition. A few minutes later she was very cyanotic, breathing heavily. Twenty-eight minutes after the injection she died.

*Clinical Diagnosis.*—Mitral stenosis and regurgitation.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Digitalis heart block (strophanthin).

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the mitral (and other valves?).

Mitral stenosis.

Chronic adhesive pericarditis. (?)

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Infarcts.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral, aortic, tricuspid and pulmonary valves, stenosis.

Slight chronic adhesive pericarditis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Hydropericardium.

Hydrothorax.

Ascites.

Anasarca.

Chronic pleuritis.

Chronic perihepatitis and perisplenitis.

Slight arteriosclerosis.

Dr. Richardson: We were not permitted to examine the head.

The abdomen was distended. There was a great amount of fluid in the peritoneal cavity. The extremities were swollen and pitted easily—ascites and anasarca. The liver was four fingers below the costal border, not very far down. The diaphragm on the right was at the fifth rib, on the left at the fifth interspace. Nevertheless the pleural cavities contained a large amount of clear straw-colored fluid —hydrothorax. There were the usual evidences of chronic pleuritis, fibrous adhesions generally.

The trachea and bronchi showed a slightly reddened mucosa and contained a moderate amount of brownish mucus material,—passive congestion. The lungs showed no areas of consolidation, no

evidences of pneumonia, but general well-marked chronic passive congestion.

The heart weighed 415 grams,—considerably enlarged for her. The myocardium was twelve mm. on the left, 3 mm. on the right. The heart muscle generally was thick. The cavities were enlarged, the auricles more especially so. The mitral, aortic, tricuspid and pulmonary valves all showed a definite chronic fibrous endocarditis. In the case of the mitral there was a small mass of fibrocalcareous material. The fibrosis appeared as irregular fibrous thickening of the valves and shortening and thickening of the chordae tendineae with consequent marked deformity of the valves and decrease of their circumferences. The mitral circumference was six cm., the aortic four cm., the tricuspid six cm., the pulmonary five and a quarter cm. Those are all decreased, most markedly so the mitral and tricuspid. On this chronic endocarditis basis there were on all the valves minute gray-red granulations which were firm. In one or two places there was some questionable fibrinous material, but all told not enough so that we could definitely state that there was an acute endocarditis. Of course these minute granulations occurring on the valves are very discouraging. If they are definitely firm and tough they of course are at least subacute or chronic. But if they are soft and fibrinous it is reasonable to assume that they are acute. It not infrequently happens however that it is difficult to say, and so it has been called "verrucose endocarditis." The only thing we have to go by is the question of consistence, the character of the material, whether firm or soft, and its relation to the underlying fibrosis. At the time this was not regarded as acute endocarditis.

The liver weighed 1047 grams. That was rather small. The organ showed very well-marked chronic passive congestion, and there were adhesions about it to the diaphragm. The spleen also showed adhesions to the diaphragm. That is, chronic perihepatitis and perisplenitis, without much definite idea as to why. It may be in association with the chronic passive congestion which was present in each of these organs, as there was some increase histologically of the interstitial tissue.

The gall-bladder and pancreas were negative. The spleen weighed 155 grams. The tissue was dark red, firm, elastic, the follicles and trabeculae visible,—a good example of chronic passive congestion.

The kidneys weighed 250 grams. The capsules stripped leaving fairly smooth surfaces, negative cortex. The tissue generally showed passive congestion.

The gastro-intestinal tract showed marked chronic passive congestion.

Between the layers of the pericardium there were membranous fibrous adhesions,—slight chronic adhesive pericarditis.

There was a slight amount of arteriosclerosis, mentioned because this woman was said to be thirty-five years of age.

A typical picture of that condition which is of course rather rarely found, all four valves of the heart affected.

DR. CABOT: That is about as near as we are going to get for the present. We can hit the mitral, we cannot the others. I am interested in the perisplenitis because she had had a very early arteriosclerosis and two miscarriages. I tend to think that perisplenitis and perihepatitis may mean syphilis.

### Necropsy 3252

An American teamster of fifty-one entered September 2 for relief of dyspnea on exertion and edema. His wife had had two miscarriages. At thirty four he first had rheumatism. Three years later he was ill four months with rheumatic fever. Since that time his joints had troubled him in bad weather. He had gonorrhea at forty-two. He occasionally urinated at night. Until three years before admission he was a steady but not very heavy drinker. For three years be had taken very little alcohol. A year ago he had slight dyspnea on exertion and slight edema of the ankles, growing worse until four months ago he gave up work. He had been in bed on and off, and for three months had at intervals slept in a chair. For two months he had had an occasional dull headache, some swelling and stiffness of the wrists, and "dancing spells" when his eyes became blurred. Six weeks ago he had some cough and vomiting. He had slight paroxysms of cough on exertion. Lately his legs had been especially swollen and a little painful, and he had occasional attacks of dyspnea without exertion.

Examination showed a well nourished man breathing rapidly. His cerebration was slow and suggested toxemia. The skin and mucosae were slightly cyanotic, with many dilated venules on the face. The sclerae were slightly injected. There was arcus senilis. A few hard cervical glands were felt. The apex impulse of the heart was in the sixth space 16 cm. from midsternum and 6 cm. outside the nipple line, coinciding with the left border of dullness. The right border was 6 cm. to the right of midsternum. The action was regular,

the sounds were of fair quality; the first and second sounds were heard best at the left sternal margin. The aortic and the mitral second were very faint. The pulmonic second sound was accentuated but faint. There was a loud explosive systolic murmur at the apex replacing the first sound, heard all over the precordia and transmitted to the axilla, as was a loud blowing diastolic which was heard best at the second and third spaces at the left sternal border and was transmitted also to the neck. At the base, loudest at the second right costal cartilage, was an early rough scraping systolic, without thrill, transmitted to the neck. The pulse was an atypical Corrigan.

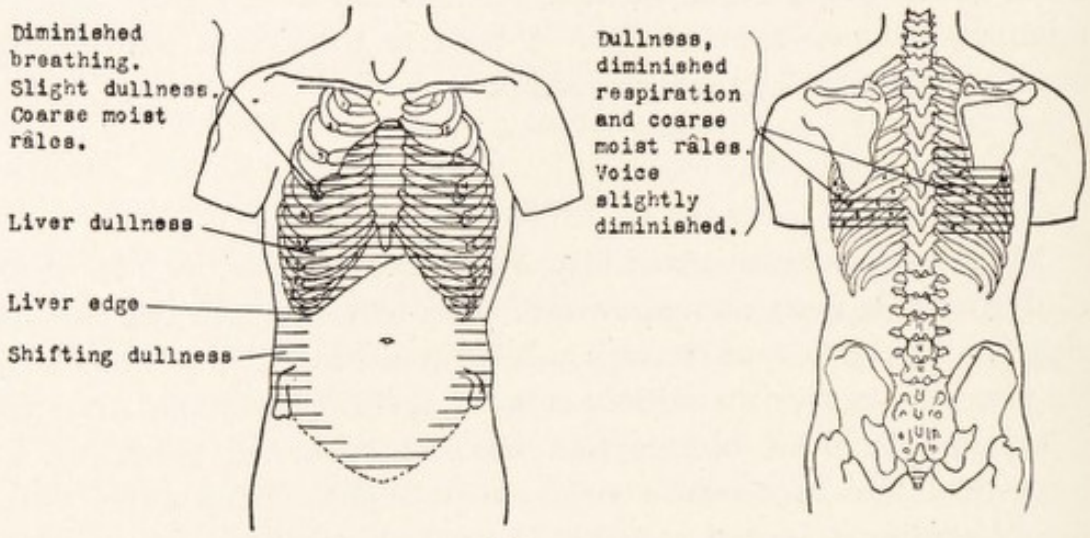



FIG. 37.

The artery walls were barely palpable. The lung signs are shown in Fig. 37. There was spasm and tenderness in the right upper quadrant and shifting dullness in both flanks. The liver dullness extended from the fifth rib to two centimeters below the costal margin, where a tender edge was felt. The lower abdominal wall, the chest wall, and the sacrum were slightly edematous. There was diastasis of the recti. The penis and right scrotum showed marked edema. There was a large right scrotal hernia, easily reducible. There was marked edema and brawny induration of the legs and thighs. The pupils were normal. The knee-jerks were sluggish, the plantars normal.

For the first three days the temperature was 97.8°–100° and the pulse 89–100; then the temperature usually 96°–98.4° and the pulse usually 80–92 until October 2. The later chart (Fig. 38) is shown. The respirations were 15–40. The systolic blood pressure was 110, the diastolic 65. The output of urine was 17–89 ounces, the specific gravity 1.016–1.021. Albumin in small amounts was shown at three

of six tests, red blood cells at three, leucocytes at two, granular casts at one. The renal function was 45%. The hemoglobin was 85%. The leucocyte count 7100–16,000, polynuclears 88%. There was achromia, some variation in size, rare poikilocytosis. A Wassermann was negative.

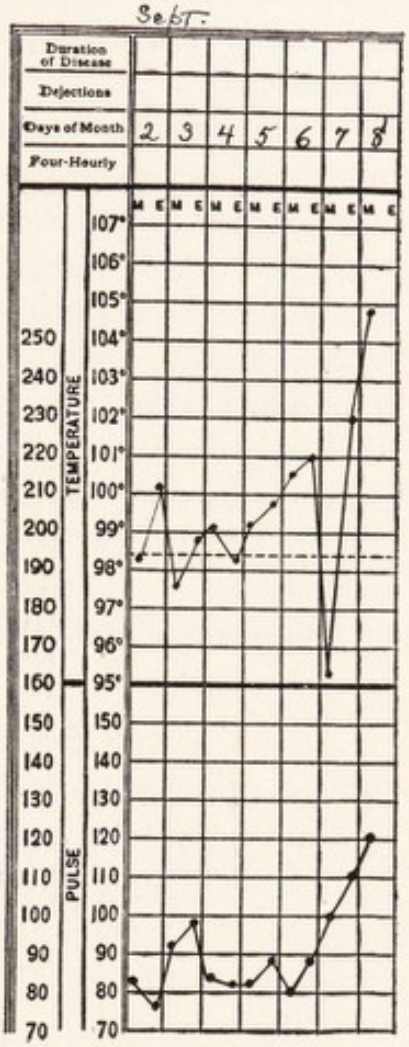



FIG. 38.

The patient was given two tablets of digipuratum a day, reduced September 5 to one. September 3 the visiting physician thought him toxic from alcohol. He heard practically no heart sounds. September 9 diuretin gr. xv was given three times. September 11 there was a systolic thrill in the manubrial and aortic areas, and the first sound at the apex and the pulmonic second sound were audible. There was increased diuresis coincident with the addition of diuretin to digipuratum. September 14 the former was omitted. September 16 the patient was much more comfortable, the toxicity gone and the edema almost gone. A week later he was having occasional short but very uncomfortable attacks of dyspnea, often nocturnal. The heart sounds were of better quality, the pulse remarkably good considering the general condition, the edema entirely gone.

From this point he grew much worse. He became increasingly delirious at night and stuporous during the day, with orthopnea and weakening heart sounds. October 6 the respiration began to grow rapid. The lungs showed a few râles at the bases, becoming many October 7 and obscuring the breath sounds so that no changes in breathing could be heard. There was no definite dullness. October 8 the lungs were full of râles. No patch of pneumonia could be made out. The patient was comatose, and died quietly that day.

*Clinical Diagnosis (from Hospital Record).*—Mitral and aortic endocarditis.

Broken compensation.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the aortic and mitral valves, with stenosis of both.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Terminal infection, probably pneumococcus.

*Anatomical Diagnosis.*—1. Origin of fatal illness. Chronic endocarditis of the aortic, mitral, tricuspid, and pulmonary valves, (stenosis).

2. Secondary or terminal lesions. Hypertrophy and dilatation of the heart. Chronic passive congestion. Very slight hydropericardium, hydrothorax, ascites and anasarca.

3. Historical landmarks. Cholelithiasis. Slightly defective closure of the foramen ovale. Slight chronic interstitial hepatitis.

The heart weighed 795 grams. The mitral valve measured 5.5 cm. The aortic valve opening was triangular in shape, each arm of the triangle measuring about 13 mm. in length. The tricuspid valve measured 11 cm., the pulmonary valve 6.5 cm.

## APPENDIX

### Fibrocalcareous Mass (Arteriosclerotic?) in the Mitral Valve—Three Cases

#### 1. Necropsy 534

A man of fifty-five died of chronic nephritis associated with a chronic adhesive pericarditis. He had had much pain in the left side of his chest for a month and some dyspnea two days before his death, but in general ran the course of a case of chronic nephritis. There were no cardiac manifestations in life not easily accounted for by the moderate cardiac hypertrophy associated with his chronic glomerulonephritis.

*Necropsy.*—The mitral valve in this case showed in its substance and projecting from its surface an irregular calcified mass the size of a split pea. This was thought to be due to arteriosclerosis, as were the similar masses in necropsies 3332 and 3999.

#### 2. Necropsy 3332

Fibro-calcareous cylindrical mass in the region of the insertion of the mitral valve. Marked arteriosclerosis in aorta and its great branches, in the cerebral arteries, in the pulmonary artery and its branches. Arteriosclerotic nephritis. Clinically the patient was a woman of 60 with chronic nephritis, uremia and cerebral hemorrhage, all of which was confirmed post-mortem. The examination of the heart in life showed nothing characteristic of the heart lesions found post-mortem. It was moderately enlarged, showed systolic murmurs at the apex and base, and nothing else.

3. Necropsy 3999

An essentially similar condition, namely a fibro-calcareous column about the size of the little finger at the base or insertion of one mitral curtain. The valve curtain over it, though wrinkled to correspond to the roughening of the fibro-calcareous material underneath, yet was otherwise smooth. The mass was situated near the insertion of the valve and did not appear to interfere with its function at all. This patient, like the last, suffered primarily from arteriosclerosis with hypertension (b.p. 235/120). She was a woman of 67 and was ill only two days, dying of cerebellar hemorrhage. Aside from a cirrhotic liver and general arteriosclerosis there were no lesions of importance. In life there was a loud blowing systolic murmur at the apex but nothing any more distinctive. The picture was one of hemiplegia, coma, and death.

**Comment.**—These data are presented because of the question which they suggest. Are the lesions arteriosclerotic? Do similar but more extensive lesions ever interfere with the heart's conduction system or with the function of the valves? In these three cases apparently the calcareous masses did no harm.

## AORTIC STENOSIS AND REGURGITATION

The rheumatic lesions of the aortic valve associated with those of the mitral and others valves have already been described in Chapter II. I tried there to make clear that the difference between "pure mitral" disease and mitral disease complicated by aortic was for practical purposes very slight and that the differential diagnosis between the two conditions was often difficult. I showed that in relation to age, sex, rheumatic, and choreic history the two groups ("pure mitral" and mitral plus aortic) were essentially the same, and that both groups differed only in unessentials from the rarer cases in which the disease had spread to the tricuspid or pulmonary valves.

But when we come to the cases of aortic disease not associated with any other valve lesion—"*pure aortic disease*" as I shall frequently call it—without any syphilitic aortitis, we face a problem in etiology.

### ETIOLOGY

The incidence of age and sex is in marked contrast with that of the rheumatic lesions of the mitral and other valves. In the pure aortic cases the males out-number the females by more than eight to one in

this series. Out of 28 cases, 25 were male and only three female, while it will be remembered that with the mitral group the females were always definitely, sometimes markedly in excess. As to age the contrast is also striking. In our 28 cases only six, or less than a quarter were under the fortieth year when they came under observation. As regards the time of onset, the data are not very clear, but appear to be as follows:

TABLE 53

| | YEAR | CASES |
|---|---|---|
| Duration of disease from first complaints to death | 1 | 1 |
| Duration of disease from first complaints to death | 2–3 | 3 |
| Duration of disease from first complaints to death | 4 | 3 |
| Duration of disease from first complaints to death | 5 | 3 |
| Duration of disease from first complaints to death | 6 | 1 |
| Duration of disease from first complaints to death | 8 | 1 |
| Duration of disease from first complaints to death | 10 | 1 |
| Duration of disease from first complaints to death | Over 10 | 2 |
| Duration of disease from first complaints to death | Several | 1 |
| Duration of disease from first complaints to death | Doubtful | 12 |
| | | 28 |
| Average duration | 6+ | |

TABLE 54.—AGE

| | |
|---|---|
| 1–9 | 1 |
| 10–19 | 3 |
| 20–29 | 0 |
| 30–39 | 2 |
| 40–49 | 7 |
| 50–59 | 7 |
| 60–69 | 7 |
| 70–79 | 1 |
| | 28 |

In Table 54 it appears that half the cases were beyond the fiftieth year when they first came under our notice, while eight out of twenty-eight were beyond the sixtieth year, and only four were under thirty. Such an incidence as regards age and sex, contrasting strongly as it does with that of the recognized rheumatic cases, cannot help making us suspect at once that these cases belong to a separate group, and are very possibly of a different etiology. Yet the history of rheumatic fever, chorea, or tonsillitis is about as frequent in these cases as in those which it is generally agreed to call rheumatic. Thus there was, in this group of 28 cases, a clear history of rheumatic fever in fourteen, two of which had also a history of tonsillitis and one of tonsillitis and chorea as well. Beyond this there were also three cases with a

questionable history of rheumatism, two with a definite history of chorea, and two with frequent attacks of tonsillitis; leaving only seven cases or ¼ without any known etiological clue. Comparing these figures with those in the mitral group we see that the two are practically the same.

If these cases are not rheumatic what are we to call them? They certainly show no evidence of being syphilitic. The usual presumption has been that some or all of them are of arteriosclerotic origin. In favor of this is the age and sex incidence already referred to, and the association with definite arteriosclerosis in other parts of the circulatory system, an association which is somewhat more frequent than in the mitral series. Out of 28 cases sixteen showed an associated arteriosclerosis, and only twelve were free from this. But these twelve cases militate strongly against the general hypothesis of an arteriosclerotic etiology for the whole group, unless one is to assume that arteriosclerosis can be confined to the aortic valve, which seems altogether improbable. In the cases associated with arteriosclerosis of the aortic arch it is very natural to assume that the process has extended on to the valves, as we remember that syphilitic aortitis does. This is further supported by the fact that in old people's heart valves a certain amount of fibrous thickening and stiffening is the rule. Why, therefore, should one not suppose that in certain cases this is exaggerated into the rigid, calcareous ring of aortic stenosis?

Against this it is to be said, in the first place, that we cannot disregard the definite rheumatic types of aortic stenosis associated with mitral stenosis in young people, which present on the valve a pathology identical with that of the group now under discussion. The same rigid, calcified ring, the same adherence and fusion of adjacent cusps, are seen in this group and in the juvenile cases without any possible suspicion of arteriosclerosis. Is it probable that two diseases as diverse as the acute endocarditis of rheumatism and the sclerosis of old age, produce identical results in the aortic valve? Moreover, five of these cases of pure aortic disease are associated with an acute aortic endocarditis such as we are very familiar with, breaking out as what we believe to be a recurrence or recrudescence of the chronic endocarditis of rheumatism. Certainly arteriosclerosis cannot cause this acute process. It is natural to suppose that the acute process is a recrudescence or relapse, in this group as in the mitral group,—in other words, to suppose it an acute infection recurring on the basis of a healed process of like type.

If one adopts the rheumatic hypothesis in these cases one has to leave largely unexplained the incidence in relation to age and sex. But on the whole the age and sex seem to me less difficult of explanation than the results of the opposite hypothesis of an arteriosclerotic origin for some or all of these cases. It may well be (1) that the heart and circulatory system accommodates itself more easily to be a pure aortic lesion than to any of the other valve lesions or combinations of such, that, (2) as a result of this the patients live on and have time to acquire in other parts of their bodies the usual arteriosclerosis of elderly people. The sex difference remains still a mystery unexplained. But we shall see in studying acute endocarditis and also in the cases of chronic non-deforming endocarditis that the aortic valve is more often hit in men and in old men. We may think of its lesions as especially men's not women's.

On the whole it seems to me best to consider these cases as due to a peculiar subtype of endocarditis belonging under the rheumatic group.

## PATHOLOGICAL ANATOMY

1. *Enlargement of the Heart.*—In Table 55 are listed the heart weights correlated with the valve circumferences and with the clinical diagnoses as regards cardiac size. To the last of these points I shall return later. But at a glance it is clear that one can find large, middle-sized, or small hearts in association with aortic stenosis and regurgitation, and that there is not much correlation between the heart weight and the size of the slit left between the edges of the stenosed valves. Thus the four hearts weighing 700 grams or more were associated with an aortic valve circumference averaging 4.5 cm. Hearts weighing from 500 to 700 grams went along with aortic circumferences slightly larger, the average being 5.3 cm. But the hearts weighing less than 500 grams were associated with valve circumferences averaging slightly *smaller* than those of the group just referred to. So that viewing the whole 28 cases I should say that there seems to be very little connection between the size of the heart and the size of the aortic orifice.

It is also particularly to be emphasized that decidedly small hearts occurred in this series, for example, 278 grams, 305 grams, and 370 grams. On the other hand, one heart weighed 1000 grams, which is among the largest of our whole series. There was nothing either in the pathological anatomy or in the history of the cases to explain these differences.

2. *Type of Endocarditis.*—In 23 cases the lesions were altogether chronic and hard. In five these chronic lesions were associated with an acute endocarditis.

3. *Size of the Aortic Aperture.*—Table 56 shows that in twenty-two cases the aperture was obviously reduced by actual measurement, many times very greatly reduced. On the other hand, one can never conclude from normal or even increased valve circumferences that stenosis is *not* present, because a rigidity of the valves, holding their margins into close approximation and immobility is quite possible even though their circumference when they are dissected apart is not diminished and even if it is increased.

TABLE 55.—CORRELATION OF CLINICAL MEASUREMENTS OF CARDIAC ENLARGEMENT WITH POST-MORTEM DATA OF HEART WEIGHTS AND WITH AORTIC VALVE MEASUREMENTS

| Case No. | Clinically enlarged | Weight | Aortic valve circumference | Mitral valve circ. | Tricuspid valve circ. |
|---|---|---|---|---|---|
| 1 | ++ | 1000 | 6.5 cm. | 12 cm. | 14 cm. |
| 2 | ++ | 740 | 8 cm. | 9.5 cm. | normal |
| 3 | ++ | 720 | 5.5 cm. | 10. cm. | 13 cm. |
| 4 | ++ | 708 | 4.5 cm. | 10. cm. | 13 cm. |
| 5 | ++ | 668 | "small" | | |
| 6 | ++ | 610 | "small" | 10. cm. | 11.5 cm. |
| 7 | ++ | 588 | 3. cm. | 9. cm. | 12.5 cm. |
| 8 | ++ | 565 | 9. cm. | 9.5 cm. | 12. cm. |
| 9 | ++ | 558 | "slit" | ? | ? |
| 10 | ++ | 553 | 5.5 cm. | 12.5 cm. | 13. cm. |
| 11 | ++ | 548 | "tip of little finger" | 10. cm. | 13.5 cm. |
| 12 | ++ | 547 | 8. cm. | 12. cm. | 13. cm. |
| 13 | ++ | 538 | 7.5 cm. | 12. cm. | 14. cm. |
| 14 | ++ | 475 | "small" | normal | normal |
| 15 | ++ | 473 | 4 × 6 mm. | ? | ? |
| 16 | ++ | 470 | 9. cm. | 11. cm. | 12.5 cm. |
| 17 | ++ | 456 | 4.5 cm. | 10. cm. | 13. cm. |
| 18 | ++ | 305 | 6. cm. | 9. cm. | 11. cm. |
| 19 | Slightly+ | 528 | 1.5 × 2.3 cm. | ? | ? |
| 20 | Slightly+ | 487 | 2 × 5 mm. | 1.8 × 2 | 3 × 3.5 |
| 21 | Not enlarged | 543 | 5. cm. | 9.5 cm. | 12.5 cm. |
| 22 | Not enlarged | 490 | 7.5 cm. | 11. cm. | normal |
| 23 | Not enlarged | 370 | 6.5 cm. | 11. cm. | 14. cm. |
| 24 | ? | 900 | 1. cm. | 10. cm. | 13. cm. |
| 25 | ? | 682 | 1.5 cm. | 9.5 cm. | 11.5 cm. |
| 26 | ? | 278 | ? | ? | ? |
| 27 | ? | 872 | "slit admitting closed blades of enterotone" | 10. cm. | 13.5 cm. |
| 28 | ? | 573<br>Average = 580 | 6.7 cm. | 10. cm. | 11. cm. |

TABLE 56.—AORTIC VALVE APERTURES IN AORTIC STENOSIS

| | |
|---|---|
| ? | 1 |
| "Small" | 5 |
| .4 × .6 cm. | 1 |
| 1. cm. | 1 |
| 1.5 cm. | 1 |
| 1.5 × 2.3 cm. | 1 |
| 2. × 5. cm. | 1 |
| 2. × 6. cm. | 1 |
| 3. cm. | 1 |
| 4.5 cm. | 2 |
| 5. cm. | 1 |
| 5.5 cm. | 2 |
| 6. cm. | 1 |
| 6.5 cm. | 2 |
| 6.7 cm. | 1 |
| 7.5 cm. | 2 |
| 8. cm. | 2 |
| 9. cm. | 2 |
| | 28 |

4. *Infarcts.*—The lungs contained infarctions in three cases, the liver in one, the kidney and spleen in one. This proportion of five out of 28 cases is somewhat smaller than that observed in the mitral group, as recorded in Table 42, page 69.

I have already mentioned the rather frequent association of *arteriosclerosis*, i.e., in sixteen out of 28 cases.

## SYMPTOMS

In Table 57 is shown the order in which symptoms were noted. There is nothing notable so far as I see about this showing or about the general incidence of symptoms (Table 58). They are the symptoms of uncompensated heart disease in general, and differ from those shown in the other groups considered in this book, only in that *precordial pain and angina are somewhat more frequent than in the other rheumatic types.* This is what we should expect from the greater average age of the patients in this group and from the preponderance of males. Obviously aortic stenosis when compensation fails, shows itself by dyspnea, cough, and cyanosis, like any other type of heart disease. It is somewhat notable, however, that *palpitation* (auricular fibrillation?) was noted so *infrequently in this group.*

The incidence of hydrothorax (1 in 8) is also somewhat less than in the mitral cases (1 in 2). On the other hand cyanosis is fully

as prominent as in any other group. I think it is very doubtful if anyone could guess, by symptoms alone, whether or not a given case was likely to turn out aortic stenosis. Only the age and sex would give one an inkling.

TABLE 57.—SYMPTOMS

| Symptoms | 1st sign | 2d sign | 3d sign |
|---|---|---|---|
| Dyspnea | 15 | 1 | 0 |
| Palpitation | 1 | 1 | 0 |
| Anginal pain* | 1 | 0 | 0 |
| Weakness | 0 | 0 | 1 |
| Dizziness | 1 | 0 | 0 |
| Cough | 2 | 0 | 0 |
| Tightness across chest | 0 | 0 | 1 |
| Edema | 2 | 2 | 0 |
| (of feet 1, legs 1, feet to chin 1, ? 1) | | | |
| Ascites | 1 | 0 | 0 |

* In 2 other cases angina appeared late in the course of the disease.

TABLE 58.—GENERAL INCIDENCE OF SYMPTOMS

| | |
|---|---|
| Total showing edema | 26 |
| Of lower extremities | 22 |
| (Of these, in addition, anasarca 1, edema of lids 1, of abd. wall 1) | |
| No record of place | 2 |
| Total showing cough | 20 |
| (cough with sputum, 16 cough with blood, 8) | |
| Cyanosis (slight 6, 1 lips only, 1 hands only) | 14 |
| Precordial pain (fairly clear angina 3) | 9 |
| Ascites (absent in 9, ? in 15) | 8 |
| Gastric symptoms | 8 |
| Hydrothorax (single 4, double 1) | 5 |
| Jaundice | 3 |

## PHYSICAL SIGNS

TABLE 59.—NUTRITION

| | |
|---|---|
| Well developed and nourished | 14 |
| Well developed, obese | 1 |
| Obese | 2 |
| Poorly developed and nourished | 3 |
| Fairly developed and nourished | 3 |
| Well developed, poorly nourished | 1 |
| Fairly developed, emaciated | 1 |
| Emaciated | 1 |
| Not recorded | 2 |
| | 28 |

Table 59 lists the data as regards nutrition and development of these patients, and calls for no special comment.

**Fever.**—Seventeen were afebrile and eleven febrile. Of these eleven cases, two died of pneumonia and may naturally be thus explained, one was associated with urinary sepsis, and one with acute endocarditis. One of the remaining seven was associated with a small auricular thrombus; in the other six fever is altogether unexplained.

TABLE 60.—LEUCOCYTES

| | |
|---|---|
| Under 6000 | 1 |
| 6000–9000 inclusive | 5 |
| 10,000–15,000 inclusive | 9 |
| Above 15,000 | 8 |
| No white count | 5 |
| | 28 |

**Leucocyte Count.**—Table 60 shows the figures in 28 cases. Correlating these with the febrile cases it appears that the highest counts (34,000, 30,000, 24,000) accompanied high fever due to streptococcus infection in all 3 cases. The more moderate counts were associated with pneumonia. For one count of 16,000 (temp. 98°–100°) no cause was found.

## CLINICAL DIAGNOSIS OF CARDIAC ENLARGEMENT

Referring again to Table 55, it appears that most of the notably enlarged hearts were recognized in life as greatly enlarged. On the other hand the same diagnosis was made on one heart which weighed

only 305 grams, and no note of enlargement is recorded in three cases weighing respectively 900, 872, and 682 grams. The note "slightly enlarged" occurs in the clinical record of cases which showed after death a cardiac weight of 528 grams and 487 grams respectively, and a definite note of "not enlarged" correlates rather poorly in three cases with cardiac weights of 543, 490 and 370. In general we got a fairly correct idea of the facts in twenty cases out of 28 cases, or about three-quarters. These facts are further shown in Table 61.

TABLE 61.—CARDIAC WEIGHTS IN 18 CASES OF AORTIC STENOSIS

18 cases showed "marked enlargement" clinically.

| | | |
|---|---|---|
| Of these, | 1 weighed | 305 grams (6 cm. aortic aperture) |
| | 4 weighed | 400–500 grams |
| | 7 weighed | 500–600 grams |
| | 2 weighed | 600–700 grams |
| | 3 weighed | 700–800 grams |
| | 0 weighed | 800–900 grams |
| | 1 weighed | 1000 grams |
| | 18 | |

2 showed slight enlargement clinically, weighing 487 and 528 grams respectively, with aortic apertures 2 × 5 cm. and 1.5 × 2.3 cm. respectively.

8 not recorded as enlarged clinically weighed the following:

| | |
|---|---|
| 278 grams | 573 grams |
| 370 grams | 682 grams |
| 490 grams | 872 grams |
| 543 grams | 900 grams |

**Cardiac Murmurs.**—I think it fair to assume that in all these cases there was some regurgitation of the blood stream as well as an obstruction to its passage from the heart. Yet in twelve out of 28 cases no diastolic murmur or other evidence of aortic regurgitation was obtained in life. The concomitant stenosis ordinarily prevents the appearance of marked peripheral vascular phenomena such as Corrigan and capillary pulse, and the pulse-pressures are not at all significant for the reason just given. The sixteen cases in which a diastolic murmur was looked for are listed in Table 62. It appears that the diastolic was widely distributed as regards its place of appearance and maximum intensity, as is apt to be the case in all types of aortic regurgitation.

TABLE 62.—DIASTOLIC MURMURS IN AORTIC STENOSIS

| | Present | Absent | Not recorded | Total |
|---|---|---|---|---|
| Diastolic at apex | 13 | 3 | 12 | 28 |
| 2 at apex alone. | | | | |
| 2 at apex and left sternal margin. | | | | |
| 5 at apex and over whole precordia. | | | | |
| 1 at apex aortic area, and left sternal margin. | | | | |
| 1 at apex, aortic area, and pulmonary area. | | | | |
| 1 at apex, aortic area and pulmonary area and left axilla. | | | | |
| 1 at apex and left axilla. | | | | |
| Diastolic at pulmonic area alone | 1 | | | |
| Diastolic at aortic area alone | 1 | | | |
| Diastolic in left axilla and back | 1 | | | |
| | 16 | | | |

TABLE 63.—SYSTOLIC MURMURS IN AORTIC STENOSIS

| | Present | Absent | Not recorded | Total |
|---|---|---|---|---|
| Systolic at apex | 22 | 3 | 3 | 28 |
| 4 at apex alone, except 1 in axilla. | | | | |
| 7 at apex and over whole precordia. | | | | |
| 4 at apex and pulmonic and aortic areas. | | | | |
| 5 at apex alone and aortic area. | | | | |
| 1 at apex and pulmonic area. | | | | |
| 10 show extension to other places: | | | | |
| 4 to axilla. | | | | |
| 2 to neck, 1 to clavicle. | | | | |
| 1 to axilla and back. | | | | |

Systolic murmurs were present in 22 cases and absent or not recorded in six. Their distribution and place of maximum intensity is shown in Table 63. In most of these cases the murmur was loudest at or near the orthodox aortic area, though four were very apt to lead to error in diagnosis because of their presence solely at or to the left of the cardiac apex instead of at the base of the heart. The extension of these murmurs to the neck, axilla, or back seems to me of no diagnostic or other significance, and merely means that in a considerable number of cases there were notably *loud* murmurs.

All loud murmurs are widely transmitted, whatever their place of production.

Presystolic murmurs at or near the apex were noted as present in six cases and absent in three. Knowing what I do of the routine of examination pursued in these cases, I think it fair to assume that such murmurs were absent in most of the unrecorded residuum of cases. So that we may say with tolerable accuracy that the "Austin Flint murmur" was here present in six out of 28 cases.

**Thrills.**—Table 64 sums our results on this point. But I am loath to believe that it represents the actual state of things in these cases. Unless one is definitely looking for aortic stenosis I know that we often fail to investigate the question of a thrill. So that I believe it may well have been present in many of the seventeen unrecorded cases.

TABLE 64.—THRILLS

| | |
|---|---|
| Systolic at base | 7 |
| Presystolic at apex | 1 |
| Presystolic at pulmonic area | 1 |
| Thrill absent | 2 |
| Not recorded | 17 |
| | 28 |

TABLE 65.—AORTIC 2ND SOUND

| | |
|---|---|
| Aortic 2d sound, absent | 9 |
| Aortic 2d sound, diminished | 2 |
| Aortic 2d sound, normal | 1 |
| Aortic 2d sound, accented | 1 |
| Not recorded | 15 |
| | 28 |

**Aortic Second Sound.**—From Table 65 it would appear that the aortic second sound was diminished or absent in only eleven of these 28 cases. In fact I believe more careful observation would have shown it to be faint or absent in a considerable number of those in which no record was made. It is worth noticing, however, that in one case the aortic second sound is recorded as definitely accentuated, a fact very difficult to reconcile with the known post-mortem findings, that is, with rigid aortic cusps. In two cases not included in this series I have faced this mystery before. So far as I see it definitely attacks the theory ordinarily held by physiologists, that the aortic second sound is due to the closure of the aortic valves and to this cause alone.

TABLE 66.—PULSE IN AORTIC STENOSIS

| | | |
|---|---|---|
| "Plateau" or "low volume and tension" | | 6 |
| "Corrigan" | with pistol shot 1 | 6 |
| | with capillary pulse 4 | |
| | with bounding pulse 1 | |
| Normal | | 10 |
| No record | | 6 |

**Pulse.**—The most notable fact about these pulse records as factors in diagnosis is the absence of any sign of "Corrigan pulse" in twenty-two out of 28 cases. This substantiates the usual belief that when aortic stenosis is associated with aortic regurgitation, as is the case in almost all non-syphilitic disease of this valve, the ordinary arterial phenomena of aortic regurgitation are neutralized or absent. On the other hand there are not many cases to sustain the classical belief in the flat-topped or plateau pulse as a frequent concomitant of aortic stenosis. We noticed it in only six out of 28 cases.

**Blood Pressure.**—The scanty observations available in our cases are shown in Table 67, which is, however, of interest because it proves that despite very considerable narrowing of the aortic valves, as for example in Necropsy 3320, a high systolic and diastolic pressure (presumably due to a complicating nephritis) may be present. In the other cases with still higher systolic pressures (Necropsies 2919, 2629) the valve stenosis is not obvious from the measurement of its circumference but was nevertheless stated to be considerable in the *post-mortem* record. In Necropsy 3928 one notes a pulse pressure of 86, which should correspond with a very tolerable degree of Corrigan pulse.

TABLE 67.—BLOOD PRESSURE IN AORTIC STENOSIS

| No. | Blood pressure | Age | Sex | Arterio-sclerosis | Valve circumference |
|---|---|---|---|---|---|
| 3928 | 168/82 | 48 | M | ..... | 7.5 cm. |
| 2919 | 210/? | 57 | M | ++ general | 8. cm. |
| 2629 | 210/?<br>150/? | 18 | M | ..... | 6. cm. |
| 3320 | 160/110 | 74 | M | ++ general | a slit<br>2 × 6 cm. |

TABLE 68.—ARTERIAL WALLS IN AORTIC STENOSIS

| | |
|---|---|
| Artery walls palpable | 1 |
| Artery walls palpable and calcareous | 1 |
| Artery walls tortuous | 1 |
| Artery walls tortuous and hard | 1 |
| Artery walls tortuous and thick | 1 |
| Artery walls thick | 6 |
| Artery walls soft | 1 |
| Artery walls just felt | 1 |
| Artery walls not felt | 2 |
| No record of their condition | 13 |
| | 28 |

**Arterial Walls.**—The condition of the artery walls as listed in Table 68 shows about the amount of arterial thickening that one would expect from the age of the patients and the amount of arteriosclerosis to be expected at that age. But it is decidedly in contrast with the condition of the artery walls in mitral disease, pure or complicated.

**Evidences of Chronic Passive Congestion.**—A good deal of the stasis present in these cases appears in the items recorded in Table 58. In Tables 69 and 70 we get some further corroboration of facts obviously to be expected.

TABLE 69.—CONDITION OF THE LIVER IN AORTIC STENOSIS

| | |
|---|---|
| Liver edge felt below ribs | 17 |
| Liver edge not felt | 9 |
| Liver edge not recorded | 2 |

TABLE 70.—CONDITION OF THE SPLEEN IN AORTIC STENOSIS

| | |
|---|---|
| Spleen enlarged | 2 |
| Spleen not felt | 8 |
| Spleen not recorded | 18 |

**The Blood.**—The condition of the leucocytes has already been explained. There was no notable abnormality about the red cells in any of these cases.

**Bacteriology.**—Streptococci were found *post-mortem* in the blood of five out of 28 cases, staphylococci in one, pneumococci in one, a total of seven, or one-quarter of the cases. Four of these positive blood cultures, all of them showing streptococci, were associated with the finding of acute endocarditis. I have already referred to these facts as tending to show that arteriosclerosis can hardly explain all of the facts in these cases.

**Urine.**—There is nothing worthy of note. Albumin was present in seventeen cases and absent in eleven. Fifteen showed casts. But

there was nothing herein to distinguish the cases of nephritis presently to be referred to from those of simple passive congestion.

**Complications.**—Obsolete tuberculosis was present in four cases. There was no case of active tuberculosis in this series. Chronic pericarditis was found in one case, acute pericarditis in one, and hyrdopericardium in four. Chronic pericarditis was also diagnosed during life in two other cases, in neither of which was the diagnosis verified. The same is true of one of the clinical diagnoses of pulmonary infarct. Nothing was found to correspond to it *post-mortem.*

*Nephritis* was present in ten cases out of 28, a surprisingly large proportion. It was of the arteriosclerotic type in four cases, of the glomerular type in four, and suppurative in two. Of the glomerular cases one was acute, two subacute, and one chronic. Some of the high blood pressures are doubtless associated with these facts.

*Jaundice* was present in three cases without any adequate explanation *post-mortem.* In one it was associated with high fever (104°) and a leucocytosis of 30,000; in the others with passive congestion.

*Angina pectoris.* Three cases out of 28 suffered from pain definitely of the anginoid type. In five others precordial pain was distressing but did not possess the characteristics distinctive of angina. Correlating these cases with the state of the coronary arteries and of the aortic arch, we find that there is no definite anatomical basis for the pain; in one case the patient was syphilitic (chronic interstitial orchitis) but with no aortitis or coronary disease.

TABLE 71.—ESTIMATED DURATION

| | |
|---|---|
| 1 year | 1 |
| 2– years | 3 |
| 4 years | 3 |
| 5 years | 3 |
| 6 years | 1 |
| 8 years | 1 |
| 10 years | 1 |
| Over 10 years | 2 |
| Several years | 1 |
| Doubtful | 12 |
| | 28 |

**Estimated Duration of the Disease.**—Table 71 lists the known facts in our 28 cases. It is of interest, as evidence of the long duration of symptoms in some cases, though it is hard, even in the doubtful cases, to picture the disease as having lasted ever since the age when rheumatic infections usually occur.

**Mode of Death.**—Pneumonia was present in four cases post-mortem, and pulmonary abscess in two. *Death was notably sudden and unexpected in six cases* though no special explanation was found for this *post-mortem*. In one case the patient died in the hospital corridor. As a rule, however, the death was preceded by the usual symptoms of passive congestion and stasis.

**Involvement of the Bundle of His.**—Necropsy 3424 showed well marked lesions in the interventricular septum including the bundle of His. During life this patient suffered typical attacks of angina pectoris but had nothing to suggest heart block.

## SUMMARY AND CONCLUSIONS

1. As in all types of aortic disease, the male sex strongly predominates, 25 out of 28 cases.

2. More than half the patients were beyond the fiftieth year when they first came for treatment at the hospital.

3. The cases appear to be of rheumatic origin, judging by the history and the *post-mortem* findings.

4. The degree of cardiac enlargement is greater on the average than that found with any single valve lesion except syphilitic aortitis with aortic regurgitation.

5. The symptoms are like those of other types of uncompensated valve lesions save that faintness, precordial pain and angina pectoris are more frequent.

6. In twelve of twenty-eight cases no diastolic murmur was heard in life though the valve lesions were such that some regurgitation seemed inevitable.

7. In six of twenty-eight cases a presystolic murmur (Austin Flint type) was heard at the apex.

8. In twenty-two of twenty-eight cases a systolic murmur was heard, though in four it was noted *only at the apex*. In eighteen it was loudest at the base of the heart, but not always on the right side.

9. As to a thrill at the base of the heart, one of the crucial points in the diagnosis of aortic stenosis, our records are defective in seventeen out of twenty-eight cases. In eight cases a basic thrill is recorded. In two it is definitely stated to be absent. In another only an apical presystolic thrill is mentioned. From these very meagre data we may say that the characteristic thrill was present in four-fifths of the cases in which a definite record was made. But the cases are too few to have value.

10. Another vital point in the diagnosis of aortic stenosis is the diminution or absence of the aortic second sound. Here again

unfortunately our records are worthless in fifteen out of twenty-eight cases. Out of thirteen cases with a definite record, nine showed absence and two more showed diminution of the aortic second sound. In one case it is noted as normal and in one *accented*,—a strange phenomenon with a rigidly calcified aortic valve!

11. The pulse is chiefly notable for the absence of any "Corrigan" shape in sixteen out of twenty-two cases definitely recorded. Since we must suppose that regurgitation as well as stenosis is present in these cases, the absence of the collapsing pulse is a point favoring the diagnosis of stenosis.

In six cases the classical flat-topped or plateau pulse-wave is recorded.

On the other hand it is notable that a "Corrigan pulse" was definitely recorded in six cases and was accompanied by a capillary pulsation in four and by a "pistol-shot sound" over the brachial in one.

Here we may conjecture that the regurgitation was more marked than the stenosis, but the explanation is rather lame.

12. It is a notable fact that despite an aortic stenosis which reduced the aortic opening to a mere slit (Necropsy 3320) a blood pressure of 160/110 is recorded. It would seem that such a stenosis would make it impossible for such a blood pressure to be maintained. This case seems to prove the contrary.

13. Nephritis of some type was found at necropsy in ten out of twenty-eight, a surprisingly large proportion. One case was "acute," two "subacute," the rest chronic.

14. Three patients suffered from angina pectoris. In one of these, a negro of forty-nine, a chronic interstitial orchitis and chronic perisplenitis were found, but no aortitis or coronary narrowing. (The connection of angina with syphilis but not always with syphilitic aortitis is noted in the chapter on Angina Pectoris.) In the other two cases there was no anatomical basis for the pain.

15. In only 1 case in the 28 was the aortic stenosis associated with an old pericarditis. This is in marked contrast with all other valve lesions of the rheumatic type, which frequently are associated with chronic pericarditis, 23 in 198 or 1 in 9—3 times as often as with aortic stenosis.

16. The average duration of the disease is probably greater than in any other valve lesion, as the age at death is greater and the supposed etiology (rheumatism) points to an infection of the valve early in life.

17. Death was notably sudden and unexpected in six cases. One patient died in the hospital corridor. In most patients, however, the ordinary congestive symptoms preceded death.

## AORTIC STENOSIS. ILLUSTRATIVE CASES

1. 2558. Slit-like orifice, pulmonary arteriosclerosis. Death from pneumonia.

2. 2238. Typical physical signs. Mouths of coronaries narrowed. Fibrous myocarditis.

3. 2603. Angina pectoris clinically prominent. Moderate coronary sclerosis.

4. 4655. Trigeminal neuralgia a major complaint. Otherwise typical.

5. 3409. Boy aet. 6. Slight acute, marked chronic endocarditis.

6. 3715. Man aet. 68. Death with pulmonary and renal abscesses.

### Necropsy 2558

An English janitor of sixty-three entered March 7, 1910. He had had a very severe case of scarlet fever in boyhood. Otherwise he had been very well. He had had gonorrhea twice when young.

Five years ago, when walking in a parade, he was suddenly seized with severe dyspnea and cough with frothy sputum. Ever since then he had had considerable dyspnea on exertion. For several years he has urinated six or eight times at night. Four weeks ago the dyspnea became much more severe, and he developed a cough and orthopnea. Two days ago these symptoms became much worse, and his legs suddenly became edematous. A few hours ago he began to raise red muco-purulent sputum.

Physical examination showed a ruddy-faced old man breathing very rapidly, with marked tracheal rattles. The apex impulse of the heart was in the sixth space, not very forcible, 15.5 cm. from midsternum, 3 cm. outside the nipple. The right border was at the sternal margin. At the aortic area a loud, rough systolic murmur accompanied by a thrill. The aortic second was not heard. At the apex a systolic murmur of higher pitch and different quality. The pulses were of very small volume and low tension, irregular, with many extra systoles. At the apex there were 96 beats, at the wrists 60. The pulse was feeble. The lungs were hyperresonant and full of sonorous râles. Expiration was very prolonged, with loud wheezes and crackles. At the bases some dullness, with great showers of fine crackles. The abdomen was obese, thick. The

liver dullness extended from the sixth rib to three fingers below the costal margin, where an indefinite edge was felt. A tremendous inguinal hernia was found. The genitals and pupils were normal. There was very marked brawny edema of the legs and ankles. The fundi showed no abnormalities.

Temperature 97.3 to 103.8 to 99.7, the pulse 71 to 131, respirations 23 to 34. The amount of urine was 31 to 21 ounces. The specific gravity was 1030 to 1025. It was pink at one of two examinations, showed albumin and granular and hyalin casts at both, many leucocytes at the first. The hemoglobin was 90%. There were 24,000 leucocytes and 90% of polynuclears. Blood culture was negative. The sputum was at first brick red, slightly foul; within twenty-four hours green, with some odor. No tubercle bacilli were found.

The heart action became more rapid, and at the same time the systolic murmur became less marked. On the evening of March 7 expiration became very difficult, respiration periodic, and the patient became comatose and almost pulseless. He responded well to medication and had a fairly comfortable night. The evening of March 9 he had the same symptoms. The veins of the neck distended upon pressure over the liver, which was definitely enlarged but not pulsating. After the withdrawal of 18 ounces of blood he was more comfortable. He afterwards spent most of the time in a chair, with much respiratory difficulty and a loose cough. March 11 the pulse suddenly failed, and he died.

*Clinical Diagnosis.*—Aortic stenosis.

Broken compensation.

Bronchopneumonia?

Emphysema and chronic bronchitis.

Chronic passive congestion.

Edema.

*Dr. Richard C. Cabot's Diagnosis.*—Aortic stenosis and regurgitation.

Chronic passive congestion of the liver.

Terminal infection.

*Anatomical Diagnosis.*—Chronic endocarditis of the aortic valve; stenosis.

Arteriosclerosis of the aorta, the coronary arteries, and the pulmonary artery and its branches.

Hypertrophy and dilatation of the heart.

Bronchopneumonia, right lung.

Focal pneumonia, superior lobe of left lung.

Purulent bronchitis.

Fibrinous pleuritis, left.
Chronic passive congestion, general.
Edema of the lower extremities.
Slight fibrous endocarditis of the mitral valve.
Chronic pleuritis, right.
Right inguinal hernia.

DR. OSCAR RICHARDSON: We were not permitted to examine the head.

There was edema of the lower extremities. We found no fluid in the peritoneal cavity. He had a right inguinal hernia. The diaphragm in this case was at the fifth rib on the right and the fifth interspace on the left. There was no fluid in the right pleural cavity and only a slight amount in the left, with a few fibrinous shreds,—chronic pleuritis on the right side and fibrinous pleuritis, left. The trachea and bronchi contained considerable muco-pus, a purulent bronchitis. In the right lung there was bronchopneumonia and in the left lung a few focal areas of pneumonia. In addition to that, chronic passive congestion.

In a passively congested lung it is sometimes very difficult to tell whether there is any pneumonia present or not. I remember a case in which I returned lobar pneumonia and it was chronic passive congestion. One has to be careful. In this particular case there were definite islands of consolidation and purulent bronchitis, and the picture was quite evident. Microscopic evidence showed that there was bronchopneumonia and a few scattered areas of focal pneumonia.

The pulmonary artery in this case showed considerable sclerosis. It is not very common. We may get a few yellowish plaques in any case at this age; but this was definite sclerosis. A curious thing was that at the junction where the ascending portion of the thoracic aorta and the pulmonary artery come together there was a fibrocalcareous plaque which extended through the conjoined walls so that we had a rough surface on the intima of the pulmonary artery and along the intima of the aorta, but the wall was essentially intact.

The heart weighed 548 grams, an enlarged heart. (Normally 280 to 300 grams.) The right ventricle wall was six mm. thick and the left thirteen mm. The columnae were hypertrophied. The mitral valve measured ten cm. and the aortic hardly anything, a slit-like orifice admitting the tip of the little finger. (See Fig. 39.) The tricuspid was thirteen and a half cm., the pulmonary nine. We cannot always believe what we see in pathological reports as to the amount of sclerosis. In this case there was moderate sclerosis of

the aorta, considerable of the pulmonary artery, and only a little of the coronaries. There was of course frank aortic stenosis of the rheumatic type. In this case we made very sure that the bundle of His was not impinged upon by the process in the aortic valve.



FIG. 39.—Necropsy 2558. Plate I.—Heart in aortic stenosis and regurgitation with arteriosclerosis of the aorta, the coronary arteries and the pulmonary artery and its branches. The heart weighed 548 grams. Shows the fish-mouth aortic valve—aortic stenosis. A little less than three-quarters actual size. (Photograph by John W. Barry. Dr. Oscar Richardson.)

The liver showed the nutmeg markings. Of course that is stasis, the blood coming back through the hepatic veins to the central veins, then into the portal system, and forming a network of distended vessels containing brown-red blood. The liver cells may show some

fatty degeneration or become atrophied and show a pale grayish brown color, and the whole thing looks like the cross section of a nutmeg. It is a convenient term for chronic passive congestion.

The spleen weighed 116 grams (normally 80 to 180), and curiously enough in this case the tissue was a little mushy, possibly on account

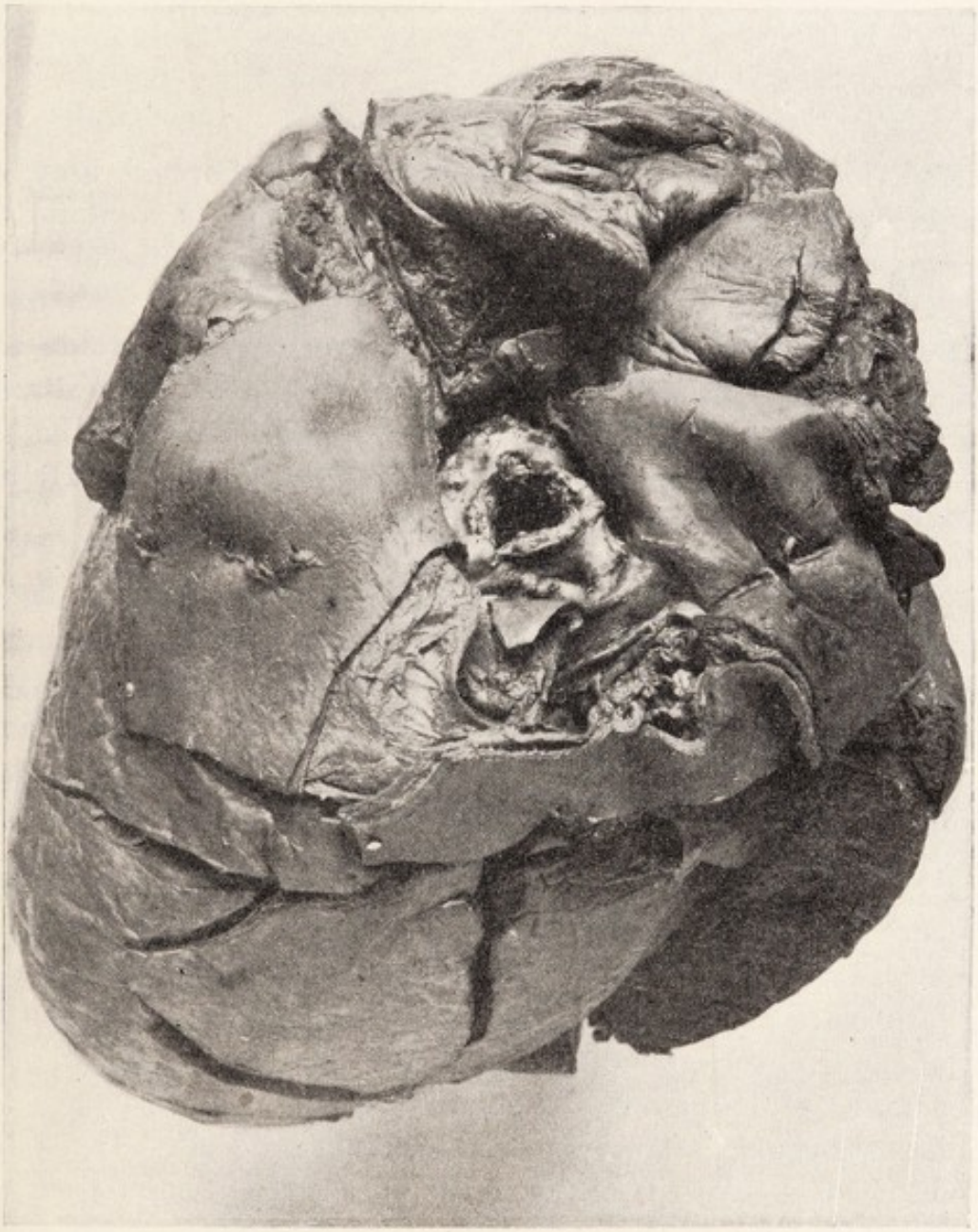


FIG. 40.—Necropsy 2558. Another illustration of the same condition. (Photograph by John W. Barry. Dr. Oscar Richardson.)

of the infection. It was rather small for a spleen in a case of chronic passive congestion.

The kidneys weighed 275 grams (normally 200 to 400), and were in good condition. They showed of course some chronic passive

congestion. I notice that the kidneys seem to stand wear and tear about as well as any organ in the body. The genito-urinary tract was out of the picture. The gastro-intestinal tract showed chronic passive congestion.

There was no growth from the heart blood. But the microscope confirmed the gross picture of the pneumonia in the lungs and the negative appearance of the kidneys.

### Necropsy 2238

An American night watchman of sixty-two entered May 14. His father died of "shock," his mother of cancer. Except for the diseases of childhood he had practically never been ill before. He occasionally drank a glass of beer.

December 13 he stopped work because of a heavy "cold" and periods of dyspnea with dizziness which prevented any exertion. The attacks lasted five minutes or less. Since December he had slept in a chair. Lying flat always brought on an attack. He had

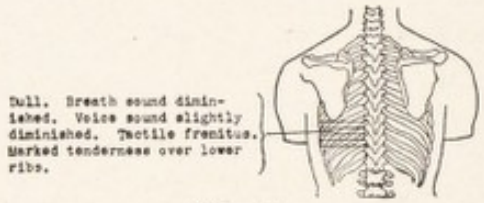



FIG. 41.

troublesome cough, constipation, and urination once or twice at night.

Examination showed a senile man with slight cyanosis and evidence of loss of weight. Only a few carious teeth remained. There was marked pyorrhea. The apex impulse of the heart was in the fifth space 12.5 cm. to the left of the midsternum, 2 cm. outside the nipple line, corresponding with the left border of dullness. The right border of dullness was at the right edge of the sternum. The acion was irregular. The first sound was long and loud. The second sounds were not heard. Starting in the third left interspace and transmitted down to the apex, entirely replacing the second sound, was a blowing diastolic murmur. Over the whole precordia was a loud systolic murmur transmitted to the vessels of the neck, with a corresponding thrill. At the apex and transmitted to the axilla and back was a blowing musical systolic murmur. To the right of the sternum in the fourth interspace and transmitted to the right was a systolic murmur of a higher pitch. The pulses were slightly irregu-

lar, of fair tension and long sustained plateau type. The arteries were palpable and tortuous. The systolic blood pressure was 135. The lung signs were as shown in Fig. 41. The abdomen was tympanitic throughout except in the flanks. The upper border of the liver was obscured by chest dullness. There was slight edema of the genitals and marked soft edema of the legs and lower back. The pupils and reflexes were normal.

The temperature was 96.5° to 99.4°, with one rise to 101.4° two days before death. The pulse was 55 to 94. The respirations were 20 to 30. The output of urine was 5 to 61 ounces, the specific gravity 1.010 to 1.030. The hemoglobin was 85%, the leucocytes 8400.

By May 19 the rough systolic murmur had extended from the base more toward the apex. The systolic murmur at the apex was blowing and higher in pitch. There was a systolic thrill in the second right interspace. The edema diminished under a course of magnesium sulphate. The patient lost strength. He dropped asleep for a few minutes at a time while sitting in his chair, and slept more and more. May 25 digitalis was begun. In three days the heart sounds were slightly stronger and the amount of urine increasing. With the rise in temperature June 2 he had a large pustule which cleared up quickly after an injection of staphylococcus vaccine. June 4 he seemed stronger and was discharged relieved, weighing 122 pounds, 22 pounds less than at admission.

After leaving the hospital he did no work, kept rather quiet and got along well until the following February, when the dyspnea became much worse. Two weeks later he had cough with yellowish sputum. He always slept in a chair on account of orthopnea. His bowels were very constipated. His urine was rather scanty. He passed it twice at night. There was much edema of the legs. For two months before his readmission, March 10, he had been taking strophanthus.

Upon examination he weighed 132½ pounds. The mucous membranes were slightly pale. The apex impulse and the left border of the heart were in the fifth space 2 cm. outside the nipple line, 12.8 cm. to the left of midsternum. The right border was just outside the right sternal margin. The action was irregular in force and rhythm, the sounds indistinct, the pulmonic second sound not strong, the aortic second absent. A coarse discordant musical systolic murmur was heard at the apex, transmitted to the axilla. At the base was a rough systolic murmur, not transmitted. Along the left

border of the sternum were a low systolic and a low diastolic. There was a systolic thrill in the third right interspace close to the sternum. The pulses were irregular, corresponding to the apex beat, of small volume and low tension. The blood pressure is not recorded. There was faint lateral excursion of the brachials. There were medium moist râles scattered throughout both lungs, and dullness with diminished respiration and fremitus at both bases posteriorly. The liver dullness extended from the fifth rib to three inches below the costal margin. The edge was not distinctly felt. There was shifting dullness in the flanks. The feet were cold and livid. The skin over the lower legs was reddened and wrinkled.

The temperature was 95.8° to 99°, the pulse 80 to 103, the respiration 20 to 31. The specific gravity of the urine was 1.011 to 1.019; otherwise the urine was negative. The blood was normal.

The patient made marked improvement with rest in bed, digitalis and purging. By March 21 he was about the ward all day, though a little weak and with a rasping cough. The heart sounds were slow and strong. The digitalis was omitted. He continued to do well without it, though he *felt a little faint when he first got up* or on sudden exertion. March 31 he was discharged relieved, weighing 119½ pounds.

He was fairly comfortable until November 10, was outdoors daily, and walked at times. He had more or less constant edema of the legs, when prone marked dyspnea, and some cough with whitish sputum. During the week before his readmission to the hospital November 17 the dyspnea was very marked, he slept poorly, passed much less urine, and felt weak.

Upon examination he was emaciated. The impulse of the heart was best felt and its sounds best heard in the fifth space, nipple line, 11 cm. to the left of midsternum. The right border was at the right border of the sternum. The sounds were entirely replaced by murmurs. A systolic thrill was felt all over the precordium, best in the second space just to the right of the sternum. A harsh systolic murmur, at times musical, was heard all over the precordium, loudest at the aortic area, transmitted into the neck, axilla and back. Both second sounds were replaced by soft diastolic murmurs, loudest over the aortic area. The pulses were irregular in force and rhythm. The systolic blood pressure was 90. The arteries were calcareous. The lungs showed dullness, diminished breathing and voice sounds and a few fine crackling râles on inspiration at both bases, at the side and back. The liver edge was felt 7.5 cm. below the right costal

margin. There was slight edema of the ankles and over the shins, and marked discoloration and pigmentation of the skin over the feet and the lower half of the legs.

The temperature was 95.8° to 97.8°, the pulse 74 to 90, the respiration 20 to 32. The specific gravity of the urine was 1.019. The sediment showed an occasional hyalin cast. The hemoglobin was 90%, the leucocytes 17,000. There was polynuclear leucocytosis.

The patient seemed fairly comfortable except for a good deal of dyspnea. Early in the morning of the 18th he had a sudden attack of dyspnea and became pulseless. In an hour he died.

*Clinical Diagnosis (from Hospital Record).*—(Probably copied from the necropsy record.) Aortic stenosis with regurgitation and broken compensation.

Arteriosclerosis.

Cardiac hypertrophy and dilatation.

Chronic adhesive pleuritis, left.

Encapsulated hydrothorax.

*Dr. Richard C. Cabot's Diagnosis.*—Aortic stenosis and regurgitation, rheumatic type.

Chronic endocarditis of the mitral valve (with some stenosis?)

Hypertrophy and dilatation of the heart.

Arteriosclerosis.

Edema of the lungs.

Ascites.

Anasarca.

Passive congestion of all the organs.

Streptococcus septicemia.

Acute pericarditis?

*Anatomical Diagnosis.*—Fibrocalcareous endocarditis of the aortic valve with stenosis.

Hypertrophy and dilatation of the heart.

Chronic interstitial myocarditis.

Arteriosclerosis.

Slight hydropericardium.

Double hydrothorax.

Compression atelectasis of the lower lobes of the lungs.

Slight chronic passive congestion.

Chronic pleuritis.

Cholelithiasis.

Old infarct of the kidney.

DR. RICHARDSON: The original anatomical diagnosis, chronic endocarditis of the aortic valve with stenosis, was replaced later by "fibrocalcareous endocarditis of the aortic valve with stenosis," because they did not wish to commit themselves. Now I think that the original diagnosis was right. I believe it was rheumatic.

They never say "regurgitation" in these reports. That is assumed, since if he had stenosis he had regurgitation. All they put down is what they can see, stenosis.

An interesting thing is an old infarct in one of the kidneys. It dates back to the point of time, probably, which Dr. Cabot referred to saying he thought there was an acute on top of a chronic condition. The infarct bears that out. It is now an old infarct. There was no acute endocarditis in this case at the time of necropsy. The other valves were negative.

The case is a pure case of chronic endocarditis at the aortic valve, rheumatic in origin. He also had some arteriosclerosis, which is to be expected at sixty-five years.

An interesting thing in regard to the aortic valve in this case is that this old material was piled up in an irregular stony mass. The upper part encroached on the sinuses of Valsalva and further upon the orifices of the coronary arteries, partially occluding them, though not altogether. As a result we find scattered through the heart areas of chronic interstitial myocarditis. One would naturally say, How about his coronary arteries? They were pretty good except that the orifices where the blood flows into them were decreased to such an extent as to interfere considerably with the blood supply to the myocardium.

The hydrothorax was so extensive as to cause compression atelectasis of the lungs.

The chronic passive congestion, in this as in many other aortic cases, does not seem to be so extensive as in the mitral cases.

Another thing of interest is that he had cholelithiasis. There were nine stones, but the gall-bladder and the bile ducts were negative.

The history says, "Except for the diseases of childhood he had never been ill before." When he came here he was at the end of trouble which occurred years ago, and the reason for not calling it arteriosclerosis is because the arteriosclerotic changes were far from the aortic valves. There were whole patches of good aorta. If one were going to demonstrate arteriosclerotic changes in the aortic cusps so extensive as this, he would have quite a job on hand if he had no

arteriosclerosis in that region. Although arteriosclerosis may be selective it does not select in such an accumulation of fibrocalcareous material on the cusps and extending well down into the myocardium as well as up into the sinuses of Valsalva. It is not a process that erects large irregular fused masses of fibrocalcareous material on the cusps.

I noted carefully its relation to the bundle of His. It approached but did not quite impinge upon the bundle.

DR. CABOT: We do not need to suppose a lesion of the bundle in order to account for the arrhythmia. The only thing tied up to the bundle is heart block.

**Necropsy 2603**

An American electrical laborer of forty-nine entered July 1. His mother died of tuberculosis when he was twenty-nine. One son died at twenty-three of "heart trouble." The patient had gonorrhea at seventeen and tertian malaria at twenty-five. At eighteen, thirty, forty and firty-five he had attacks of rheumatic fever. Practically all the joints were affected at some time, but chiefly the knees and ankles. The attacks lasted from a few weeks to two or three months. For fifteen or twenty years he had had slight dyspnea on exertion, not increasing.

While convalescent from the last attack of rheumatism four years before admission he began to have attacks of sharp pain starting in the left shoulder and shooting down the extensor surface of the arm to the elbow. These attacks came three times a day or oftener and lasted three or four minutes, being at times severe enough to make him cry out. "Dull soreness" was practically constant. At about the same time he began to have slight soreness over the precordia with the attacks. For three years he had had a cough with considerable "green" sputum, bloody on three occasions. For two or three weeks he had had attacks of sharp pain over the precordia lasting three to five minutes. This pain did not radiate from the precordia to the arm, but occasionally did from the arm to the precordia, although the attacks were often independent. His legs had been swollen for a week.

Examination showed a rather obese man weighing 186 pounds, orthopneic, with pale skin and mucosae and slightly cyanotic lips. The apex impulse of the heart was in the sixth space 14 cm. to the left of midsternum, 3.5 cm. outside the nipple line, corresponding with the left border of dullness. The right border was 2.5 cm. to the right of midsternum. The action was slightly irregular in

force and rhythm. No second sound was audible except in the pulmonic area, where it was faint and followed by a short diastolic whiff. At the apex the first sound was rather forcible, partly replaced and followed by a soft blowing systolic murmur transmitted to the axilla and toward the base. In the aortic area a loud, harsh, rough, rumbling, systolic murmur was heard, transmitted to the neck and toward the apex and heard over the carotids on both sides. At times a faint thrill was felt in the second right space. The pulses were equal, slightly irregular in rhythm and force, of good volume, slightly increased tension, suggestive of plateau. The artery walls were palpable, a little thickened. The lungs showed numerous fine and medium moist râles at both bases, with occasional moist râles; hyperresonance throughout. The abdomen was very tympanitic and was held rather rigid. There was an epigastric hernia the size of a peach, with impulse on cough, half-way between the ensiform and the navel. There was dullness, not shifting, in the flanks. The liver dullness extended from the sixth rib to the costal margin. There was moderate soft edema over the sacrum and considerable edema of the lower legs, ankles and feet. The pupils were very slightly irregular, equal. Their reactions and the other reflexes were normal.

The temperature was 97° to 98.6°, the pulse 70 to 90, the respirations 18 to 22. The output of urine was 10 to 40 ounces, the specific gravity 1.015. There was a slight trace of albumin at all of three examinations, many hyalin and granular casts at two, a few pus and red cells at one. The hemoglobin was 80%, the leucocytes 10,000, the smear normal. A Wassermann was very slightly positive.

Under treatment the edema decreased, the heart grew steadier and more regular, and the dyspnea was relieved. Compensation being fairly well established, on July 15 he was discharged.

After leaving the hospital he was able to move slowly about the house and to go out a few times with care. Slight exertion caused cough and pain in the region of the heart and the left shoulder and arm. He usually had several attacks of this pain during the day. He urinated at night rarely.

February 7 he returned because of weakness and increasing frequency and severity of pain. He now weighed 166 pounds.

Examination showed no cyanosis. A forcible, thrusting apical impulse was seen and felt in the sixth space. The left border of dullness was 14 cm. from midsternum, 3.5 cm. outside the nipple line. The right border as 2.5 cm. to the right. The supracardiac dullness

was 8 cm. There was a loud rough systolic murmur, accompanied by a rough thrill, all over the precordia, loudest in the aortic area, transmitted to the neck and axilla, entirely replacing the first sound. No second sounds were heard at the base. The action was slow, with many well marked extrasystoles. The artery walls were not sclerotic. The systolic blood pressure was 110 to 118. The lungs showed slight dullness with diminished breathing and occasional râles at the extreme bases. There was shifting dullness in the flanks. The liver dullness extended from the sixth rib to a handbreadth below the costal margin, where an indefinite edge was felt with a rounded mass the size of an egg on it in the nipple line. There was some tenderness over the liver; no pulsation. The genitals and extremities were normal.

The temperature was 97° to 99.8°, the pulse 70 to 105, the respiration 17 to 24. The output of urine was 20 to 50 ounces, the specific gravity 1.016 to 1.019. There was a slight trace to the very slightest possible trace of albumin at all of three examinations, no sugar, many hyalin casts with small red cells attached, occasional fine granular casts and red blood corpuscles at one, rare hyalin casts at one. The hemoglobin was 90%, the leucocytes 4000, the polynuclears 75%.

In the hospital the patient was able to lie almost flat in bed. He had several attacks of mild cardiac pain relieved in a few minutes by nitrites. On February 13 he had a chill. The next day he had pain and moderate tenderness in his knees and ankles, redness and swelling in the right ankle only. This was relieved by aspirin. By February 20 the joints were entirely clear. He had no further attacks of cardiac pain, and was discharged relieved February 22.

He was up and about feeling fairly well until April 25, when his cough began to increase in severity and he raised considerable frothy sputum. He became more dyspneic and orthopenic. His legs swelled. April 28 he had pain across the upper abdomen, relieved before his return to the hospital May 2.

Examination was as before except for the points noted. He had occasional hard dry cough during which he became cyanotic. The apex impulse of the heart was not seen or felt. The percussion borders were 11 cm. to the left, 4 cm. to the right, 8 cm. in the supracardiac region. The whole precordia moved with systole. A short systolic thrill was felt in the second right space, stronger during expiration. The veins of the neck were full but did not pulsate. At the apex a systolic murmur replaced the first sound, transmitted to the axilla and heard all over the precordia. As the

base was approached a harsh rough systolic was heard, loudest over the aortic area, transmitted to the vessels of the neck. A faint diastolic murmur was heard all over the heart. The pulmonic second sound was faint; comparison with the aortic second was not possible. The pulses were of the plateau type, occasionally irregular. The artery walls were thickened. The systolic blood pressure was 100. The lungs showed equal expansion and good resonance throughout. There were normal breath sounds, a few inspiratory squeaks, and dullness below the angles of the scapulae posteriorly, with fine moist râles at both bases. The abdomen showed no masses or tenderness. The liver dullness extended from the fifth space to the costal margin. The edge was not felt. The knee-jerks were equal, lively. There was slight soft edema of the legs and ankles.

The temperature was 98.6°, the pulse 85, the respiration 22. The amount of urine is not recorded. The specific gravity was 1.017. There was ½% of albumin, many hyalin and granular casts, some with cells and blood attached, some laden with fat, a few free cells. The hemoglobin was 85%, the leucocytes 7000, the polynuclears 72%.

The patient voluntarily walked upstairs, was put to bed and given a quarter grain of morphia. He seemed fairly comfortable except for occasional attacks of vomiting. In one of these attacks the morning after admission he suddenly fell back and died.

*Clinical Diagnosis (from Hospital Record).*—Aortic stenosis.

Slight aortic regurgitation.

Hydropericardium.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the aortic valve with stenosis and regurgitation.

Possibly slight endocarditis of the mitral valve.

Hypertrophy and dilatation of the heart.

Chronic passive congestion of all the organs.

Possibly slight acute glomerulo-nephritis.

*Anatomical Diagnosis.*—Chronic endocarditis of the aortic valve. Stenosis.

Arteriosclerosis of the coronary arteries.

Fibrous myocarditis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Hydropericardium.

Incarcerated hernia, peritoneal fat tissue of the anterior abdominal wall.

DR. WRIGHT: This was a case of aortic stenosis with a very marked "fish-mouth" aortic valve, the valve fibrocalcareous, thickened, fused, and forming a truncated cone. It was a typical chronic aortic stenosis. The mitral valve showed no evidence of primary affection, but there was some extension of the fibrous process to it.

There was no arteriosclerosis of the aorta to speak of, but it was well marked in the coronaries, especially the descending branch of the left, with fibrosis of the myocardium, so that he had an excuse for anginoid pain. This fibrosis was not obvious or gross. The heart was very much hypertrophied.

There was no other significant or important lesion beyond chronic passive congestion.

The kidneys grossly were negative. No note was made on the microscopic examination. This is one of the few cases where it has been omitted.

### Necropsy 4655

An American masseuse of fifty-four entered November 5, 1918, for relief of trifacial neuralgia. Her father and mother died of consumption, and many relatives were tuberculous. She had measles, diphtheria, scarlet fever and whooping cough in childhood, and frequent mild sore throats. She had been deaf in the left ear since the scarlet fever and had had occasional abscesses in both ears. She had not been really well since a very mild attack of influenza at fifty-one. She passed the menopause the following year. For two years she had had marked pyrosis and gas, ending with the onset of the present illness and accompanied by severe dyspnea, cyanosis and stabbing precordial pain half an hour after meals, relieved by belching or passage of gas. She had lost fifty-eight pounds (230 to 172) in two years, probably because of lack of proper food.

Beginning six months before admission she had had increasingly frequent and severe attacks of pain in the right side of the face over the malar bone. Vomiting of food seemed to be an occasional cause of the attacks.

Upon examination the apex impulse of the heart was not found. The percussion measurements were as shown in Fig. 42. The sounds were distant and of fair quality, the action slow. There was a rough loud musical systolic murmur at the apex, but heard best at the base over the aortic area, where a diastolic was heard. The pulses were of fair volume and tension. The blood pressure was 100/70 to 100/65.

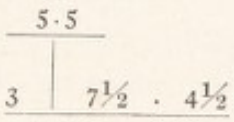



FIG. 42.—Measurements by percussion.

The abdomen was negative except for a possible palpable liver edge at the costal margin. Genital, pelvic and rectal examinations were not made. The extremities and the pupils were normal, the knee-jerks sluggish.

The temperature was 97.4° to 99.2°, the pulse 69 to 94, the respiration 16 to 28. The amount of urine was normal when recorded, the specific gravity 1.024 to 1.032. The urine was alkaline at one of six examinations, cloudy at two and showed leucocytes at two. The renal function was 40%. The blood was normal. A Wassermann was negative. The stools were negative. X-ray showed the heart measurements as shown in Fig. 43. There was a pus pocket from a bicuspid supporting extensive bridge work and apparently communicating with the antrum, which was dull.

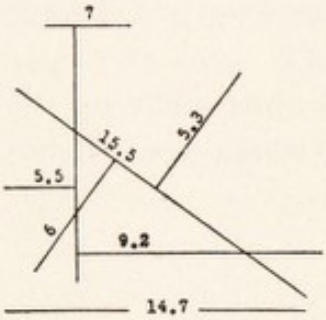


FIG. 43.—Measurements by X-ray.

November 20 the entire bridge work was removed and the abscessed tooth roots were curetted. The pain became less frequent and the patient had good nights until November 30, when severe paroxysms of right supraorbital pain started.

December 3 under ether and oxygen through a small incision in the right eyebrow about 2 cm. of the supraorbital nerve was evulsed.

For the next four years the patient remained in a general run-down nervous condition, taking care of an insane aunt, after whose death she was considerably better. Eight weeks before her readmission she found that the area of anesthesia, which had gradually grown less, had completely disappeared. Soon she had sharp stabbing pain in the right eyebrow very similar to the pain at her first entry, radiating out to the temple and to the top of her head like a red-hot spray, the attacks coming twenty times a week. A week before readmission she had all her upper teeth removed with only partial relief.

On examination September 22, 1922, she appeared old for her age. There was evidence of loss of weight. The apex impulse of the heart was in the fifth space well outside the nipple line. There was questionable enlargement at the base. (The measurements are not recorded.) The sounds were of poor quality or replaced by a prolonged soft blowing systolic murmur heard all over the precordia and in the axilla, also a short diastolic murmur at the aortic area. The blood pressure was 110/90. The lungs, abdomen, genitals, extremities, pupils and reflexes were not remarkable.

The temperature was 97° to 101.1°, the pulse 71 to 91, the respiration normal. The amount of urine is not recorded. The urine was cloudy at one of two examinations, the specific gravity 1.025. There was a very slight trace of albumin once. The sediment showed rare leucocytes at both examinations. X-ray showed the left antrum distinctly less radiant than the right. The outline was practically obliterated. The frontal sinus on the left was smaller than that on the right, but showed no marked change in density. Plates of the mastoid region showed no definite pathology.

October 2 under gas and ether careful search for the nerve was made but no definite evidence of it found. Several fragments of tissue looked suspicious and were excised. Pathological examination showed medullated nerve bundles surrounded by fibrous tissue. The patient was discharged October 7.

Very little relief followed the second operation. She continued to have attacks of shooting pain lasting two or three minutes, sometimes none for days, sometimes throughout one or more days. Talking or eating or any movement of the face tended to bring on the attacks. During the attack the eyelid closed and sharp shooting pains passed through it, sometimes making her cry out, always making tears come, with accompanying relief of symptoms.

In October, 1922, after being hurt in a train wreck she began to have pains and soreness of the chest, with extreme dyspnea and orthopnea. She gradually improved after ten months. After the accident she had occasional attacks of palpitation. When she lay on her left side she soon became uncomfortable, felt her heart beating forcibly against her ribs, and had some sense of being smothered. In May, 1923, she had edema of the legs for three weeks, during which time she was in a hospital. In December for about ten days she had some urinary retention, dark urine, and her doctor told her 6% albumin. She urinated twice at night. December 23 her legs became swollen and itched. By the middle of January, 1924, she was able to breathe comfortably lying down, but had dyspnea on exertion. In five years she lost about 80 pounds, perhaps in part by dieting.

The hospital in which she was treated from the 14th to the 26th of May, 1923, reported: "Heart enlarged to the left. Murmur heard at aortic and mitral areas. Sounds weak. Slight edema of the ankles. Dyspnea and cyanosis on slight exertion. Blood pressure 140/92. Arteries thickened. During her stay in the hospital she was very short of breath and quite purple on the least

exertion. Had several severe attacks when pulse became imperceptible and body very cyanotic. Responded well to stimulation at these times. The blood pressure came down to 112/94."

At her third admission, January 16, 1924, she gave additional history of tonsillitis and of a "nervous breakdown" in 1916, with shooting pains in both arms and weakness.

Examination showed her emaciated and slightly cyanotic. During the examination she had a severe attack of pain lasting four minutes, during which she sat up in bed, her face distorted with pain and cyanotic, with distended veins. The chest expansion was somewhat limited. The lungs were normal. The maximum heart impulse was seen and felt in the fifth space 10 cm. from midsternum and 1.5 cm. outside the midclavicular line, coinciding with the left border of dullness. The right border of dullness was 5 cm. from midsternum. The supracardiac dullness was normal. The action was regular. There was a loud rough systolic murmur, loudest in the third right interspace near the sternum, transmitted to the vessels of the neck and the rest of the precordium. An early diastolic murmur was heard along the left sternal border, blowing, short and high pitched. There was well marked thrill over the aortic area. The blood pressure was 105/80 to 90/60. To the left of the umbilicus was a swelling in the abdominal wall which felt like a lipoma. The liver edge was felt rather deep in at the level of the umbilicus in the midclavicular line, firm. No nodules or irregularities were felt. The extremities showed very slight pitting over the shins. The knee-jerks were sluggish. The vessels in the fundi were varied in size in different parts of their course. In the left fundus one vessel almost disappeared over a short distance as though covered by edema. The disc margins were clear cut.

The temperature was 96° to 99.4°, with one rise to 101° February 16. The pulse was 70 to 120, the respiration 15 to 30. The amount of urine was normal when recorded, the specific gravity 1.014 to 1.022. The urine was cloudy at one of four examinations, showed the slightest possible trace to a trace of albumin at three, 6 to 50 leucocytes per high power field at both. The renal function was 5 to 40%. The blood was normal January 16 except for slight increase in platelets and 2% of reticulated cells. January 18 the hemoglobin was 95%, the reds were 4,600,000, with slight achromia, the platelets normal. A Wassermann was negative. The non-protein nitrogen was 36.3 mgm. X-ray (Fig. 44) showed the heart shadow distinctly enlarged both to right and left.

. . . There was no definite abnormality of the sinuses, although the plates were not entirely satisfactory.

The day after admission another examiner found a low pitched rumbling middiastolic murmur at the apex after exercise; the first sound replaced by a systolic murmur; an aortic diastolic murmur at the aortic area, the left sternal border and the apex; a faint diastolic thrill at the apex; the aortic second sound absent; a loud rough sys-

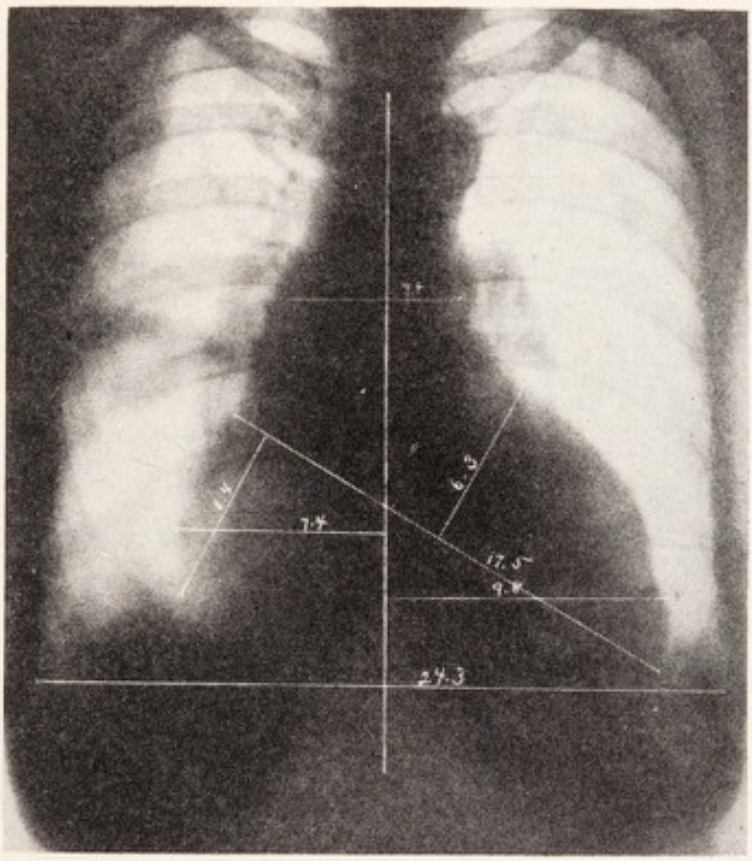



FIG. 44.—Heart shadow distinctly enlarged both to right and left. Left ventricle large and round. Supracardiac shadow shows moderate increase in width and is prominent on the right side. Root glands large and show evidence of calcification. Across the right chest in the region of the interlobar fissure is a dense band about a centimeter wide extending from the root to the periphery. Right lung field below this considerably diminished in radiance, although not dull, suggesting a loss of expansion rather than consolidation. Costophrenic angles on both sides somewha nazy but not so dull as to suggest fluid. Apices relatively clear.

tolic murmur and thrill at the aortic area; sustained pulse. He found many loud crackling inspiratory râles at the left border of the heart and in the left axilla, and noted systolic retraction of the sternum, though no definite Broadbent. Dr. White noted, "Murmurs at lower end of sternum probably transmitted aortic murmurs, although there is also a slight diastolic rumble there . . . Liver large. Face very cyanotic. Cause of pulmonary râles in left axilla near heart? Bases clear behind. Operation will probably be borne all

right. I should advise full digitalization in any case, with maintenance of it thereafter."

January 25 divulsion of the posterior root of the fifth nerve, right, was done under novocain. Next day she had some dyspnea and marked general bodily soreness and stiffness. The right eye was ecchymotic, with a corneal abrasion, and on the 28th was swollen shut. There were râles at the left base. She still had some pain in the right face. She was up in a chair for a few minutes at a time, but was always rather dyspneic on slight exertion. She had an attack or two of dyspnea, cyanosis, and slight increase in heart rate lasting three hours. The morning of February 16 she had a severe attack, unrelieved by adrenalin, morphia or digifolin. The cyanosis was marked, the dyspnea variable. There was a red area over the flank and back. February 17 the general cardiac storm continued unrelieved by vagal pressure. The heart continued regular, the rate about 120. In the attacks her breathing was shallow, rapid, and slightly irregular. The pulse could not be felt at the wrist. The attacks began and ended suddenly. She was drowsy during them. February 17 she was irrational. The blood pressure was 110/70. February 22 she was discharged to the Eye and Ear Infirmary for care of the eye.

During the next three weeks she had no definite attack of heart symptoms. At times she had to sit up in bed to breathe with comfort. She took one digitalis pill a day. March 10 she had "stomach ache" followed by vomiting of food.

At her fourth admission, March 11, 1924, no additional examination is recorded except some notes upon the eye condition. The corneal epithelium had broken down during the past few days.

During her two weeks in the hospital the temperature was 96.7° to 99°, the pulse 80 to 102, the respiration 20 to 36. The amount of urine is not recorded. The specific gravity was 1.034 to 1.028. The urine was cloudy at both of two examinations, showed a slight trace to a trace of albumin and 5 to 50 leucocytes at both and very rare red blood cells at the second. The hemoglobin was 85%. There were 14,600 to 12,500 leucocytes, 80% polynuclears. The reds and platelets were normal.

The patient had a good deal of vomiting. She showed increasing cyanosis and dyspnea, went steadily downhill, and died March 24.

*Clinical Diagnosis (from Hospital Record).*—Rheumatic heart disease with aortic stenosis and regurgitation and congestive failure.

Operation, posterior root of fifth nerve section.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the aortic valve, with stenosis.

Possibly chronic endocarditis of the mitral valve, with stenosis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Double hydrothorax.

Ascites.

Arteriosclerosis, general, and in the aorta and great branches.

Slight arteriosclerosis of the kidneys.

Chronic pleuritis.

*Anatomical Diagnosis.*—Chronic endocarditis of the aortic valve. Stenosis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Edema of the lower extremities.

Fibrous myocarditis.

Mural thrombus of left ventricle of the heart.

Chronic pleuritis.

Obsolete tuberculosis or bronchial glands.

Slight arteriosclerosis.

Cholelithiasis.

Old infarct of left kidney.

Dr. Richardson: The body looked as though weight had been lost. We were not permitted to examine the head.

The legs, ankles and feet were swollen and pitted on pressure. The subcutaneous tissues were a little wet.

The peritoneal cavity contained a small amount of fluid, the beginning of ascites.

The mucosa of the stomach and intestines was reddened, velvety, juicy, weeping thin bloody fluid,—the typical picture of chronic passive congestion.

The diaphragm on the left was at the sixth rib, a little low. The right pleural cavity contained a small amount of thin pale fluid; none on the left. The bronchial glands were slightly enlarged, and several of them showed small areas of fibrocalcareous degeneration. The left lung was bound down by old adhesions. The right lung showed many scattered old adhesions. The lung tissue was spongy to a little leathery, showing what we call varying shades of salmon color, varying shades of brown and red intermingled. The tissue yielded a moderate amount of thin brownish-red frothy fluid,—chronic passive congestion.

DR. CABOT: Is there nothing to say about that patch in the right lung?

DR. RICHARDSON: The only thing of course is the pleural adhesions.

DR. CABOT: That is the sort of result that makes me cautious. We cannot make anything of that X-ray shadow.

A PHYSICIAN: Interlobar septum?

DR. RICHARDSON: That shadow might be due to an adhesion between the wall of the chest and the lung, especially after Dr. Holmes' careful demonstration of the shadows and of the different angles at which he takes the picture. He might, I should think, pick up the edge of an adhesion and possibly get a shadow of that.

DR. HOLMES: I don't believe that you could get a picture of adhesions to look like that. You could get a thick interlobar septum which needs no more than the thickness of a knife-blade to show. I mean a thickening of the pleura between the lobes.

A PHYSICIAN: Interlobar pleurisy that had healed?

DR. CABOT: But the pathologist does not find it. That is the trouble.

DR. RICHARDSON: I should have been a little more careful and put down just where those scattered adhesions on the right side were; but they were not of course of great extent.

She was fifty-nine, with fairly developed frame and muscles. If her heart had weighed 240 to 260 grams that would have been normal. It weighed 750 grams. Of course that is very great enlargement. The myocardium generally was thick, more especially that of the left ventricle, which was 15 mm. The right ventricle wall was 4 mm., slightly thickened. The marked increase was in the left ventricle wall. The columnae carneae were well marked. There was considerable dilatation of the left ventricle, nothing very definite of the left auricle. On the right there was slight dilatation of the auricle and the ventricle. The mitral valve measured 9 cm. There were no definite lesions of this valve. The orifice of the aortic valve presented as a slit about 1½ cm. long and 1 mm. wide. The cusps were fused into a fibrocalcareous mass. The tricuspid valve measured 13 cm., negative, the pulmonary 7½ cm., negative. This is a case, then, of pure aortic stenosis. The coronaries were negative. The ascending thoracic aorta was negative. The descending thoracic and the abdominal portions showed a slight amount of fibrous sclerosis. That aorta at fifty-nine is a pretty good one. The great branches, however, showed a slight to moderate amount of fibrous sclerosis.

The liver weighed 1200 grams. The margin of the liver was well down below the costal border, 6½ cm. below. This liver was rather small, and showed typical nutmeg markings,—chronic passive congestion.

The gall-bladder was slightly distended with thin bile. It contained nine stones which were facetted, 1 cm, in greatest dimension, and crushed under the thumb, showing brownish crystalline material. One of the stones obstructed the cystic duct.

The spleen weighed 150 grams. The organ generally was thick, the tissue elastic, rubbery; passive congestion.

The kidneys combined weighed 315 grams. The capsules stripped. The surfaces were dark bluish brown-red, as was the tissue. In the left kidney there was a small old infarct. I should have mentioned that in the left ventricle opposite a place in the myocardium where there were several areas and streaks of myocarditis there was a mural thrombus.

The microscope comfirmed the presence of the fibrous myocarditis in the ventricle wall and that of the thrombus. The examination of the other tissue showed chronic passive congestion, and in the case of the kidney there was some arteriosclerosis of the renal vessels. There was no definite nephritis.

DR. CABOT: Dr. Richardson says this is a perfectly typical aortic stenosis. There is no hole in the valve opening at all, as far as you can see. The astonishing thing to me is that she lived on.

It is very common to find gall-stones with no symptoms, no harm, no reason to pay any attention to them.

A PHYSICIAN: For two years she had marked pyrosis and gas.

DR. CABOT: A surgeon of a certain type would find that evidence of gall-stones. But you see it in hundreds of people with no trouble in the gall-bladders.

A PHYSICIAN: Do you still think the leucocytosis came from the eye?

DR. CABOT: We have this heart clot in case there isn't enough in the eye.

### Necropsy 3409

A boy of six who had been treated at the Eye and Ear Infirmary for "mastoid" entered September 4. The history is taken from the records of the Out-Patient Department. At his first visit, March 28, one brother was also being treated at the Infirmary for "mastoid." The patient had chicken-pox, tonsillectomy and adenoidectomy at three, scarlet fever and mumps at four, measles at five. Cardiac

symptoms followed the scarlet fever. His appetite and sleep were poor, his bowels constipated. He wet the bed. During the past week he had had stiff neck, better now that his ear was discharging again.

Examination in the Out-Patient Department March 28 showed a fairly well nourished, pale boy with torticollis and a stiff jaw. The precordia bulged. The apex impulse of the heart was in the sixth space. The percussion borders were ten cm. to the left of midsternum, four to the right. The action was regular, rapid, (124). There was a musical systolic murmur at the base transmitted widely, and a double murmur at the base. The pulmonic second sound was accentuated. The pulses were Corrigan.

Under treatment in the Out-Patient Department he made continued improvement until the second week in May, when he had an attack of dyspnea and palpitation. After a rest of five hours he was as well as usual. June 12 he was, his father said, equal to any game. September 4, however, he had been in bed with dyspnea and edema for six weeks.

Examination in the wards showed him poorly nourished, with marked pallor of the skin and mucous membranes. There was marked pyorrhea. The neck was not stiff. The apex impulse of the heart was in the sixth space, with visible wave-like pulsation from the base toward the apex. The left border of dullness was thirteen centimeters from midsternum, six centimeters outside the nipple line, the right border four centimeters from midsternum. At the apex a long systolic thrill was felt, and all over the precordia a questionable presystolic. The action was rapid. The first sound was strong at the apex, where there was a loud blowing musical systolic murmur, heard all over the precordia and followed by a rough rolling diastolic sound which lasted up to the next systolic. The diastolic (and presystolic?) were louder at the third and fourth left interspaces and also up to the pulmonic area. The pulmonic second sound was sharp and accentuated. The cardio-hepatic angle was obtuse. The pulse was Corrigan. The blood pressure was 135/55–100/50. The abdomen was protuberant. The liver dullness, extended from the fifth rib to three centimeters below the costal margin, where the edge was indefinitely felt. The knee joints were rather prominent, suggesting epiphyseal enlargement. The right ear drum showed considerable cerumen which was removed in part, showing a thickened drum—no landmarks—with a small patent perforation in the upper portion. The right mastoid showed the scar of a fairly

recent complete mastoid operation. There as no tenderness or discharge.

During his first eleven weeks in the hospital the boy's temperature was normal except for the period of elevation shown in Fig. 45. During the last three weeks it was usually subnormal, 95.4° to 98°, and "picket-fence." The pulse was 90 to 155. The respiration was constantly rapid, 25 to 40, after November 18 40 to 70. The urinary output was usually twenty to fifty ounces daily until October 15,

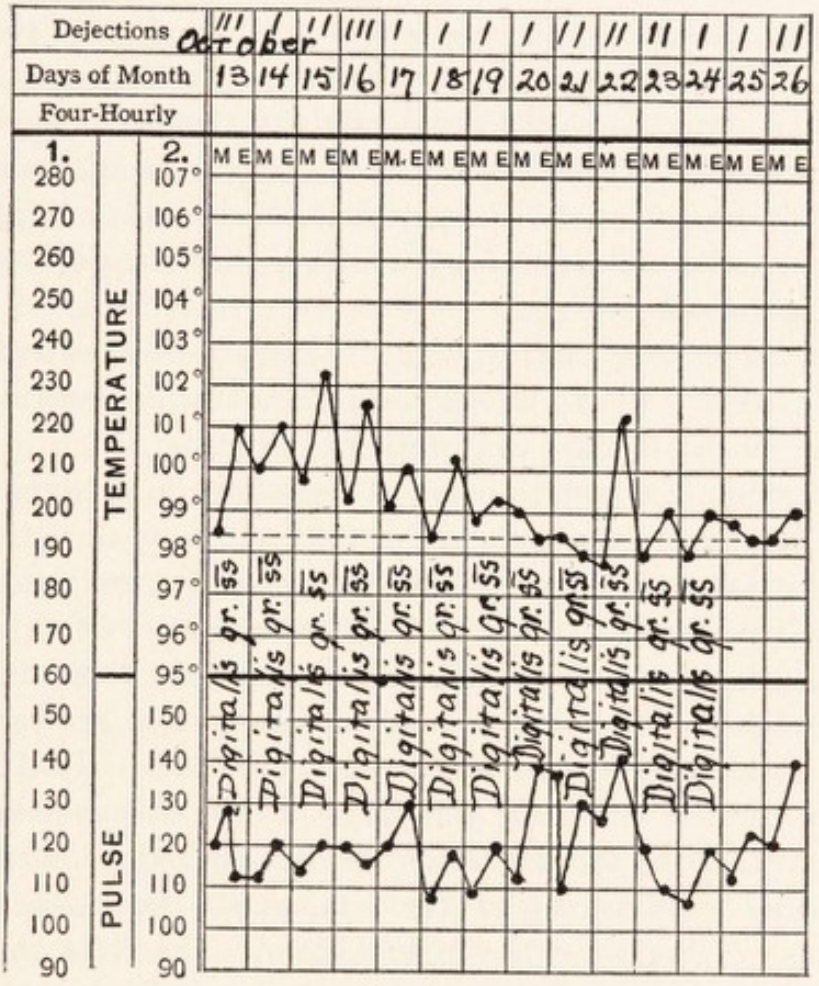



FIG. 45.

then gradually decreasing until after December 1 it was usually five to ten ounces. The specific gravity was 1.010 to 1.030. There was a slight trace of albumin at three of six examinations. The renal function was 55%. The hemoglobin was 60 to 75%, the leucocyte count 12,400, rising to 23,600 October 16, then falling to 11,000; polynuclears 47 to 62%, red, 4,560,000 to 6,088,000, some irregularity of size and shape, considerable achromia; blood culture negative.

By September 6 the pulse had come down from 140 to 120 and was regular. The electric flash light showed capillary pulsation

in the finger tips. The liver dullness was only 1 cm. below the costal margin. The edge was not felt. The left cardiac border was now 11 cm. from midsternum. Systolic and presystolic murmurs replaced the first sound at the apex, diastolic and systolic at the base, where the pulmonic second sound was much increased and the aortic second sound faint. No thrill was felt. September 10 the pulse was about 110, and the child looked and felt better.

By September 18 there was marked arrhythmia. There were pauses of apparently two seconds, and some extra systoles. The boy was nauseated, and the digitalis was stopped. By the time a machine could be got to take a heart tracing the rate was perfectly regular. This was repeated for several days, the rate being irregular in the morning but becoming regular again in the afternoon. No tracing could be obtained. September 22 there was systolic blowing at the apex, and a diastolic, rough at the fourth left space and blowing at the pulmonic area.

October 14 the boy began to gain. There were râles at the right base which persisted until October 19, when the lungs seemed clear.

October 20 he seemed well until the evening, when he showed signs of distress, did not respond to questions, and coughed hard. The heart borders were 4.5 cm. to the right, 15 cm. to the left. There were many coarse râles in the left base, a few at the right, but no dullness. The cardio-hepatic angle was normal. Two days later the cough was very troublesome. The respiration was diminished at the left base, and there was a suspicious spot in the right axilla with harsh respiration, increased voice, and râles. That morning the boy was roused with great difficulty. In the afternoon he seemed bewildered. During the next few days he vomited frequently.

November 5 the temperature was 98.6°, the pulse 120, the respirations 30 ever since the night before. The child was feeling very well and had been sitting up on the edge of the bed and entertaining the entire ward. The right cardiac border was 4 cm., the left 14 cm. For the next two weeks he continued to grow stronger and more talkative.

November 19 the respirations shot up to 52, and he again became unwilling to speak. Nothing definitely wrong appeared during the next week except two attacks of vomiting and the subnormal temperature and unsteady pulse. November 27 the respiration was nearly 60 and the pulse 154. The child continued to grow weaker. December 8 his face and legs showed edema and there was fluid in the abdomen. He seemed to be failing very rapidly. His heart

reached the midaxillary line. No precordial or pleural fluid was found. December 15 his penis and scrotum became edematous. He was very restless and coughed a great deal. That night he died.

*Outcome.*—The clinical diagnosis was myocardial insufficiency, mitral and aortic insufficiency with possible stenosis, and general anasarca. The anatomical diagnosis was chronic and acute endocarditis of the aortic and mitral valves, hypertrophy and dilatation of the heart, slight subacute glomerulonephritis, chronic passive congestion, right hydrothorax, ascites, anasarca, and streptococcus septicemia.

DR. RICHARDSON: The head was not examined.

The body was limp, the lips purplish, and the skin very pale, somewhat waxen in appearance. The hands were puffy and the body and extremities pitted on pressure. The left side of the anterior thoracic wall protruded slightly. The subcutaneous fat was in small amount, the subcutaneous tissues wet and of a dull coppery color. The muscles were of fair consistence and pale brown red.

The peritoneal cavity contained about 200 c.c. of thin pale clear fluid.

The stomach and intestines showed well marked chronic passive congestion.

The anterior margin of the liver was four finger breadths below the costal border in the right mammillary line. The diaphragm was at the fifth interspace on the right, the sixth rib on the left.

The right pleural cavity was half full of thin clear pale fluid. The left pleural cavity contained a few cubic centimeters of similar fluid. There was no pleural adhesions.

There was a small portion of the thymus gland still present.

The trachea and bronchi showed a brownish-red mucosa and contained much frothy brownish-red mucus.

The apices of the lung tissue showed well marked chronic passive congestion.

The pericardium contained a few cubic centimeters of thin pale clear fluid. The heart weighed 473 grams, greatly enlarged. The myocardium was of good consistence and pale brown-red. The right ventricle wall measured 4 mm., the left 10 mm. The columnae carneae were fairly well marked. The cavities were enlarged. The mitral valves presented a moderate amount of chronic endocarditis and several minute to small patches of acute endocarditis. There were minute similar masses scattered along the chordae tendineae. The cusps of the aortic valve showed considerable chronic endocar-

ditis. The cusps were fused about an opening 4 mm. by 6 mm. The endocardium just below the cusps showed a few minute patches of acute endocarditis. The tricuspid and pulmonary valves were negative. The coronary artery was negative. The sino-auricular node was negative. The foramen ovale was closed. The auricular appendices were negative. The aorta and great branches, the pulmonary artery, veins, venae cavae, and the portal vein and radicles were negative.

The spleen showed chronic passive congestion.

The kidneys combined weighed 152 grams. The organs showed a well marked chronic passive congestion and a slight amount of subacute glomerulonephritis.

The culture from the heart blood showed a growth of an organism of the streptococcus-pneumococcus group.

### Necropsy 3715

A man of sixty-eight, formerly a caterer, for the past six years unoccupied, entered March 12. His mother and father both died of consumption. He had always had good health, though he had never been strong. From the age of twenty-five to forty-five he had "sick headaches" every three or four weeks associated with vomiting and lasting a day. For twenty-three years he had had none. He used to be nervous. Four years before admission he had one convulsion or "fit." His eyes were "perfect." He denied venereal disease.

Five years before admission he began to have dyspnea on exertion. He also had occasional dizziness for a month. He was told he had heart trouble. The dyspnea persisted, so that he had been unable to do hard work. Two months before admission he was obliged to use three or four pillows. He had considerable gas and gastric distress. Two weeks before admission he had a severe cold with exacerbation of all the symptoms, marked orthopnea, obliging him to sit up all night, and frequent night attacks of inspiratory "croup," with cough and considerable sputum. March 9 his ankles became edematous. He was very weak and tired.

Examination showed a fairly well developed, emaciated man with dry, yellow skin and slightly cyanotic mucous membranes. The apex impulse of the heart was seen and felt, strong and heaving, in the sixth space 13.5 cm. to the left of midsternum, 4 cm. outside the nipple line. The right border of dullness was 4 cm. to the right, the supracardiac dullness 4 cm. The percussion measurements

are shown in Fig. 46. The action was normal. The pulmonic second sound was faint, the aortic second was not heard. The first sound was replaced by a loud low-pitched blowing systolic murmur loudest at the apex and the aortic area, transmitted to the vessels of the neck and slightly to the axilla. A softer diastolic was heard at the apex, at the left border of the sternum, and very faintly at the aortic area. There was no thrill. The pulses were normal. The blood pressure was 145/65. The lungs showed a few fine crackles above the angle of the left scapula. Below this and extending to the base were marked dullness with diminished breathing, whisper, and tactile fremitus; no râles. Near the angle of the right scapula was a small area over which all signs were increased. The spine showed marked left dorsal structural scoliosis. The abdomen was normal except that the bladder was two finger-breadths above the symphysis and a sharp liver edge was felt at the costal margin. Rectal examination showed the prostate somewhat enlarged, the left lobe more than the

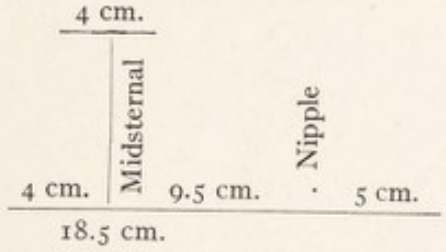



FIG. 46.—Measurements by percussion.

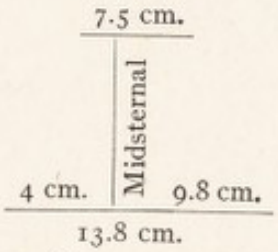



FIG. 47.—Measurements by X-ray.

right. The bladder was full and slightly tender. The genitals were otherwise negative. The legs showed slight edema to the knees. The pupils were irregular. The left was fixed; the right was contracted, but reacted to light and distance.

The temperature was 98°, the pulse 78, the respiration 24. The urine occasionally showed a little albumin. The hemoglobin was 80%, the leucocyte count 7700. A Wassermann was negative. The stools gave a positive guaiac at one of two examinations. The renal function March 14 was 10%, March 21 55%. A lumbar puncture gave 10 c.c. of fluid, at first slightly blood-tinged, not under increased pressure; no cells. The blood nitrogen was 40 mgm. per 100 c.c. of blood. X-ray showed enlargement of the left side of the heart and dilatation of the arch. (See Fig. 47.)

The patient was put on constant drainage. He showed little change except that the systolic blood pressure rose to 170 and the diastolic to 70, and that he grew progressively weaker. March 30 his abdomen became distended and he died.

*Clinical Diagnosis (from Hospital Record).*—Aortic regurgitation.
Hypertrophy and dilatation of the heart.
Arteriosclerosis.
Urethritis.
Cardiac failure.

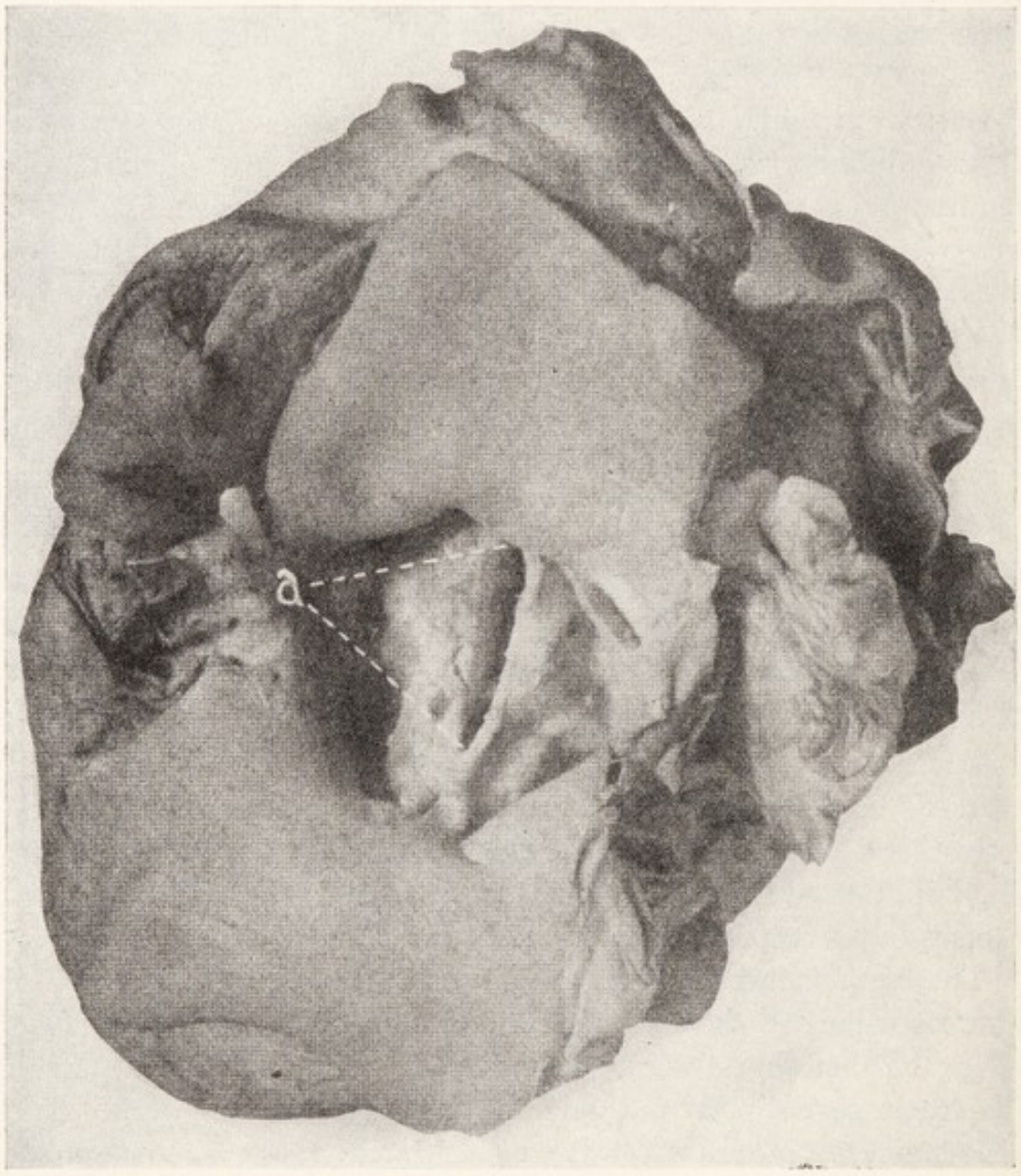



FIG. 48.—"Fish-mouth" aortic valve. (Actual size.) Aortic stenosis in a man of sixty-eight. No history of rheumatism. (Photograph by Lewis S. Brown. Dr. Oscar Richardson.)

Epididymitis.

*Dr. William H. Smith's Diagnosis.*—Probably arteriosclerosis involving the arch.
Hypertrophy of the heart.
Passive congestion of the liver and kidneys.

Arteriosclerotic degeneration of the kidneys.

Dilatation of the bladder.

*Anatomical Diagnosis.*—Chronic endocarditis of the aortic valve. Stenosis.

Arteriosclerosis.

Hypertrophy and dilatation of the heart.

Slight chronic interstitial myocarditis.

Slight chronic passive congestion, general.

Hydrothorax, slight.

Hydropericardium, slight.

Focal pneumonia, lower lobe of the left lung, with small abscess.

Serofibrinous pleuritis, left.

Suppurative nephritis.

Infarcts of the spleen.

Abscess of the left epididymis.

Obsolete tuberculosis, apices of lungs.

Slight chronic pleuritis.

DR. RICHARDSON: This is a perfectly typical case of aortic stenosis, the result of an infection a great many years ago. The arteriosclerosis and aortic stenosis have caused the hypertrophy and dilatation of the heart. The heart weighed 456 grams, (normally 200–300); in other words, it was considerably enlarged for a man of his size. The valve circumferences were interesting in this case. The aortic was a small slit. The mitral measured 10 cm., the tricuspid 13, and the pulmonary 7 cm.; all three were negative. There was some arteriosclerosis of the coronary arteries, and a few small scattered areas of myocarditis. By myocarditis we do not mean myocardial weakness, but the definite replacement of heart muscle by fibrous tissue. The aorta and the great branches showed arteriosclerosis. We have, then, the old infectious process ending in stenosis, and the added arteriosclerotic process. There was moderate dilatation of the left ventricle and slight of the left auricle. The right ventricle was negative and the right auricle slightly dilated. The aortic orifice presented as a small crevice-like slit with irregular margins, about 2½ cm. long, what I call the fish-mouth valve. (See illustration.)

The spleen showed a few infarcts. How did these get there? They seemed rather recent. I think that they were septic.

The kidneys were infected. Unfortunately our culture in this case was contaminated. With suppurative processes in the kidneys we are more inclined to think of the staphylococcus aureus as being the organism, but sometimes the abscesses are produced by organisms

belonging to the streptococcus-pneumococcus group or the colon group. In arteriosclerotic conditions the kidneys, as a rule, are apt to be rather large, and there may be more or less arteriosclerotic nephritis. As a matter of fact it is often astonishing in examining the kidneys of elderly men to find how good they are. The kidneys in this case had fair cortex and good tissue generally, but scattered through them were the abscesses which probably caused the low renal function.

In the prostate, in the region of the so-called middle lobe, there was a small nodule of prostatic tissue which obstructed the urethra slightly. The epididymis showed an abscess which was a part of the expression of the infection.

In the region of the apex of each lung was an area of obsolete tuberculosis. There was nothing in the bronchial lymphatic glands. It is more common perhaps to find these obsolete areas of tuberculosis in the glands than at the apices. Sometimes you find them in both places, and again you find them scattered through the lungs, but as a rule the preferred seat is in the bronchial lymphatic glands. The right lung showed some chronic passive congestion, the area of obsolete tuberculosis, and nothing else. In the left lung the focus of pneumonia was situated in the region of the lower third of the lower lobe, and in one place showed a small abscess. The pleura over the focus of pneumonia showed sero-fibrinous pleuritis.

This case shows very well indeed the end results of acute infections occurring years ago and a terminal infection finding expression in the lung, spleen, kidneys and epididymis.

## Pure Aortic Regurgitation without Stenosis (Rheumatic Type)

The rheumatic form of aortic disease ordinarily produces adhesions of the cusps in such a form that *stenosis* as well as insufficiency is the result. This has been the case in the forty cases of combined mitral-and-aortic disease and in the twenty-eight cases of pure aortic disease already analyzed in Chapter II. On the other hand, *syphilis* (in forty-four cases analyzed under Chapter III) *produces aortic regurgitation without stenosis*, although there were also three cases in that series of ninety-two in which syphilitic aortitis and aortic stenosis coexisted, and in which the stenosis *may* have been due to the syphilis, though as I there point out, it is quite possible that syphilitic aortitis might be engrafted upon an earlier rheumatic lesion of the aortic valve, or that the reverse sequence might take

place. But at any rate, in the overwhelming majority of cases, the rule is:

**Syphilis Produces Pure Aortic Regurgitation While Endocarditis Produces Stenosis and Regurgitation at the Aortic Valve.**—This statement is contradicted by 11 cases in this series (see Table 72). Seven of these are essentially of the ulcerative, acute, destructive type. In only six of them a rheumatic history is clear. But as I have been unable to make a clear separation between the rheumatic group and the other types of infection of heart valves, there does not seem any good reason for insisting upon this distinction here. All that can be said is that the rule already formulated: "rheumatism produces combined aortic stenosis and regurgitation, syphilis gives regurgitation alone" is contradicted by only 6 cases out of 93 in this series, if we confine our attention to cases of *chronic* valve lesions. *It is chiefly when acute endocarditis is present that there has been an exception to this rule in our series.* Even including the "acute" cases it remains true that in 87 cases out of 93, or 93%, the recognition of an aortic regurgitation supposedly due to endocarditis has meant that we should assume the presence of stenosis as well as regurgitation, whether we have the physical signs of the former or not. The eleven exceptional cases I give below in detail.

These cases do not differ in clinical essentials from the twenty-eight cases of pure aortic stenosis and regurgitation just discussed. There we found twenty-five men to three women. Here we find eleven men and no women at all. Half of the cases in each group (five of eleven and fifteen of twenty-eight) are beyond the fiftieth year. There is one very large heart among the eleven (1205 grams), the hypertrophy produced chiefly, I believe, by the associated chronic pericarditis. One other heart in this group is also much enlarged (778 grams), and all show *some* hypertrophy. Ten out of eleven died a congestive death, associated with sepsis in six and with angina in two. Angina was the sole cause of death in another case free from passive congestion.

A diastolic murmur was recognized in every case but one, a presystolic also in three. Systolic murmurs were invariably present. Corrigan's pulse was recorded in six cases, diminution or absence of the aortic second in seven. But strange to say, *an accented aortic sound is recorded in three cases.* In the six cases free from acute endocarditis, acute pericarditis occurred once and chronic pericarditis once.

TABLE 72.—AORTIC REGURGITATION (RHEUMATIC?) WITHOUT STENOSIS

| No. | Age and sex | Rheum. syph. arterio-sclerosis | General Stasis | | Heart weight | Heart in life | Mur-murs | Corri-gan etc. | Thrill | A-2 | Angina | Death | Blood pressure | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Be-fore death | After death | | | | | | | | | | |
| 819 | 51 M | .... | 0 | 0 | 460 | sl. + | 2 | + | 0 | ? | 0 | Septic | ..... | Acute endocarditis. Dermatitis multiformis. |
| 1020 | 42 M | Arterio-sclerosis | + | + | ++ | sl. + | 3 | .. | 0 | dim. | .. | Congestive | ..... | Atheromatous endocarditis aortic valve. |
| 1107 | 25 M | Rh. | + | + | 778 | + | 3 | .. | .. | dim. | + | Congestive and septic | ..... | Sepsis, pseudo-pneumococcus. Possibly aortic stenosis also. Lobar Pn. Pain in epigast. and right shoulder radiating down arm. |
| 1345 | 35 M | Rh. | + | + | 455 | + | 2 | + | .. | abs. | 0 | Septic and congestive | ..... | Focal Pneumonia. Streptococci. |
| 2288 | 29 M | .... | + | + | 511 | + | 2 | + | 0 | dim. | 0 | Congestive | ..... | Clubbed fingers. Sclerosed radials. Acute endocarditis, aortic-and-mitral. Holes in aortic valve. |
| 2318 | 77 M | Rh. | + | + | 576 | + | 2 | + | 0 | dim. | 0 | Septic and uremic | ..... | Latent General Peritonitis Subac. nephritis. General arteriosclerosis. Chronic and acute endocarditis. |
| 2613 | 50 M | Rh. | + | + | 565 | + | Syst. | 0 | 0 | dim. | 0 | Pn.-strep. sepsis | ..... | Red cells 3,000,000. Fever, sepsis and purpura. A.9 cm. nodular fibrous thickening and acute polypoid vegetations. |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 3022 | 52 M | ..... | 4 | + | 470 | + | 2 | 0 | 0 | + | Congestive and septic | 160/130 | Strep. sepsis. Acute endocarditis. Polyps on aortic. Died in a week. |
| 3290 | 31 M | Rh. 5x | + | + | 1205 | + | 2 | + | 0 | + | Congestive | 180/120 | Paralysis left recurrent laryngeal nerve. Chr. pericarditis. M 12, A 8.5, T. 15.5. |
| 3333 | 45 M | Rh. | + | + | 595 | + | 2 | + | .. | abs. | Septic and congestive | 165/ 80 | Acute endocarditis; aortic valve 8.5, yet probably some stenosis. Double empyema. |
| 3527 | 74 M | .... | 0 | 0 | 368 | + | 3 | 0 | 0 | + | In angina | ..... | Intense precordial agony lasting 4 hours, finally relieved by morphia ⅓ gr. Similar attacks past 4–5 weeks. Coronary arteries free. No infarct p.m. No cause for pain discovered. |

**Does Arteriosclerosis Ever Produce Aortic Regurgitation?**—In the entire series of cases analyzed in this book, that is in the *post-mortem* examinations extending from October 1896 to November 1919, our pathologists recorded but one case, Necropsy 1277, as "atheromatous endocarditis of the aortic valve with aortic insufficiency." In this case "the aortic cusps and sinuses are larger than normal. Two of the cusps are to some extent fused together and at the point of fusion there is much thickening and some calcification. These cusps also show some fibrous thickening in places, and the remaining cusp shows an irregular nodular thickening." This patient had no history of syphilis or of rheumatism, and showed no lesions suggesting either of these diseases. He exhibited for seven weeks before his death the usual evidence of decompensated heart trouble. In the hospital he lived but five days. There was an apical systolic murmur; a diastolic murmur was heard all over the precordia, loudest in the fourth left interspace near the sternum. The aortic and pulmonic second sounds were of equal intensity. Corrigan's pulse was present.

He had a generalized arteriosclerosis and a heart weighing 870 grams. There was a slight chronic fibrous endocarditis of the mitral valve, described as follows: "The valve is but a little deformed. At one point near its edge there is considerable thickening and some puckering over a restricted area. The chordae tendineae associated with this are markedly thickened." The circumference of the aortic valve was 9 cm., of the mitral 10.5 cm. The left ventricle was 20 mm., the right 5. All the cavities were dilated. There was chronic fibrous myocarditis, general passive congestion, and anasarca.

I follow here the practice to which I have adhered throughout this book, that is, I submit to the pathologist's judgment as the fundamental basis of classification. But it is certainly striking that in this whole series only one case was found by our pathologists to illustrate the common belief that arteriosclerosis can produce disease of the aortic valve. In view of the very great commonness of arteriosclerosis in our series it is strange that it should affect the aortic valve so seldom, if it is capable of producing this effect at all. From the description of the valves, in the absence of any microscopic examination, I can quite see how a view contrary to that of our pathologists might be maintained. On the records as I accept them, however, the figures about aortic regurgitation stand as follows:

TABLE 73.—TYPES OF AORTIC DISEASE

| | CASES |
|---|---|
| Endocarditic type, stenosis and regurgitation | 93 |
| Endocarditic type, regurgitation alone (some acute) | 11 |
| Syphilitic type, aortic regurgitation alone | 44 |
| Syphilitic type, stenosis and regurgitation | 3 |
| Arteriosclerotic type (?), regurgitation alone | 1 |
| | 152 |

## SUMMARY

1. Possibly the absence of a thrill in the aortic area may give some clue to the diagnosis of this rare condition. In other respects it is essentially like the usual rheumatic lesions of the aortic valve.

2. The rule borne out to some extent in all the different aortic lesions—that males greatly predominate—is strikingly emphasized in this small group: eleven males, no females.

3. The heart is enlarged in all the cases, markedly so in nine out of eleven.

4. A diastolic murmur was heard in ten cases; a presystolic also ("Flint murmur") in three.

5. Corrigan's pulse was present in six, absent in three, not noted in two.

6. Death was of the congestive type in ten of eleven cases. But in four of these there was manifestation of general septicemia as well, due to the accompanying acute endocarditis. In three other cases sepsis seemed to be the main cause of death, while angina was apparently the chief lethal factor in one case.

7. Blood pressure was unfortunately not recorded in eight of the eleven cases. In all the rest, systolic pressure was high and in two of these three the diastolic was also high. No reason for this is apparent.

8. A single case of aortic regurgitation is recorded by the pathologist as apparently of arteriosclerotic origin, though he does not feel quite certain of this. Against this we have 104 cases of aortic regurgitation due to endocarditis, and forty-seven due to syphilis, so that the arteriosclerotic (?) case is in a minority of one out of 152.

## PURE AORTIC REGURGITATION. ILLUSTRATIVE CASES

### Necropsy 819

An engineer of fifty came to the hospital in August for relief of hemorrhoids of twenty-five years' standing. He gave a history also of painless jaundice of a year's duration.

Examination showed a well nourished man with dull anemic yellow skin and pale mucous membranes. The apex impulse of the heart was in the fifth space just outside the nipple line. There was a high pitched short musical diastolic murmur, loudest over the pulmonic area, transmitted upward, across the sternum and into the mitral area. The pulse was Corrigan. The liver edge was palpable on inspiration. On deep pressure there was some tenderness in the lower epigastrium in the median line. The blood showed marked secondary anemia.

The hemorrhoids and prolapsed rectum were cauterized. The patient made a good convalescence and was discharged eleven days after operation "well."

January 16, five months later, he reentered for relief of a skin eruption. He now gave a history of the death of a brother and a sister from phthisis. He had had the diseases of childhood, scarlet fever, pneumonia, gonorrhea at seventeen and three or four later attacks, the last two accompanied by soft chancres and buboes in each groin. He had rheumatic fever at eighteen. Two years before admission he had shingles. Two months before admission he had nasal catarrh. He drank a glass of whiskey daily. A little over a week before admission, after having a few itching pimples on his chest for a few days, blisters appeared on his lips. The following day blisters appeared all over the body. There was not much itching except from the few lesions on the chest. His nasal catarrh became worse. He had no appetite, was very thirsty, and felt sore all over. For two days his legs had been very stiff.

His skin and mucous membranes were now of good quality. The uvula was somewhat enlarged. The pharynx was reddened. There was an expiratory rub in the left lower axilla and back. The intercostal spaces were wide and separated. The apex impulse of the heart was in the fifth space 4½ inches from the median line. The right border of relative dullness was ⅞ of an inch to the right of the median line. The action was regular, the sounds of good quality. At the apex was a systolic murmur transmitted slightly into the axilla. Over the aortic area was a systolic murmur, loudest in the third left interspace and transmitted down to the apex. In addition to the Corrigan pulse there was visible pulsation in the brachials and carotids, with capillary pulse in the finger nails. Nothing abnormal was found in the abdomen. The under surface of the foreskin showed scars. Widely distributed over the entire body, except the hands and feet, was a discrete eruption of sharply defined circular lesions

varying from the size of a pinhead to an inch in diameter. In places they were confluent in very large areas. Small areas were elevated and hemorrhagic, firm but not painful. After reaching about a quarter inch in diameter the areas became blebs in the center, vesiculation spreading to the periphery, the center drying as the vesiculation spread. On rupture the vesicles discharged clear sticky fluid. The face was not affected, but the scalp showed many lesions resembling those on the body.

During his ten days' stay in the hospital the patient had a temperature of 99° to 103.6° with the exception of one drop to 96.7° January 21 and a terminal drop to 97°. The pulse was 88 to 110. The respirations were not remarkable. The urine was smoky, specific gravity 1.018–1.021, albumin 1/10 % to a trace at both examinations, frequent fresh and decolorized red blood corpuscles, occasional fine and coarsely granular casts at one, occasional fine and brown granular casts and casts with adherent fat drops and blood at the other. The hemoglobin was 85%, the leucocytes 23,000.

The patient felt very well for the first two days except for the irritation due to the eruption, which seemed to fade in places, though a few new spots appeared. He grew weaker, did not retain enemata and January 26 died. His heart continued to beat for some time after the respirations had ceased.

The clinical diagnosis was dermatitis multiformis.

Necropsy showed:

Extensive superficial losses in the epidermis of the head, trunk, and extremities. Acute and chronic endocarditis of the aortic and mitral valves.

Hypertrophy and dilatation of the heart.

Hydropericardium.

Small area of ecchymoses in the visceral pericardium.

Ecchymoses in the mucosa of the stomach.

Parenchymatous hemorrhages in the kidneys.

Hyperplasia of the spleen.

Hemorrhagic edema of the lower lobes of the lungs.

Old pleural adhesions of the left lung.

Edema piae.

Interstitial orchitis of the right testicle.

Right inguinal hernia.

**Necropsy 1020**

A meat cutter of forty-two entered November 11. His family history was good. He had measles, mumps, scarlet fever and

typhoid fever in childhood, and at twenty-two pneumonia and rheumatic fever of eight months' duration and a chancre followed by sore mouth and throat with loss of hair. He had gonorrhea once. He drank all the gin he could get, about ten glasses daily. For a year and a half he had been troubled a good deal with gas. He was almost always nauseated in the morning and occasionally vomited. Six weeks before admission he became very short of breath and unable to lie down, his appetite failed, and he vomited almost every morning. Three weeks before admission his legs swelled, and a week later his face became puffy. He coughed much, especially at night, raising a good deal of white mucus. Recently he had passed little urine.

Examination showed a well nourished man with a flushed face. The mucous membranes were slightly pale. The throat was reddened and showed considerable mucus. There were frequent sonorous and sibilant râles scattered throughout the lungs. The breathing was labored. The heart sounds were best heard in the fifth space in the nipple line. The left border of dullness was five inches to the left of the median line, one half inch outside the nipple. The right border of dullness was two inches to the right. The sounds were of fair quality, the action somewhat irregular. The pulmonic second sound was accentuated. There was a double first sound at the apex followed by a systolic murmur heard also in the axilla. A slight diastolic murmur was heard at the second and third right interspaces. At the apex there was a suggestion of a presystolic roll. The pulses were of rather high tension, otherwise normal. The abdomen was tympanitic except for dullness in the flanks shifting with change of position. A fluid wave was present. The liver dullness was from the sixth rib to two inches below the costal margin in the nipple line. The edge was indistinctly felt. The spleen apparently was not enlarged. There was edema of the genitals and much brawny edema of the feet and ankles. The right pupil was greater than the left; both reacted. The knee-jerks were slight, the plantars normal. The temperature was 97.2° to 103.3°, the pulse 142 to 105, the respirations 40 to 20. The output of urine was ten to twelve ounces, the specific gravity 1.020–1.024. There was a large trace to a very slight trace of albumin at both of two examinations. The single sediment examination showed many hyalin casts with oil globules adherent, numerous finely granular and occasional coarsely granular casts.

February 13 only the diastolic murmur in the aortic area remained. The heart was still rapid and irregular.

The night of February 14 the temperature rose to 102° with no discoverable cause. At the right base there were numerous fine moist râles. February 17 the heart sounds were weak, the action slow. The temperature was still elevated. That day the patient died.

*Clinical Diagnosis.*—Aortic regurgitation.

Broken compensation.

Pneumonia.

*Dr. Maurice Fremont-Smith's Diagnosis.*—Cirrhosis of the liver, alcoholic or syphilitic.

Probably syphilis of the aortic valve and aorta.

Cardiac failure.

Anasarca.

Terminal pneumonia.

Possibly tuberculosis of the lungs.

Chronic passive congestion of the kidneys.

*Anatomical Diagnosis.*—Chronic endocarditis of the aortic valve.

Hypertrophy and dilatation of the heart.

Chronic passive congestion of the lungs, liver, spleen, kidneys, stomach and intestines.

Hydrothorax, double.

Ascites.

Anasarca.

Chronic perihepatitis.

Chronic perisplenitis.

Anomaly of the right kidney.

Dr. Richardson: We were not permitted to examine the head. The muscles were pale and wet. A moderate amount of ascites was present. The gastro-intestinal tract was negative except for passive congestion.

There was marked chronic perisplenitis and perihepatitis, but no cirrhosis of the liver. There was well marked chronic passive congestion.

The aorta was smooth.

There was considerable thin brownish fluid in the pleural cavities, and a few old adhesions about the right lung. The lungs were rather voluminous, rather solid and spongy to leathery, showing a well marked salmon color, and in the case of the right one, the lower half of the upper lobe was so much firmer than usual and grayish that I thought it was pneumonia. It was simply chronic passive congestion.

There was a slight excess of fluid in the pericardium. The heart was considerably enlarged and there was considerable dilatation of all the cavities. The mitral valve was negative. The aortic cusps however generally showed some fibrous thickening and deformity and on one of them there was a rather large thick patch, fibrous to fibrocalcareous, more or less degenerated, and at one point a small ulceration,—a chronic endocarditis of the aortic valve with ulceration.

The liver showed marked chronic perihepatitis and the spleen marked chronic perisplenitis, otherwise they showed chronic passive congestion.

Here again we find variation in the weight of the kidneys. In some instances, here at least, there is considerable variation in the weight of the kidneys. One can be very much smaller than the other and have nothing else the matter with it. The individual was born with one kidney small, the other larger. It was so in this case. The right kidney was the smaller one. In addition there was some arteriosclerotic degeneration, a few foci of atrophy, but other than for this and the passive congestion the kidneys were negative. The ureters, bladder and prostate on section were negative. The testes were negative.

No organism was recovered from the heart culture.

DR. FREMONT-SMITH: Did you find any sign that he had ever had syphilis?

DR. RICHARDSON: No.

### Necropsy 1107

A teamster of twenty-five entered July 24. Five years before admission he had gonorrhea. Seven months before admission he had pleurisy and pneumonia followed by rheumatism in the shoulder, knees and feet. He was in a Boston hospital for two months. A month after leaving the hospital he noticed shortness of breath, palpitation, and cough with frothy sputum, occasionally accompanied by attacks of vomiting. Three weeks before admission he noticed swelling of the feet, two weeks later swelling of the abdomen. The symptoms gradually increased until the day before admission, when he suddenly became much worse and on reaching home had to go to bed. The chief complaint was dyspnea. He was also troubled with a feeling of distension in the upper abdomen. The night before admission he was not able to lie down, and slept very little. His appetite was poor. In the past twenty-four hours he had taken only milk.

Examination showed a well nourished man with brownish-yellow skin and pale mucous membranes. The apex impulse of the heart was in the sixth space four and a half inches to the left of the median line. The right border of dullness was two and a half inches to the right. The sounds were somewhat weak, the action regular. The pulmonic second sound was accentuated. In the aortic area and over the third and fourth left cartilages was heard a high pitched diastolic murmur. The pulse was regular, of large volume, and ill sustained. There was capillary pulse. Pistol shot was heard in the groin. The systolic blood pressure was 160. The lungs were negative except for an occasional sonorous râle. The abdomen was tensely distended in the lower portion, soft above the umbilicus. There was considerable tenderness in the right upper quadrant. There was a fluid wave. The liver dullness extended from the fifth space to two inches below the ensiform in the median line, where the edge was felt. There was considerable edema of the legs. The pupils were normal. The knee-jerks were lively. The temperature was 97.9° to 102°, the pulse 81 to 130, the respirations 24 to 50. There were fourteen to seventy ounces of urine daily, the specific gravity 1.013–1.016, turbid at one of three examinations, high colored at the other two, a slight trace to a trace of albumin at all. At the single sediment examination there were a few abnormal red blood corpuscles. The hemoglobin was 65%, the leucocytes 8700. The fundi were normal.

The dyspnea continued severe through the day of entrance. Afterwards it diminished. The fluid in the abdomen increased through the day of entrance, but two days later was somewhat less. The chief complaint July 26 was hemorrhoids, which were tense and thrombosed. The heart action was much stronger. A marked presystolic murmur was heard at the apex. July 29 the hemorrhoids were incised with ethyl chlorid and clots partially removed. After this the patient complained less of pain. By August 1 the presystolic murmur had disappeared at the apex. The diastolic persisted, and a marked systolic was heard in the aortic area, transmitted to the neck. The dyspnea and cough were less. The patient was constantly drowsy and complained of weakness. The following night he had an attack of nausea and repeated vomiting. The fluid in the abdomen increased. Under digitalis the condition rapidly improved until the night of August 10, when the dyspnea became severe but was somewhat relieved by oxygen. Edema of the legs and scrotum, dyspnea and malaise increased. He had dis-

comfort in the epigastrium, pain in the left shoulder radiating down the left arm, and numbness in the left hand. August 19 Southey's tubes were inserted in the left leg. They failed to withdraw much fluid. The next day the respirations became considerably more rapid and the pulse and temperature rose. The patient became delirious. The morning of August 21 he died.

*Clinical Diagnosis (from Hospital Record).*—Aortic regurgitation. Hemorrhoids.

1. *Anatomical Diagnosis.*—1. Primary fatal lesion.

Chronic endocarditis of the aortic valve (with regurgitation and probably some obstruction).

2. Secondary or terminal lesions.

Hypertrophy and dilatation of the heart.
Septicemia (pseudopneumococcus).
Lobar pneumonia of upper lobe of right lung.
Chronic passive congestion of the lungs, liver and spleen.
Anasarca.
Ascites.
Hydropericardium.
Edema of the kidneys.

3. Historical landmarks.

Small accessory spleen.
Arteriosclerotic patch in aorta.
Infarcts of the spleen and kidneys.

Dr. Richardson: A well developed and nourished white man. We were not permitted to examine the head. There was anasarca, ascites, and hydropericardium, no definite hydrothorax. On the left side there was a small amount of pale clear fluid in the pleural cavity, on the right a small amount of cloudy fluid and fibrin and a thin coating of exudate. The lungs generally showed chronic passive congestion, but the upper lobe of the right lung, about two-thirds of it, showed frank pneumonia. The organism is what we called in those days the pseudopneumococcus, now known as the streptococcus mucosus capsulatus. This was one of the first cases in which I recovered it from a case of pneumonia.

The heart weighed 778 grams,—markedly enlarged. The only lesions on the valves were those of the aortic valve, where there was a polypoid mass of chronic endocarditis.

In the spleen and the kidneys were infarcts. The liver showed chronic passive congestion. The kidneys, although they weighed

415 grams, which of course is a considerable weight, showed nothing but edema, no evidence of any glomerulo-nephritis.

I recovered from the heart blood the organism mentioned above.

DR. CABOT: Will you tell us a little more about the aortic valve?

DR. RICHARDSON: In the situation of the free edges of the cusps and at their points of juntion there is a row of irregularly rounded yellowish tag-like masses, the largest about one cm. in greatest dimension. At one point there extends down from the junction of two of the cusps on to the endocardium a grayish-yellow, fibrous, somewhat shaggy, firmly adherent patch of material. The material of these masses in the situation of the aortic cusps is slightly soft in places, but on section it is in many places finely calcareous in character.

DR. CABOT: So that is all chronic, no acute?

DR. RICHARDSON: Yes, all chronic. There was some question, but the histology showed the same thing.

DR. CABOT: Was there any stenosis?

DR. RICHARDSON: The circumference was eight cm.; no stenosis in one sense, but the mass when the valve closed would leave less room for the blood to go through.

DR. FREMONT-SMITH: What can we call the etiology? This is not a typical rheumatic chronic endocarditis.

DR. RICHARDSON: Yes, I should think it was rheumatic chronic endocarditis.

### Necropsy 1345

A colored houseman of thirty-three entered March 24. He gave a history of boils in childhood, whooping cough, gonorrhea years earlier, a sore on the penis twelve years before admission, and an attack of "rheumatism" the previous winter. His habits were good. Three months before admission he began to have heavy dull ache across the upper abdomen and dyspnea on exertion. The ache persisted and the dyspnea grew steadily worse until at admission it was continuous and at times he had to sit up to get his breath. During the past two months he had cough and raised a great deal of bloody sputum. For the past month his legs had been swollen. He had some palpitation, slept poorly, had little appetite, and during the past week had vomited food immediately after eating. He had been using salts with small movements. For four weeks he had been unable to work and for three weeks had been in bed.

The examination showed a well nourished man with cyanotic mucous membranes. His throat was covered with mucus. The apex impulse of the heart was felt in the sixth space five inches to the left

of the midline. The left border of dullness was four inches (?) to the left. The right border of dullness was an inch and a half to the right of the median line. The action was rapid. The pulmonic second sound was accentuated, the aortic second sound absent. A short soft systolic murmur and a soft diastolic murmur were heard at the apex, a loud diastolic and a systolic all over the precordia, loudest at the third left interspace. There was a diastolic murmur in the aortic area and also in the back. There was Corrigan pulse, pistol shot and Duroziez's sign. The lungs showed slight dullness at both bases and medium moist râles throughout, more numerous at the bases. Expiration was prolonged and harsh. There was slight shifting dullness in the flanks and general tenderness throughout the abdomen, more marked over the liver, which extended to three inches below the costal margin, where the edge was felt in the mammillary line. The splenic dullness was not made out. The edge was indistinctly felt. A few bean-sized glands were felt in the axillae and groins. The pupils were normal. The knee-jerks were unsatisfactory, the plantars normal. There was marked edema of the ankles, legs and thighs. There was a subcutaneous mass, four by six inches, over the lower ribs in the left back.

During his twelve days in the hospital the temperature gradually rose from 97.1° to 103.2°. The pulse ranged from 101 to 135, the respirations from 16 to 39. The urine was from 34 to 92 ounces, specific gravity 1.012–1.016, a slight trace to the slightest possible trace of albumin at all of three examinations, a rare blood cell at one. The hemoglobin was 100%, the leucocytes 14,000 to 19,300.

The patient failed from admission. After the first day the sputum was almost pure blood. No localizing sign of an infarct could be found. The edema of the legs and genitals increased. March 29 there was a patch of bronchial breathing just below the angle of the right scapula which increased, extending to the midscapula by April 3. That day, after he had been delirious for several days, he died.

*Clinical Diagnosis.*—Aortic roughening.
Aortic and mitral regurgitation.
Failing compensation.
Possible pericarditis.
Chronic passive congestion.
Infarcts of the lungs and bronchopneumonia.

*Anatomical Diagnosis.*—Marked fibrous endocarditis of the aortic valve and slight fibrous endocarditis of the mitral valve.

Hypertrophy and dilatation of the heart.
Focal pneumonia.
Hemorrhagic edema and infarcts of the lungs.
General chronic passive congestion.
Anasarca.
Hydropericardium.
Ascites.
Chronic perisplenitis and hepatitis.
Streptococcus septicemia.
Foci of necrosis in the fat tissue of the pancreas.

**Necropsy 2288**

A musician of twenty-nine entered January 20, 1909.

He formerly had taken a good deal of whiskey and beer and had an occasional spree. His past history was negative except for indefinite rheumatic pains in the calves of his legs at times and an attack of pneumonia the previous November. A week after he thought he was entirely over this, a cough developed which persisted. He had remained weak, tired, and unable to work, or to sleep without three or four pillows, as he choked up and coughed when lying flat.

Five weeks before admission he began to be very short of breath, especially on exertion. Three weeks ago he was obliged to sleep for several nights in a chair, and afterwards often had to finish the night in a chair. He had very little cough. For ten days his abdomen had increased steadily in size. For a week his legs had been edematous. The dyspnea had now become extreme.

Examination showed a well nourished man, orthopneic, with short hacking cough and husky voice. Skin pale and pasty. Mucous membranes cyanotic. Fingers markedly clubbed. Nails rounded and very cyanotic. Considerable throbbing of arteries in supraclavicular space and in neck. Apex impulse of the heart and left border of dullness in the fifth space 2½ inches outside the nipple line, 6½ inches to left of midsternum. Right border of dullness 1¾ inches to the right. Action very irregular in force and rhythm. At the apex three sounds equidistant from one another followed by a pause. The $P_2$ sharp and greater than $A_2$. Loud harsh blowing diastolic murmur heard best in third space just to left of sternal edge, transmitted upward and toward the apex but not to it. At the apex a soft blowing systolic murmur occupying the greater part of the first two sounds at the apex and the single systole elsewhere (but to a less marked degree) and transmitted to the axilla, where it was heard

better that over the cardiac area. Pulses of fair volume, low tension, Corrigan. Double sharp booming sounds heard over femorals. Blood pressure not recorded. Arteries palpable and sclerosed. Dullness, diminished respiration, voice sounds and fremitus, many fine and medium moist râles at both bases posteriorly, more extensive on the right. Abdomen full. A transverse ridge from the right iliac crest across the umbilicus marked the lower border of the liver. In the right flank was dullness, non-shifting. Liver dullness from the sixth rib to four inches below the costal margin in the right nipple line. Edge felt at level of umbilicus as a firm hard somewhat tender body. Spleen apparently enlarged downward to precussion but impossible to palpate because of tenderness. Marked soft edema of everything from a point just below the knee. Temperature and respiration normal. Pulse 104–115. Amount and specific gravity of urine not recorded. A trace of albumin, bile, a large number of red blood corpuscles, a few hyalin and finely granular casts.

The patient was very uncomfortable at admission and had a good night only when being given sufficient morphia. Next day he showed extreme pallor, marked distress and dyspnea. After an intravenous injection of strophanthin the pale color and distress remained the same, until he suddenly became livid and died at once.

*Clinical Diagnosis.*—Aortic roughening and regurgitation (fibro-calcareous endocarditis).

Hypertrophy and dilatation of the heart.

Broken compensation.

*Anatomical Diagnosis.*—Ulcerative polypous endocarditis of the aortic valve; slight fibrous endocarditis of the mitral valve; small parietal thrombi of aorta; hydropericardium; infarcts of lungs, spleen, and kidneys; obsolete tuberculosis of apex of left lung; hydrothorax, slight; chronic pleuritis, left; hypertrophy and dilatation of the heart; chronic passive congestion; ascites; anasarca; icterus, slight. Head not examined.

The heart weighed 511 grams. The right ventricular wall measured 4 mm., the left 11 mm. Valve circumferences: mitral 11 cm., aortic 7 cm., tricuspid 15 cm. All cavities were dilated, especially the left ventricle.

*Mitral Valve.*—"The curtain at one point near the free margin and at another a short distance from it shows small, circular, distinctly isolated fibrous-like areas. They measure about 3 mm. in diameter and the auricular surface of the one nearest the free margin of the valve is reddish and coated with a slight amount of

fibrin-like material. The circumference of the valve is slightly increased and other than mentioned, this valve is not remarkable."

*Aortic Valve.*—"The aortic cusps are largely transformed into very irregularly surfaced grayish yellow, softer and firmer polypoid masses, to the surfaces of which fibrin and blood-clot-like material are adherent. In places the vegetations are slightly gritty and at two or three points there is a complete interruption in the continuity of the masses, leaving small openings through which the cavity of the sinuses of Valsalva are continuous with the cavity of the ventricle. The endocardium just below one of the cusps and for a short distance downward shows an area of slight discoloration, the surface of which is slightly granular and to which a slight amount of fibrin and blood-clot-liked material is adherent.

*Tricuspid valve*, "except for its edge is not remarkable." The pulmonary valve is not mentioned.

No microscopic examination recorded.

### Necropsy 2318

A clergyman and civil engineer of seventy-seven entered February 27. His mother died of "hemorrhage from the mouth." He had rheumatic fever at fourteen, and was refused for the army at the time of the Civil War. At thirty-one he had "country fever." At forty-four while skating he fainted and had great palpitation of the heart. Since that time he had had a number of attacks of weakness, dizziness and palpitation lasting a few hours, never laying him up for more than a day. He had always led a strenuous outdoor life and had done a great deal of walking. His habits were excellent. In January, two months before admission, he began to be short of breath, especially after exertion, and found that his heart beat fast, jerkily and forcibly. He became noticeably weaker and had a little cough with dark brown sputum. At the end of January his legs and feet became swollen. He would wake at two or three o'clock in the morning suffocated and weak and be obliged to sit up with his hands and arms resting on a table. In an hour or two he would feel relieved. He had some epigastric discomfort. He had vomited a few times. A month before admission he was in a hospital for nine days with considerable benefit.

Physical examination showed a slight, much emaciated old man with marked distension of the external jugular and the veins along the left clavicle and visible bounding of the carotid, brachial and femoral arteries. The left chest showed marked prominence in the

precordial region. The mucous membranes were very pale. Pea sized glands were palpable in the axillae and groins. The apex impulse of the heart was visible, beating forcibly in the fifth and sixth spaces outside the nipple line, six and a quarter inches to the left of mid-sternum and in the third space to the left of the sternum. The impulse and dullness corresponded in the sixth space. The right border was at the sternal margin. The supracardiac dullness was not increased. The action was slightly irregular, chiefly in rhythm. The first sound at the apex was sharp. The second sound could be heard only about half way toward the base, not at the apex. The pulmonic second sound was accentuated. Following the sharp first sound at the apex was a high pitched musical systolic murmur and a diastolic murmur transmitted to the axilla. In the third space to the left a harsh rough systolic was heard, and a loud blowing diastolic murmur transmitted upward to the second space at the right of the sternum, downward to the apex and into the axilla, where a to-and-fro murmur was heard distinctly. The pulses were slightly irregular, Corrigan. The arteries were palpable and tortuous. Sclerosed plates were easily felt. The systolic blood pressure was 135 to 145. The lungs showed slight dullness, diminished respiration and voice sounds, with fine moist râles at both bases posteriorly, rising higher on the right, and fine moist râles at the bases in front. The right flank showed dullness, not shifting. The liver dullness extended just below the costal margin, where the edge was felt. There was slight edema of the genitals and marked soft edema of the legs, ankles and feet. The venules on the legs were prominent. The pupils and knee-jerks were normal.

During the patient's stay in the hospital his temperature was subnormal, 98° to 94.6°, until the day of death, when it rose to 101.1°. The pulse was 61 to 94 with a terminal rise to 135. The respirations were 23 to 36. The daily output of urine was 10 to 28 ounces, the specific gravity 1.020 to 1.022, a slight trace of albumin at one of two examinations, rare pus cells at both, rare red cells at one. The blood was not remarkable.

The patient was comfortable except at night, when he tended to be rather delirious. At times he was very short of breath. March 7 he developed Cheyne-Stokes respiration. That night he gradually failed. His pulse grew rapid and weak. The next morning with the rise in temperature his lungs were full of moist râles. That morning he died.

*Outcome.*—The clinical diagnosis was aortic and mitral regurgitation, broken cardiac compensation, arteriosclerosis, and hypertrophy and dilatation of the heart.

Necropsy showed arteriosclerosis, subacute glomerulonephritis with arteriosclerotic degeneration, fibrocalcareous endocarditis of the aortic valve and fibrous endocarditis of the mitral valve, hypertrophy and dilatation of the heart, thrombosis of the left auricular appendix, slight chronic passive congestion, papillary adenomata of the kidneys, teratoma of the kidney, infarct of the kidney, general peritonitis (terminal), foci of obsolete tuberculosis in the apex of the left lung, chronic pleuritis, edema piae, and scoliosis.

Dr. Richardson: The head in this case showed edema piae and some arteriosclerosis of the vessels of Willis but was otherwise negative.

The peritoneal cavity showed an acute peritonitis,—terminal. Terminal infections occur in any of the cavities,—pleuritis, pericarditis, or peritonitis. In the streptococcus epidemics streptococcus peritonitis is common.

The gastro-intestinal tract was negative, no perforations, no ulcers or appendicitis.

The lungs showed chronic passive congestion and some obsolete tuberculosis in the apex of the left lung.

The pericardium was negative. The heart weighed 576 grams. Considerable hypertrophy. There was marked general arteriosclerosis. The valve measurements were, for the tricuspid 14.5 cm., the pulmonary 9, the aortic 9.5, the mitral 13.5. These valve circumferences are all increased. On the mitral valve there was a moderate amount of rather diffuse fibrous endocarditis, and on the aortic a moderate amount of fibrous thickening with calcareous degeneration regarded as chronic endocarditis and of the rheumatic type. In the left auricular appendix there was a thrombus and in one kidney an infarct. In the lower pole of the right kidney there was a tumor, a small teratoma, and in each kidney some very small papillary adenomata. The kidneys otherwise showed a combination of conditions, subacute glomerulonephritis, with some arteriosclerotic degeneration.

The bladder, prostate, seminal vesicles and testes were negative. The gastro-intestinal tract showed passive congestion. There was some curvature of the spinal column, but it was otherwise negative.

Dr. Cabot: We are sometimes asked by a patient with rheumatic heart disease, "How long may I live with this?" We should put it

down in the tablets of memory that we have here read a necropsy of a man who had rheumatic fever at fourteen and presumably got his heart lesion then. He died at seventy-seven. He lived sixty-three years with that lesion. That is a point of value in the practice of medicine,—to be able to say truthfully to patients, "We have known a case necropsied with a rheumatic heart lesion that probably lasted sixty-three years."

### Necropsy 2613

In September, 1905, at his first admission to the hospital, the history and examination of this patient, an American shopkeeper of forty-five, were negative except for hemorrhoids and the heart. The apex impulse was one and a half inches outside the nipple line. There was a loud whistling systolic murmur transmitted to the axilla. The hemorrhoids were cauterized. A year later he reported a perfect result.

In May, 1906, he reentered. He now gave a history of chancres two years earlier. For a week he had not felt well. For six days he had had cough. Then he had severe pain in the back of the left thigh and calf running up into the small of the back. Two days before admission this became so severe that he had to go to bed. He had also had severe pain in the left knee-joint, and of late had had some abdominal pain radiating to the back.

Examination showed a barrel-shaped chest. The apex impulse of the heart was in the sixth space just outside the nipple line. There was no enlargement to percussion. The action was regular, the sounds of good quality, the second sound at the apex ringing. A systolic murmur at the apex replaced the first sound and was transmitted to the axilla. The aortic second sound was greater than the pulmonic second. The pulses were normal, the arteries palpable. The lungs were hyperresonant. The breath sounds were loud. Expiration was prolonged. There were occasional moist râles. There was considerable general tenderness in the abdomen. The genitals were normal. The left knee-joint contained an excess of fluid, the patella floating. There was great tenderness about the joint. The left leg was everted. There was much tenderness over the course of the left sciatic nerve and calf. The pupils and reflexes were normal except that the left knee-jerk was not obtained.

The temperature at entrance was 102.5°, after May 9 normal. The pulse was 108–60. The respirations were normal. The urine was normal in amount. The specific gravity ranged from 1.010 to

1.023. There was the slightest possible trace of albumin at both of two examinations. The hemoglobin was 75%, the leucocytes 6600.

Under salicylates the pain and swelling of the knee-joint disappeared. May 15 the patient was discharged "well."

He was admitted for the third time January 8, 1910. For the past few years he had urinated four or five times at night. For two months he had had considerable cough which disturbed his sleep, loss of appetite, dry and slightly sore throat, and occasional vomiting. He had lost some weight and strength recently. January 6 he had soreness at the base of his neck, all over the chest, and along the right costal margin, worse on cough or deep inspiration. For a day or two he had been raising bloody sputum.

Examination showed a well nourished, full-blooded man with frequent paroxysms of violent coughing, considerable dyspnea, and slight periodic audible respiratory wheeze. The skin was somewhat pale. The teeth were a few decayed snags, with pyorrhea. The apex impulse of the heart was in the fifth space fifteen centimeters from the midsternal line, five centimeters outside the nipple line, corresponding with the left border of dullness. The right border of dullness was four centimeters from midsternum. A systolic scratchy murmur was heard at the apex. The pulmonic second sound was accentuated. The pulses were normal, the artery walls moderately thickened. The systolic blood pressure was 130. Expiration was prolonged throughout and accompanied by many wheezes, especially at the apices in front. There were a few inspiratory crackles. The liver dullness extended from the sixth rib to two centimeters below the costal margin; the edge was not definitely felt. The urinary output was normal. The urine was light brown to black-brown at the first seven of sixteen examinations, cloudy or smoky at all the rest, high colored at three. The specific gravity was 1.009 to 1.013. There was albumin at all but the last examination, a large trace at the first six, a trace to a very slight trace later. The sediment showed blood decreasing from much to rare red cells, then none; at the end many red corpuscles to much free blood. The first eight examinations showed many brown granular and blood casts and hyalin casts with cells and blood attached. Later there were few, and the blood disappeared. At entrance the hemoglobin was 80%, the leucocyte count 34,200; the reds showed slight achromia and numerous large polychromatophilic cells. Later the leucocyte count fell, reaching normal January 17.

During the first week in the hospital the patient ran a temperature of 99.7° to 104.2°; the pulse was 71–100, the respirations 24–48. During the next week the temperature was 98.2°–101°, the pulse 64–95, the respirations 18–37. The third week the temperature gradually reached normal and the pulse and respirations were normal throughout. During the first three days the patient had violent paroxysms of coughing, raising considerable thick tenacious brown sputum showing some fresh blood and considerable pus; no tubercle bacilli. He had slight nosebleed much of the time from entrance. A large number of bloody crusts formed on the nose and upper lip. He bled easily also from the ear. A purpuric spot was left after the injection of morphia. After January 11 the cough was much less severe. January 14 there was marked pain on inspiration and a friction rub in the left axilla. He made good general improvement. The tendency to bleed diminished. The urine showed much less color than at first. January 18 he had a small bloody stool. January 22 there was a diastolic blow transmitted down the left border of the sternum and a slightly collapsing pulse. The apex impulse was localized and forcible. The patient said he felt strong and well. He continued to improve rapidly. January 29 his lungs were clear. That day he was discharged.

After leaving the hospital he felt fairly well, though he had severe paroxysms of cough. He was able to do a little work by resting every few minutes to ease his dyspnea. When walking he had to rest every few steps. His bowels were constipated. He slept poorly because of attacks of coughing. There was no sputum. He had much dizziness. In the late spring his feet and ankles began to swell at night and he had severe cramps in his legs. He urinated three times at night. By May 9 the cough had become almost incessant and dull abdominal pain had developed.

At his fourth admission to the hospital, May 12, 1910, he was well nourished but looked old. He sat propped up in bed breathing very rapidly, with frequent attacks of severe cough during which he became cyanotic and after which he gasped for air. The skin was pale and moist. Over the arms and in great numbers on the legs were purpuric areas. The mucous membranes and throat were pale and dry, the throat and pharynx crusted. The tongue was dry, furrowed and cracked, with a brown crust. Lymph glands the size of peas were felt in the neck and axillae and the size of beans in the groins. The apex impulse of the heart was seen and felt in the fifth space fourteen centimeters from midsternum and six and a half centimeters

outside the nipple line, corresponding with the left border of dullness. The right border was four and a half centimeters to the right. The action was regular, not rapid. The sounds were clear and of good quality. At the apex was a loud harsh systolic murmur, heard also over the precordia and transmitted to the axilla and back. The pulmonic second sound was slightly accentuated. There was slight lateral excursion of the brachials. The systolic blood pressure was 105. The lungs were rather hyperresonant except at both bases posteriorly, where there was slight dullness. The expiratory murmur was rather prolonged. A few squeaks were heard throughout, and below the angles of the scapulae numerous fine moist râles. The abdomen was held rigid. The liver dullness extended from the sixth rib to the costal margin. A tender edge was felt indistinctly at the level of the umbilicus in the nipple line. Both shins and ankles showed considerable soft edema.

During his eleven days in the hospital the patient ran an irregular temperature from 97.9° to 101.6°, with two intervals of normal temperature for two days and a terminal rise to 103.7°. The pulse ranged from 85 to 110, with a terminal rise to 135. The respirations were 24 to 43. The output of urine was normal except on May 14, when it was ℥ 73. The urine was smoky or cloudy at all of five examinations, bloody at two, high colored at the other three. The specific gravity was fixed at 1.011–1.012. A trace to the slightest possible trace of albumin and a few to rare red blood corpuscles were found at all examinations. At one a guaiac test on the supernatant fluid was strongly positive. Hyalin, granular and cellular casts were found at all examinations, with blood cells attached at the first two. The hemoglobin was 50 to 60%, the reds 3,000,000, showing achromia at one of three examinations, macrocytosis at two, variation in size at one, in shape at another. The leucocytes and differential count were normal until the day of death.

The patient seemed at times rather childish. He had sudden attacks of severe dyspnea which usually could be easily quieted without medicine, though at times there was very marked genuine dyspnea requiring morphia. This grew much worse. He had several very severe nosebleeds. The gums and lips bled slightly at times. May 19 an ulceration on the nasal septum was touched with chromic acid. May 21 a localized patch of large vesicles developed on the upper right eyelid. The stool was hard costive pellets imbedded in a little fresh blood. May 22 the sputum was a membranous looking material showing a few Gram-positive diplococci.

The patient was unable to swallow. A throat culture was taken. The leucocyte count was 14,000. The heart was very irregular the pulse was weak. May 22 he suddenly collapsed and died.

*Bacteriological Report.*—A throat culture reported after death was positive for Klebs-Loeffler bacilli.

*Clinical Diagnosis.*—Chronic bronchitis and emphysema.

Mitral regurgitation.

Chronic passive congestion, general.

Purpura.

Hematuria.

Secondary anemia.

Diphtheria.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic nephritis.

Hypertrophied and dilated heart.

Acute endocarditis.

Chronic passive congestion.

Diphtheria.

*Anatomical Diagnosis.*—Chronic fibrous and acute vegetative endocarditis of the aortic and mitral valves.

Hypertrophy and dilatation of the heart.

Chronic passive congestion of the lungs and liver.

Arteriosclerosis.

Lobar pneumonia of the upper lobe of the left lung.

Soft spleen.

Acute degeneration of the liver.

Secondary anemia.

Arteriosclerotic degeneration of the kidneys.

Streptococcus septicemia.

DR. RICHARDSON: The head was not examined. There is nothing about diphtheria in our record here. Scattered over the lungs were numerous irregular discrete and confluent purpuric-like areas. The culture taken from the heart blood yielded a good growth of the streptococcus. There was a slight amount of pale fluid in each pleural cavity and a few adhesions. The right lung showed chronic passive congestion, no pneumonia. The lower two-thirds of the upper lobe of the left lung showed frank pneumonia with the usual pleuritis.

DR. CABOT: No emphysema?

DR. RICHARDSON: No.

DR. CABOT: You would have put it down if it had been there, wouldn't you? It is an obvious thing.

DR. RICHARDSON: Yes, it is obvious.

Scattered along the aorta there was a slight to moderate amount of fibrous sclerosis.

The pericardium contained a slight amount of pale fluid. The heart weighed 565 grams,—considerably enlarged. The myocardium was thick. The mitral valve measured 9.5 cm., the aortic 9 cm. The tricuspid and pulmonary valves were negative. Scattered along the curtain of the mitral valve were numerous fibrous areas and nodules, causing some deformity, and planted on these were smaller and larger masses of soft frank spongy vegetations,—acute endocarditis on a basis of chronic.

The coronaries were free, capacious, and showed only a slight amount of fibrous sclerosis.

The liver and the other organs showed chronic passive congestion.

There was some question as to anemia in this case. It was considered at that time that there was some anemia, but it was secondary and not primary.

The kidneys weighed 345 grams. From the description there was nothing but arteriosclerotic degeneration, that is, arteriosclerosis of the vessels and some of the capillaries of the glomeruli and scattered foci. At that particular time it was not regarded as sufficient to call it arteriosclerotic nephritis.

A PHYSICIAN: What about the Klebs-Loeffler?

DR. CABOT: It was a terminal infection. I have no doubt it contributed to the death. But you see there is plenty more infection, with the acute endocarditis throwing emboli around. I suppose it perfectly might have been in the upper air passages, and if you had not been told about it you would not look.

DR. RICHARDSON: No, but a case of Klebs-Loeffler has to be taken care of in a proper fashion.

DR CABOT: One would think they would have made more of a fuss about it. The chief mystery to me is that Dr. Richardson so far refuses to back us up on the diagnosis of nephritis.

### Necropsy 3022

An American plumber of fifty-two entered March 9, 1912, for observation. He had gonorrhea at twenty-three. He recalled no illnesses until 1900, when as a soldier in the Philippines he had shortness of breath and edema of the legs during forced marches. A year later he had malaria. Two years after this he was discharged from the army on account of "chronic dysentery, chronic gastritis, heart trouble, chills and fever." He continued to have severe diar-

rhea until a year before coming to the hospital, sometimes forty movements a day, of late years four or five. During the past year he had had only three or four short attacks. The movements were slimy and bloody. He drank considerable beer and whiskey until five months before admission.

Eight months ago on a hot day he was "sunstruck," was unconscious for a few minutes, and had high fever. Ice water treatment revived him. Since that time he had been in poor health. For three months he did a little work. For the past five he had done none. He had had marked dyspnea and at times orthopnea, considerable precordial distress, and occasional sharp attacks of precordial pain lasting a few minutes. His legs and ankles had been markedly edematous at times. For three days he had had moderate pain with some swelling at the base of the left thumb.

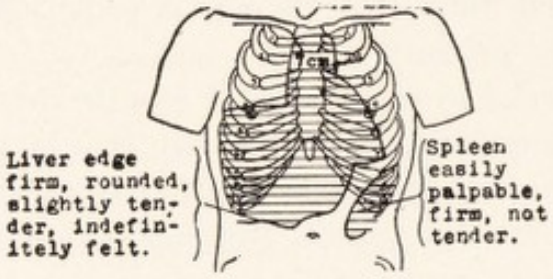



FIG. 49.

Examination showed a well-nourished man. The heart borders are shown in Fig. 49. The action was regular, slightly rapid, the sounds of good quality. Systolic and diastolic murmurs were heard over the entire precordia, the systolic short and rough, faint except over the region of the apex, the diastolic long, loud, rough in the aortic area and along the right sternal margin, elsewhere softer. There were no thrills. The left pulse was greater than the right. The artery walls were not felt. The blood pressure was 160/130–110/80. The lungs showed fine crackles at both bases. There was slight swelling, redness, and tenderness about the left carpophalangeal joint of the thumb.

After the day of entrance the temperature was 96° to 99°, the pulse 80 to 98, the respirations 17 to 32. The urinary output was 8 to 27 ounces, the specific gravity 1.022–1.013. There was the slightest possible trace to a very slight trace of albumin. Rare granular casts were seen at both of two examinations, hyalin casts at one. The hemoglobin was 85%, the leucocytes 12,000, the polynuclears 67%. The reds showed slight achromia and a number of large "transition-like" cells with nucleolae and granular pseudopodic-like

protoplasm. Two Wassermanns were negative. The fundi were normal. The stools were negative. X-ray showed the heart shadow symmetrically enlarged; no evidence of pathology in the lungs.

The joint condition cleared up rapidly, and the patient was comfortable until March 14, when he complained of slight sore throat and weakness. Next morning he suddenly became very dyspneic. His color was poor and his pulse barely felt at the wrist. He was given one-thirtieth of a grain of strychnia and soon afterwards one-hundredth of a grain of strophanthin intravenously. The pulse and heart sounds improved markedly, and with morphia he passed a fairly comfortable day. Next day the heart borders, which had gone 3 cm. to the left and 2 cm. to the right beyond the entrance examination, were back to the earlier measurements. During the night he sank gradually, and the morning of March 17 died.

*Clinical Diagnosis (from Hospital Record).*—Chronic endocarditis, aortic and mitral regurgitation.

Syphilitic aortitis?

Chronic dysentery. Amebic?

*Dr. Richard C. Cabot's Diagnosis.*—Acute endocarditis, aortic stenosis and regurgitation.

Hypertrophied and dilated heart.

Chronic passive congestion of the liver.

Slight chronic passive congestion, general.

*Anatomical Diagnosis.*—Acute and chronic endocarditis of the aortic valve, stenosis and regurgitation.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Slight ascites.

Slight anasarca.

Septicemia, streptococcus.

Obsolete tuberculosis of a bronchial lymphatic gland.

Adenocystoma of kidney.

Dr. Richardson: The patient was a well developed and fairly well nourished man. He had a slight amount of ascites, and the gastro-intestinal tract showed passive congestion.

There is a note that we measured the intestine; the small one was twenty feet six inches, the large one five feet three inches. At that time there was some talk about the varying length of intestine in different individuals, and they were trying to type into groups those who had a very long or a very short intestine to see if there was any relationship to length of life or liability to disease.

The liver showed chronic passive congestion in moderate amount.

The spleen was enlarged, weighing 480 grams, and so far as we could tell anatomically was like any spleen in chronic passive congestion except that it was rather larger than they usually are. That may be accounted for by his malaria.

The kidneys were large, one 214 grams and the other 190, (normally 200–400 grams together), but that excess in weight was due to edema. There was no glomerulo-nephritis and no arteriosclerotic nephritis.

The circulatory apparatus, the aorta and great branches, the pulmonary artery and veins were all negative. The heart weighed 470 grams,—moderate hypertrophy with slight dilatation. The myocardium was of good consistence, with no evidence of myocarditis, and the mitral, tricuspid and pulmonary valves were negative. The aortic valve had the usual three cusps, two of which were larger than usual, and all the cusps showed considerable fibrous deformity and erected on this chronic endocarditis an acute endocarditis. So that the heart valves showed acute and chronic endocarditis of the aortic valve, rheumatic in type.

There was no evidence anywhere in the body of syphilis.

There was obsolete tuberculosis of one bronchial lymph node.

There was 300 c.c. of fluid in the pericardium. It was otherwise negative.

In one of the kidneys was a very small tumor, an adenocystoma.

This case shows very well the anatomical basis for the rheumatic picture in the aortic valve.

DR. CABOT: There was nothing to account for his dying so suddenly?

DR. RICHARDSON: Nothing except the streptococcus infection. We did not examine the head. He had a source on the aortic valve from which emboli might have gone to the brain. There were no infarcts elsewhere.

### Necropsy 3290

A New England carpenter of thirty-one entered November 11th for the relief of dyspnea and precordial pain.

The family history was good except that his mother died at twenty-eight of "brain fever."

He took half a glass of whiskey, three to eight glasses of beer, and two boxes of cigarettes a day until a year and a half ago. Since then he had used none. He denied venereal disease.

A year before entrance, following an attack of "rheumatic fever," he was obliged to give up regular work on account of dyspnea, transitory edema, and sharp precordial pain. He had nycturiâ and polyuria, which disappeared when he took to bed, as he was obliged to do three weeks ago on account of aggravation of his symptoms. He now has marked dyspnea on slight exertion; orthopnea, eructation of gas, occasional distension of the abdomen, cough with clear, watery sputum sometimes containing purulent material, cold hands and feet, precordial pain at intervals, often sharp, sometimes dull, a "leaky-pump" feeling over the precordia, hoarseness, and dark urine. For the past three or four days he has had edema below the knees.

His best weight was 172 pounds, six years ago. Three months ago he weighed 153.

Physical examination showed a poorly nourished man, hoarse, dyspneic, and cyanotic. His skin was yellowish-pale, with many papules and pustules scattered over the back, chest and arms. There was also a bluish erythema over the lower back. The head and throat were not remarkable except for some pyorrhea and caries. The chest was barrel-shaped. There was systolic retraction of the nipple, also under the ensiform and posteriorly in the tenth and eleventh spaces.

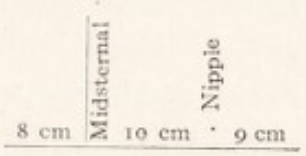



FIG. 50.—Borders of dullness.

The heart did not seem to shift with change of position. The precordia moved with every beat. The apex impulse was irregular, diffuse and heaving, faintly felt in the seventh space, 19 cm. to the left of the midsternum. The maximum impulse was in the sixth space, 15 cm. to the left. There was no supracardiac dullness. A presystolic thrill was felt over the apex. The sounds at the apex were very irregular, and not synchronous with the radial pulses. The first sound was replaced everywhere by a harsh systolic murmur, transmitted to the axilla and the back. There was also a short blowing diastolic murmur at the apex, and a diastolic over the aortic area. The aortic second was greater than the pulmonic second sound, and *both were greatly accentuated.* At the third left space the second sound was very loud and ringing, and there was also a diastolic murmur. The pulses were equal, irregular, not Corrigan, of poor volume and fair tension, and lost many beats of the heart. There was marked pulsation of the carotids and capillaries, and pistol-shot sound over the femorals.

The lungs showed nothing in front; slight dullness in the left lower back; in the right back flatness extending from the angle of the scapula downward, with diminished voice and breathing. Over both backs there were medium and coarse râles, most marked on the right. The abdomen showed liver dullness from the fifth rib to nine cm. below the costal margin, where a smooth, rounded, tender edge was felt. The spleen was barely felt. The genitals, pupils and reflexes were normal. The lower extremities and the sacrum showed edema. The fingers were cyanotic and slightly clubbed.

The temperature was usually 97. to 94.2. Pulse normal except for drops to 40 Nov. 30 and the day of death. Respirations 30 to 20. Systolic blood pressure 180 to 140, diastolic 120 to 60. Urine, amount usually 9 to 17 ounces, pink at entrance, turbid. Specific gravity 1026–1013. Albumin at four of five examinations, leucocytes at the first, granular casts at two, hyalin at the last three. No red blood corpuscles until the fifth. Renal function, in two hours, 45%. Blood: hemoglobin 80%, leucocytes 12,400–8800, polynuclears 50%. Blood culture, no growth. Two Wassermanns negative. Feces, guaiac negative. Laryngoscopic examination showed complete paralysis of the left recurrent laryngeal nerve.

The heart sounds became a little more regular, and the patient grew more comfortable and made a little general improvement, though he was troubled by gas, vomiting, and cramp-like pain in the abdomen, relieved by soda. After he had been sitting up because of these symptoms his legs became badly swollen, and by Nov. 22 showed peculiar reddish patches on which a skin consultant could not make a definite diagnosis, but which faded in a few days.

The patient was given two digipuratum tablets daily at entrance, reduced to one tablet Nov. 20. After the drop in pulse Nov. 30 this was stopped, and he felt better than for some time. By Dec. 3 he had been worrying and was considerably worse, so weak and dyspneic that he could not be taken home. The pulse was very irregular and the heart action extremely arrhythmic. The digipuratum was started again. The arrhythmia increased, the patient grew more and more restless, the skin at the ankles broke. Dec. 5 the temperature rose from 94.2 to 98.4. The murmurs were not so constant, but there was no change in the size of the heart. Dec. 6 he suddenly sank back and died.

*Clinical Diagnosis (from Hospital Record).*—Chronic and acute aortic and mitral endocarditis.

Adhesive pericarditis.

Chronic passive congestion.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the mitral valve, stenosis.

Slight if any change in the aortic valve.

Hypertrophy and dilatation of the heart. It may be due to adhesive pericarditis possibly. But I think it is due to chronic glomerulonephritis, because of the high blood pressure, though this is a dangerous amount of weight to put on one support.

Chronic passive congestion, general.

*Anatomical Diagnosis.*—1. Chemical or physical origin of fatal illness.

Chronic endocarditis of the mitral and aortic valves.
Chronic adhesive pericarditis.

2. Secondary or terminal lesions

Hypertrophy and dilatation of the heart.
Chronic passive congestion, general.
Infarct of the lower lobe of the right lung.
Hydrothorax.
Ascites.
Anasarca.

The heart and pericardium together weighed 1205 grams, four times the normal weight. The heart alone weighed 1140 grams. The myocardium was rather thick, 12 mm. on the left ventricle and 5 on the right. The auricular walls also were thickened. All the cavities were greatly enlarged. The mitral valve measured 12 cm. Its usual measurement is about 10. The aortic valve measured 8.5 cm. Both the tricuspid (15.5) and the pulmonary (10) were increased. The mitral curtain showed considerable thickening. The aortic showed considerable fibrous change in the cusps, which were stiffened, giving regurgitation without stenosis.

The liver and kidneys were large, the liver nutmeg. There was no nephritis.

### Necropsy 3333

A teamster of forty-five came to the hospital February 24th for relief of dyspnea and hemoptysis.

His family history was good.

Two children had died at birth. His wife had had two miscarriages. His wife and two children were well.

At eighteen he was in bed three weeks with "slow fever" accompanied by abdominal pain. He had had no chorea or tonsillitis. Fourteen years ago he had "rheumatism" in ankle and toe-joints,

with some fever. He was unable to work for two or three months but was not sick in bed.

He drank five to seven glasses of ale daily, not much whiskey during the day, occasionally one before breakfast. He had gonorrhea at twenty-two. He denied syphilis.

Four years ago he first noticed dyspnea on carrying furniture upstairs. This increased until any carrying or excitement made him so weak and dyspneic that he had to stop for breath. For the past year he had had occasional violent cough, with sputum. For the past six months he had had palpitation. For six weeks he had been so weak and dyspneic that he had worked only three days, was in bed most of the time, and orthopneic. He had had edema of the legs but not of the abdomen.

The night before admission he had a sense of pressure in the epigastrium. The following morning he awoke with severe precordial pain, which continued two hours. The dyspnea and palpitation were worse. He had hemoptysis for the first time.

His appetite was fair. His bowels moved daily with cathartics. Nycturia once. His best weight was 145 pounds; he then weighed about 138.

Physical examination showed a poorly nourished man with labored breathing. The skin was slightly cyanotic, the mucous membranes pale. There was much pyorrhea.

The entire precordia pulsated. The left chest was more prominent than the right. The apex was in the sixth space. The left border of dullness was 17 cm. to the left of the midsternal line in the anterior axillary line. The right border of dullness was 4 cm. to right of the midsternal line. In the left lateral position the apex shifted 2 cm. The sounds were rapid, regular, of rather poor quality at the apex, where the first sound was weak and followed by a harsh systolic murmur transmitted to the axilla. At the base was a diastolic murmur loudest at the third left space, transmitted toward the apex and along the sternal border. A high-pitched systolic almost replaced the first sound at the lower sternum. The second sound at the base was absent. There was a systolic at the base transmitted toward the carotids. The pulses suggested the water-hammer. The vessel walls were barely palpable.

The lungs showed evidence of congestion, especially at the right. The abdomen was slightly rigid. There was shifting dullness in both flanks, no masses, but spasm in the right upper quadrant. Liver dullness was from the fifth rib to four cm. below the costal

margin. The edge was not felt (but see below), There was slight varicocele on the left, where the testis was small and tender, and slight edema of the scrotum. There was marked edema of the lower legs. Both legs showed slight varicosites. The left pupil was greater than the right. The reflexes were normal.

The temperature was usually subnormal until the 19th day; then 100–102 until death three weeks later, except for four days subnormal. Pulse 90–140. At times many beats did not reach the wrist. Respirations 20–40. Systolic blood pressure 135–165, diastolic 70–80. Two weeks before death, systolic 130, diastolic 65. The urine was normal in amount until the 19th day, then usually 6–15 oz. Specific gravity 1025–1032. The slightest possible trace to a trace of albumin, occasional hyalin and granular casts with cells attached, and a few red blood corpuscles. Renal function, first hour 30%, second hour 15%. The blood showed leucocytes 12,000 to 78,000, polynuclears 83%. The Wassermann was negative. Luetin was negative. Blood culture gave no growth. The sputum was bloody at two of three examinations, showed pneumococci at three examinations, also streptococci at one of these. On the 28th day there was typical rusty sputum. X-ray of the chest on the ninth day showed opacity at both bases, the angles between the diaphragm and the chest wall obliterated. A seven-foot plate of the heart showed the heart enlarged to the left, its greatest diameter 15.5 cm. The greatest transverse diameter of the great vessels was five cm. The apex could not be made out. X-ray on the twenty-second day showed the left chest opaque throughout, especially at the base. That side of the chest was contracted. The heart was displaced to the left. There was considerable thickening about the right lung root. The diaphragm could not be seen on the left, was indistinct on the right. There was no positive evidence of tuberculosis. X-ray on the following day was the same except that the opacity at the left base rose as high as the middle chest. The outline of the diaphragm was indistinct on both sides and the angles between it and the chest wall were obliterated. No movement could be seen with respiration. The heart shadow was much enlarged and triangular in shape. The cardiohepatic angle was obliterated. Very little pulsation was made out. (Evidently the radiologist suspected chronic pericarditis.) The lung apices were normal. Chest tap on the twentieth day gave 1200 c.c. of bloody fluid with a specific gravity of 1015, 2% of albumin, and abundant cells nearly all polynuclears. Culture gave no growth.

On entrance the patient was very restless and dyspneic. He was put on digipuratum and much morphia. The edema disappeared and the liver edge became less tender. Amyl nitrite and nitroglycerine gave only slight and transient relief to the sudden attacks of dyspnea accompanied by pain in the chest.

Under strophanthin, however, there was marked improvement. The pulse and heart action steadied somewhat, and the dyspnea and cyanosis became much less marked. Morphia was no longer needed. The patient then began to vomit, and the diastole was seen to be lengthened. The strophanthus was omitted, with cessation of vomiting. The first sound at the apex became sharp, and there was an occasional presystolic murmur. The fluid in the right back decreased.

On the eighteenth day the temperature began to rise. Dullness increased in the left lower back, where there was faint bronchial breathing, many fine râles, and egophony. The chest tap on the twentieth day gave much relief. On the twenty-third day hemoptysis began.

During the next week the temperature remained elevated. The signs in the left back were essentially the same, and there seems to have been only slight accumulation of fluid. The heart sounds became much more distant, except the first, which was lost. The murmurs became variable, with a presystolic at the apex. There were many extra systoles. The patient raised large amounts of sputum showing blood and pus. The urine output was very low, the temperature high, and the general condition poor. The patient had become very pale and much thinner, although he ate and slept well without morphia, and was up in a chair.

At a second chest tap on the twenty-eighth day of 200 c.c. of bloody straw-colored fluid was recovered. Six days later the chest was again tapped, with recovery of 30 c.c. of brownish turbid fluid. A smear showed polynuclear predominating.

On the thirty-fifth day Dr. W. H. Smith's examination of the sputum showed moderate numbers of pneumococci and streptococci, no tubercle bacilli. Such a sputum in Dr. Smith's opinion would correspond with chronic passive congestion or infarction. Three days later the left back showed tympany at the extreme base, above that flatness, egophony, faint bronchial breathing, and many fine râles extending up to midscapula. The heart sounds were very faint. The fluid last withdrawn from the chest showed on culture pneumococcus. Dr. Smith considered the signs in the left back as due to a septic infarct or a pneumonia with abscess.

On the fortieth day the patient died.

*Clinical Diagnosis (from Hospital Record).*—Chronic valvular heart disease, aortic and mitral.

Empyema.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis with aortic stenosis and regurgitation and mitral stenosis and regurgitation.

Acute endocarditis and pericarditis, with effusion.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Pneumonia.

Probably infarcts of the lungs, liver, kidneys, and spleen (but no peripheral embolism).

Empyema (beginning).

*Anatomical Diagnosis.*—Chronic endocarditis of the aortic and mitral valves. The aortic measured 8.5 cm. in circumference, the mitral 10.5 cm. The aortic showed fibrocalcareous deposit extending into the mitral, reducing their opening function,—really stenosis, despite normal measurements.

Acute endocarditis of the aortic valve, slight.

Hypertrophy and dilatation of the heart. (Weight 595 grams.)

Serofibrinous pleuritis, right.

Empyema, left. (500 c.c. at base and in the interlobar fissure.)

Chronic passive congestion, general. Ascites. Anasarca.

Small infarct of the right kidney.

Infarcts of the right lung.

Compression atelectasis of the left lung. (500 c.c. of pus.)

Defective closure of the foramen ovale.

Chronic pleuritis.

Diverticulum of the esophagus (congenital).

Arteriosclerotic degeneration of the kidneys (i.e. foci of arteriosclerosis. Together they weighed 350 grams.)

### Necropsy 3527

An Irishman of seventy-four entered December 4 for relief of a skin condition.

His family history was good so far as known.

He had had "smallpox," "scrofula," and whooping cough in childhood. Fifty years ago the lens of his right eye was injured by a blow. For a year he had been slightly deaf. For three weeks he had had edema of the feet.

His habits were good, except for nycturia one to two. He used no alcohol or tobacco, and denied venereal disease.

Two years before entering the hospital a small itchy, scaling area appeared on his left instep, called eczema by his doctor. It remained confined to this area for a year. Then it appeared on both upper arms. Local treatment was ineffectual. Two months ago his whole body became involved in the scaling. The itching, however, had become much less intense. For two weeks he had had alternate periods of three or four days of improvement and relapse.

Physical examination showed a fairly nourished old man, slightly dyspneic. The skin of the face, neck and scalp was slightly red and infiltrated, and was desquamating large flakes. On the left side of the neck was a crusted lesion the size of a twenty-five cent piece. The borders were pearly and indurated. (This lesion was of a year's duration.) The skin of the rest of the body was dark red, of porky consistency. On the abdomen and chest it was moist and oozing, with several small scattered vesicles. On the back it was infiltrated, but showed less redness and oozing. Over the left clavicle was an irregular scar three inches long (remains of scrofula). At the level of the fifth thoracic spinous process was attached a pedunculated cyst the size of a walnut. The skin of the legs was infiltrated, red and scaling. On the dorsum of the right foot was an old scar an inch long. The apex impulse of the heart was in the fifth space. At the apex was a loud, rough, systolic murmur, transmitted to the axilla; also a short, rough presystolic murmur. In the fourth space was a loud, blowing, diastolic murmur. The aortic second sound was slightly accentuated. The rate was normal, but there were occasional extra systoles. The lungs were normal except for slight dullness at the left space. The liver dullness extended from the fifth rib to the costal margin. The edge was felt two finger-breadths below the costal margin on deep inspiration. The genitals were negative. There was slight hard edema of the lower legs and feet. The pupils were equal, regular, reacted slowly. The knee-jerks were equal and active.

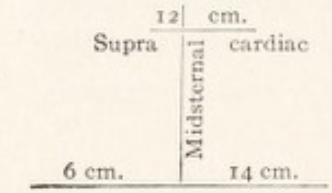


FIG. 51.—Dullness.

Temperature normal. Pulse 100 to 118. Respiration 19 to 28. Blood pressure not recorded. The amount of urine was not recorded. The specific gravity was 1015, the findings negative. The blood showed hemoglobin 80%, leucocytes 19,800, polynuclears 72%. The reds showed considerable achromia.

On the night of December 5 the patient was slightly dyspneic and complained of some abdominal distress, which was attributed to

the skin condition. At one a.m. he had an attack of sharp pain in the heart region. Half an hour later he was in intense agony, with P. 140, R. 50. He was given morphia gr. 1/6 and nitroglycerin gr. 1/100. The attack continued until 5:40, when morphia gr. 1/6 was again given. On being questioned he now said he had had attacks of this sort, but much less severe, for the past four or five weeks. A medical consultant advised giving nitroglycerin gr. 1/200 or 1/400 every hour or two for the attacks, increasing dosage and frequency for acute pain; if still uncontrolled, morphia. The prognosis he considered entirely uncertain.

December 6 the patient vomited clear greenish fluid at intervals all day and all night. He became semi-conscious and irrational, later restless and dyspneic, requiring morphia. Then the breathing became Cheyne-Stokes, and in ten minutes he died.

*Clinical Diagnosis*( *from Hospital Record*) Dermatitis exfoliativa.

Angina pectoris.

Toxemia.

*Dr. Richard C. Cabot's Diagnosis.*—Arteriosclerosis.

Hypertrophy and dilatation of the heart.

Incompetent aortic valve.

Coronary sclerosis, plugging one or more coronary arteries.

*Anatomical Diagnosis.*—Dermatitis exfoliativa.

Fatty metamorphosis and siderosis of the liver.

Fibrous and verrucose endocarditis of the aortic valve with some deformity.

Hypertrophy and dilatation of the heart. (Weight 368 grams.)

Hydropericardium.

Edema of the lungs.

Chronic peritonitis (diffuse).

Chronic perihepatitis.

Chronic perisplenitis.

Healed appendicitis.

Chronic pleuritis.

Slight arteriosclerotic degeneration of the kidneys.

Small accessory spleen.

The coronary arteries were free. The aortic valve measured 7.5 cm. There was no general arteriosclerosis. The aorta above the cusps was smooth. The anginoid attacks were not explained.

## MITRAL REGURGITATION

**Does It Exist?**—The commonest of all diagnoses in the field of heart disease is mitral regurgitation. This statement can be easily

verified by reference to the reports of any hospital that lists the diseases treated there, or to almost any statistical record dealing with the different varieties of heart disease. Yet most of these diagnoses are not in my opinion justified for it is a lesion almost never to be verified post-mortem. In our 4000 necropsies we have been able to find but seven cases, three of which are more or less doubtful.

What is the reason of this astonishing discrepancy? (1) The chief reason is, I think, that physicians diagnose mitral regurgitation when they hear loud apical systolic murmurs, which are extraordinarily common in all sorts of non-cardiac disease as well as in health. Thus in Roger I. Lee's experience, gained by examining healthy Harvard students over a considerable period of years, 70% of unselected normal individuals showed a definite systolic murmur over the heart in the recumbent position after strong expiration. In children the percentage of such murmurs is I believe even greater.

(2) When a systolic apical murmur happens to be associated with pain about the heart, with breathlessness, fainting spells, or with any of the various nervous manifestations summed up under the term of "the effort syndrome," a diagnosis of mitral regurgitation is very likely to be made by the incautious, even though it can be proved beyond all question that the heart is in fact sound.

(3) In elderly people systolic murmurs are also very frequent and are sometimes heard best at the apex of the heart, though usually loudest at the base. It is impossible to say how far these murmurs may be related to arteriosclerosis or to stretching of the aortic arch, which is usually sclerotic in the elderly. But at any rate there is no good reason to suppose that these systolic murmurs indicate any insufficiency of the mitral valve.

(4) Besides these conditions in which systolic murmurs with normal valves are so common, it is well known that a great variety of physiological or pathological states such as *fever*, *anemia*, *muscular exertion*, *nervousness*, are also associated with the transient appearance of systolic murmurs or with an increase of intensity in murmurs already present.

The pernicious habit of diagnosing mitral regurgitation in people with sound hearts became of great importance during the war of 1914–1918. In the early years of that conflict large numbers of men were rejected, especially in England, because a systolic murmur was heard over the heart, a murmur often associated with pain, breathlessness, tachycardia, or something else which misled the examiner. Such exemptions became so frequent as to constitute a serious handicap to a country needing all her healthy young men in the crisis of war.

Fortunately the mistake was discovered, and before long the rule was put into effect that no man should be rejected *merely* on account of a systolic murmur, no matter how loud it might be. Diastolic or presystolic murmurs were still considered proper grounds for rejection. But these, on account of their relative rarity, made no serious inroad upon the body of available recruits. When America at last entered the war the special cardiac examiners appointed in the United States to judge the American draft army, were able to profit by the experience of the English examiners and to avoid the bugaboo of "mitral regurgitation" as a cause for rejection. So far as I know no bad results have been reported as a result of carrying out this policy either in England or the United States.

Similar conclusions have been arrived at by competent physicians who have been called upon to judge the fitness of athletes for competitive contests. The presence of a systolic murmur without other evidence of cardiac disease has been found to involve no functional weakening of the heart, no lessening of the athlete's powers, and no evil results either immediately after the contest or in later years. Judged by these most searching and practical tests—war and athletic contests—which certainly bring upon the heart as great a strain as any of life's exigencies, it is now clear that *systolic murmurs without other signs of cardiac disease are of no importance as evidence of valve lesions*. They may of course be helpful auxiliary evidence of hypertension, or of arteriosclerosis.

It is easier to settle this point than it is to decide beyond peradventure what *is* the cause of these murmurs, or whether they are *ever* associated with a genuine regurgitation through the mitral valve. On this point the evidence may be summed up as follows:

(1) *Post-mortem* findings show that the scarring and deformity produced by endocarditis on the mitral valve leads almost invariably to *stenosis* of that valve, and only in the rarest instances to regurgitation *without* stenosis.

(2) Relative mitral insufficiency due to stretching of the valve orifices from muscular weakness or other causes remains a *possibility* which even *post-mortem* examination cannot exclude,—a hypothesis which can never be proved or disproved. But against it one may say:

(a) That many cases of enlargement of the mitral orifice are demonstrated *post-mortem* when no murmur or arrhythmia has been detected during life and no cardiac enlargement, passive congestion, or other evidence of poor heart function is found after death.

(b) That in many cases a loud apical systolic murmur, even though transmitted to the axilla and back and occasionally even

musical in quality, goes along with a mitral valve orifice of normal size, with an absolutely sound valve and no passive congestion as revealed at *post-mortem* examination. This does not disprove the possibility of a *temporary* stretching of this valve with transient regurgitation during life. This could never be disproved so far as I see unless one could somehow visualize the movements of the cardiac blood during life. But I think it can be asserted that there is no evidence for such relative and temporary insufficiency, and that even if it exists there is no reason to believe that it does any harm.

. . .

It is really astonishing how long the legends about mitral regurgitation have persisted and on what good authority they have rested. Thus in Osler's textbook of medicine we find the following description of the *post-mortem* lesions believed by him to underlie mitral regurgitation:

"The common lesions producing (mitral) insufficiency result from endocarditis, which causes a gradual thickening at the edges of the valves, contraction of the chordae tendineae, and the union of the edges of the segments, so that *in a majority of the instances* there is not only insufficiency but some grade of narrowing as well. Except in children we rarely see *the mitral leaflets curled and puckered without narrowing of the orifice* . . . In longstanding cases the entire mitral structures are converted into a firm calcareous ring."* (Italics mine.)

For these "mitral leaflets curled and puckered without narrowing at the orifice," I have been searching for the last twenty years (a) at necropsies, (b) in the records of necropsies which I have not myself seen, and (c) by questioning various pathologists. Osler's description aroused a vivid picture in my mind. But I have rarely if ever found it, and know of no one else who has seen a reality corresponding to it except in the rarest instances. Perhaps in ulcerative endocarditis such a state of things may occasionally exist. But *in chronic non-ulcerative heart disease it is certainly so rare as to be negligible clinically.*

One may inquire why it is that as a matter of observation in any series like the present, mitral endocarditis leads so commonly to stenosis of the valve and so rarely if ever to regurgitation. The reason appears to be that most of the bacteria and platelet thrombi, in other words the soft vegetations, in which endocarditis begins, are deposited *near the free margin of the valve.* When these vegetations become organized and healed into scar tissue, the traction of the adhesions so produced, pulls together the contiguous parts

* Osler and McCrae: The Principles and Practice of Medicine, p. 810 (1923).

of the mitral orifice like a purse-string, and so produces, not pure regurgitation but stenosis with (presumably) regurgitation as well.

In many cases there are also small vegetations and scars upon the papillary muscles and upon the tendinous cords as well as upon the valve curtains. If mitral regurgitation ever exists it is probably in the rare cases when the endocardial vegetations are *confined largely or wholly to the papillary muscles*, so that in the end we get no adhesion of the cusps to each other, but only a shortening and thickening of these muscles and of their tendinous cords. Such a change would pull the mitral curtains back towards the ventricular wall, so that they could not rise up to close. Yet it would produce no stenosis in them.

Something of this kind was probably present in Necropsy 2341, possibly in 3320 of our series (see below). I have myself seen one case (not in the Massachusetts Hospital series) in which almost exactly the conditions above described were fulfilled. There was no ulceration and almost no thickening of the mitral curtains, but the papillary muscles and tendinous cords were so much shortened and thickened that the valve could hardly float out from the heart wall. In Necropsy 3092 the pathologist evidently believed that a similar condition was present. In Necropsy 2825 the lesion was distinctly different from that which I have described and represents a very peculiar, perhaps unique, state of things. (See below.)

**Can Mitral Regurgitation Be Diagnosed in Life?**—But granting that mitral regurgitation probably exists as a great rarity, it is not a clinical entity, for it cannot, so far as I see, be recognized in life. The classical trio of signs (systolic murmur widely transmitted from the apex, accented pulmonic second sound, enlarged heart) which have been supposed to point to mitral regurgitation, have occurred many times in this series without any evidence *post-mortem* that the mitral valve was diseased or incompetent. In most of the cases just referred to, necropsy showed a hypertrophied and dilated heart, often associated with arteriosclerosis or chronic nephritis, but with a normal mitral valve and no dilatation of its ring.

Mackenzie has told us how he gradually came to recognize that no one ever *died* of mitral regurgitation. Even if one admits that there *may* be in many cases a relative mitral reflux by muscular relaxation around a sound mitral valve, even then the main cause of death will usually be found to lie in the nephritis, the hypertension, the general infection, or the anemia which brought about this relaxation.

The old point of differentiation between functional and organic murmurs heard best at the cardiac apex, namely the transmission of

the supposedly organic murmurs to the left axilla and back, has been proved to my satisfaction, in the necropsies on which this book is based, to be false. I have heard many such murmurs which showed nothing particular to account for them at necropsy, not even cardiac enlargement, no increase of the valve circumference, and no disease of the valve itself. Indeed the wide transmission of a murmur is merely an evidence of its loudness. All loud murmurs are widely transmitted. Some of the murmurs in congenital heart disease can be heard over the extremities and even on the top of the head. A few of them can be heard at a distance from the patient. Yet such murmurs may have very little effect upon the cardiac function and be therefore of very little practical importance. On the other hand, some very faint diastolic murmurs are evidence of crippling heart disease. The worse the heart grows the fainter, in many cases, the murmur, while if improvement occurs one may watch the murmur grow louder and louder as the patient regains his powers. (In all probability this means that a faster and stronger current of blood will make more noise than the feeble current in an exhausted heart, just as a powerful river current makes more noise than a feeble one.)

In a recent article by Breed and White* organic mitral regurgitation is considered the proper diagnosis when one hears "a loud systolic murmur at the apex masking the first sound." To this belief I see the following objections:

(1) Most if not all cases of mitral stenosis must be conceived as having regurgitation also. But they often have clear, or even exaggerated, first sounds. Hence mitral regurgitation under the only circumstances wherein we can often feel reasonably certain that it exists, usually does not abolish the first sound.

(2) In many cases of this same group the systolic murmur referred to by Breed and White is not loud but is faint or absent.

(3) In our rare proved or suspected cases of mitral regurgitation the first sound was sharp in one, poor (like the second sound) in two, not definitely recorded but probably present in two and definitely replaced by the murmur in only one out of seven.

(4) In many of our cases proved post-mortem to have normal heart valves and normal valve orifices, there has been heard in life a loud apical systolic masking the first sound.

(5) In many cases of mitral stenosis in which we find post-mortem such rigidity of the half-open mitral orifice that we must believe that regurgitation (as well as stenosis) existed in life, no systolic murmur

* Boston Medical and Surgical Journal, June 21, 1923.

has been audible in life though it is the murmur most eagerly sought for and most easily recognized by most examiners.

. . .

I have gone over these points at considerable length and in detail because I know nothing of greater practical importance in the field of cardiac diagnosis than to diminish, as far and as fast as we can, that very considerable number of persons whose lives are now rendered ineffective and miserable by a false diagnosis of heart disease, usually of mitral regurgitation, when in fact their hearts are perfectly sound. I believe such false diagnoses to be extremely common. I know that they produce great mental torture and bodily discomfort. Out of eighteen successive cases coming under my observation recently with a diagnosis of heart disease, only four had, in my opinion, any good evidence of such disease. The other fourteen had systolic murmurs or cardio-respiratory murmurs, systolic in time, with no history of rheumatism, no enlargement of the heart, no hypertension, no arrhythmia, no thrill, and no evidence of passive congestion either in the history or in the physical examination.

In life insurance examinations this certainly is a point of real importance. It is a serious thing to be refused life insurance because of the false interpretation of a systolic murmur. It is also serious for a growing boy to be refused permission to enter athletics and to gain all the advantages, mental, moral, and physical, which athletics may bring. All this boys often lose now because an athletic instructor or an ill-trained physician has told them that their hearts are diseased. Nearly 10% of a recent freshman class at Harvard College was composed of men who believed themselves, quite falsely, to be suffering from heart trouble. They had been told so by their physicians. Yet on careful study their hearts showed nothing significant of disease, or nothing but the systolic murmurs about which so much has been said in this chapter.

## SUMMARY AND CONCLUSIONS

1. Mitral regurgitation without stenosis is the commonest diagnosis now made by American physicians in cases of real or suspected heart disease.

2. Yet this lesion is exceedingly rare *post-mortem;* only 7 cases, 3 of them doubtful, were found in 1846 necropsied cases of heart disease. In the same series there were 107 of mitral *stenosis*.

3. Pathologically the lesion is probably due to an endocarditis confined or nearly confined to the chordae tendineae and papillary muscles.

4. Even in rare cases wherein mitral regurgitation without stenosis does exist, there are no physical signs by which it can be recognized or reasoned out, so that a diagnosis of mitral regurgitation without stenosis is never justified.

5. For the proper evaluation of national strength in time of war, for the proper education and development of growing boys and girls, for the fair and profitable adjustment of life insurance, and for the happiness and comfort of all concerned, it is essential that we should get this matter straight. I am very glad to believe that with the rapid development of cardiac clinics in many cities of this country, and the better teaching of physical diagnosis which is now, I think, in sight, we may see a notable improvement in this matter which will date, I think, from the war of 1914–1918, and constitutes one of its best results.

## SEVEN CASES OF THAT GREAT RARITY—MITRAL REGURGITATION

Because of the beliefs just expressed and because I believe pure, uncomplicated mitral regurgitation to be an exceedingly rare lesion, if indeed it exists at all, I have given in detail all the cases of our series which show any evidence of such a lesion. These cases follow herewith.

Of the seven cases of this series, 5 occurred in males and two in females. Five were above the thirty-sixth year. A rheumatic history was obtained in only one case. The patients presented themselves with the ordinary symptoms of decompensated heart trouble. In the physical examination cardiac enlargement with a systolic murmur were the chief data bearing upon a possible mitral lesion. Two cases were thought in life to have mitral stenosis also (282, 2825). In one case there was another murmur referable to an associated aortic stenosis. I have been interested to note that while textbook descriptions—including those given in the earlier editions of my own book on *Physical Diagnosis*—list an accentuated pulmonic sound as one of the cardinal points in the diagnosis of mitral regurgitation, this sign was present in only three cases of our series and one case observed outside the hospital. In three cases the pulmonic second sound was notably weak or absent, as is often the case in mitral stenosis. Possibly the explanation is the same in both groups. Detailed histories of these cases follow.

TABLE 74.—ANALYSIS OF 7 CASES OF MITRAL REGURGITATION (ACTUAL OR POSSIBLE)

| Case No. | Necroposy No. | Age | Sex | Rheum. history | Heart enlarged in life | 1st heart sound | P2 | Apical systolic murmur | Heart weight in grams | Post-mortem |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 2341 | 49 | M | 0 | 0 | ..... | 0 | loud | 422 H + D | M.* 14.5. Tendinous chords short and thick. Doubtful case. |
| 2 | 3092 | 48 | M | 0 | sl. + | gone | + | loud | H + D | M. 12, otherwise normal. Chronic passive congestion. Chronic pericarditis. Doubtful case. |
| 3 | 2825 | 37 | M | 0 | much + | sharp | ? | loud | 600 | M. 17.5. Calcareous column at base of one cusp. |
| 4 | 3320 | 74 | M | 0 | mod. + | ..... | faint | loud | 558 | M. 9.5, curtains mod. thick, chords sl. thick. Aortic stenosis. Doubtful cases. |
| 5 | Outside case | 17 | F | + | much + | poor | + | soft | 500 | M. 13, its curtain and chords thickened, chronic passive congestion. |
| 6 | 797 | 37 | F | 0 | 0 | double | sl. + | sl. | 360 | M. 10. Chords shortened and thickened. Acute endocarditis. |
| 7 | 282 | 13 | M | ? | + | ..... | + | rough also diast. & presyst | 625 | M. 13. One-half of curtain much shortened, slightly thickened; minute acute granulations along its edge. |

*In this column, M. means "mitral circumference in centimeters."

### Necropsy 2341

An American carpenter of forty-nine came to the Accident Room of the Massachusetts General Hospital, March 31, 1909.

He had had the diseases of childhood, gonorrheal urethritis as a youth, chancre followed by sore throat and eruption at twenty, "inflammation of the bowels" with vomiting, fever and tender abdomen laying him up for two weeks at twenty-two, and "in early life." an attack of jaundice.

Two and a half weeks before admission ne was seized with general abdominal pain and diarrhea. March 25 the diarrhea was "checked by blackberry wine." Since then his bowels had had to be moved by enemata. The pain had continued, not localized. For forty-eight hours he had been vomiting continuously, for the last twelve hours a greenish material.

Examination showed the apex impulse of the heart in the nipple line, fifth space. At the apex was a loud systolic murmur, heard also over the whole precordia and transmitted to the axilla. The second sounds were not made out. The abdomen was slightly distended. On the left side was some deep seated tenderness, but very little spasm. On the right there was considerable rigidity of the abdominal wall, and tenderness which was extreme in the right lower quadrant, where there was an area of dullness. The knee-jerks were lively. There were varicose veins in the left leg. The rest of the examination showed nothing of importance. The temperature before operation was 101.4°, the pulse 102, the leucocytes 25,000.

Operation, which was immediately done, showed a great deal of thin very foul-smelling pus in the peritoneal cavity. The appendix was found buried in a mass of adhesions, perforated near its tip, and very friable, as was the surrounding tissue. In removing the appendix it was torn in two. In making a stab wound for drainage the deep epigastric artery was cut and so tied off. The pathological report on the appendix was gangrenous appendicitis. Culture of the pus from the peritoneum showed a scum of growth of bacilli and micrococci.

The patient was sent to the ward in good condition and was put in semi-sitting posture with rectal seepage. He spent a comfortable night, but the following night became very much worse. The pulse became more rapid, 122–135, poorer in quality, and intermittent. The temperature rose from 99° to 104°. The respirations ranged from 36 to 40. April 3 he died.

The clinical diagnosis was appendicitis with abscess and general peritonitis.

Necropsy showed

Acute diffuse fibrinopurulent peritonitis,

Acute pleuritis,

Fibrous endocarditis of the mitral valve,

Syphilitic aortitis,

Hypertrophy and dilatation of the heart,

Hypoplasia of the left kidney,

Compensatory hypertrophy of the right kidney.

The heart weighed 422 grams and showed hypertrophy especially of the left ventricular wall. The four cavities were all enlarged. All the valves were enlarged: tricuspid 14 cm., aortic 8 cm., pulmonary, 9 cm.

The mitral valve measured 14.5 cm. Its circumference greatly enlarged, its curtains generally thickened. "Much fibrous thickening produced rather marked areas of elevation and depression *with shortening and thickening of the chordae tendineae.* (Italics mine.)

The other valves were not remarkable.

*Discussion.*—We have here no evidence of passive congestion *post-mortem* and no symptoms suggesting it in life. Yet the heart is enlarged and none of the ordinary extracardiac causes of hypertrophy are present. It is notable that the hypertrophy is if anything more marked on the left than on the right side. Yet both ventricles are thickened, and we have abundant evidence that such *general* cardiac enlargement is often associated with mitral disease. The syphilitic aortitis which was present did not involve the aortic valve or produce any other change which should cause cardiac hypertrophy.

Was the mitral valve a cause of trouble in this man's circulation? It is hard to say. Since all the valve orifices probably became enlarged with the terminal infection following operation, the cardiac murmur may have been produced at one of the other orifices and may have had no connection with the demonstrated fibrous endocarditis of the mitral. The latter may belong with our series of non-deforming valve thickenings (Chapter VIII).

Yet on the whole and especially in view of the lesions in the chordae tendineae, it seems to me that this *may* be a genuine case of mitral regurgitation, though slight in degree.

### Necropsy 3092

An American railway engineer of forty-eight entered August 21, 1912. His father died with dropsy of unknown origin. The

patient had gonorrhea at nineteen, double pneumonia in 1898, measles, mumps, and chicken-pox between the ages of thirty and forty. Until eight months before admission he was a heavy smoker. He also chewed tobacco. He occasionally drank beer. He slept poorly.

Ever since his pneumonia in 1898 he had had slight edema of the ankles at night, some cough with a little yellowish sputum, and had slept on four pillows. For the past four years he had had some dyspnea on exertion. For two years he had urinated twice at night. A year and a half before admission he began to have edema of the legs. He stopped work for two weeks. The swelling gradually subsided and he was able to resume work. Beginning in December, 1911, he was laid up for three months with a return of the edema. After working again for a few days the swelling returned and had persisted, growing more extensive and brawny. His hands had been slightly swollen for six weeks. He had been in bed most of the time for the past five weeks. The least exertion caused dyspnea. He belched considerable gas. The morning of admission he noticed cyanosis. In 1900 he weighed 185 pounds, five months before admission 146.

Examination showed a very poorly nourished man with cyanotic skin and mucous membranes. The respiration was somewhat labored, with both expiratory and inspiratory dyspnea. There was some pyorrhea. The apex impulse of the heart was faintly felt in the fifth space 12 cm. to the left of the midsternal line, one cm. outside the nipple line. There was no enlargement to the right. The action was regular. The first sound at the apex was replaced by a loud blowing systolic murmur heard over the entire precordia, loudest at the apex, transmitted to the axilla. There was slight dullness above the precordia on both sides of midsternum. The sounds at the base were very distant, the pulmonic second sound accentuated. The pulses were of fair volume and low tension. The artery walls were not felt. The blood pressure was 100/80. The expansion of the lungs was poor but equal. There was marked retraction of the interspaces with inspiration. The accessory muscles of respiration were employed. There was hyperresonance throughout, with diminished "emphysematous" breathing. Fine to coarse moist râles, piping and squeaking râles were heard throughout. The abdomen was full, distended. There was marked ascites. (See Fig. 52.) In the flanks was shifting flatness and a fluid wave. The liver dullness extended from the sixth space to 4 cm. below the costal margin. A non-tender edge was indefinitely felt. There was great edema of the

scrotum, slight edema of the penis, very marked brawny edema of the upper and lower legs, slight soft edema of the hands, wrists, and abdominal wall, moderate soft edema of the lower back. The fingers were clubbed, the nails cyanotic. The pupils and reflexes were normal.

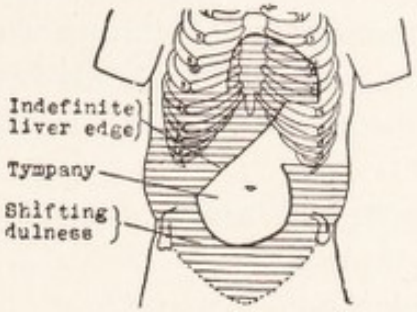



FIG. 52.—Dullness, abdominal and thoracic, in case 3092.

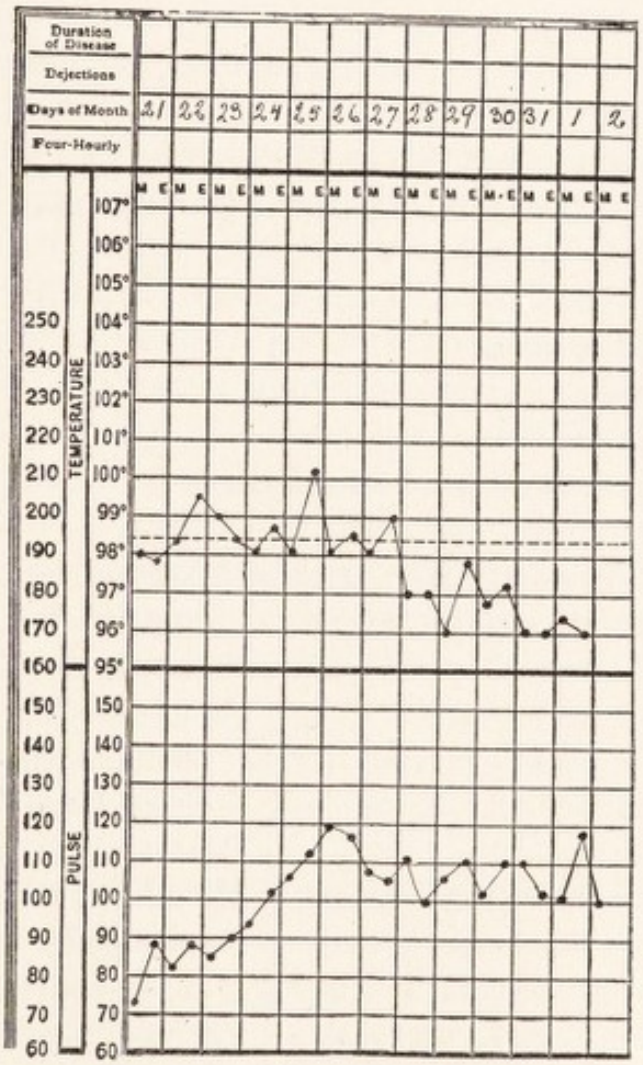



FIG. 53.

The temperature and pulse were as shown in Fig. 53. The respirations were 20 to 33. The amount of urine was 10 to 33 ounces, the specific gravity 1.014 to 1.022. There were slight traces of albumin at five of six examinations, occasional hyalin casts at three.

The hemoglobin was 100%. The other blood findings were normal. A Wassermann was negative.

The abdomen was tapped the day of entrance and a quart of clear straw-colored fluid removed before the cannula was shut off. The specific gravity of the fluid was 1.012. The sediment showed 6% polynuclears, 94% lymphocytes. A culture was negative.

The patient did not respond to stimulation or diuresis. Venesection was also tried with no benefit. He failed and September 2 died.

*Clinical Diagnosis (from Hospital Record).*—Chronic bronchitis with emphysema.

Mitral regurgitation.

Hypertrophy and dilatation of the heart.

Decompensation.

Anasarca.

Ascites.

Chronic passive congestion of all the organs.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic broncho-pulmonary infection.

Bronchitis.

Cirrhosis of the lung.

Arteriosclerosis.

Myocardial weakness.

Hypertrophy and diltation of the heart.

Passive congestion.

*Anatomical Diagnosis (Dr. Oscar Richardson).*—

| | |
|---|---|
| 1. Chemical or physical origin of fatal illness. | Chronic bronchitis. |
| 2. Secondary or terminal lesions | Focal pneumonia.<br>Focus of chronic interstitial pneumonitis, inferior lobe of left lung.<br>Edema of the lungs.<br>Mitral insufficiency.<br>Hypertrophy and dilatation of the heart.<br>Chronic passive congestion, general.<br>Hydropericardium.<br>Ascites.<br>Anasarca. |
| 3. Historical landmarks. | Chronic pleuritis. |

The mitral valve was 12 cm. in circumference (normally 10 cm.). Except for the increase of circumference the valve was not remarkable.

The hypertrophy of the cardiac walls was more marked on the right side. The organ was enlarged. Its cavities were enlarged.

There was no emphysema. There is a possibility that the chronic interstitial pneumonitis was much more extensive in this case than is indicated, as it may have been disguised in the section surface by the chronic passive congestion.

The case is instructive in several ways as we look back upon it with the wisdom of hindsight. In the first place it is a good example of the heart lesion often diagnosed simply as mitral regurgitation. The true diagnosis was cardiac hypertrophy, especially of the right side, from overwork in overcoming the pulmonary obstruction due to chronic pneumonitis or cirrhosis of the lung. As a minor feature of this cardiac dilatation we have a mitral orifice measuring 12 cm. instead of 10 cm., and perhaps somewhat incompetent.

A second point of interest is the absence of any pulmonary emphysema post-mortem, despite the fact that he had during life hyperresonance, a breathing of the type usually associated with emphysema, and râles such as usually go with that disease. This proves that the physical signs often supposed to be diagnostic of emphysema may accompany either that disease or other diseased conditions, among which passive congestion from cardiac weakness in a barrel-chested individual is perhaps the commonest.

The valves were all enlarged in this case (tricuspid 13 cm., aortic 8, mitral 12) but the pathologist definitely calls attention in his diagnosis to *mitral regurgitation* and to the congested lungs, so much engorged that other and more chronic changes were masked.

There is evidently more hypertrophy of the right ventricle than of the left, though both sides are dilated. (Right ventricle 7 cm., left ventricle 10.)

In the absence of hypertension, nephritis and arteriosclerosis I think we must say that this is one of the cases of true *chronic bronchitis* with broncho-pulmonary infection and chronic pneumonitis gradually developing after an (imperfectly resolved?) pneumonia and that as part of the cardiac hypertrophy due to this cause the mitral valve orifice was enlarged and very possibly incompetent. But the systolic murmur might also be explained as due to incompetency of the tricuspid valve especially as the pulmonary disease must have brought unusual strain upon the right ventricle.

### Necropsy 2825

An American motorman of thirty-seven entered March 30, 1911. His past history was negative except for dyspnea on considerable exertion for fourteen years. He had had his present trouble for four years and a half and was refused admission to the police department because of it. Six weeks before admission he became very dyspneic on running for a car, and felt very cold all day. For the next five days he had dyspnea on slight exertion and pain in the left shoulder. Medical treatment gave relief. Since the acute onset he had had cough and edema of the legs. His dyspnea, palpitation, and edema had grown gradually worse unless he stayed in bed. He gave up work three weeks before admission, and had been most of the time in bed. The cough was now hacking, occasionally with white sputum. For two days his trousers had felt too tight. He slept poorly with two pillows because of dyspnea and nervousness. Cold affected him unfavorably. Two days before entrance he vomited. He had lost flesh, but thought he had gained weight.

Examination showed a fairly well nourished man, tired-looking and dyspneic. The mucous membranes were slightly pale and cyanotic. The teeth showed many carious stumps. The apex impulse of the heart was diffusely seen and felt as far down as the fifth space and as far out as the border of dullness, 15 cm. to the left of the midsternum and 4 cm. outside the nipple line. The right border of dullness was 4.5 cm. from midsternum. The sounds were irregular in force and frequency, fair in quality. The first sound at the apex was rather sharp. There was a slight systolic, possibly presystolic, thrill at the apex. A blowing rough systolic murmur was heard all over the precordia and in the axilla and both backs, loudest over the apex. The aortic second sound was scarcely audible. The pulses were irregular in force and frequency, fair in volume and tension. The blood pressure was 195/90 to 115/90. The lungs were negative. The liver dullness extended from the fifth rib to 5 cm. below the costal margin. The edge was indistinctly felt. There was great soft edema of both legs and slight edema of the sacral region. The genitals, pupils, and reflexes were normal.

The temperature was 96.4° to 98.5°, the pulse 59 to 120, the respiration 26 to 31. The amount of urine was 4 to 71 ounces. The urine was turbid, the specific gravity 1.034 to 1.020. There was the slightest possible trace of albumin at two of three examinations, a few hyalin casts at all, fatty casts at one. The hemoglobin was 95%. There were 14,000 leucocytes. The smear showed

polynuclear leucocytosis. A Wassermann and a throat culture were negative.

The patient was restless and dyspneic, requiring morphia and atropin. Tracings of the jugular vein and the apex showed auricular and ventricular waves occurring synchronously, i.e. nodal rhythm. He grew steadily worse, vomiting everything. All medication except strychnia was omitted, and he was given intravenous strophanthin ¼ to ½ mg. on April 4, 5, and 6, with temporary slowing of the pulse. He continued to fail steadily, and April 6 died.

*Clinical Diagnosis (from Hospital Record).*—Mitral insufficiency.

Dilated heart.

Infarction of lung.

*Dr. W. H. Smith's Diagnosis.*—Mitral stenosis and regurgitation.

Probably aortic stenosis and regurgitation.

Possibly adhesive pericarditis.

Acute glomerulonephritis.

*Anatomical Diagnosis.*—Chronic fibrocalcareous endocarditis of the mitral valve.

Hypertrophy and dilatation of the heart.

Septicemia, streptococcus.

Thrombosis of the right auricular appendix.

Thrombosis of branches of the right pulmonary artery.

Infarct of the right lung.

Septic staining of intima of all the great blood vessels.

Chronic passive congestion, general.

Slight hydropericardium, hydrothorax, and ascites.

Anasarca.

Dr. Richardson: The heart weighed 660 grams,—considerably enlarged and all its cavities markedly dilated. (Normal weight 200–300 grams). The right ventricle wall measured 3 mm., the left 11 mm. The heart muscle was thin, flabby, dirty brownish-red,—anatomically a wall in harmony with the marked dilatation. The dominant feature at necropsy was the dilatation of the heart. The right auricular appendage was occluded by a mural thrombotic mass, a typical source for the embolic thrombosis of the branches of the right pulmonary artery and the infarct of the right lung. The mitral valve measured 17½ cm. (normally 10)—nearly twice the normal circumference. The valve was the seat of what was called chronic endocarditis. The mitral valve has two cusps. Along the entire length of the insertion of the posterior cusp was a fibro-calcareous columnar mass, its surface irregular, nodular, and fibrous.

This mass ceased abruptly in the region of the anterior cusp and was 6 cm. long, 1.5 cm. wide and 1.2 cm. thick. The small strip of curtain of the posterior cusp which lay anterior to the mass and included the free portion of the cusp showed little if any fibrous change. The chordae tendineae showed little if any thickening. The remaining 11½ cm. of the valve curtain was negative. One portion of the valve showed chronic fibrous deformity, and the rest was so stretched that between the two the valve was almost twice its normal circumference. One would expect to find hypertrophy of the myocardium of the right ventricle, yet it was only 3 mm.; but probably it was once thicker, and had stretched.

The other valves were negative, except that their circumferences were increased, especially the tricuspid, which was 16 cm. (normally 12–13) aortic 8.5, pulmonic 10.5; of course in association with the dilatation of the heart. The rest of the circulatory apparatus was negative except for deep purplish staining of the endocardium and the intima of the great vessels which is found at times in association with streptococcus septicemias.

The liver, spleen, etc., showed chronic passive congestion, and the gastro-intestinal tract some swelling and reddening of the mucosa associated with the chronic passive congestion.

Culture from the heart blood showed a good growth of streptococci.

This was a very unusual lesion of the mitral valve. The only other lesion I have seen like it was in a heart in which there was found in the same cusp a similar mass, which had extended across into the region of the bundle of His, giving a typical picture anatomically of Stokes-Adams disease. The clinical diagnosis also was Stokes-Adams disease.

A PHYSICIAN: Would the valve close?

DR. RICHARDSON: I do not know what a valve like that would do under pressure. Under the anatomical condition present in the valve at necropsy it would seem probable that regurgitation would be more or less marked.

### Necropsy 3320

A machinist of seventy-four entered the Mass. General Hospital February 28, 1914.

His past history was excellent except for gonorrhea thirty years ago.

Four years ago he had to give up work for seven months on account of dyspnea, weakness and swelling of the feet. He then had

a sore on the right leg for five months, but was not confined to bed. Since then he had been well and working every day until seven months ago, when he was waked one night by dyspnea. Next day he felt weak and noticed that his feet were swollen. He soon began to cough and raise a little frothy sputum. He has not worked since then; he tried it but found that he was too weak. Sleep, appetite and bowels were normal. He has urinated two or three times at night for several months, and has had a little dribbling recently, but no polyuria. He has not been confined to bed.

On examination he was well developed and poorly nourished. Respiration rapid, shallow. The skin showed many papules on chest and some looseness. Head not remarkable. Throat reddened. Much yellow exudate. Apex impulse of heart in the fifth space 1.5 cm. outside nipple line. No increased supracardiac dullness. Action slow; occasional extrasystole. A loud musical systolic murmur was heard at the mitral area referred to axilla and audible faintly all over the precordia. No diastolic heard. $A_2$ not heard. $P_2$ faint. Pulses slow, occasional extrasystole. Arterial walls tortuous and hard. The lungs showed slight dullness at the right apex and dullness at the right base with diminished breathing, high-pitched inspiration and prolonged expiration, many coarse râles and diminished voice sounds. Signs in left not so marked. Abdomen slightly resistant. Slight shifting dullness in flanks. No spasm or masses. Slight edema of the abdominal wall in flanks. Genitals negative. Hands slightly cyanotic. Skin over shins and ankles raw, reddened, scaling, brawny, loose. Slight edema of ankles. Pupils and reflexes negative.

Temperature 99°–101.5° until the day of death, then falling to 97.8°. Pulse 99–81. Respiration 25–45. Blood pressure 160/110. Urine, ℥6–34. Specific gravity 1.024–1.020. A slight trace of albumin at both examinations. A rare red blood corpuscle. Blood: Hemoglobin 95%. Leucocytes 34,600–24,000. Polynuclears 91%.

Much morphia was used to give rest. The patient had Cheyne-Stokes breathing almost constantly. March 4 a reddened, brawny, tender area had appeared at the inner right thigh, pronounced by a surgical consultant streptococcus lymphangitis. The general condition was worse, the patient almost comatose at times. The following day he was weaker, with very feeble and irregular heart action and very poor pulse. March 6 he died.

The clinical diagnosis reads "chronic endocarditis of the aortic and mitral valves. Arteriosclerosis."

*Anatomical Diagnosis.*—I. Chronic fibrinocalcareous endocarditis of the aortic valve, stenosis.

Fibrous sclerosis of the mitral valve.
Arteriosclerosis.
Hypertrophy and dilatation of the heart. (558 grams.)

II. Septicemia, streptococcus. (Terminal.)
Erysipelas of the right thigh.
Hydrothorax, double.

III. Ulcers of the legs.
Slight arteriosclerotic nephritis.
Slight chronic pleuritis, right.
Obsolete tuberculosis of the apices of the lungs.
Chronic perisplenitis.
Hypertrophy of the middle lobe of the prostate.
Hypertrophy of the trabeculae of the bladder.

The aortic valve was reduced to a slit like orifice 2 cm. × 6 cm. The tricuspid 13.5. Pulmonary 8.

"The mitral valve measures 9.5 cm. Its curtains present a moderate amount of diffuse fibrous thickening which is slightly nodular along the free margins. The chordae tendineae are slightly thickened and the upper portions of the papillary muscles show small yellowish fibrous areas.

"The heart weighs 558 grams. The organ is considerably enlarged. On section the myocardium generally is thick and pale brown red. Right ventricle wall 5 mm., left ventricle wall 13 mm. The columnae carneae are thick and prominent on each side. The auricular walls are thickened. The cavities of the ventricles are but little enlarged, if any. The auricular cavities are enlarged."

*Comment.*—Obviously aortic stenosis with regurgitation is the main lesion. There *may* have been some mitral regurgitation also though this is by no means certain.

A *17-year-old-housewife* was seen March 13, 1922, complaining of "cough and breathlessness."

Had childrens' diseases and frequent tonsillitis until tonsillectomy and adenoidectomy 5 years ago. Severe attacks of rheumatic fever frequently (every 2–3 years) until three years before admission. Nycturia, lately 2–3 times a night.

Twelve years before admission, after an attack of acute rheumatic fever complicated by chorea, the patient noticed breathlessness and swelling of legs. Four years later it was necessary for her to go to

bed for three months because of an increase in breathlessness and swelling of legs with substernal pain. Since then she has spent about a half of each year in bed because of these symptoms, up to the present time. One year ago she was married and immediately became pregnant. Her symptoms grew worse until she had a miscarriage at five months, since which her condition has been much worse. She has been in bed, suffering with a persistent cough, dyspnea, orthopnea, palpitation, and substernal pain.

On examination the patient was poorly developed and poorly nourished, in extreme discomfort, markedly orthopneic and coughing almost incessantly. *Heart:* The apex impulse was diffuse; pulsations were palpable in the midaxillary line 18 cm. from mid-sternal line. Percussion note dull to flat over base of left lung. Absolute arrhythmia. Heart rate 142 with a pulse deficit of 60. The sounds were poor in quality, the 1st sound approaching the second in character. Soft blowing diastolic and systolic murmurs were heard over entire precordium. P2 was greater than A2 and was accentuated. No thrills. Blood pressure 92/54. Evidence of left hydrothorax. Abdomen showed a fluid wave and shifting dullness. The liver edge, firm, smooth and tender, was palpable 8 cm. below the costal margin. There was marked edema of the legs.

The blood Wassermann was positive on two tests. Renal Function 39%. Urine 1022 and 1014 in two specimens with traces of albumin, rare red cells and a few hyaline and granular casts. Leucocytes 12,000 to 16,000. Temperature 100 to 102. Pulse averaged 90.

Six days after admission the dullness at both bases disappeared and only a few crackling râles could be heard. On the twelfth day after admission the temperature rose to 104, the patient became weaker, with increased edema and dullness at lung bases. She died on March 25th.

*Necropsy.*—Heart 500 grams. 250 c.c. in pericardium. Mitral 13 cm., tricuspid 12, pulmonic 7.5, aortic 7.5. *Left auricle big and stretched.* Mitral curtain and its tendinous chords thickened. Aortic valve normal except for *acute endocarditis. No stenosis.* Acute endocarditis on aortic valve (streptococci). Patchy fibrosis in myocardium.

Lungs normal except for chronic passive congestion with infarcts and thrombosed vascular branches.

Liver and spleen showed chronic passive conjestion.

Calcified (Tb.) mesenteric nodes.

Calcified nodes (Tb.) in liver and spleen.
Kidneys normal.
*Pure mitral regurgitation with acute aortic endocarditis.*

### Necropsy 797

A woman of 37 was in the ward from December 16th to December 24th with what appeared to be pneumonia. She had been ill 15 days with dyspnea and fever beginning with a chill. The pulse was regular at 120 to 160, finally 220. The leucocytes were 16,000, 21,000, 24,000, the temperature 101 to 105°. The heart's action was regular. There was a slight systolic murmur at the apex. The pulmonic second was slightly accentuated.

Post-mortem the heart weighed only 360 but considering the size of the individual was hypertrophied and dilated. The aortic valve though quite normal measured only five cm. in circumference. The mitral circumference was ten cm. The cordae tendineae leading to the curtain were shortened and thickened. The free edge of its curtain presented a row of irregular nodular masses fibrous and gray-white. A soft, gray-red, shaggy, mushy mass, two cm. in diameter, extended from the curtain of the valve up on to the wall of the left auricle where it was weakly adherent. There was chronic passive congestion of the lungs and pneumococcus septicemia.

The diagnosis was not suspected in life, but from this history and symptoms, pneumonia was in all probability believed to be present, and may well have been present though it was not shown at necropsy, for there was a fibrinous pleurisy and a pneumococcus sepsis in addition to the acute and chronic endocarditis of the mitral.

### Necropsy 282

A thirteen-year-old schoolboy, with good family history and past history, entered June 8.

He thought he had never had any children's diseases, rheumatism or chorea. Last October he had mumps. All winter he had a cold with cough so violent that he sometimes vomited; not much sputum, never blood. Two months before admission he noticed a bunch on the right thigh, painful on lying down and when walking. A month ago he had pain in the knee when lying down. The bunch was now growing smaller.

Three years ago and a year and a quarter ago he was treated in a hospital for "heart disease." He was not particularly short of breath on going upstairs until lately. He sometimes had pain

over the precordia and over the left shoulder. Four days before admission, after a night of hard coughing, his scrotum suddenly began to swell and became bigger than his fist and painful. The pain and swelling were now growing less.

A rather poorly developed and nourished, pale boy with slightly cyanotic lips and fingers, breathing rapidly with effort and coughing often and in paroxysms, more violently when lying down, without much sputum. Cervical and right epitrochlear glands enlarged. Apex impulse of the *heart* in the sixth space (see Fig. 54), strong and diffuse. Action irregular. $P_2$ accentuated and reduplicated. At the apex a rolling presystolic, a rough systolic, and a short diastolic murmur, presystolic and systolic thrills; second sound audible. At the base a faint diastolic murmur. At the right border of the sternum below the costal cartilage of the fifth rib a systolic

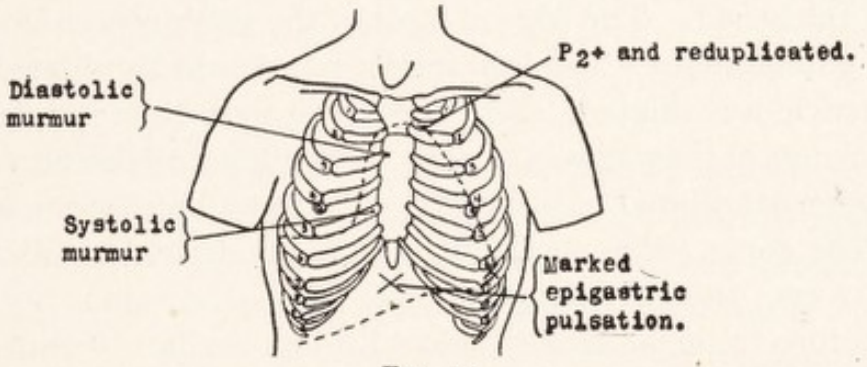



FIG. 54.

murmur. Marked epigastric pulsation. A boat-shaped prominence over the lower sternum. Pulse rapid, rather irregular, fair volume, compressible. *Lungs* not recorded. *Abdomen* rather tense. Wall edematous above the pubes. *Genitals*. Scrotum edematous, translucent, rather tender. *Extremities*. Slight edema of the legs and ankles, more on the right. Both patellae floating, the right more than the left; also more tender than the left. On the right thigh was an irregular, hard, slightly tender mass the size of a butternut attached immovably or growing from the bone.

T. 97°–102.3°. P. 82–102. R. 41–79. *Urine*. Normal amount, sp. gr. not recorded, a trace of albumin. *Blood*. Leucocytes 18,400.

The boy coughed very hard all the afternoon, raising light bloody sputum. He suffered a good deal. After being given morphia and castor oil he vomited. Next day he was quieter. The temperature, however, was up. He still coughed considerably. June 11 he had pain over both chests and in the epigastrium, and was very restless.

The sputum was considerably blood stained. The temperature dropped to 97°, then rose to 102.3°. The morning of June 12 he died.

*Clinical Diagnosis.*—Chronic and acute endocarditis.

*Anatomical Diagnosis.*—Acute and chronic endocarditis of the mitral valve.

Chronic adhesive pericarditis.

Hypertrophy and dilatation of the heart.

Chronic passive congestion of the lungs, liver, spleen and kidneys.

Edema of the scrotum.

Slight double hydrothorax.

Edema of the lungs.

The heart was large, weighing, with the pericardium adherent, 625 grams. The parietal pericardium was everywhere adherent to the heart by old fibrous adhesions. The mitral valve was 13 cm. in circumference. One half the curtain was much shortened and slightly thickened. The edge of most of the valve was studded with minute granulations. The left auricle was markedly dilated. The left ventricle was dilated. The trabeculae were thin and flattened. The thickness of the wall was 14 mm. The right auricle and ventritle were somewhat dilated. The right ventricle wall was 4 mm. in thickness. The aortic valve measured 5½ cm. in circumference, the tricuspid 13 cm., the pulmonary 6½ cm.

A culture from the kidney showed a few bacilli. Cultures from the heart blood, the liver and the spleen were sterile.

## FATAL CHOREA

Necropsies are so rare in Sydenham's chorea that I have added here the full clinical and pathological record of three fatal cases, occurring in unmarried girls aged thirteen, sixteen, and twenty, respectively. The duration of symptoms from the first complaint until death was: eight weeks, six weeks, and four and a half weeks. Two of the patients had had previous attacks of chorea. In one patient there was also an acute rheumatic arthritis and tonsillitis. In the others the chorea was the only obvious clinical sign, the movements being in one case so violent as to lead to fracture of two ribs.

At necropsy all the hearts showed an acute endocarditis in the form of "small, soft, grayish-yellow red-tipped vegetations" placed in rows along the free margins of the mitral curtains; also to a less extent on the aortic and in one case the tricuspid also. In two of the cases there was also a chronic fibrous endocarditis on the aortic valve.

In one case the heart was slightly enlarged; in two it was of normal size. The myocardium showed no lesions. In one case there was an acute pericarditis.

Complications of interest were:

*Case* 1.—Double parotitis. Slight cystitis. Slight bronchopneumonia.

*Case* 2.—Pulmonary tuberculosis with empyema (left). Fibrinous pleurisy (right). Fracture of 6th and 7th ribs with purulent infiltration about the fractures.

*Case* 3.—Pulmonary tuberculosis. Serofibrinous pericarditis. Status lymphaticus.

The occurrence of pulmonary tuberculosis in two of these three cases is of interest, especially as it was apparently acute and gave no signs or symptoms until the last few weeks of life unless we are to count the rcord: "five months ago threatened with pneumonia" as evidence of tuberculosis.

### Necropsy 2563

An American schoolgirl of thirteen entered March 9, 1910. Two years after her birth her mother died of tuberculosis. The child was always very nervous. She had a few sore throats, with removal of adenoids several years before her admission. At ten she had a bad attack of chorea. Three weeks before admission to the hospital she began to have twitching, which March 8 became much worse and in the evening very violent. For two days she had had slight cough.

Examination showed a well nourished girl unable to sit in a chair, screaming at times, with violent choreiform movements of all the muscles. At times she had to be held in bed. The left tonsil was enlarged. The apex impulse of the heart was best felt in the fifth space in the anterior axillary line 6 cm. outside the nipple, 13 cm. from midsternum. There was no enlargement to the right. The action was rapid. A faint systolic murmur was heard at the apex transmitted to the axilla. At the base were sounds of an entirely different quality. In the pulmonic area a late systolic murmur was heard, with a loud sharp pulmonic second, greatly accentuated and louder than the aortic second. The pulses were of fair volume and tension. The lungs, abdomen, pupils and reflexes were normal. The genitals and extremities are not recorded. The skin of the back and the exposed areas was reddened and roughened. A skin consultant pronounced the condition irritation due to constant movements.

The temperature was 99.8° to 105°, with continuous general rise after March 10. The pulse was 119 to 151, the respiration 29 to 58. The output of urine was 9 to 29 ounces, the specific gravity 1.012 to 1.026, cloudy at all of four examinations, the slightest possible trace of albumin at the last, slight acetone at two. The hemoglobin was 75%, the leucocytes 17,000, the polynuclears 85%.

The patient continued to be very violent and was not quieted except for periods of half an hour to an hour by any of the sedatives used until large doses of veronal made her sleep most of the time for thirty-six hours. After this the motions were easily controlled by morphia. March 16 the heart seemed larger than at entrance. During the next two days the jerks increased, and the patient became quite cyanotic and had a good deal of cough and pain in the chest. A pleural rub developed over the left chest and also a to-and-fro cardiac rub loudest over the area of relative dullness and hardly heard over the area of flatness. March 19 there was some dullness in the middle of the left back, with a moderate number of fine respiratory crackles and a dry rub over the right back. The heart's apex impulse became more forcible and localized, now in the fifth space as far out as the anterior axillary line. The pulmonic second sound seemed louder.

March 19 the patient became suddenly worse and died.

*Clinical Diagnosis (from Hospital Record).*—Acute chorea.
Acute endocarditis.
Acute pericarditis.
Mitral regurgitation.
Dry pleurisy
Bronchopneumonia (?)

*Dr. Richard C. Cabot's Diagnosis.*
Chorea.
Acute endocarditis.
Acute pericarditis (?)
Acute pleuritis (?)
Bronchopneumonia or tuberculosis of the left lung.

*Anatomical Diagnosis.*—(Acute chorea.)
Chronic and acute endocarditis of the aortic valve.
Acute endocarditis of the mitral valve.
Serofibrinous pericarditis.
Hypertrophy and dilatation of the heart.
Chronic passive congestion, general.
Hydrothorax, double.
Tuberculosis of the upper lobe of the left lung.

Status lymphaticus.

Foci of obsolete tuberculosis in the bronchial lymphatic glands and lungs.

Chronic pleuritis.

Chronic appendicitis.

DR. RICHARDSON: We were not permitted to examine the head.

The appendix showed a well-marked stage of chronic appendicitis.

Each pleural cavity was half full of thin, pale fluid.

The right lung was bound down generally by old adhesions, and the left lung presented a few scattered old adhesions, some in the region of the apex. The trachea and bronchi contained a moderate amount of dirty yellowish mucopurulent material.

The thymus gland was still present and weighed 12 grams. The bronchial and mediastinal glands, generally, were more or less enlarged and pigmented, and three of the bronchial glands on section showed small fibrocalcareous masses.

The tissue of the right lung generally was rather leathery, dark salmon red, and yielded a moderate amount of dark red, frothy fluid. In the substance of the middle portion of the upper lobe there was a small fibrocalcareous mass situated just beneath the pleura, which was slightly retracted over it. In this lung elsewhere there was no good evidence of tuberculosis of any sort. The upper two-thirds of the upper lobe of the left lung was resistant and lumpy to the touch. In the substance of the upper part of this lobe there was a small cavity 2 cm. in diameter lined with a grayish yellow membrane 1 mm. thick and contained a small amount of caseo-purulent material. At one point in the wall of the cavity a small bronchus opened into it. The lung tissue about the cavity showed fibrosis and induration, and within it were several small fibrocaseous and fibrocalcareous masses, and in their region were numerous minute to small discrete and confluent tubercles. These tubercles extended pretty well down into the substance of the middle portion of the lobe. Just beneath the pleura on the upper part of the anterior lobe there was a very small fibrocalcareous nodule. The tissue of this lung elsewhere was generally leathery, dark red, and yielded a moderate amount of dark reddish, frothy fluid.

The pericardium contained a considerable amount of pale, cloudy fluid supporting fibrinous shreds and masses. The vascular and parietal layers were coated with a thin layer of membranous fibrinous material which weakly bound the two layers together. Here and there on the pericardium there were discrete and confluent dark reddish hemorrhagic spots and areas.

The heart weighed 257 grams, enlarged. The myocardium was negative. The cavities showed some enlargement. The valve circumferences were: mitral 9 cm., aortic 6 cm., tricuspid 10½ cm., pulmonary 6½ cm. The mitral curtain showed scattered along its free margin an irregular row of reddish coxcomb-like granulations. The curtain elsewhere showed but little if any fibrous thickening. The aortic cusps presented a moderate amount of diffuse fibrous thickening which slightly contracted and deformed at least two of the cusps. Along the free margin of the cusps there was an irregular band of grayish granulations. The other valves were not remarkable. The coronaries were free and negative. The aorta, however, showed in a few places fibrous streaks and areas in an extent not to be expected at this age. The great branches were not remarkable.

The spleen showed a dark red, elastic tissue with prominent follicles.

The kidneys weighed 252 grams and showed passive congestion. The gastro-intestinal tract was negative except that the solitary and agminated follicles showed marked prominence.

The retroperitoneal and mesenteric lymphatic glands were moderately enlarged, up to 2 cm. in greatest dimension.

Dr. Cabot: It is of interest to note that there was a fibrous and therefore chronic endocarditis on the aortic valve in addition to the acute process there and on the mitral. This means that at previous time, very possibly in the attack of chorea three years ago, the aortic valve was involved. The chronic pleuritis also gives us reason to believe that the tuberculous process had extended beyond the bronchial lymphatics at an earlier time. Indeed this seems to be proved by the presence of obsolete foci of tuberculosis in the lungs themselves.

One may speculate a little perhaps upon the question whether this old tuberculosis, presumably acquired in her second year, was lighted up by the attack of chorea, or whether the chorea itself represents an infection made possible by diminished resistance, itself a result of tuberculosis. It would be interesting to collect cases of this association and trace the order of development so as to determine whether the chorea determines the out-break of tuberculosis, or *vice versa*.

What relation, if any, has the condition of status lymphaticus found here at necropsy to the rest of the lesions? We have ordinarily supposed, on rather insufficient evidence, I think, that the presence of status lymphaticus makes any other disease or injury, such as

operative insult, much more serious. But one needs to trace further the relation between this mysterious condition and the two infections which were present in this case.

Since no organisms were demonstrated in this case except those of tuberculosis in the lung, it might be argued that the lesions in the heart and pericardium were really manifestations of tuberculosis. Against that we can merely say that from their morphology it is reasonable to suppose that they represent another infection, since we often see such lesions in connection with rheumatic disease, and do not so far as I know ever see them demonstrated to be of tuberculous origin. That is, we never find tubercle bacilli in the vegetations on the valves in such case. Until such are demonstrated we may well refuse to believe that tuberculosis is ever a cause of endocarditis.

### Necropsy 2564

A schoolgirl of sixteen entered March 13, 1910. She had had an attack similar to the present illness at the age of twelve. She had been ailing since she had a streptococcus sore throat five weeks before admission. Three weeks before admission she had rheumatism in her feet, ankles, hip, and wrist. Two weeks later her face began to twitch. She left school five days before admission, four days before began to have trouble with her speech, three days before began to throw her head from side to side. The following night she was found in her nightclothes squatting on the register talking to herself about being chilly. Her bed was pulled to pieces and the room generally torn up. She talked incoherently and later could not speak. She had not slept for five or six nights, and the night before entrance was very restless in spite of chloral and the care of a nurse. Her bowels were regular, her appetite poor. Catamenia were regular as a rule. She had had no Fowler's solution.

Examination showed a well developed and nourished girl with flushed skin and mucous membranes. There was marked reddening, not decolorizing on pressure, in the form of discrete and confluent papules of the skin over all prominences and convex surfaces of the body. The skin was dry and harsh. The lymph nodes were not enlarged. The apex impulse of the heart was felt in the fifth space, eight cm. from midsternum. The sounds were rapid, regular, of fair quality. No murmurs were heard. At the base the pulmonic second was greater than the aortic second and accentuated. The lungs showed good resonance, normal breathing, no râles. The abdomen was held rigid, and was tympanitic. No masses were felt.

The knee-jerks were not obtained. The plantars were normal. There was no edema. There were marked incoordinate movements of the entire body, requiring forcible restraint to keep the patient in bed. She did not speak when questioned. No Kernig, abdominal, wrist or elbow reflexes obtained.

The temperature was 100° to 102° or 103° daily until March 19 when there was a steady rise to 108°. The pulse was 108 to 160, the respirations 15 to 28. The urine was cloudy at one of two examinations, slightly alkaline at two, the specific gravity was 1022. There was a very slight to the slightest possible trace of albumin, no sugar, and acetone present at one. The hemoglobin was 85%, the leucocytes 14,000 to 19,000. Smear showed polynuclear leucocytosis. Lumbar puncture gave fifteen c.c. of fluid, part of which was very bloody, part nearly clear, and which reduced Fehling's solution slightly. The sediment from the clearer tube was mostly blood corpuscles. The cell count showed 80% polynuclears, 20% mononuclears. "The number of these cells seems to be no more than the white cells to be expected with the amount of blood present."

The patient's movements were only partially controlled by chloral and an occasional dose of morphia. On March 18 a high-pitched systolic murmur appeared at the apex. The temperature began to rise. The patient grew weaker, the choreiform movements subsiding as her strength failed. On March 20 strophanthin was given without response and the patient died without developing any new symptoms except those of double parotitis.

*Clinical Diagnosis.*—Chorea major.

Endocarditis.

*Anatomical Diagnosis.*—(Acute chorea.)

Verrucose endocarditis of the aortic and mitral valves.

Hemorrhagic areas of the lungs.

Slight bronchopneumonia.

Fatty metamorphosis of the liver.

Slight cystitis.

Parotitis.

The pericardium was not remarkable. The heart weighed 247 grams. The myocardium was pale, slightly yellowish and a little lax. The right ventricle wall measured three mm., the left twelve mm. The columnae carneae were fairly marked. The mitral valve measured nine cm., the aortic five and a half, the tricuspid eleven and a half, the pulmonary six and a half. The mitral curtain showed along its free margins in two or three places small short rows of

minute reddish granulations. The valve was otherwise not remarkable. The aortic cusps showed stretching across the cusps from side to side, a little below the upper margins, a narrow band of fine, minute, rather smooth reddish granulations. The cusps were otherwise negative. The tricuspid and pulmonary valves were negative. The coronaries were free. The intima of each artery showed scattered along its course a few very minute yellowish plaques, and similar plaques were found in the intima of the great branches.

Blood cultures from the heart and spleen gave no growth.

**Necropsy 2260**

An unmarried American girl of twenty was sent from the Out-Patient Department for "chorea" and entered November 23, 1908.

Her father had died of pneumonia. There as no family history of tuberculosis. She had had mumps. She had not had rheumatic temple. Five months ago she received a severe blow on the right temple. Five months before admission the patient was "threatened with pneumonia."

Five weeks before entrance, "nervousness" was first noticed. She went to bed four weeks before and had shown some improvement, at the time of entrance lying more quietly and feeling better. Since the blow on her head the patient thought she had lost in weight, color, and strength.

Examination showed her to be well developed and nourished, with skin and mucous membranes of good color, showing some loss of weight. The pupils were equal, regular, reacted normally. There was slight bilateral enlargement of the inguinal glands. The apex impulse of the heart was in the fifth space, eight and a half cm. from midsternum in the nipple line. There was no enlargement to the right. The sounds were regular, forcible. At the apex a soft systolic murmur was heard. The pulmonic second was greater than the aortic second but not accentuated. The pulses were synchronous, equal, regular, of fair volume and tension. There was slight prominence of the sternal articulation of the clavicles.

There was dullness throughout the left lung, marked above the fifth rib, with many consonating râles, bronchial breathing and somewhat increased voice sounds. The left back was dull throughout, with fine and medium moist râles. At the apex behind there was bronchial breathing.

The abdomen was level, soft, tympanitic throughout. The liver extended from the sixth rib to the costal margin. The spleen was not

felt. The right kidney was palpable *in toto*. The knee-jerks were present but slight, the plantar reflexes normal. The hands showed several encrusted abrasions (from hitting them on the bed before entrance), and small traumatic hemorrhages beneath the nails. Choreiform movements were marked, especially of the fingers and arms, but also involving the legs, neck, and other muscles. There was a slight cough without sputum.

The temperature was 101 falling gradually to 98, rising again on December 6 to 105.2. The pulse was 80 to 170, the respirations 22 to 58. The hemoglobin was 90%, leucocytes 9,700 to 25,100. The urine at two examinations was acid, cloudy, with a specific gravity of 1028, no albumin, no sugar, no pus, blood, or casts. Sputum examination showed streptococci, a few intracellular diplococci, no tubercle bacilli. Chest tap gave a small amount of bloody fluid. The needle did not feel as if it were in a free space. The smear showed pure blood with no excess of leucocytes.

The choreic movements were less severe on November 29. There was intense bronchial breathing throughout the left lung, with consonating râles. Hot baths were tried with good result. The patient was quieter and slept without bromides. On December 6 the patient complained of severe pain in the region of the heart. Nothing was found on physical examination to account for it. Bromides, swathe, and an ice-bag gave temporary relief, but the patient again became restless, tossing about violently and crying out. During the afternoon and evening three-fourths of a grain of morphia in all was given, with hyoscin once a little ether and chloral by mouth, which was vomited. Then chloral suppositories, after which she passed a fairly good night. An area of tympany to the left of the sternum, under the clavicle, discovered on December 5, had now disappeared.

On December 8 a medical consultant wrote: "Left back flat throughout (like front), with distant tubular breathing. The percussion note is so woodeny that fluid is strongly suggested but with the heart in normal position fluid seems impossible. No râles to-day. Over the sixth right rib near the nipple there was felt some days ago for the first time a hard painless enlargement, apparently the callus of a rib broken in her thrashing previous to entry."

On December 10 the left lung showed vesicular breathing, becoming more bronchial on the 11th, with many coarse crackling râles. The temperature was very high and there was a troublesome cough. The patient was unable to rest quietly even with drugs. Three days later there was labored breathing. An area of tympany was found over the left front from the clavicle to the third rib, extending

to the anterior axillary line. The heart action was irregular. On December 14 she suddenly coughed up a large amount of thick yellow pus, the pulse was poor, she became dyspneic and cyanotic. The condition was helped by strychnia and strophanthin intravenously, but the pulse remained poor and the patient was restless and at times irrational. On December 15 the breathing was very difficult, her color and pulse were poor, there was no response to stimulation. After another attack of coughing up large amounts of pus she became worse and died.

*Clinical Diagnosis.*—Chorea major.
Caseous pneumonia (tuberculous).
Four broken ribs, sepsis near one fracture.
Streptococcus sepsis with interlobar empyema.
*Anatomical Diagnosis.*—(Chorea.)
Verrucose endocarditis of mitral, tricuspid and aortic valves.
Slight fibrous endocarditis of the aortic valve.
Empyema, left.
Acute pleuritis, right,
Tuberculosis of the left lung with cavity formation.
Fracture of sixth and seventh ribs on each side.
Purulent infiltrations of tissues of thoracic wall on the right side.
The head was not examined.

The heart weighed 226 grams. The myocardium was fairly good, a little pale. The valve measurements were: mitral eight and a half cm., aortic six cm., tricuspid eleven cm. The curtain of the mitral showed scattered along its free margin several shorter and longer rows of rather soft grayish-yellow granular warty material, the surfaces of which were reddish-tinged. Together these rows of vegetations extended along nearly the entire margin of the valve. The valve was otherwise negative. The aortic cusps showed a moderate amount of fibrous thickening which very slightly deformed the valve. At one point one of the cusps showed a minute grayish-red granular adhering mass. The margin of the curtain of the tricuspid valve showed in several places minute to small grayish granular masses similar in character to those already described. The coronary arteries and aorta were negative.

Cultures from the heart gave no growth; from the spleen, a scum of growth of colon-like bacilli.

Microscopic examination of the lung showed typical tubercles, discrete and confluent. "The vegetation on the mitral valve indicates an old process, for the vegetation seems to consist of more or less degenerated fibrous tissue."

## CHAPTER III

## SYPHILITIC HEART DISEASE

In the material studied for this book syphilitic heart disease means essentially *syphilitic aortitis and its results*. I do not forget that gumma of the heart is not very rare. I remember vividly the case of a huge gumma of the interventricular septum demonstrated by Dr. William H. Welch at the Johns Hopkins Hospital a few years ago, from a case of heart-block. Nevertheless in this particular group of cases, that is, in the twenty-three years from October 1896 to November 1919, we have had at the Massachusetts General Hospital but one case of cardiac gumma. (Reported in detail on p. 375. See reference to one other dubious case under Myocarditis, p. 496.)

Neither do I forget the studies of Warthin and the frequency with which he has found the organism of syphilis in the heart muscle. As we have not been able to follow his methods or to devote the generous allowance of time given by him to the finding of organisms in the heart, our negatives in no way contradict his positives. But I do not know that he has ever been able to show that the presence of syphilitic organisms in cardiac muscle proves that that muscle is *functionally impaired*. In this, as in many other parts of the body, the organisms may well be present without doing any harm. Their presence does not prove *disease* of the organ in which they are discovered, though it certainly raises the fear that such disease if not already present may be coming later.

Syphilitic myocarditis therefore may well be the end-result of the triponema's presence. But in our necropsies there was no notably frequent association of fibrous myocarditis with syphilitic aortitis nor with any other demonstrable form of syphilis. What our cases might have shown could we have pursued Warthin's methods I cannot say.

**Syphilitic Aortitis a Late Syphilitic Lesion.**—In thirty-one cases of our series we had reliable evidence as to the date when the syphilitic infection began. Seventeen of these cases were in the end of the aneurismal type, and in them the average duration of life from

the original syphilitic infection to death was *eighteen years*. Eight were of the uncomplicated or latent type of aortitis, and averaged twenty years of life after infection. Five were of the type first manifested by aortic regurgitation and its results. In these cases the average duration since the original infection was fifteen years. In only one case out of 31 in our series did the serious symptom follow swiftly upon the original infection, after an interval of only fourteen months.

**Associated Syphilitic Lesions.**—Though our studies are rather fragmentary and incomplete on this point, we have obtained in this series but little evidence of syphilitic lesions outside the circulatory system. Only six cases showed such evidence. Two of these were tabetics, three showed syphilitic orchitis accompanied in two cases by syphilitic hepatitis and amyloid disease, and in another case by syphilis of the kidney and lung. I recognize, however, that with our methods of examination we cannot challenge such evidence as Warthin has brought to light.

**Four Types of Syphilitic Aortitis.**—Our 92 necropsied cases of syphilitic aortitis are divisible into four groups: (1) *Aneurismal type.* In 41 cases of our series the disease produced aneurism of the aorta (with or without aortic regurgitation.)

(2) In 26 cases *aortic regurgitation without aneurism* was the outstanding feature of the syphilitic process in the aorta.

(3) In 22 cases the disease was *latent;* that is, it had involved the aortic arch or other portions of the aorta without producing aneurism, aortic regurgitation, aortic stenosis or angina pectoris. I shall call this group *latent or uncomplicated aortitis.*

(4) Finally, in three cases the aortitis was associated *with aortic stenosis,* whether as cause or concomitant will be discussed later.

There is some reason to believe that at any one time the largest number of cases of syphilitic aortitis is of the uncomplicated or unmanifested type. In the very numerous necropsies on young negro soldiers which I saw in France during 1918 a small patch of syphilitic aortitis was the rule. It did not involve the aortic valves or the coronary arteries. It had not produced aneurism or any known symptoms. Judging from the age of these men, one may suppose that it represented the earlier portion of that long period of development which I have already referred to as characterizing syphilitic aortitis. Presumably the natural history of syphilitic aortitis is represented by fifteen to twenty years of silent, symptomless progress or regress during which it may well happen that the patient

dies of some other disease, so that *for him* syphilitic aortitis is apparently a harmless as well as a symptomless malady. *Manifest syphilitic aortitis*, on the other hand, represents probably the terminal stages of this long latent disease.

*When once the disease becomes known it kills in most cases within two years from the first symptom.* Thus in the 41 cases of the aneurismal type, the course of the *manifest* disease from the first symptom to death was under two years in all but three cases. In the 26 cases associated with aortic regurgitation all but seven had less than two years of life after their disease became manifest. In the cases of uncomplicated aortitis we can say nothing as to duration, since there were no symptoms clearly referable to the disease itself. In these latent cases the patients died of non-circulatory diseases such as pneumonia, general peritonitis, brain tumor, etc. Hence we cannot speak of the duration of the symptoms in this group. There were no symptoms.

**Age of Manifestation.**—It has often been noted that syphilitic aortitis is characterized by the appearance of *heart symptoms* or *aneurism symptoms in a middle-aged man*, previously free from such and not subject to rheumatism. This familiar observation is entirely borne out in our series. In 30 of our 41 aneurismal cases and 13 of those showing themselves by aortic regurgitation the disease appeared within the years from thirty-six to forty-nine. Only in one case of the entire series did the disease appear before the thirtieth year, only in five cases out of 92 before the thirty-fifth year. On the other hand only fourteen cases out of 92 were over fifty. Hence

TABLE 75.—AGE AT ONSET OF SYMPTOMS

| Age | Aneurismal type | Type with aortic regurgitation |
|---|---|---|
| Unknown | .. | 4 |
| 29 | .. | 1 |
| 30–34 | 3 | 2 |
| 35–39 | 9 | 7 |
| 40–45 | 11 | 7 |
| 46–50 | 11 | 1 |
| 51–55 | 2 | 2 |
| 56–60 | 4 | 0 |
| 61–66 | 1 | 2 |
| | 41 | 26 |

we may say: Roughly—the disease appears between 35 and 50. A more detailed picture of the age incidence is shown in Table 75.

It appears that *in men the symptoms usually appear earlier than they do in women.* Thus before the fiftieth year there are fifty-two men to 10 women; after fifty, eighteen men to eight women. But these figures are not of much significance, since the total number of cases is not large enough.

These ages are in manifest contrast with those of the two other main groups of heart disease;—the rheumatic which appears much earlier, usually before the twenty-first year, and the hypertensive type, which usually shows itself much later.

**Color.**—Few colored patients seek the assistance in the Massachusetts General Hospital. Hence the *small number* of colored patients in the aneurism series ( 5 out of 41) is not of any significance. There is good reason to believe that this, like most manifestations of syphilis, is decidedly commoner in negroes than in whites.

**Sex.**—All but six of the 41 aneurismal cases, all but 7 of the 26 cases of aortic regurgitation, and all but three of the uncomplicated group were in men. Of the three cases producing aortic stenosis two were female and one male. From these figures it is clear that the disease is *six times as common in men as in women*, if we leave out of account the three stenoses. The fact that two of these occurred in women raises a slight suspicion that in them there may be some other cause at work. I shall refer to this point later.

### *Occasions of Discovery*

The grounds of diagnosis in this disease will be discussed in detail later. But here it is convenient to point out that the disease is discovered:

(1) most frequently by the evidence of aneurismal pressure (pain, aphonia etc.);

(2) less often by the evidence of aortic regurgitation with or without broken compensation;

(3) by the appearance of angina pectoris in a young or middle-aged syphilitic man. In the vast majority of all cases the disease will be manifested, if at all, in one or more of these three fashions.

(4) Occasionally an X-ray of the chest taken for some other purpose reveals an unsuspected aneurism. I have seen one such case. Beyond this, one may say that in a known syphilitic patient the appearance of *any* cardiac symptoms raises the suspicion, perhaps the presumption, that these symptoms are syphilitic in origin.

**Position of the Disease in the Aorta.**—22 of 80 cases in our series affected the ascending aorta alone, 20 were confined to the region of the arch, and 5 to the vicinity of the aortic valves, 16 affected the whole aorta, 10 affected the whole thoracic aorta, 7 affected the arch and upper thoracic aorta. Obviously the most dangerous part of this disease is the part which attacks the aortic arch, where it can produce aneurism, angina pectoris, and aortic regurgitation. In the remainder of the aorta the disease *may* occasionally produce aneurism, but usually produces no recognizable disturbance of any sort.

**Associated Arteriosclerotic Lesions.**—In eight of the cases associated with aortic regurgitation, in fourteen of the aneurismal cases, and in nine of the uncomplicated cases—making a total of thirty-one cases, ⅓ of all, arteriosclerosis complicated the syphilitic process. In six of these cases the sclerosis affected the aorta generally, in three the aorta and its branches, in two the sclerosis is recorded as "general." Besides these cases there were four cases of arteriosclerotic nephritis and two of focal renal arteriosclerosis. I know no reason to believe that the associated arteriosclerosis was of any importance.

## DIAGNOSIS OF THE DISEASE IN LIFE

1. As already said, most cases, probably, are recognized by the presence of aneurism. In 23 of the 41 aneurismal cases in this group it was aneurismal symptoms which brought the disease to light. Sixteen of the aneurisms were not diagnosed at all, but a study of these cases shows that seven of these *should* have been diagnosed and three more perhaps suspected, leaving only 6 out of 41 that were quite impossible to reach, unless a purely fortuitous X-ray examination had brought them to light. In these six patients there were no pressure signs, no evidences of syphilis. One of them entered the hospital for general peritonitis due to perforated gastric ulcer; another was seen only in his final collapse; two others met their death from pneumonia, having had, so far as we can learn, no trouble from their aneurism. Another case dying of pneumonia should have been suspected of aneurism, however, in addition to the pneumonia, since he had had dysphagia, hard dry cough, and evidences of aortic regurgitation.

2. In the 26 cases manifested chiefly by aortic regurgitation the diagnosis of syphilitic aortitis was made in twelve, aortic and mitral endocarditis in 1, aortic and mitral insufficiency in 1, a diagnosis

merely of aortic regurgitation in five, a diagnosis of aortic endocarditis in three, a diagnosis of arteriosclerosis and angina pectoris in one, a diagnosis of cancer of the vagina in one, and no diagnosis at all in the 2 remaining cases. Probably all but one case of this group could have been diagnosed in life.

Of the 22 uncomplicated or latent cases, one was recognized in life upon the evidence of a luetic history, Argyll-Robertson pupils, and poor cardiac function. It is doubtful however whether in this case the diagnosis was well founded, since the patient had also a chronic nephritis which might have accounted for his cardiac troubles. In the remaining 21 cases of this group there was nothing in the patient's history on which diagnosis could be based.

**X-ray Diagnosis.**—Of the 41 aneurismal cases X-ray diagnosis was not attempted in 26. In the remaining 15 the disease was identified in seven, mistaken in five, and missed altogether in one. In two cases the radiologist remained in doubt. The five mistaken X-ray diagnoses predicted that a neoplasm would be found, largely, it would appear, because of the absence of demonstrable pulsation in the aneurismal sac. Evidently pulsation is not at all a reliable differential point in the distinction between neoplasm and aneurism by the X-ray.

In 24 out of the group of 26 cases manifested by aortic regurgitation there is no record of any X-ray examination. In the remaining *two* cases the radiologist reports enlargement of the aortic arch. But as this formula is a very common one accompanying a diagnosis of simple hypertrophy and dilatation of the heart (which was also present in these cases) we have no reason to believe that the radiologist thought these cases syphilitic.

Of the 22 uncomplicated or latent cases of aortitis, an X-ray examination is recorded in *three*, two of which are quite negative while in the third there is recorded a slight dilatation of the aorta,—a phrase which, like that just referred to in the last group, is equivocal and does not indicate any clear recognition of syphilitic disease on the part of the radiologist.

**Wassermann Reaction.**—The Wassermann reaction began to be done as a matter of routine only in the 5 year period covered by the last 1000 necropsies in this series. Hence in 56 out of our 92 cases no test was made. In the remaining 36 cases there were 29 positive (80%) and 7 negative which is about the figure reported by other observers.

These 36 cases were divided as follows:

TABLE 76

| Lesion | Wassermann | |
|---|---|---|
| | Positive | Negative |
| Syphilitic aortitis with aneurism | 11 | 2 |
| Syphilitic aortitis with aortic regurgitation | 13 | 1 |
| Pure syphilitic aortitis without aneurism or aortic regurgitation | 5 | 4 |
| | 29 | 7 |

In 19 of 29 positive cases the diagnosis was made (10 out of 13 with aneurism, 9 out of 11 with aortic regurgitation, none out of 5 "pure" aortitis) doubtless in part on the basis of the reaction. In some of the cases the details are of interest.

(a) *Aneurism Cases.*—In No. 3038 the diagnosis was suspected but not positively made. There was a syphilitic history 14 years ago, a *recurrens* paralysis, a suspicion of tracheal tug, sluggish and irregular pupils, a failing heart with a basal systolic murmur. Here the presence of a positive Wasserman was undoubtedly of value in supporting the diagnosis.

In No. 3369 a man of 38, with syphilitic history 22 years ago, with signs of a failing heart for a year, an apical systolic murmur, an unexplained cough and edema of the left arm, was apparently not suspected of aortitis in life. The positive Wassermann might have aroused such a suspicion.

In No. 3469 a man of 59, dying of prostatic obstruction in a surgical ward, had apparently very little cardiovascular examination. The aneurism was not suspected. Possibly the indication of the positive Wassermann might have led to some suspicion, as he had previously had cough and substernal oppression.

(b) *Cases with Aortic Regurgitation.*—In No. 2982 no diagnosis of syphilitic aortitis was made though there was a diastolic murmur along the *right* sternal margin with a Corrigan pulse. There had been definite symptoms of congestive failure at intervals for three years.

The Wassermann certainly should have suggested the diagnosis. In No. 3033 and in No. 3557 the conditions are essentially the same.

In No. 3499 the diagnosis was simply cancer of the vagina, but there was ample evidence of aortic regurgitation as well and the presence of a Wassermann *should* have clinched the diagnosis of aortic syphilis.

In No. 3748 the Wassermann was negative and there was good evidence of aortic regurgitation but without any proof of a syphilitic origin. There had been decompensation for a year with pain suggesting angina pectoris, which in a woman of 42 who had been divorced from one husband and deserted by another (one miscarriage, no children) is certainly enough to suggest syphilis strongly. But the absence of the Wassermann reaction probably led to the ward diagnosis of aortic regurgitation and stenosis, without syphilis.

(c) *"Pure" Cases.*—No. 3759. Clinical diagnosis: "femoral embolism, myocardial weakness, fibrillation. Wassermann ++. Chancre 25 years ago. Probably syphilis somewhere. Heart weak." Aortitis might have been suspected, I think.

No. 3619. Colored, female, 60. Died of acute pericarditis and chronic nephritis after an operation for cancer of cervix. Wassermann ++. Aortitis, as one of the commonest sites for syphilis, might have been suspected.

No. 3607. Male, 36, died of post-operative hemorrhage after an operation for intestinal obstruction. X-ray recorded "slight dilatation of arch." Wassermann suspicious. At 36 these two facts might have led to a suspicion of aortitis.

No. 3526. Male, 35. Aneurysm of superior mesenteric with syphilitic aortitis. Diagnosis made in life apparently from diastolic thrill at the base (no murmur recorded). Suspicious Wassermann. At necropsy no aortic valve involvement. Wassermann may have helped here.

### Cardiac Hypertrophy in Syphilitic Aortitis

*In the aneurismal group* there was no record of cardiac hypertrophy in 28 out of 39. The heart weights were recorded as normal in five cases and under 290 grams in five more. In six cases the weights were between 300 and 400 grams; in eleven the weights were from 400 to 1000 grams; in two the only record is "slightly hypertrophied," and in 12 we have no definite figures.

It is of interest to seek the cause of hypertrophy in the group of 11 out of 39 aneurismal cases in which it was marked. 8 of 11 cases are accounted for by aortic regurgitation. Of the remaining three

TABLE 77.—HEART WEIGHTS

| Weight | Aortitis with aneurism | Aortitis with aortic regurgitation | Uncomplicated aortitis |
|---|---|---|---|
| "Large" | .. | 1 | |
| Normal | 5 | 0 | 0 |
| Under 290 grams | 5 | 0 | 3 |
| 300–320 grams | 3 | 1 | 4 |
| 340–400 grams | 3 | 1 | 5 |
| 400–450 grams | 4 | 2 | 4 |
| 450–500 grams | 0 | 1 | 0 |
| 500–600 grams | 3 | 5 | 4 |
| 600–700 grams | .. | 7 | 2 |
| 700–800 grams | 4 | 5 | 0 |
| 800–1000 | .. | 3 | 0 |
| "Slight" | 2 | 0 | 0 |
| Not recorded | 12 | 0 | 0 |
| | 41 | 26 | 22 |

cases, two show only moderate enlargement, 402 and 450 grams respectively. One of these patients died after an operation, without cardiac symptoms. The other had had dyspnea for two years and died with evidences of passive congestion. The third case had also had dyspnea for six months and died of passive congestion. In this case the heart weighed 553 grams. *In none of these cases, it seems to me, do we need to suppose that there was any connection between the aneurism and the cardiac hypertrophy.* Aortic regurgitation accounts for most of them, and in the rest the hypertrophy may well have been associated with a previous hypertension. All the heaviest hearts—1000 grams, 950 grams, 940 grams—were associated with aortic regurgitation in addition to the aneurism.

*Of the 26 cases manifested chiefly by aortic regurgitation*, all showed, according to the pathologist, a greater or less degree of hypertrophy. In 20 cases this produced hearts weighing more than 500 grams, and of these 20, 5 weighed more than 600 grams.

*Of the 22 cases of uncomplicated or latent aortitis*, 17 showed hypertrophy, though in only six did this hypertrophy produce a weight of more than 500 grams. In the cases with hypertrophy there was, we may assume, a hypertension, since all showed an associated arteriosclerosis or nephritis, the latter accounting for three of the six cases with hearts weighing more than 500 grams.

From the whole group of cases studied in this chapter, then, we may conclude that *syphilitic aortitis does not produce cardiac hypertrophy unless it involves the aortic valves*, producing regurgitation or, rarely, stenosis. *Aneurism does not produce hypertrophy of the heart*, nor does uncomplicated aortitis. But of course these diseases do not inhibit the action of the commonest of all causes for cardiac hypertrophy, namely hypertension with or without nephritis.

## GENERAL CARDIAC SYMPTOMS OF SYPHILITIC AORTITIS

(1) *Dyspnea*, the commonest of all heart symptoms, was present in most of these cases except in the "uncomplicated" group. Of the 41 aneurismal cases 31 showed more or less dyspnea. This was apparently of the cardiac type in 18 cases and due to local pressure in 10. In 14 of these 18, aortic regurgitation was present, in the rest only hypertrophy and dilatation of the heart. Dyspnea was due to aneurismal pressure or obstruction in ten cases. Seven of these included paralysis of the recurrent laryngeal nerve. This *pressure-dyspnea* was usually of the "*asthmatic*" type, that is, it produced wheezing and difficult expiration. In three cases this was definitely paroxysmal and had been diagnosed as asthma over a considerable period of years. In one, the "asthma" showed (naturally) a considerable improvement under iodide of potash, which fact was considered during life a proof that the diagnosis of asthma was correct.

In two case dyspnea had lasted but a few days and was due presumably to the pneumonia with which the patient died.

These three causes: pneumonia, pressure on respiratory structures, and cardiac failure, account for the dyspnea in all the cases except one, in which a man of forty-five died with symptoms of passive congestion, although the heart weighed but 286 grams and showed no hypertrophy. He had complained of dyspnea for twelve months and no explanation for this was found after death.

In the 23 cases associated with aortic regurgitation dyspnea was absent in only four cases, doubtful in one. All the others showed it for varying periods, but in 16 out of 20 cases this period of dyspnea—coinciding with the patient's awareness that he was ill—was two years or less, and in only three of the cases did it extend beyond one year. In contrast to this, one case gave a history of dyspnea for five years and one for eight years. In this type of the disease the dyspnea was ordinarily the first symptom, though it was preceded in five cases by other symptoms; by angina in two, palpitation in one, edema in one, and cough in one.

In the uncomplicated or latent cases dyspnea was present only when an associated hypertrophy and dilatation had weakened the heart. Only in one case of this group was there death from passive congestion. The other causes of death will be mentioned later.

(2) *Angina.*—Pain of the anginoid type was present in five of 41 aneurismal cases, in two of the uncomplicated cases, and in 8 of those associated with aortic regurgitation, making *a total of* 15 *cases* out of 92. Yet evidently this was not the revealing symptom which brought the disease to light in most cases of this series.

The coronary arteries were blocked either at their mouths or in their course in five of the regurgitant cases. In the uncomplicated cases the coronaries were normal except in two. In other words, in this relatively early or mild form of the disease the coronaries ordinarily escaped, as they did in all the aneurismal cases.

These cases bring out a fact elsewhere noted, that angina pectoris is prone to be associated with syphilitic aortitis, whether there is any coronary blocking or not.

The ages and the sex in these 15 cases of syphilitic angina were as follows:

TABLE 78

| No. | Necropsy No. | Age | Sex | Type of aortitis |
|---|---|---|---|---|
| 1 | 3222 | 29 | M | With aortic regurgitation |
| 2 | 2604 | 30 | F | With aortic regurgitation |
| 3 | 1143 | 32 | M | With aortic regurgitation |
| 4 | 1816 | 36 | M | With aortic regurgitation |
| 5 | 3607 | 36 | M | Uncomplicated |
| 6 | 448 | 38 | M | With aneurism |
| 7 | 1280 | 43 | M | With aneurism |
| 8 | 1810 | 43 | M | With aneurism |
| 9 | 916 | 49 | M | With aneurism |
| 10 | 2534 | 49 | M | Uncomplicated |
| 11 | 2488 | 50 | M | With aortic regurgitation |
| 12 | 2980 | 50 | F | With aortic regurgitation |
| 13 | 3918 | 52 | F | With aneurism |
| 14 | 3837 | 57 | F | With aortic regurgitation |
| 15 | 3998 | 71 | M | With aortic regurgitation |

In 8 of these 15 cases, the angina comes at a relatively early age. One notices, however, that 3 of the 4 females in this list are from 50 to 57 years old.

## CAUSE OF DEATH

Naturally, in the cases with aortic regurgitation, that lesion and its results usually led to death from passive congestion. Only in two was angina undoubtedly the lethal event. In two others it was associated and perhaps dominant, though passive congestion was also well marked.

In the aneurismal group 14 died apparently of cardiac failure see above), though in four of these there is some doubt. Only 6 out of 41 died of rupture of the aneurism. Four were post-operative deaths; four died of respiratory failure (choking), three of pneumonia, three of uremia, one of general peritonitis, one of intestinal obstruction, and four without known cause.

Of the cases of uncomplicated or latent aortitis, four died of nephritis, four of post-operative conditions, three of pneumonia, one each of empyema, pulmonary cancer, brain tumor, acute pericarditis, sepsis, general peritonitis and prostatic disease. In one an unexplained passive congestion was apparently the main cause of death, and in another it was associated with sepsis.

The fourteen aneurismal deaths from cardiac failure *seem* to correspond with the fourteen cases of aortic regurgitation included in that group. But as a matter of fact the two numbers do not precisely correspond. In fact, eight of the aneurismal cases dying a congestive death had no regurgitation. On the other hand, one of the patients in whom aortic regurgitation was present died of pneumonia, two of uremia, a third of rupture of the aneurism, another of respiratory pressure, while in two more the nature of the death was never explained.

## SPECIAL SYMPTOMS OF ANEURISM

*An abnormal area of pulsation* was noticed in fourteen out of 41 cases, not counting pulsation seen in the neck or at the xyphoid. The pulsation involved the left upper chest in four cases, the right upper chest in four, the sternal and parasternal region in three, in the rest is not clearly described. This abnormal pulsation was associated with *tumor in eight out of fourteen*, with *thrill* in only three—a surprising point. Of the total five cases showing thrill two were not associated with any pulsation. *Percussion dullness* was associated with the pulsating area in only three cases, and diastolic shock in three. *Tracheal tug* occurred in association with a pulsating area in five cases and without it in three.

*A systolic thrill* was present in five cases only, three of them, as has already been mentioned, without any abnormal area of pulsation.

*Paralysis of the recurrent laryngeal nerve* was noted in twelve cases. Its most common associations were with unequal pupils in six cases, and with tracheal tug in five cases.

*X-ray examination* gave the correct diagnosis in seven cases. In two of these it was the only sign of importance, and in another was the only sign of importance except a diastolic murmur.

*Tracheal tug* was noted in eight cases. In was associated with *recurrens* paralysis in five, with abnormal pulsation in five, with unequal pupils in four, with tumor in two.

*Inequality of the pupils* was noted in ten cases. It was associated with *recurrens* paralysis in six and with diastolic murmur in four.

*Systolic murmurs* at the base of the heart were noticed in twenty-seven cases and absent in only four. *Diastolic murmurs* were heard in fourteen cases.

The actual association post-mortem of aortic regurgitation was fifteen cases out of 41. Thirteen of these proved cases had diastolic murmurs; three others not so proved also had diastolic murmurs. If we count these three cases as having aortic regurgitation we have a total of 18 cases out of 41, showing regurgitation, and these with 26 other cases make a total of 44 cases (out of 92) in which aortic regurgitation was also present. In the three cases of stenosis a regurgitation may be assumed.

### THE DIAGNOSIS OF ANEURISM

It has already been said that all but six of the 41 aneurisms either were diagnosed or should have been diagnosed during life. Thirteen of those which were recognized during life had no evidences of aortic regurgitation. But in the majority this lesion was present, including four of those not recognized. Hence it appears that *in syphilitic cases with aortic regurgitation the presence of aneurism should always be suspected* and sought for by all the other diagnostic means at our disposal. *Summing up the physical signs of aneurism:*

TABLE 79.—PHYSICAL SIGNS OF ANEURISM

| | |
|---|---|
| None | 5 |
| Some | 36 |
| Dull area | 22 |
| Diastolic murmur | 14 |
| Pulsating area | 14 |
| Arterial phenomena of aortic regurgitation | 12 |
| Recurrens paralysis | 12 |
| Unequal pupils | 10 |
| X-ray evidence | 7 |
| Tracheal tug | 8 |
| Tumor | 8 |
| Thrill | 5 |
| Diastolic shock | 3 |

I have not mentioned here the 27 cases showing *systolic* murmurs, because these do not seem to be of diagnostic importance. Of the fourteen cases with diastolic murmurs, it may be said that this was the only significant sign in two cases, that in two others this with the peripheral arterial phenomena characteristic of aortic regurgitation were the only striking signs, and in one other the association of diastolic murmur and X-ray evidence was the only fact on which we had to build. Arterial phenomena were associated with the diastolic murmur in ten cases. A presystolic murmur was present with the diastolic in two cases and without it in one, making a total of three cases that can be said to have had the "Austin Flint murmur" at the apex of the heart.

*Pressure Signs.*—Eleven cases showed evidence of pressure on respiratory structures. In eight of these the pressure was evidenced by stridor or wheezing, in two by so-called "asthma," and in one by laryngeal obstruction.

*Dysphagia* was noted in only one case in the whole group. I think this scantiness was due to insufficient questioning of the patients.

*Edema in the Aneurismal Region.*—One arm was swollen in three cases, both arms in two cases, the face in one case, the chest and arms in one, the neck and jaws in one.

*Pain in Aneurism.*—Excluding pain of the anginoid type we find pain present as an early symptom of aneurism in *seventeen* cases; dyspnea had preceded it in ten cases, and accompanied it in six; edema, cough and hoarseness had preceded it each in one case. Ten cases were noticeably free from pain and were obviously of the type due to pressure on respiratory structures, which might be called "*the asthmatic type.*" One case (No. 878) occurred in a man of forty-five who got his primary syphilitic infection thirteen years before we saw him and began to suffer from "asthma" in the following year. Post-mortem examination in this case showed that the aortic arch was dilated into a huge sac of irregular shape compressing the left primary bronchus, eroding the manubrium and the 2nd and 3rd ribs to the left of the sternum. The bronchial stenosis had led to bronchiectasis with chronic interstitial pneumonitis and abscesses. He had no aneurismal symptoms except "asthma" until a year before death, when he began to notice pain in the left chest and shoulder.

In ten cases the symptoms were those of *cardiac failure* without other notable symptoms, or anything characteristic of aneurism. Two showed angina as their first symptom and might be said to belong to the "angina type."

## CARDIOVASCULAR COMPLICATIONS

TABLE 80

| | |
|---|---|
| 1. In 39 out of 94 cases—nearly half—there were no other cardiovascular lesions | 39 cases |
| 2. The commonest complication was arteriosclerosis, present in | 23 cases |
| 3. Next comes acute (terminal) pericarditis | 8 cases |
| 4. Next comes nephritis | 7 cases |
| 5. Next comes chronic non-deforming endocarditis | 7 cases |
| 6. Valve lesions (rheumatic type) | 4 cases |
| 7. Chronic adhesive pericarditis | 3 cases |
| 8. Myocarditis, acute endocarditis, and arteriosclerotic degeneration of the kidneys, each | 3 cases |
| | 94 |

The most interesting point is *the rarity of rheumatic valve lesions*, only *four cases in* 94. This means that if in our diagnostic examination we are convinced that any valves other than the aortic are involved, the case almost certainly is not syphilitic aortitis.

## CONCLUSIONS

1. So far as our studies go, cardiac syphilis means a syphilitic aortitis which is usually confined to the arch of the aorta.

2. This is often, perhaps usually, harmless, a mere *post-mortem* finding.

3. It becomes serious (a) when it produces aneurism, (b) when it deforms the aortic valves and makes them leak, and (c) when it obstructs the coronary orifices in the aorta so as to produce angina or cardiac infarction.

4. It is recognized

(a) by the presence of aneurism,

(b) by evidence of aortic regurgitation appearing usually in a middle-aged man with a syphilitic history.

5. It is six times as common in men as in women, and usually makes itself felt between the 35th and the 50th year. The aneurismal type appears later in life than the type manifested by aortic regurgitation.

6. The disease appears from fifteen to twenty years after the original syphilitic infection.

7. When symptoms finally appear they usually lead to death within two years.

8. Enlarged heart is found only when the aortic valves are incompetent or when some complication (such as chronic nephritis) is present. Aneurism does not produce cardiac hypertrophy.

9. Rheumatic valve lesions rarely accompany syphilitic aortitis.

10. An isolated aortic regurgitant lesion in a man under the forty-fifth year is usually syphilitic. Only eleven of our 41 cases of "pure" non-syphilitic aortic disease were in persons under forty-five years old, while only 18 of 41 cases of syphilitic disease in the aortic valves occurred after the forty-fifth year.

11. Pain suggesting angina pectoris in a young or middle-aged man (i.e. under forty-five) is often evidence of syphilitic aortitis. Severe epigastric pain (perhaps of the anginoid or infarction type) is a feature of some cases.

12. Death in the cases with aortic regurgitation is usually from passive congestion. In the aneurismal cases only 14% died from rupture of the sac, and only in 24% (including the cases of rupture) was the aneurism the main cause of death.

13. Among the physical signs of aneurism abnormal percussion dullness is more often of value than any other single sign. Abnormal pulsation or bulging is next in value, and *recurrens* paralysis next.

14. X-ray evidence is in some cases our best help towards diagnosis, yet in five cases the radiological diagnosis was mediastinal tumor, and in one case the aneurism was missed altogether.

15. Chronic aneurismal "asthma" may be due to compression of a primary bronchus or of the trachea by an aneurism.

## POINTS OF SPECIAL INTEREST TO THE WRITER

1. The frequency of syphilitic aortitis in a wholly latent and apparently harmless form.

2. The infrequent association of syphilitic with rheumatic heart disease.

3. The long interval between the syphilitic infection and the appearance of aortic symptoms.

4. The fact that aneurism does not cause cardiac enlargement.

5. Isolated aortic disease or angina pectoris under the forty-fifth year is generally syphilitic.

## ILLUSTRATIVE CASE OF SYPHILITIC AORTITIS

### Necropsy 4734

A Canadian railway engineer of thirty-nine entered September 3 complaining of nervousness, loss of speech and pain in the stomach. His memory was very defective, especially for symptoms relating to the central nervous system. The story given in the wards differed in most respects from that given in the Out-Patient Department.

One brother died of kidney disease.

The patient's past history was negative except for some dyspnea and pain across the chest upon exertion.

For six years he had had running of the nose. At one time he gave this as his chief complaint, though he said he was not troubled with head colds. For eight months he had had attacks of motor aphasia every week or two lasting one or two days. For three or four months he had been growing weaker. His dyspnea and chest pain on exertion had increased. For six months he had had colic with each stool. For three months the pain in the stomach had been getting worse. It was continuous and partially relieved by food. He was becoming more nervous and irritable. His sister reported temporary amnesia. The day before admission he was dizzy for the first time. During the illness he had gained weight.

In the Out-Patient Department June 3 his blood pressure was found to be 128/84. The abdominal reflexes were present but sluggish, especially the cremasterics. The deep reflexes of the upper extremities and the knee and ankle reflexes were normal. Babinski was present both sides. Oppenheim, Chaddock and Gordon were also present. There was no ankle clonus. Finger counting was awkwardly done. He did not recognize the smell of acetic acid, alcohol or ammonia. The skin and mucous membranes were pale. The right pupil was greater than the left and did not react to light. The left reacted poorly. Both reacted normally to distance. The fundi were normal. August 30 the red blood corpuscles numbered 2,080,000, the polynuclears 74%, the hemoglobin 50%, the platelets were somewhat decreased. There were some microcytes and occasional macrocytes, not much change in the shape of the cells, which were often well laden with hemoglobin. There was striking sallowness and pallor of the mucous membranes. The tongue showed slight atrophy of the marginal papillae.

Examination showed a well nourished, anemic looking man whose mind functioned rather slowly. All the mucous membranes were pale. Pus was expressed from the root of one incisor. The tonsils were moderately enlarged on the left. The heart sounds were of fair quality. The action was normal. The apex impulse was 8 cm. from midsternum in the midclavicular line, the left border of dullness one cm. outside the midclavicular line. There was no other enlargement to percussion. The sounds were of fair quality, the action normal. There were no murmurs. The blood pressure was 105/72. The lungs were normal. The abdomen showed voluntary spasm.

A possible liver edge was felt 7½ cm. below the costal margin. The bladder dullness was two finger-breadths above the symphysis. The genitals and rectal examination are not recorded. The extremities were normal. The pupils were unequal, the right greater than the left. The right reacted to distance but not to light. The right knee-jerk was greater than the left. There was inconstant Oppenheim. The gait was that of a weak man rather than anything else. Rectal examination showed a sphincter which seemed to have lost some of its strength. The fundi showed slight arteriosclerosis. The right disc showed a vein extending a short distance into it on the temporal side and then seeming to disappear.

The temperature was 101.9° to 102.5° at entrance, then 98.1° to 100.3° until September 8, when it rose to 103.5°; afterwards not remarkable until a terminal rise beginning September 17 and reaching 107.8°. The pulse was 78 to 104, with a terminal rise to 161. The respirations were normal except for a terminal rise to 44. The amount of urine is not recorded. The specific gravity was 1.008 to 1.018. There was the slightest possible trace of albumin at both of two examination, leucocytes at both, one or two red blood corpuscles per high power field at the second. September 4 he was catheterized. The residual urine was 32 ounces. The hemoglobin was 60 to 55%. There were 6500 to 10,600 leucocytes, 75% polynuclears, 2,720,000 to 1,929,000 reds, some polychromatophilia and variation in size, with a tendency to large cells, slight variation in shape, occasional achromic cells at one of three examinations, reticulated cells 2% at another. A Wassermann in the Out-Patient Department three days before admission was strongly positive. Three Wassermanns in the ward were strongly positive. The bleeding time was 2 minutes, coagulation time 12 minutes. Retraction was normal in one hour and marked in three hours. The serum dilution was 1:20; this was a surprise. The non-protein nitrogen was 40 mgm. September 5 the fasting contents of the stomach were 30 c.c. of slightly blood tinged white material showing no free HCl, total acid 3 c.c. A test meal gave 50 c.c. of blood tinged white material, total acid 9 c.c. Both showed fresh blood and a very strongly positive guaiac. Lumbar puncture September 4 showed an initial pressure of 180, oscillation normal, jugular compression 260, respiration 170. After withdrawal of 5 c.c. the pressure was 150, after withdrawal of 5 more c.c. 140. There were 6 cells. Alcohol and ammonium sulphate were positive, the total protein 63, goldsol 5555553110, Wassermann strongly positive. September 17 the initial pressure was 270, after

withdrawal of 5 c.c. 210, after withdrawal of 5 c.c. more 150. The jugular compression is not recorded. There were 7 cells. Ammonium sulphate and alcohol were positive, Wassermann strongly positive, total protein 55, goldsol 5555542000. X-ray September 6 showed the frontal sinuses practically absent; no anatomical variation. The antra showed no definite evidence of pathological change, although the plates were not very satisfactory. The calvarium was normal in outline. The sella showed no abnormalities in size or shape. There

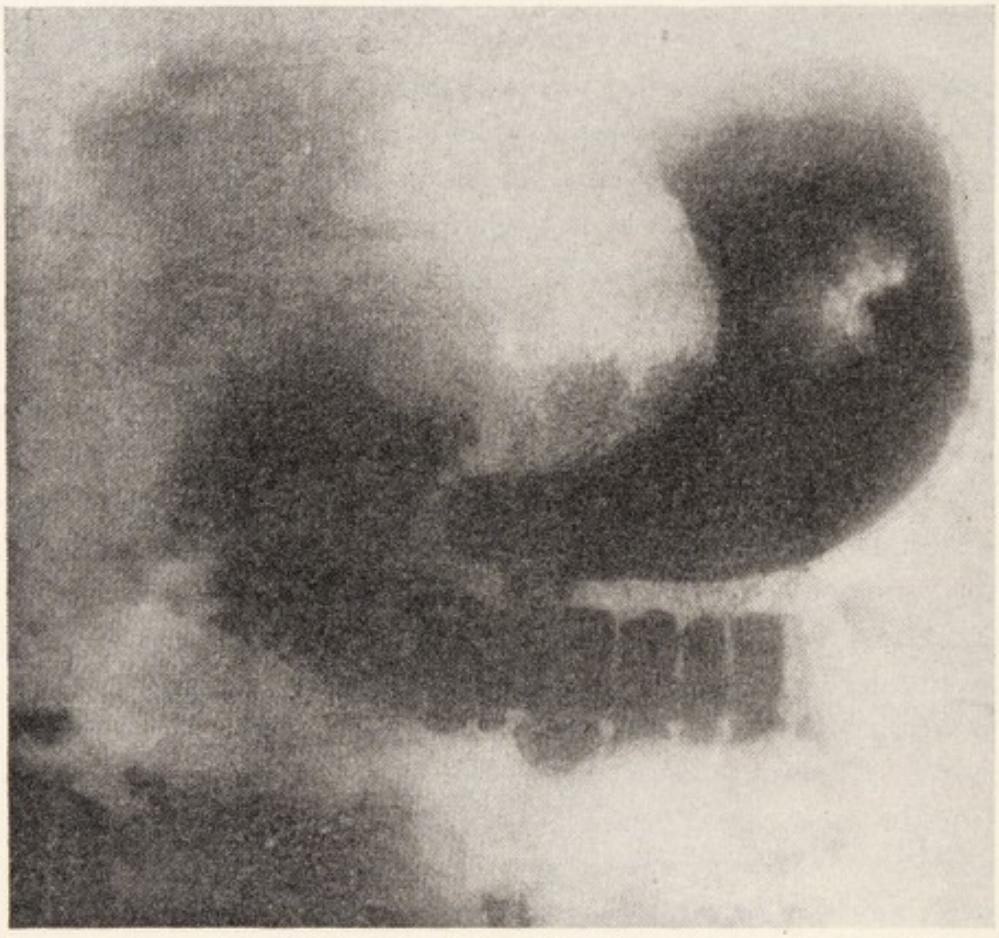


FIG. 55.—Area of diminished density in midportion of stomach suggesting a mass nearly as large as the palm arising from the wall of the stomach, freely movable with the stomach.

was little calcification in the region of the pineal, which was normal. A plate of the chest was not entirely satisfactory. There was no definite evidence of pathological change; no evidence of periostitis of the clavicles. Another examination September 12 confirmed the previous findings. There was an area of diminished density in the midportion of the stomach suggesting a mass arising from the wall of the stomach nearly as large as the palm, freely movable with the stomach. (See Fig. 55.) There was no peristalsis over this region. The shadow was constant on repeated examinations. There was a

small six-hour residue. The first portion of the duodenum, the pyloric sphincter, and the colon were not remarkable.

During the first two nights in the hospital the patient was disoriented and laughed easily. He looked myxedematous. He made no general improvement until the 9th, when he was mentally a little clearer, although still disoriented as to time and place in the immediate past. He was very restless, with poor insight.

The morning of September 17 he suddenly saw double and had complete disassociation of the eyes, one moving upward, the other downward. The left eye showed a lateral nystagmus with quick component to the right. There was no ptosis. Vomiting soon started, but was not projectile in type. The vomitus contained brown changed blood at first, one pint three times, and later bright red blood in the stomach fluid, one pint twice. Donors were collected and large doses of morphia given. The vomiting stopped and counts were found to be normal, the pulse steady. The eyes were bandaged. They were widely divergent and moving ceaselessly, even while he was deeply asleep. The blood pressure was 140/75 all that day and evening. The temperature, which had been practically normal for a week, began to rise, reaching 107.7° the evening of the 18th. The pulse rose to 162, the respirations to 44. Examination on the 18th showed no evident paralysis, although the left arm was found spastic the night before. His whole body was now flaccid. The arm reflexes were active, the knee-jerk very active on the right, normal on the left. There was marked Babinski on both sides, not present the night before. No clonus. The eyes were now divergent and the right pupil greater than the left. The heart was very rapid. The lungs were clear except for fine crackling râles in the right back between the angle of the scapula and the spine. The patient was in mild shock, unconscious. The breathing was Cheyne-Stokes. He continued in coma. The left pupil was pinpoint, the right dilated. The right fundus showed dilatation of the veins but no choking of the discs. The eyeballs continued divergent. On the 19th there was edema of both bases. Bubbling râles were heard well up to both angles of the scapulae. The temperature rose steadily. The breathing was very shallow, rapid and stertorous and continued Cheyne-Stokes. The breath was foul. The pulse was rapid and thready. The patient sweated profusely. A soft short systolic murmur was heard at the apex and the base. $A_2$ was greater than $P_2$. The right side of the heart was slightly enlarged to percussion, possibly dilatation. The abdomen was slightly distended with gas, not relieved by

an enema. The patient was catheterized. The Babinski persisted. There was no Kernig. September 19 he died.

*X-ray Interpretation September 12.*—Findings probably represent new growth in the wall of the stomach.

*Clinical Diagnosis (from Hospital Record).*—Syphilis and malignancy of stomach.

Cerebral hemorrhage?

Syphilis, tertiary stage?

*Dr. Richard C. Cabot's Diagnosis.*—Syphilis of the central nervous system.

Gastric, tumor, probably benign, with hemorrhage.

Secondary anemia.

*Anatomical Diagnosis.*—Embolism of the basilar artery.

Anemic infarction of the pons.

Adenomatous polypi and adenocarcinoma of the stomach.

Embolism of a minute branch of the left coronary artery.

Infarcts of the spleen and kidneys.

A few areas of bronchopneumonia, right.

Wet brain.

Slight chronic pleuritis, right.

Luetic aortitis with small aneurism and mural thrombus.

DR. RICHARDSON: This was a very unusual case.

The pia was wet, infiltrated with thin pale clear fluid. The vessels of Willis showed a very slight amount of sclerosis in places, nothing remarkable. The basilar artery was occluded at its distal portion by a frank embolic columnar mass which was prolonged a little into the branches at its bifurcation. (See Fig. 56.) Practically the entire pontine tissue was softened, pasty, pale and homogeneous,—frank anemic infarction. The brain tissue generally was a little wet.

The esophagus was negative. Hanging off from the anterior wall of the stomach midway was a large adenomatous-like mass, as seen in Fig. 57. Macroscopically it looked like an adenoma. There were several hemorrhagic areas in the peripheral portion of the mass, and areas of ecchymosis in the mucosa of the stomach. Microscopic examination showed at one place in this adenomatous tissue a slight invasion of the wall of the stomach. That would make it of course cancerous in that portion. So we have a mass the greater portion of which is adenomatous but at one place an area of cancerous degeneration.

The intestines were negative save that they contained a little bloody mucus and blood-like material.

The lungs were negative except that on the right side at one place there were a few small areas of bronchopneumonia.

The heart weighed 370 grams, a little large, a little dilated. The aorta in the region of the ascending thoracic was the seat of a definite luetic aortitis. (See Fig. 58.) One of the areas of luetic aortitis was capped by frank thrombotic material. Across from this larger area were two or three smaller ones capped with thrombotic material.



FIG. 56.—Embolism of the basilar artery. ×3. (Photograph, with enlargement, by Louis M. Adams. Dr. Oscar Richardson.)

The spleen showed two or three infarcts and the kidneys showed several infarcts.

Microscopic examination of the aorta showed luetic aortitis.

Every once in a while we find mural thrombi on the aorta. Sometimes we find infarcts associated with them.

DR. CABOT: How big was the pedicle of the stomach tumor?

DR. RICHARDSON: The pedicle ran along the base of the mass without much thickening of the wall underneath, and it was only at that one point microscopically that it showed carcinoma. The mass was 7 cm. × 4½ cm. × 3½ cm.

DR. CABOT: It was not a papillary mass not like a papilloma of the bladder?

DR. RICHARDSON: No. It did not have a small pedicle, but a pedicle running all along the base of the mass.



FIG. 57.—Adenomatous polyp of the stomach. (Photograph by Louis M. Adams. Dr. Oscar Richardson.)

These adenomatous masses along the gastrointestinal tract are not uncommon. I have seen a stomach that was practically covered with them, like the knotted fringe of a shawl.

The large intestine of one of our former cases presented in the upper portion of the descending colon adenomatous masses while in the lower part of the same colon there was frank cancer.

DR. CABOT: Is adenoma the ordinary benign tumor of the stomach?

DR. RICHARDSON: Adenoma, fibroma, fibrolipoma—I should say adenoma is probably the commonest.

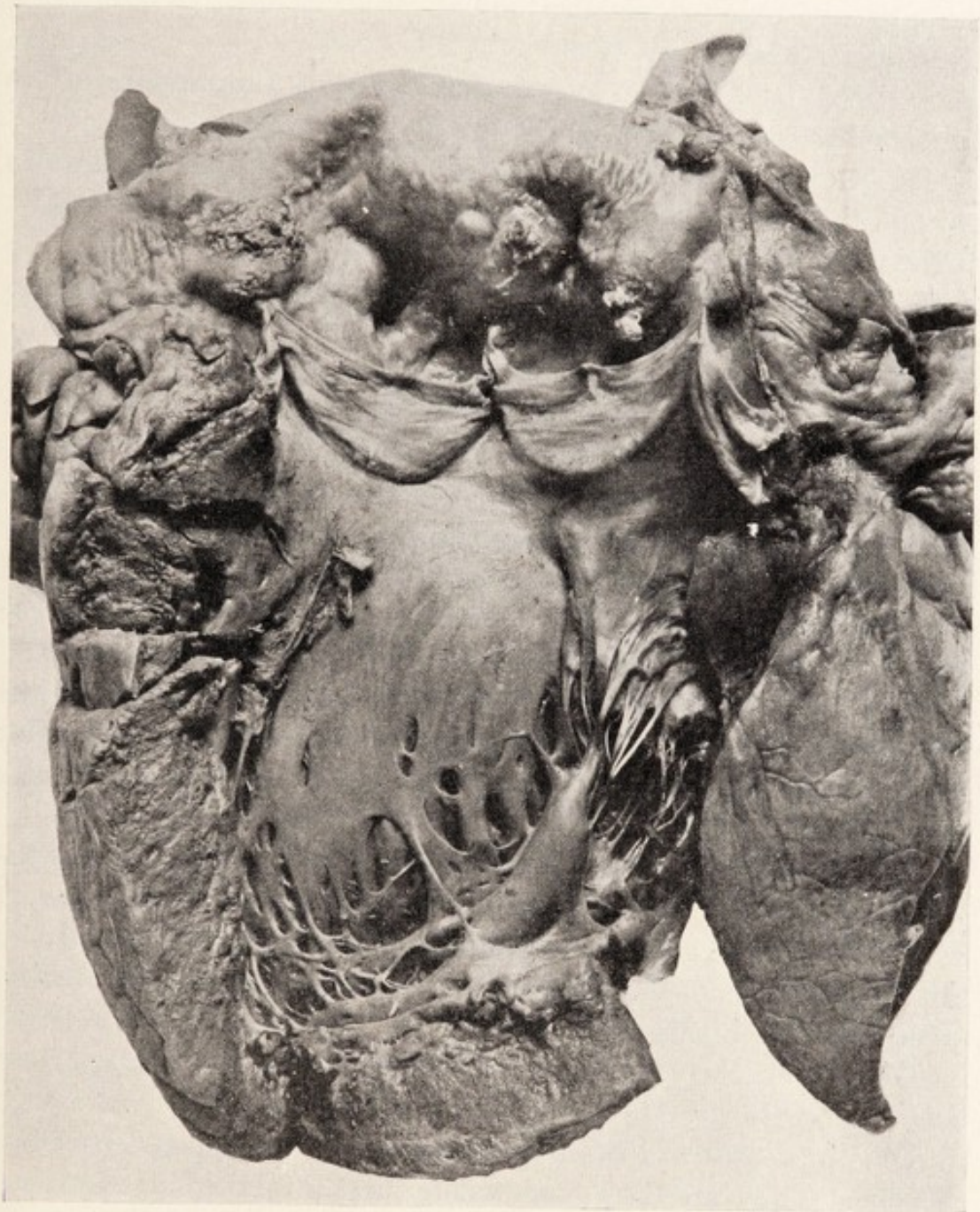


FIG. 58.—Syphilitic aortitis with small aneurisms. Aortic valves stretched close to aortic wall so as to be incompetent. (Photograph by Lewis S. Brown. Dr. Oscar Richardson.)

The anatomy of the heart is beautifully shown in Fig. 58. There was but little if any decrease in the orifices of the coronary arteries. In the first portion of the aorta, just above the aortic cusps, which were not invaded by the luetic process, the thrombotic caps are well

shown. One cap was about one and a half cm. across and consisted of brown soft thrombotic material. The other smaller caps are apparent. The black shadow is a small aneurism. Along the aorta we can see irregular streaks and areas of thickening and elevation and thinning and depression, longitudinal striation, etc., characteristic of luetic aortitis.

A PHYSICIAN: Do you ever find evidence of the presence of the spirochetes?

DR. RICHARDSON: Once in a while. When the process is old we do not find them. But if the material was all cut up in serial sections we might find a few.

A PHYSICIAN: We might conclude from that that the spirochetes at times are floating in the blood?

DR. RICHARDSON: Yes. It is or was a septicemia.

In this case then a bit of that material from the mural thrombus of the aorta was swept up into the basilar artery, shut if off, and produced anemic infarction of the pons. Then bits were swept down into the splenic and renal arteries and produced the infarcts of the spleen and kidneys. In addition to that, in speaking of the heart I forgot to mention that a very minute branch of the coronary artery was also occluded by a very minute embolus.

DR. LINCOLN DAVIS: I should like to ask a question. Don't you think it is extraordinary that an embolus can occlude the basilar artery? The basilar artery is larger than the two arteries that make it up.

DR. RICHARDSON: No. The basilar artery is larger than the vertebral arteries, but the basilar artery gives off branches and at its distal end bifurcates into two smaller arteries, and so anatomically this distal portion of the basilar is well arranged for the lodgement of an embolus, as in this case.

DR. DAVIS: Have you ever had an embolus of the basilar artery before?

DR. RICHARDSON: I do not remember at the moment a case like this one. There are those cases where there is marked arteriosclerosis of the vessel with arteriosclerotic thrombotic occlusion at the site. We must remember that this embolus in the plate looks huge; it was of course roughly only twelve and a half mm. long, two mm. in diameter.

DR. CABOT: I should interpret this man as having syphilis of the nervous system. I do not think Dr. Richardson would in any way contradict that. He did not examine the cord, and the changes

in the cerebral arteries in a man of thirty-nine are such as we think of with syphilis. There is nothing in any of the findings post-mortem which would account for a spinal fluid such as was found in life.

The tumor in the stomach seems to be partly benign and partly malignant, so I think we did pretty well on that. Dr. Holmes said malignant, I said benign. Did you look at the marrow? He had a great anemia and I discussed and excluded pernicious anemia.

DR. RICHARDSON: The vertebral marrow was negative macroscopically.

DR. CABOT: I do not believe there was any pernicious to it.

## SYPHILITIC AORTITIS WITH AORTIC REGURGITATION. ILLUSTRATIVE CASES

### Necropsy 3912

An unoccupied negro of thirty-six entered December 27 for relief of sleeplessness and nausea. His mother died of heart trouble. His health had been fair. At eighteen he had typhoid fever. For five years he had had hemorrhoids. At thirty-two he had acute pain in the right lower quadrant. For six months he had been very thirsty. His bowels were constipated. For the past week they had been moving hourly as a result of catharsis. He had a little cough with dark colored sputum and occasionally hemoptysis. His best weight was 145, his usual weight 135.

Four months before admission he became dyspneic and had increasing edema of the ankles and sleeplessness. Soon afterward he began to sweat at night. In order to rest at all he required three pillows. After slight exertion he had attacks of boring precordial pain sometimes lasting three or four hours. For two months he had been taking veronal every third night with no appreciable relief. For three weeks he had had nausea and vomiting, caused he thought by his medicine. He now had continual nausea, partially relieved by vomiting. For ten days there had been pain localized at the umbilicus.

Examination showed a well nourished man. The apex impulse of the heart was seen and felt in the fifth space 14 cm. to the left of midsternum. The action was rapid, the sounds of fair quality. The aortic second sound was accentuated. There was a diastolic murmur at the aortic area and a systolic at the apex, transmitted to the axilla. The murmurs were heard in the left back. There were loud sounds over the vessels in the neck. The pulses were of high

tension, the artery walls palpable, the brachials tortuous. The blood pressure was 165/55. There was a sound over the brachial with no pressure. The lungs were normal. The abdomen was tympanitic except in the right upper quadrant. The liver dullness extended from the fifth space to 5 cm. below the costal margin, where the edge was felt. There was slight bilateral costovertebral tenderness. The genitals were negative. The legs showed edema up to the knees. The pupils and reflexes were normal.

The temperature was 95.3° to 98.7°, the pulse 80 to 85, the respiration 20 to 49. The amount of urine is not recorded until December 30, when it was 24 ounces. The specific gravity was 1.010 to 1.018. There was the slightest possible trace to a slight trace of albumin at four examinations, rare pus cells at one, occasional to many hyalin casts at the last two. The renal function was 15%. The hemoglobin was 90%. There were 10,000 leucocytes, 75% polynuclears. The urea nitrogen was 40 mgm. per 100 c.c. of blood. A Wassermann was strongly positive

The patient was much troubled with dyspnea and epigastric distress and slept little. The night of December 31 the pulse began to be slower and he complained of difficulty in breathing. About twelve o'clock the heart rate was 50 per minute, later 40–42 per minute, although still regular There appeared to be a 1–2 heart block. Within two hours he died.

*Discussion by Dr. Richard C. Cabot.*—Sleeplessness and nausea are not a usual combination in the cases coming into this hospital. Very few, I should say, come in for those symptoms, and they have so many causes that I do not think it is worth while to discuss them at any length. Sleeplessness and nausea are caused by alcoholism, general peritonitis, uremia, and psychoneurosis. But I think it is unlikely that we can hit the cause in this case in our preliminary consideration.

Apparently the pain in the right lower quadrant was not serious, for no further account of it is given.

Dyspnea and edema look like cardiac trouble. We get no idea from the past history as to why he had this present group of symptoms.

The history of the present illness points strongly to cardiac or renal trouble, and a good deal more on the whole to cardiac than to renal. In cardiac trouble in a negro of thirty-six one's snap diagnosis is syphilitic aortitis. Seeing a large number of autopsies on negroes as I have in the past two years, I can say that syphilitic aorti-

tis in the American negro seemed to be almost invariable, and almost every negro had a positive Wassermann.

The vomiting and the pain that this patient had I have seen a number of times in connection with syphilitic aortitis. We must remember that the coronary artery goes off just above the aortic valve, and that syphilitic aortitis usually begins within an inch of that artery. A very slight extension will close the mouth of the artery and produce just as great obstruction as if we had coronary sclerosis. When one gets pain of the anginoid type in young people, especially in a negro, we think of syphilitic aortitis because of the known relation of anginoid pain to disease of the coronary artery, and because of the position of syphilitic aortitis.

Properly speaking there are two aortic areas, the second right and fourth left interspaces. I presume this note means the second right.

The pulse pressure, 110, is such as we get in three conditions: (1) aortic regurgitation, the commonest and most marked; (2) arteriosclerosis without regurgitation; (3) exophthalmic goitre. These are the three conditions which give the largest pulse pressure. All may give rise to a capillary pulse, a phenomenon that we ordinarily associate with aortic regurgitation.

"There was a sound over the brachial with no pressure." Presumably a systolic sound but no sufficient evidence. They were looking for Duroziez's sign, which is a diastolic murmur over the peripheral arteries heard on pressure with the stethoscope.

Bilateral costovertebral tenderness under these conditions I should say means nothing. It is a very hard thing to make any deductions from, because it is hard to tell how sensitive the individual is in that region. It is not the pleasantest place in the body to have pressure exerted. It is only when a patient is very much more sensitive on one side than the other that I get anything out of it. Do you agree?

Dr. Hugh Cabot: Yes, provided the examination is made at the costovertebral angle; but not twenty-five out of a hundred house officers know where the costovertebral angle is. The valuable point of tenderness is high up, really in the angle between the rib and the spine. If present, I believe it is very important evidence of trouble in the kidney. In the flank it may mean anything, but usually means nothing.

Dr. Richard Cabot: If you had this tenderness in the real costovertebral angle on both sides, would it be important?

DR. HUGH CABOT: I think it would. I have no idea that he had it in this case.

DR. RICHARD CABOT: When we say "pupils and reflexes" we mean that the pupils were tested for reaction to light and accommodation, their size was looked at, and their shape. By reflexes we mean the knee-jerk and the plantar. There are many other reflexes which are not tested as a matter of routine in this hospital, but these two we do in every case, because they can be done so quickly, and if anything is wrong with them, it is usually important to find it out.

I should say there is nothing important in that urinary examination. The renal function was 15%. In a cardiac patient in bed with the dropsical conditions which we find in that state of things I do not think 15% is enough to give us any particular information about the kidney. It shows that that kidney is not doing much work, but a normal kidney under those conditions often will not do much work. So I should conclude nothing from that examination.

The urea nitrogen findings are above the average, but under those conditions do not seem to me significant. The blood is normal.

*Differential Diagnosis.*—The essentials of that case, I should say, are a history of cardiac trouble in a young negro who shows a diastolic murmur, a large heart, a positive Wassermann and anginoid pain. That is the typical picture of syphilitic aortitis.

I want to stress particularly the occurrence of pain and vomiting in syphilitic aortitis. It is sometimes so great that we think the patient must have a perforated gastric ulcer or something of that kind. Why they have so much vomiting I do not know. They vomit distinctly more than other cardiac patients, and they suffer more. The worst suffering that I have seen in cardiac patients has been in this type of case. In other words, syphilitic aortitis is the worst kind of cardiac trouble one can have, not merely in prognosis. It is the shortest; it is the most resistant to treatment; but is also the most agonizing. If anybody wants to use the end results of syphilis as a terror he certainly should point out syphilitic aortitis as one of the most torturing results of that disease. Few people who die of syphilitic aortitis have any other than a painful death.

I have mentioned only one diagnosis; I can see only one reasonable diagnosis in this case. It is perfectly true that a man who already had a rheumatic type of endocarditis and a cardiac lesion resulting from it, acquired in childhood, might get syphilis on top of it and have a positive Wassermann for that reason. But this combination is rare; experience has taught us that when we can explain all the

facts of a case by one diagnosis, we are more likely to be right than if we explain them by two. There is only one diagnosis that will explain all the facts in this case, and that is syphilitic aortitis.

Shall we find aneurism? That is the natural, I suppose the invariable result of syphilitic aortitis if the patient lives long enough. But most patients with syphilitic aortitis do not get aneurism, otherwise it would be a much commoner disease than it is. We have no symptoms of aneurism here that I see. He has pain which might be due to aneurism, but might perfectly well be produced in other ways.

There are no pressure symptoms, as differences in the pulses, pupils, aphonia due to laryngeal paralysis. The majority of aneurisms show pressure symptoms unless the aortitis is very slight.

Is there anything in the lungs? I do not see any reason to believe there is anything other than passive congestion.

The liver was enlarged, presumably by passive congestion, possibly by syphilis in that organ. But syphilis of the liver is rather rare, and we do not need to suppose it.

I do not think we shall find anything in the kidneys. If that turns out to be true, it will support what I have said about the functional test and the urea nitrogen.

If the diagnosis is correct we should find a very large heart with a so-called basin-shaped left ventricle, with an aortitis which has dilated the aorta and bound back the aortic valves flat against the wall in such a way that the blood regurgitates through them, but without stenosis. I have no evidence that syphilitic aortitis often produces aortic stenosis. If it produces any valve lesion, it usually produces aortic regurgitation. The mouth of the coronary artery will be wholly or partially obstructed. We say that because of the pain that he had. There will probably be some arteriosclerosis. Most of these cases show it, but not as a cause of death.

A PHYSICIAN: Do you expect to find anything corresponding to heart block?

DR. CABOT: He had at the end what may have been heart block. But that is always a dangerous diagnosis to make without either a well-taken polygram or an electrocardiogram. So I am not at all sure that he had heart block. If he had it, what should we find in addition to what I have said? We should find a lesion in the interventricular septum where the conduction fibers known as the bundle of His carry the impulse from the auricle to the ventricle. Syphilis is the disease which most often produces heart block. We know we have

disease not more than an inch from the bundle of His, and it is quite possible that at that point we shall have either fibrous or fibrocalcareous degeneration; if so we should have heart block.

A PHYSICIAN: Don't you think this was a terminal heart block?

DR. CABOT: It might perfectly well be. On the other hand the existence of true heart block might have been the cause of death. The lesion back of it might exist for a good while without being sufficient to produce heart block. I have seen a gumma of the heart as big as a man's thumb, a real tumor, which must have been there a long time, but without symptoms enough to produce heart block until near death.

Heart block may be produced by rheumatism as well as by syphilis. I took care of such a case in France; the patient got entirely well.

A PHYSICIAN: Was it rapid or merely hard breathing in this case?

DR. CABOT: I assume it was rapid, because we should have called it air hunger otherwise.

A PHYSICIAN: The sputum was not frothy?

DR. CABOT: I assume not. I assume no edema of the lungs.

A PHYSICIAN: Why should aortic regurgitation give such a high blood pressure?

DR. CABOT: High systolic pressure comes from the great power of the left ventricle. The left ventricle is hypertrophied because of the leak of the aortic valve. It will send a bigger volume of blood into the arteries and therefore raise systolic pressure.

DR. HUGH CABOT: I think I must disagree about the kidney function test. If your view is sound it seriously undermines the position of the functional test. When it was introduced it was believed that the test would enable us to distinguish in cardiorenal cases between the cases which were primarily renal and those which were primarily cardiac. In essentially cardiac cases it gives a pretty normal result, 30 or 40% instead of this very low %. I should be surprised if simply poor general condition with more or less starvation would give this result, the kidney functioning poorly simply because the body is functioning poorly. Added to that there is the slightly high blood nitrogen. I think there is a lesion of the kidney here.

DR. RICHARD CABOT: Other than passive congestion?

DR. HUGH CABOT: I think so.

DR. RICHARD CABOT: I think not.

*Clinical Diagnosis (from Hospital Record).*—Chronic nephritis (specific?).

Endocarditis.

Myocarditis.

Heart block with coronary spasm.

*Dr. Richard C. Cabot's Diagnosis.*—Syphilitic aortitis, aortic regurgitation, obstruction of coronary artery.

Passive congestion of the liver.

Hypertrophy and dilatation of the heart.

Slight arteriosclerosis.

*Anatomical Diagnosis.*—Syphilitic aortitis with coronary obstruction and involvement of the aortic valve (aortic regurgitation).

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Hydropericardium.

Ascites.

Anasarca.

Chronic pleuritis.

Obsolete tuberculosis of a bronchial lymph node.

DR. RICHARDSON: This was a typical case of syphilitic aortitis. Anatomically the picture was as follows:

The heart weighed 760 grams, very marked hypertrophy. The dilatation, which was also marked, was greatest in the left ventricle, the cavity of which was basin-shaped. The valves other than the aortic were negative. The aortic cusps presented a certain amount of fibrosis, and in the region of their contiguous margins were bound down to the aortic wall by thick fibrous bands which were continuous with a syphilitic process in the aorta. This same process extended about the orifices of the coronary arteries, causing marked decrease in their circumferences. The aorta presented an irregular luetic band 3 or 4 cm. wide, extending around just above the aortic cusps and continuous with the process mentioned, and from that point up there were scattered areas of syphilitic aortitis as far along as the arch. In the descending portion of the thoracic aorta the process faded out, and in the abdominal portion and in the great branches there was no aortitis or any definite arteriosclerosis.

DR. RICHARD CABOT: That is in contrast with what you would have found if there had been arteriosclerosis.

DR. RICHARDSON: Yes. One of the favorite seats of arteriosclerosis is in the abdominal portion of the aorta with involvement of the great branches. We seldom have an opportunity to examine the peripheral arteries, but when we do the clinical observations of

sclerosis in these vessels are not always borne out unless it is so evident that we do not need to examine.

The liver weighed 1440 grams, the spleen 170 grams, the kidneys 295 grams. These organs all showed chronic passive congestion.

So far as I could find in this case there was no other evidence of the presence of syphilis except in the aorta and the extension to the aortic cusps.

The gastro-intestinal tract showed the usual picture of chronic passive congestion,—thickened, reddened, velvety, juicy mucosa.

The head was not examined.

DR. CABOT: Is there anything to explain the heart block?

DR. RICHARDSON: No. Death would seem related to hypertrophy and dilatation of the heart, with the added burden of the considerably obstructed coronary arteries. There was no anatomical evidence at all of the presence of anything impinging upon the bundle of His.



FIG. 59.—The dark narrow strip above shows the ordinary thickness of an artery. The wide band below it is the thickened wall of the syphilitic aorta. Natural size. (Dr. Oscar Richardson.)

A PHYSICIAN: Do you see as many aneurisms as you used to?

DR. RICHARDSON: We get them every once in a while; yes.

A PHYSICIAN: All the old text-books seem to be full of aneurisms. I have not seen any in four or five years.

DR. CABOT: I get the impression that they are not so common, but it is very hard to be sure. It is only an impression.

DR. RICHARDSON: The astonishing thing to me is the absence of syphilitic lesions elsewhere in the body. The man must have been infected at some time, possibly unknown to himself, and syphilis is a septicemia. Why the spirochetes in these cases should select the first portion of the aorta as the favored seat of residence seems difficult to explain.

### Necropsy 4438

An Irish-American chauffeur of thirty-six entered November 20, 1922. He had difficulty in seeing in the dark and stumbled easily. For these conditions a physician gave him "five drops for three months." He had possibly once been jaundiced. His weight was never better.

Fifteen years before admission he had a syphilitic infection. The first symptom was trouble with his eyes. He also lost some hair. He thought he had some 606, but "not much."

Six weeks before admission he had a head cold, then a cough with scanty white, sometimes brownish, tenacious sputum. Since the onset he had developed dyspnea on exertion. Four weeks ago he began to have edema of the ankles, disappearing at night. In the past week, during which he had been in bed, he had had palpitation and urination once at night.

It was learned from another hospital that since May 8, 1922, he had been given weekly doses of arsphenamin at the Out-Patient Department there, 0.2 grams for the first five doses, then 0.5 grams for 12 doses. Between September 4 and October 9 he had five doses of intramuscular "mercury solution," ten minims each dose. Following this he had four doses of arsphenamin, each of 0.4 grams. Total arsphenamin 21 doses, total 7.4 grams in seven months.

Examination showed a well nourished man with some smoothing out of the nasolabial folds. The mucous membranes were slightly pale. The tongue and throat were very dry. There was some pus in the nasopharynx. The heart was greatly enlarged with diffuse irregular heaving impulse in the left sixth interspace 12 cm. from midsternum. The left border of dullness was 13 cm., the right border 4 cm., the substernal dullness 10 cm. The midclavicular line is not recorded. The sounds were mostly obscured by murmurs. There were systolic and diastolic murmurs over the base, a mitral diastolic over the apex. Each beat was followed by an extrasystole. There was a very coarse diastolic thrill after the extrasystole at the apex. There was Corrigan pulse, pistol shot, bigeminy, and visible capillary pulsation. Electrocardiogram showed the rate 110, bigeminy due to ventricular premature beat occurring every other beat, possibly slight aberration. The blood pressure was 140/80. The lungs showed many moist râles and slight dullness at both bases, more on the left. (The left side was down during examination.) The abdomen was slightly full, otherwise normal. The knee-jerks were equal. There was no clonus or Babinski. Romberg's sign is not recorded. The right pupil was greater than the left. The right was oval, the left irregular. Both reacted to distance but poorly to light.

The temperature was 97.3° to 99° until November 28, 98.1° to 101° until December 4, then rising, 99° to 106°. The pulse was 50 to 140; after November 24 only once below 100, after November 30 not below 108, and after December 4 not below 120. The respirations were 17 to 30. The amount of urine was 22 to 25 ounces on the three occasions recorded, the specific gravity 1.020 to 1.030. The

urine was alkaline at one of two examinations. The sediment showed leucocytes at both examinations, red blood corpuscles at one, much bile at two. The hemoglobin was 80%, the leucocyte count 8600 to 6800, the polynuclears 76 to 85%, the reds and platelets normal. A Wassermann was unsatisfactory on account of the presence of bile. The non-protein nitrogen was 39.3 mgm. The stool was negative to guaiac. Lumbar puncture gave 15 c.c. of clear fluid. The initial pressure was more than 240 (the fluid rose very slowly). After the withdrawal of 5 c.c. the pressure was 240, after 5 c.c. more 230, after 5 c.c. more 210. Jugular compression raised the pressure only 20

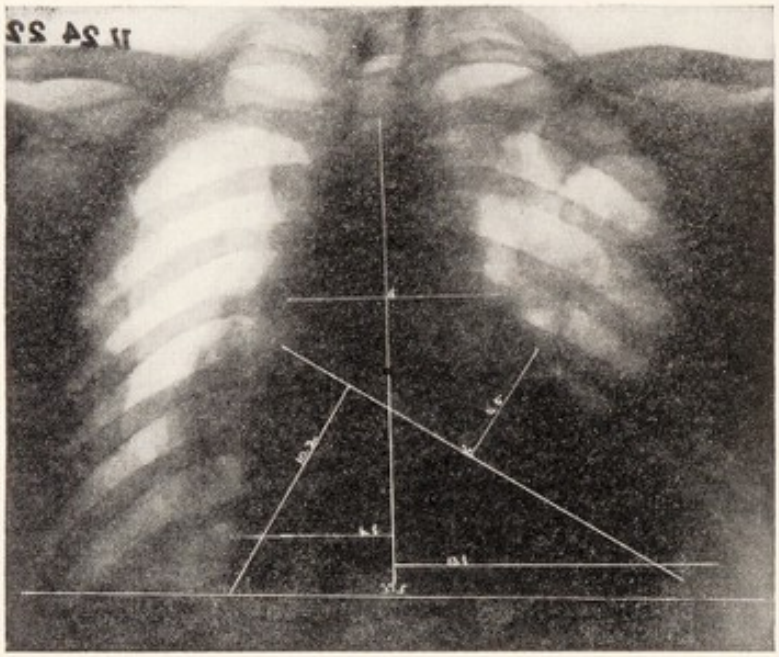


FIG. 60.—Heart shadow much enlarged in all diameters; greatest increase to the left. Also marked increase in supracardiac dullness. There is no definite tumor-like formation.

mm. Cough caused no increase. Normal pulse and respiratory oscillation. There were 26 cells. Alcohol was positive, ammonium sulphate negative, Wassermann strongly positive, goldsol 555555-3210, total protein 65. A heart consultant reported November 20, "Apparently well marked Austin-Flint murmur with mitral diastolic thrill . . ." An eye consultant reported November 20. "Apparently beginning changes in the macular regions of both eyes. Retinae show a suggestion of pigment deposits with a few vague areas suggestive of involvement. The condition is slightly more marked in the left eye. Pupils not dilated well enough for the best view. Vitreous opacities in both eyes." A syphilis consultant reported, ". . . Further antisyphilitic treatment contraindicated." X-ray November 25 (see Fig. 60) showed the outline of the various

chambers of the heart indistinct. The examination was rather unsatisfactory on account of the patient's condition.

The patient seemed to be holding his own. By December 2 the jaundice was about the same (*sic*). The digitalis was discontinued. After three days he seemed less jaundiced. The pulse was poor several times on the 5th, and there was much mucus in the throat. There was edema of the right forearm and hand for which no cause was found, and which was much reduced by December 7. December 8 there was a great change for the better. The pulse was good, the heart vigorous. The patient was restless, not stuporous, and talked with his friends, though he would not answer the doctor's questions. December 10 the condition was not so good. The jaundice was increased. The spleen was readily palpable. Percussion of the liver showed it to be possibly smaller than at the previous examinations. December 11 the patient became comatose and died.

*Clinical Diagnosis (from Hospital Record).*—Syphilis.

Aortitis and acute atrophy of the liver.

Central nervous system syphilis.

*Dr. Richard C. Cabot's Diagnosis.*—Syphilis of the central nervous system.

Syphilitic aortitis.

Aortic regurgitation.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

*Anatomical Diagnosis.*—Luetic aortitis with small aneurism.

Fibrous endocarditis of the aortic valve.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Thrombosis of the portal vein.

Atypical cirrhosis of the liver.

Hypertrophy of the spleen.

Icterus.

Ascites.

Decubitus.

Hemorrhagic areas in the serosa of the intestines and the peritoneum.

Serofibrinous pleuritis, right.

Slightly defective closure of the foramen ovale.

DR. RICHARDSON: What were the clinical data as to the liver?

DR. CABOT: They do not say. They say it was enlarged and then less enlarged. But we do not know any more.

DR. RICHARDSON: It is curious, because the history given before the necropsy inclined the house officers to think there was *atrophy*, and it was so stated on the necropsy permission.

Unfortunately we were not permitted to examine the head. The conjunctivae and skin showed a well marked saffron discoloration. There was quite an extensive bedsore in the sacral region.

The peritoneal cavity contained 1500 c.c. of thin bile-stained fluid. The appendix was negative, and the peritoneal cavity in general, other than for the fluid, was negative.

The mucosa of the esophagus showed a well marked meshwork of varices running up and down. The stomach showed some areas of reddening in the mucosa but was otherwise out of the picture. The intestines were negative except as stated below, and the fecal material in the large intestine brown. It is to be mentioned that scattered along the mucosa of the intestine in numerous places were small hemorrhagic areas, and we might as well clear that up now. The portal vein at its lower end presented an area of chronic phlebitis to which a long columnar thrombus was attached, extending up towards the liver and in the direction of the stream. This thrombus only partly occluded the portal vein, but the obstruction was sufficient to produce the hemorrhagic areas in the mucosa of the intestine which otherwise were unaccounted for.

The mesenteric and retroperitoneal glands were out of the picture. The right pleural cavity contained about 400 c.c. of thin pale cloudy fluid. On the left there were only a few c.c.—one of those not very uncommon cases where we find fluid on one side and not on the other. The bronchial glands were soft and juicy. The lungs showed hemorrhagic edema, and on the lower lobe of the right lung a little fibrinopurulent pleuritis. On the left there was no evidence of any exudate.

The pericardium was negative. The heart weighed 645 grams,—considerably enlarged. The myocardium was pale brown-red and a little lax, or what is called flabby, although the right ventricle measured 3½ and the left 14 mm., a good thickness. On the right side the chordae tendineae were well marked. The left ventricle showed much dilatation and was basin-shaped,—that peculiar dilatation and configuration which we associate with syphilitic aortitis. The left auricle was of full size. On the right side there was moderate dilatation. The valve circumferences were: mitral 12 cm., aortic 9 cm., tricuspid 15 cm., pulmonary 8 cm. Those were large circumferences, a little increased. But the valves, other than the aortic valve, were out of the picture. We have a very good picture of the

aortic valve here; and we should note that one of the cusps is in pretty good condition. The other two speak for themselves. They are markedly deformed, wrinkled down, curled up, plastered back against the wall. So that two of them are not cusps at all, but simply flattened, wrinkled masses of fibrous tissue.

That condition we took to be syphilitic aortitis, and it accounts very well for the clinical manifestations.

We noted that the right coronary was frankly negative; the left was just below a little aneurism shown very well in the picture. There was no apparent obstruction to the left coronary. Of course with the distension of the sac there might have been a little, but nothing marked. So that we have syphilitic aortitis with extension

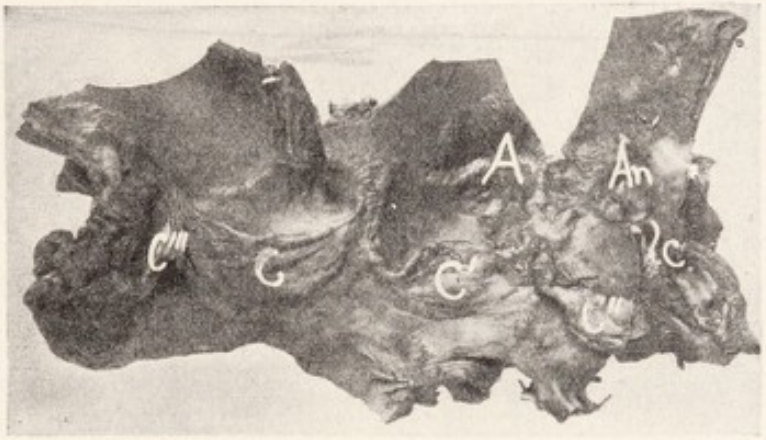



FIG. 61.—Syphilitic aortitis. C, fairly good aortic cusp. C′ and C″, deformed cusps. C‴, tag of C″. A, area of aortitis. An, aneurism. l.c., beginning of left coronary artery.

to the aortic valve, with deformity of the cusps and with irregular luetic areas for a short distance on the aorta just above the aortic cusps. Beyond that the aorta over its entire length and the great branches were negative. This position just above the aortic cusps is the typical zone for luetic aortitis.

DR. CABOT: How big was the aneurism?

DR. RICHARDSON: It was not bigger than the end of the little finger.

DR. CABOT: You won't blame us for not diagnosing that.

DR. RICHARDSON: The venae cavae were negative. The portal vein we have spoken of. The liver weighed 990 grams. That means some atrophy in a man such as is described here. His liver should have weighed at least 1300 or 1400 grams. There was chronic passive congestion. There was some sclerosis. So far as we have gone there was no definite evidence of the destruction of the liver tissue such as we find in some of these cases where salvarsan has been

pushed and we get conditions which simulate acute yellow atrophy of the liver.

In association with this the spleen weighed 995 grams. Here again there was chronic passive congestion and a certain amount of sclerosis in the background.

The gall-bladder and bile-ducts were frankly negative, so that the icterus cannot have been from obstruction unless of the very minute ducts within the liver substance itself. But that is not what we usually mean by obstruction, although the current belief is that all icterus is mechanical obstruction of the bile-ducts *somewhere*.

The kidneys were a little large but showed no definite lesion. The bone-marrow of the lumbar vertebrae was rather red, but was negative.

DR. CABOT: Did that portal thrombus have anything to do with the sclerotic process in the liver?

DR. RICHARDSON: No. There are two or three possibilities: sometimes with chronic passive congestion we get a condition which simulates cirrhosis; the second is a straight ordinary cirrhosis; and the third is the question whether it is syphilitic or not. There was chronic passive congestion in this case, which is responsible for a part of the picture. The bile ducts were negative. That throws out the possibility that any infection went up and produced the so-called biliary infections with cirrhosis. The liver tissue finally showed no such definite picture as we get in the over-salvarsanized liver cases.

DR. CABOT: It might, however, be due to syphilis?

DR. RICHARDSON: It might. The anatomical evidence is fibrosis or sclerosis of an atypical kind. If the other things all point to that, then it might be syphilitic.

## ILLUSTRATIVE CASES

1. Syphilitic Aortitis with Aneurism
2. Malignant Lymphoma Simulating Aneurism

### Case LX

A Russian Jewish court interpreter of fifty-three entered August 30 for relief of loss of voice, hoarseness, and "inability to breathe."

Thirty years before admission he was shot in the left elbow. About the same time his nose was fractured. Since that time he had had almost constant catarrh and sinus involvement. A year after the extraction of the bullet in his elbow he had paralysis of the vocal cords and possibly syphilis, though there were no secondary symptoms. While in Vienna taking a nine months' course of treat-

ment for syphilis the elbow became intensely and increasingly painful, so that large amounts of morphia had to be used. He acquired the habit, and had it for a year and a half, taking eleven grains a day. Then he was treated at the Mt. Sinai Hospital, New York, for numerous boils caused by his hypodermics, and was cured of his morphinism. For twenty-five years he had had bleeding hemorrhoids. Five years before admission he had gastric distress, heartburn, gas and nausea for several months, relieved by careful dieting. The following year he had an attack of chills and fever followed by enlargement of the heart, and was laid up in bed six weeks. For six months he had had a dull aching across the small of his back, not radiating, at times catching him up suddenly. Two years before admission he weighed 210 pounds, his best weight. His usual weight was 200.

He drank a rare glass of whiskey and smoked twenty or twenty-five cigarettes a day.

The first attack of loss of voice and difficulty in breathing came on suddenly over night twenty-nine years before admission. The attacks previous to the present one lasted from a few hours to five days, rarely over three days. Cold, dry winter air seemed to free him from them. He talked in an undertone and only on forced expiration after a very deep breath. With each attack there was increased difficulty of inspiration. During the present attack, which had lasted three weeks, it had seemed at times as though he could not get breath, try as he might. Loss of voice had been marked, with cough, sputum, and hoarseness. Talking tired him out and caused a dull pain beneath the base of the sternum. During the past few weeks he had had a good many headaches, with some dizziness. His appetite was poor. His bowels were constipated. At the Out-Patient Department, August 23, 1915, a Wassermann was negative. X-ray (Fig. 62) showed the entire right chest less radiant than the left; marked peribronchial thickening; enlarged and calcified glands; the aortic arch dilated; the heart shadow enlarged to the left. Examination in the Nerve Department was negative except that the left biceps and triceps reflexes were greater than the right. In the Throat Department operation was advised. The patient was unwilling to have it done immediately, but agreed to consider it.

Examination showed a pale, well-nourished man, speaking hoarsely. Glands were palpable in the neck and axillae. The apex of the heart is not recorded. The width of the arch was 7 cm. The other dimensions are shown in Figure 63. The sounds were of poor quality. X-ray showed the dimensions as in Figure 64; the

length of the heart 17.1 cm. There was a systolic murmur at the apex and a faint diastolic at the second right interspace. The pulses were of fair volume. The artery walls were just palpable, the brachials slightly tortuous. The abdomen, genitals, extremities,

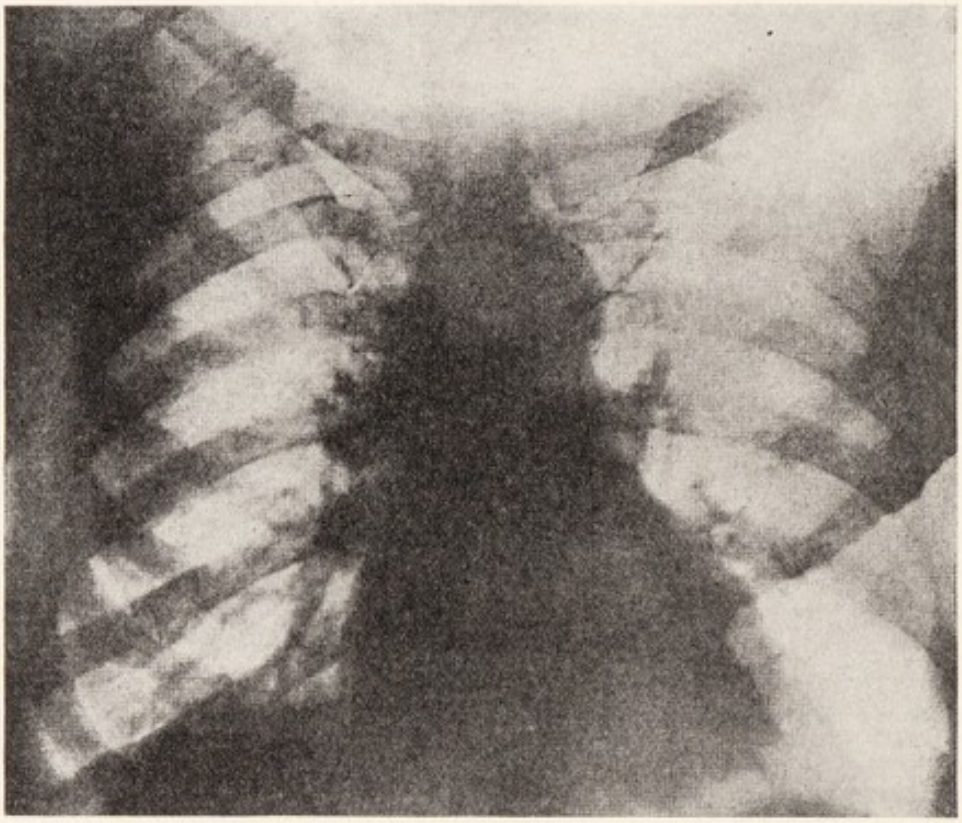


FIG. 62.—Hypertrophied heart in syphilitic aortitis.

and reflexes were normal. The pupils were irregular, otherwise normal.

The temperature was 97° to 99.7°, the pulse 60 to 100, the respiration normal except for two rises to 29. The amount of urine was

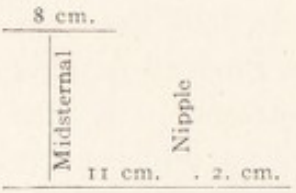



FIG. 63.—Measurements by percussion.




FIG. 64.—Measurements by X-ray.

normal, the specific gravity 1.018 to 1.010. There were leucocytes at both examinations. The renal function was 50%, the hemoglobin 75%. The other blood findings were normal. A Wassermann was negative. The spinal fluid gave a cell count of three lymphocytes. Noguchi and Nonne were negative. A Wassermann was anti-

complementary. Gold chlorid test was suspicious of syphilis. The stool gave a negative guaiac.

Two throat surgeons advised tracheotomy. September 11 it was done. The patient was sent to the ward in good condition and breathing well. Next day the tube slipped out of the trachea and could not be reintroduced because of swelling around the wound. The tube was too short. There was considerable emphysema of the tissues of the neck and face. The sutures were therefore removed and the wound left wide open. Next day the emphysema was much reduced, and the tube was reintroduced. September 14 it was replaced by a longer tube after a larger opening had been made in the trachea by punching out part of the rings with a tonsil punch. The wound was moderately septic. This tube also slipped out, but was put back a few hours later. September 15 a still larger tube was put in, slipped out, and was put back. The patient was much depressed, and complained a great deal. September 16 an extra long tube was put in, fitting better than any of the others, but it also slipped out. September 17 a new, extra long tube was inserted and strapped tightly to the neck with adhesive. It stayed in place until next morning, and was reinserted with ease. The wound was draining a little pus. After this the tube remained in place. September 22 the patient was discharged, feeling very comfortable, the wound granulating.

*Dr. Richard C. Cabot's Diagnosis.*—Syphilitic aortitis.

Hypertrophy and dilatation of the heart.

Paralysis or syphilitic ulceration of the vocal cords.

*Interpretation of X-ray, August* 23.—Dilatation of the arch.

Enlargement of the heart.

Thickened pleura.

*O. P. D. Throat Department Diagnosis, August* 23.—Complete paralysis of left vocal cord, abductor paralysis of right vocal cord.

*Diagnosis in Medical Department, September* 11.—The same, with

Dilated aorta.

*Operation.*—No pre-operative diagnosis but that above is recorded. Two-inch median incision over the trachea at the upper limit of the first tracheal ring. The deep tissue was infiltrated with novocain. The sterno-thyroid and sterno-hyoid muscles were split and retracted, the isthmus of the thyroid gland defined by powerful blunt dissection and retracted upward. The trachea below this was exposed for about an inch. A few drops of 10% cocain with adrenalin were introduced into the lumen of the trachea by means of a hypodermic

needle. A longitudinal incision ¾ inch long was made in the trachea, cutting two rings, which were found to be partially calcified. A silver tracheotomy tube was inserted.

*Clinical Diagnosis at Discharge (from Hospital Record).*—New growth of lung?

*Dr. Richard C. Cabot's Diagnosis.*—(See above.)

*Later History (from Out-Patient Record).*—September 24 the wound looked well. September 29 it showed exuberant granulation, and was cauterized. Oct. 4 it was again cauterized. It was doing well at this and the next visits, October 18 and 29. At three later visits there is no entry except that once the tube was cleaned, as it had been at most previous visits. March 3, 1916, the patient was found to be doing well. July 8, 1916, there was slight movement in the left vocal cord. There were no signs of growth. He kept the tube closed but in place for emergency use. September 23, 1916, there was practically no movement in either cord. There was sufficient space, however, to allow the tube to be cleaned. The voice sounds were fairly good.

April 11, 1922, he reported that he had been wearing the tube plugged and breathing with a fair degree of comfort through the mouth until a month before the visit, when he had an attack of grippe. Since that time the breathing had been gradually becoming more difficult. Laryngoscopic examination showed both cords fixed in adduction. The left cord showed a polypoid mass apparently growing from the margin and inferior surface of the cord. This flapped up and down with respiration. He was recommended for admission to the Eye and Ear Infirmary for study and removal of the growth. No record of his having visited the Infirmary could be found February 27, 1925.

### Case CIX

(Copyright, 1918, by Oscar Richardson, M.D., and William H. Smith, M.D.)*

A Swedish carpenter of forty-four entered October 15 for relief of shortness of breath. He had always been well and strong. He had possible scarlet fever at ten. He had urinated once at night all his life. A year before admission he vomited every morning and was constipated. Since that time he had taken Russian oil every morning. He was having no more stomach symptoms. He was last employed by a large ship-building company, and worked nine to ten hours a day. The work was noisy, damp, and too hard for him.

* This and the following case and discussion are used by permission of Dr. Richardson and Dr. Smith.

During the past year he had worried about his health. At twenty-one he weighed 176 pounds, his best weight. His usual weight was 175, his present weight 154 pounds. He denied venereal disease and the use of alcohol and tobacco.

Two months before admission he noticed that after undue exertion he became breathless and dizzy and had a sense of pressure over the precordia. A physician gave him medicine which slowed his pulse. He was able to work somewhat better for a week. A week before admission he became weak and breathless and was told by a doctor to stay in bed.

Examination showed a well-nourished man, his head nodding with each heart beat. There was marked pulsation of the arteries in the neck and a systolic thrill over both carotids. There were bean-sized inguinal glands. The apex impulse of the heart was seen and felt only in the left lateral position in the sixth space 14 cm. to the left of midsternum, 5.5 cm. outside the midclavicular line. The left border of dullness was 13 cm. from midsternum, the right border of dullness 4 cm. to the right, the supracardiac dullness 6.5 cm. The action was regular, slightly rapid. The aortic second sound was replaced by a murmur. At the apex was a blowing systolic murmur transmitted to the axilla. At the base was a systolic somewhat rougher in character, transmitted to the carotid. There was a blowing diastolic over the precordia replacing the aortic second sound, best heard at the aortic area and along the left sternal border. The pulses were Corrigan. Pistol-shot and Durosiez's sign were heard over the peripheral vessels. The artery walls were just felt. The blood pressure was 135/25 to 115/15. The lungs, abdomen, genitals, pupils and reflexes were normal, as were the extremities, except for slight roughness of the left shin.

The temperature was 100.8° to 96.7°, the pulse 88 to 64, the respiration 32 to 13. The amount of urine was 17 to 28 ounces. The urine was cloudy at two of four examinations. The specific gravity was 1.022 to 1.026, The sediment showed rare leucocytes at two of four examinations, hyaline casts at one. The renal function was 40%. The hemoglobin was 80%. There were 7200 to 10,000 leucocytes, 71% polynuclears. The urea nitrogen was 22 mgm. per 100 c.c. of blood. Two Wassermanns were strongly positive. The stools were negative.

There was nothing to account for the elevated temperature at entrance. October 16, after the report on the Wassermann, he was put on potassium iodid. By October 19 the temperature was lower.

There were no signs of decompensation, and he felt better. He continued to gain, and October 31 was discharged relieved.

### Necropsy 164

A Negro Pullman porter of thirty entered October 15. His past history was unimportant. He used alcohol in moderation, chewed a twenty-cent plug of tobacco a week and constantly had a cigar in his mouth. Seven years before admission he had chancre with double buboes; no secondary manifestations

Four weeks before admission he was suddenly seized with shortness of breath upon exertion and cough with whitish sputum. The dyspnea had increased, coming in spasms during which he perspired freely. For three weeks he had been unable to lie down. There had been much palpitation and throbbing of the heart. He had had no sleep for a week or more. He passed half a pint of urine at night, about the usual amount by day.

Examination showed a fairly well nourished negro. The mucosae were pale. The lips were at times somewhat cyanosed. During the paroxysms of dyspnea the veins stood out prominently in the head and forehead. The apex impulse of the heart was in the fifth space. There was no enlargement to percussion. A roughened heart sound was heard through into the back near the vertebral column. There was dullness over the sternum up to within an inch of the suprasternal notch. Pulsation was felt on palpation in the notch. Throughout the cardiac area a vibratory systolic thrill was felt and a harsh systolic murmur was heard, loudest at the third cartilage, transmitted into the aortic region downward. The second sound was very indistinct. In the region below this, loudest at the fourth space, was heard a diastolic murmur, blowing in character. At the apex the first sound was somewhat churning in character. The lungs were normal. The liver was just below the costal border. Pressure on it filled the veins of the neck. The pupils reacted. The knee-jerks were present. The extremities showed no edema. On the under surface of the penis was a cicatrix, said to be from a sinus. A crust was seen upon it in one place. In both groins were cicatrices, said to be from buboes.

The temperature was 95° to 98°, the pulse 95 to 120, the respiration 25 to 48 The amount of urine is not recorded. The specific gravity was 1.020. There was the slightest possible trace of albumin. The sediment showed abnormal blood cells and leucocytes at both of two examinations, hyalin and granular casts at both, with a few cells

adherent at one. The hemoglobin was 54%. There were 8900 leucocytes.

During the afternoon the patient had several attacks of dyspnea, relieved by nitroglycerin. There was no change in the way the air entered the lungs during the attacks. Two doses of morphia gr. ¼ and ⅙ gave relief. He slept most of the night and had fewer paroxysms next day. He complained of pain in the cardiac region, relieved by an ice bag. A few medium moist râles were heard in the right back. After another fairly comfortable night there were more râles at the right base and some altered blood in the sputum. A few râles were heard over the apex. An increasing number of fine râles

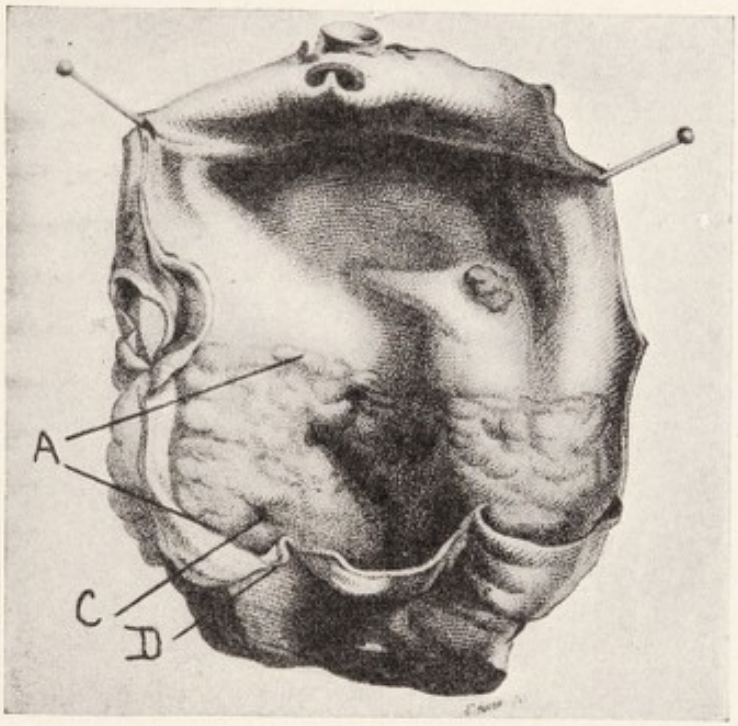



FIG. 65.—Syphilitic aortitis. From a woodcut made about a hundred years ago. A-area of aortitis. C-coronary. D-aortic cusps.

appeared in the backs, especially on the right, and there was bright blood in some quantity in the sputum. The murmur was somewhat softer. The legs became increasingly edematous. Bright red, frothy blood was raised in considerable amounts. The sclerotics became much ecchymosed. A hemorrhagic area appeared on the tip of the nose, though there was no history of trauma. The dullness in the upper part of the sternum became more marked, extending more to the left. Respiration became more and more difficult. October 23 he began to have difficulty in swallowing. Next day there were some transient pains in the upper sternal region. The lungs began to fill up with coarser râles. These could be felt through

the chest wall. At ten o'clock he was fairly comfortable, with no marked dyspnea. Three-quarters of an hour later he died.

*Discussion* by Dr. Oscar Richardson.*—DR. RICHARDSON: Definite syphilitic lesions in a case follow infection through definite portals of entry, but there are also cases in which there is no such definite portal of entry and the patient knows nothing whatever of having had any

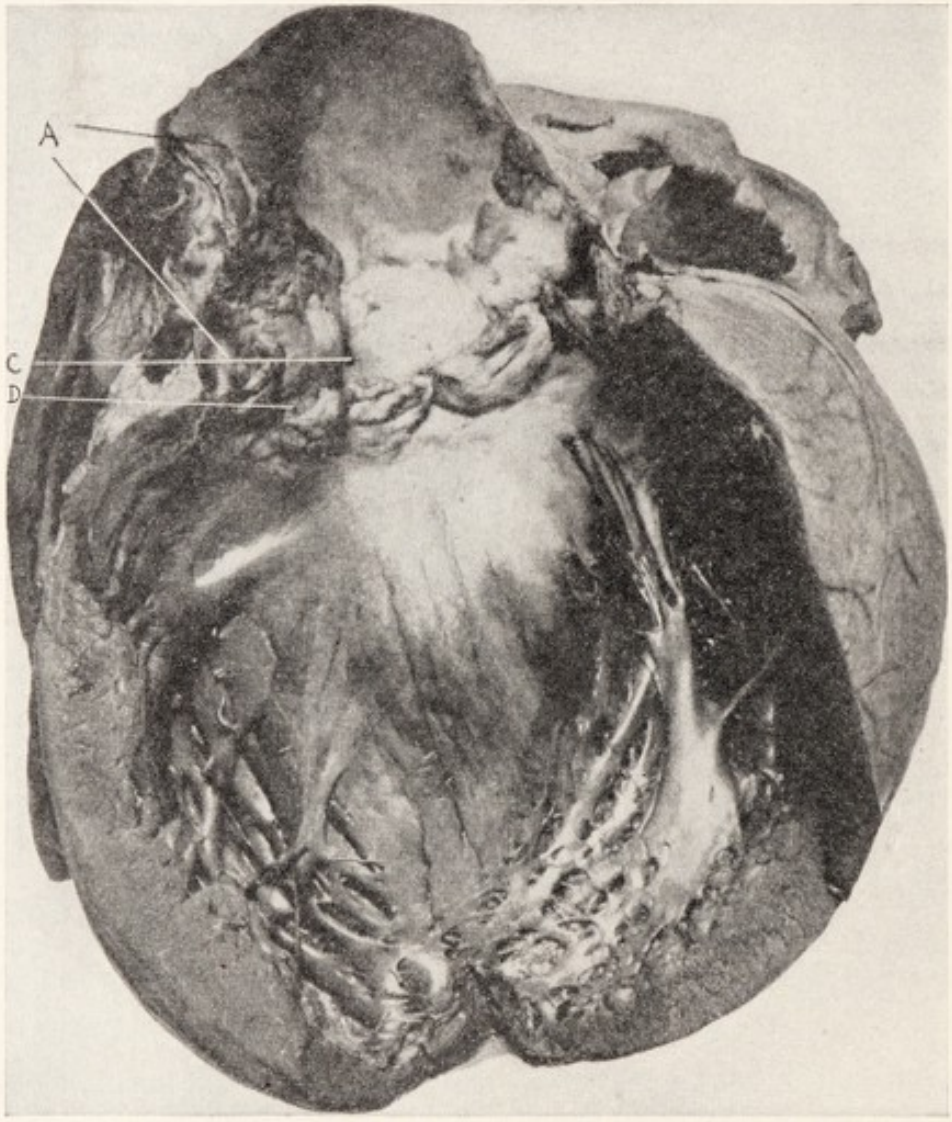



FIG. 66.—A-area of aortitis. C-coronary. D-aortic cusps.

infection at any time in his life. This statement of the patient can be regarded as truthful in many instances, for the simple reason that he wishes to know himself and is anxious to give as truthful a history as he can for his own benefit. He does not know he has had syphilis, and yet he will die of luetic aortitis.

Fig. 65 is from a photograph by Mr. Brown of a woodcut of a portion of the heart and aorta made about a hundred years ago. It

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might be regarded as the fossil remains of an observation made in a previous age of a condition which we now know to be syphilitic aortitis. The picture is irresistibly convincing. Note the broad band-like area (A) extending around the aorta just above the aortic cusps, how sharply it it differentiated from the natural-appearing aorta above, and how it extends down about the coronary artery (C) and involves the aortic cusps below (D). Again, the intimal surface of the band-like area shows the discrete and confluent, irregularly

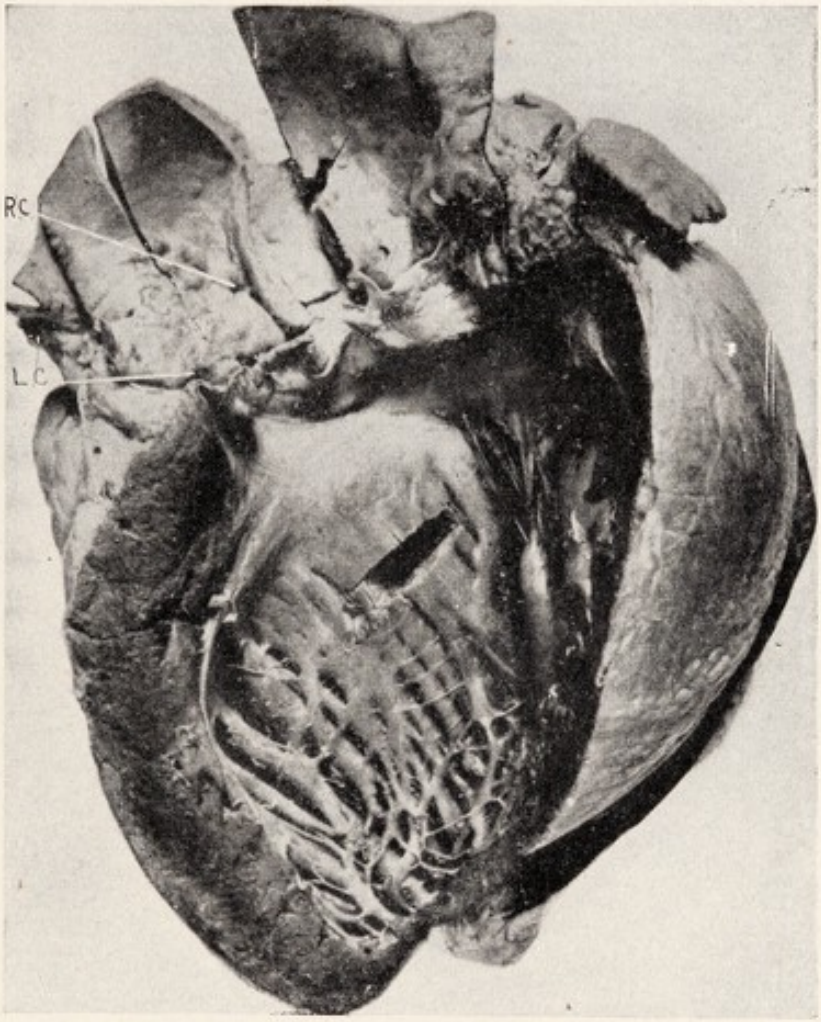



FIG. 67.—L. C.-left coronary. R. C.-right coronary.

shaped, more or less elevated, plaque-like masses with intermingling smaller and larger irregular areas of depression which give the gristly, scar-like aspect to the surface so characteristic of syphilitic aortitis. The aortic cusps show the usual fibrous wrinkling and deformity with narrowing of the width of the cusps and pinning back of their contiguous margins. This region of the aorta is the favorite seat of syphilitic aortitis.

The following pictures are photographs made by Mr. Brown in this laboratory and are taken from our own cases.

Fig. 66 shows the syphilitic lesion in the same situation as it was in Fig. 65. The lesion in this case is a duplicate of the other, and as an added fact the basin-like dilatation of the left ventricle is to be noted.

In Fig. 67 we see again a case with the lesion in the same region as in the others. The small gaps in the aortic wall are where pieces were removed for microscopical examination. This was the very remark-

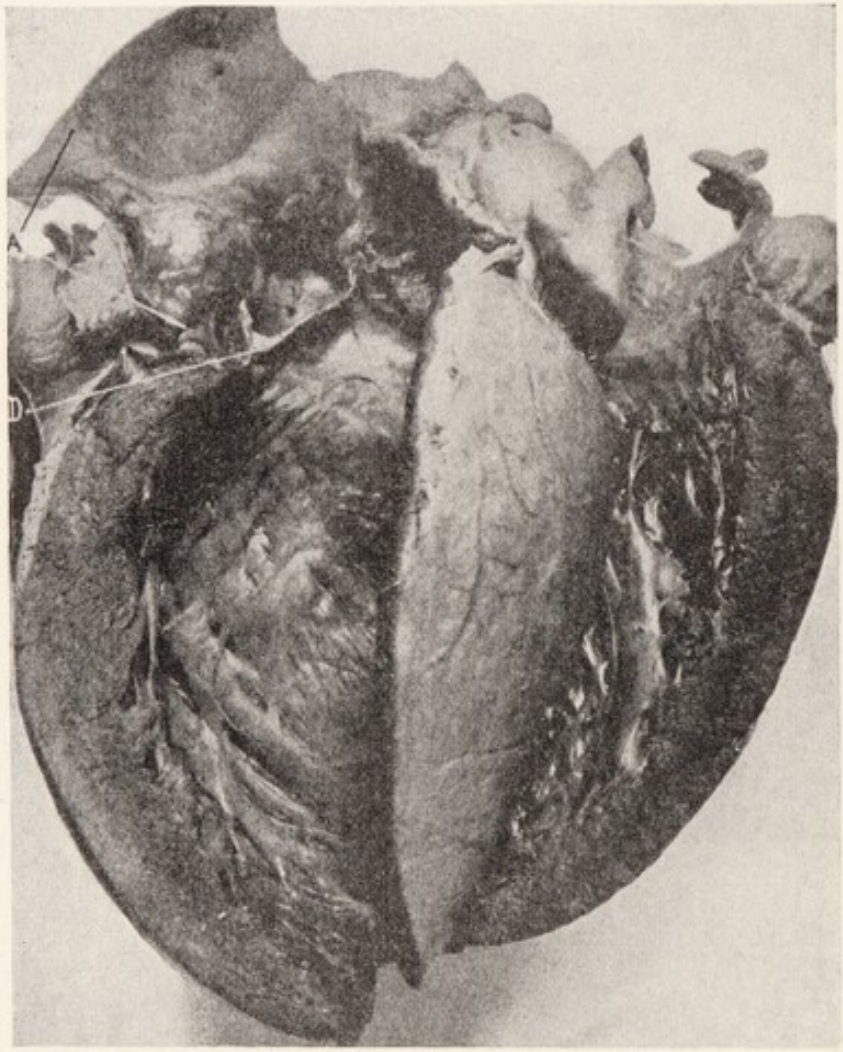



Fig. 68.—A-area of aortitis. D-aortic cusps. (Dr. J. H. Wright and Dr. Oscar Richardson.)

able case of a young man twenty-one years of age who died suddenly and was thought to have been poisoned. There was nothing unusual in the previous history, and so far as his family knew he had always been perfectly healthy. His death had been associated with distress and pain in the epigastric region and vomiting. I found at the necropsy, however, that his death was due to occlusion of the orifices of the coronary arteries associated with luetic aortitis.

Fig. 68. This case dates back to 1909 and is the one in which the spirochetes were found for the first time in the wall of the aorta. The lesion is in the first portion of the aorta, as in the other cases. The basin-shaped dilatation of the left ventricle is well shown.

Fig. 69. This picture shows very well indeed the marked involvement of the aortic cusps in syphilitic aortitis. This fibrous wrinkling and deformity of the cusps (C, C), and the pinning down of their contiguous margins, which shortens their width and decreases their

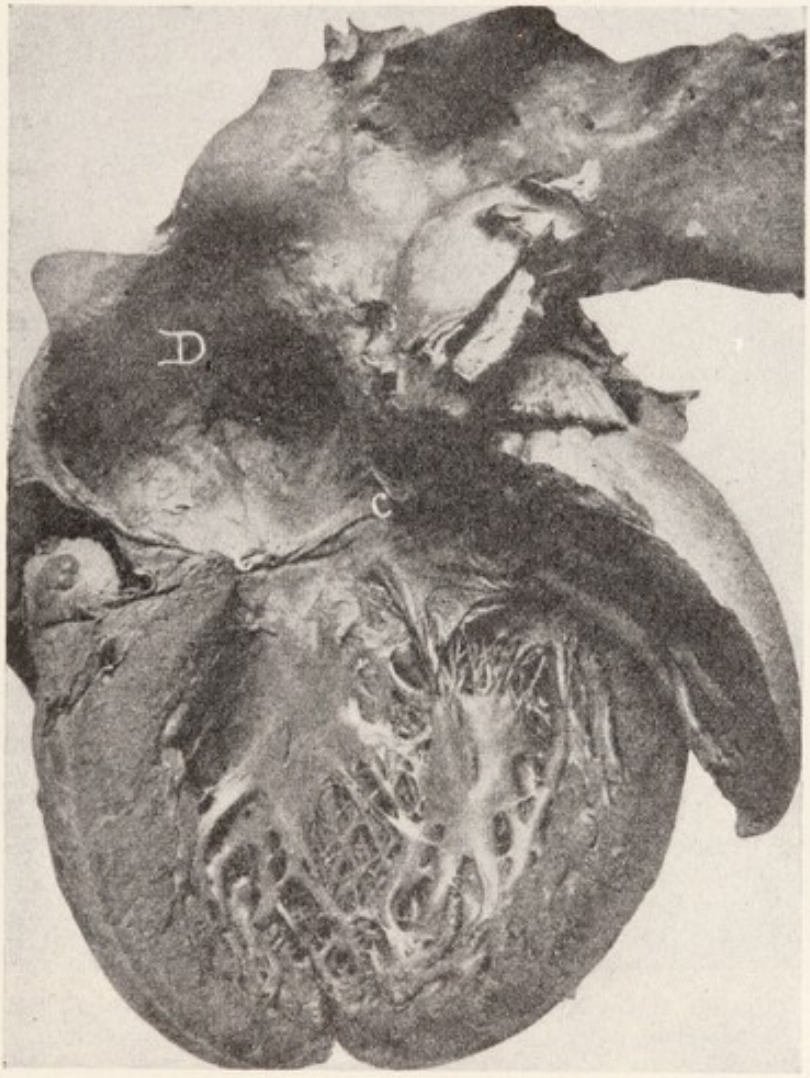



FIG. 69.—C, C-deformed cusps. D-aneurismal dilatation.

excursions, are the important factors in the production of the aortic regurgitation so characteristic of luetic aortitis. Aneurismal dilatation (D) of the first portion of the aorta is also well shown.

When we open an aorta which is the seat of syphilitic aortitis, the first thing which strikes our attention is the gristly, scar-like aspect of the surface of the intima. Closer inspection shows this to be due to smaller and larger discrete and confluent, irregularly shaped

areas and streaks of depression and longitudinal furrows, extending about and between smaller and larger, more or less elevated plaque-like masses. Many of the plaques show rounded, rather smooth, somewhat translucent surfaces. If the process is a long-standing one or if there is accompanying arteriosclerosis there may be areas of calcareous change, but commonly this is absent. On cross section of the wall of the aorta you will find varying degrees of thinning in the

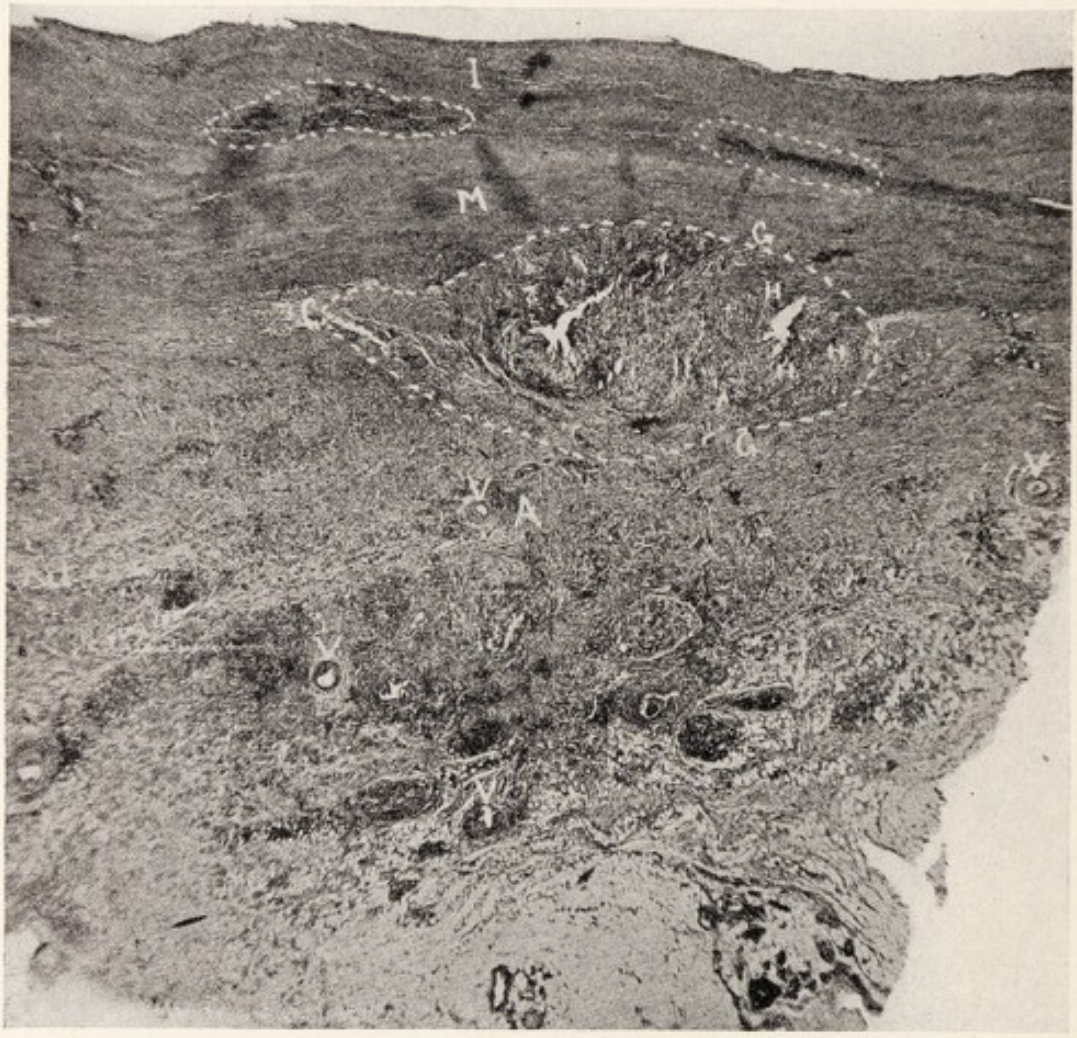



FIG. 70.—Section of the wall of the aorta in Fig. 69. I-intima. M-media. A-adventitia. Enclosed in dotted lines are areas of cellular infiltration. The largest, G, G, is a gummatous focus; great cellular infiltration, with three giant cells, H, H, H. V, V, V, some of the vasa vasorum, with partial occlusion, and in instances with surrounding cellular infiltration, consisting of cells of the lymphocyte series, plasma cells, and a few polynuclears.

regions of the areas of depression and of thickening in the regions of the plaques. In instances the wall may be thickened up to 7 or 8 mm. This great thickening is one of the typical characteristics.

All three coats of the aortic wall are thickened with more or less fibrous fusion and scarring. The media is also apt to show in places small yellowish, rather soft, necrosis-like areas, while the adventitia

is thickened and reddened. It is apparent that the areas of extensive fibrosis with great thinning of the wall are the points where aneurisms, which occur rather frequently in this disease, may form.

Syphilis is a septicemic disease; and the spirochetes gaining entrance through an obvious or an unknown lesion in a certain number of the cases are located in the wall of the aorta and possibly in those minute vascular caverns, the vasa vasorum. They multiply, produce their toxins, and as a reaction to this, inflammatory and



FIG. 71.—Aortic wall. Showing spirochetes of syphilis. From the same case as Fig. 70. × 1500. (Dr. J. H. Wright and Dr. Oscar Richardson.)

gummatous processes arise in the aortic wall on which as a basis the gross anatomic conditions found in the aorta rest.

Fig. 70. We see here a low-power microphotograph of the wall of the aorta in Fig. 69. It shows a section of the thickened syphilitic wall of the aorta with the cellular infiltration in full tide.

Fig 71 is a high-power microphotograph of the spirochetes in the aortic wall shown in Fig. 70, and is taken from the original section in which they were first found.

Syphilitic aortitis is usually confined to the first portion and the arch of the aorta, fading out in the region of the thoracic portion. It is to be noted, however, that it is present in the abdominal portion at times, and is seen in cases of abdominal aneurisms.

In all probability the smaller arteries of the body may at times be the seat of syphilitic arteritis. We have one case in which sections from the vessels of Willis show this condition.

A PHYSICIAN: Then death from syphilitic aortitis is purely mechanical?

DR. RICHARDSON: Death in syphilitic aortitis is commonly due to the following conditions:




FIG. 72.—Pulmonary artery in Case 4122. G-gummatous focus. Areas of cellular infiltration are well shown at C, C. Cellular infiltration about a blood vessel at V.

(1) The luetic process extends down and around the openings of the coronary arteries and finally occludes them.

(2) Aneurism with rupture and hemorrhage.

(3) Aortic regurgitation, hypertrophy and dilatation of the heart, myocardial insufficiency.

In (1) and (2) death is usually sudden in character. Of course all of this excepts any intercurrent cause of death. It is worthy of note that patients with well-established cases of syphilitic aortitis usually die before fifty years of age Thirty-five to forty-five is probably the decade in which death is most common.

A PHYSICIAN: In what percentage of cases do you find spirochetes?

DR. RICHARDSON: Only in a small percentage of the cases, but it is probably true that if we made serial sections of the lesions the percentage would be larger.

A PHYSICIAN: Is there any particular location in which you find the spirochetes?

DR. RICHARDSON: Usually in the region of or nearby the areas of necrosis and cellular infiltration.

Fig. 72 shows a low-power microphotograph of a section of the first portion of the pulmonary artery. The areas of cellular infiltration and the gummatous focus are well indicated.

Fig. 73. Here we see an almost perfect picture of the gumma in the heart muscle.




FIG. 73.—Heart muscle in Case 4122. G-gumma.

Long before the discovery of the spirochete of syphilis the association of peculiar lesions in the circulatory apparatus in cases of syphilis had been noted. The discovery of the spirochete, however, and the finding of spirochetes in the lesions settled the question, just as the discovery of the tubercle bacillus and the finding of tubercle bacilli in tuberculous tissue gave an etiological basis for tuberculosis. So although the necropsy in this case was made in 1897, some time before the spirochetes were found in the lesions, yet from the character of the lesions and the clinical history the anatomic diagnosis was written as given.

The pathologic work herein set forth is due to my association with Dr. James Homer Wright, the Director of the Pathological Laboratory at the Massachusetts General Hospital.

*Clinical Diagnosis (from Hospital Records).*—Syphilitic aortitis.
Decompensated heart.
Aortic stenosis. Insufficiency.
Aneurism of the aortic arch.
*Anatomical Diagnosis.*
Syphilitic endoaortitis.
Gummata of the heart and pulmonary artery.
Obliteration of the opening of one coronary artery.
Heart thrombi.
Hypertrophy and dilatation of the heart.
Chronic passive congestion, general.
Syphilitic scars on the penis and groin.

### Case CV

An army man thirty-three years of age came to Boston, went to a hotel, and for a week went about his affairs, was well liked, and made no complaints.

At the end of a week he came in about 5:45 p.m., spoke to the hotel clerk as usual and went upstairs to his room. He had been there only about five or ten minutes when the chambermaid came down and said that he had called for a glass of water, said that he was "feeling pretty bad," and asked her to call a doctor. They tried to get the doctor, but he was not in. Then they called in a police officer, who went with the clerk up to the guest's room. They found him dead on the bed. The whole thing happened within fifteen minutes.

The dead body lay on its back, completely clothed except for the jacket. It showed full rigor mortis, but otherwise was negative. He was a well set-up man weighing about 135 pounds.

The landlord said the only unusual things that he had noticed about the man were that when he hired the room he said he wanted one where there was plenty of air, and that he seemed to be what is called a "hard breather."

This man from time to time had been subjected to physical examinations by physicians.

*Dr. William H. Smith's Diagnosis.*—Probably syphilitic aortitis, with coronary sclerosis and occlusion.

*Anatomical Diagnosis.*—Luetic aortitis.
Aneurism of the aorta with rupture into the pericardium.
Hemorrhage into the pericardial cavity.

DR. RICHARDSON: There was some hypertrophy and dilatation of the heart, not very marked however. The valves were negative. The coronary arteries were negative. The aorta itself was the seat of an extensive aneurism which extended up into the region of the arch, involving it to a certain extent. At *s* (see Fig. 74) you will note that the aneurism gave off a little pocket-like sac and that this sac pressed on the wall of the trachea. The man was a "hard breather."



FIG. 74.—Aneurism showing sac at *s*. Photograph by L. S. Brown. (DR. Oscar Richardson.)

A seemingly insignificant point like that in a history may be of great aid in diagnosis. The capacity of his trachea had been reduced one-half from the pressure of this aneurism upon it. (See *s*, Fig. 75). The sac was wadded around with thrombus. Of course that made a good buttress, and it would have taken a long time before the aneurism would break into the trachea. What it did do was to

break through a thinned-out area in its wall and to rupture into the pericardium, which was found full of fluid blood with a thick layer of blood clot enveloping the heart. There was about 500 c.c. of fluid blood. The lesion itself, judging from its situation and from its character, is due to syphilis. It is syphilitic aortitis with an aneurism in the situation of the first portion of the aorta and the arch with rupture of the wall and hemorrhage into the pericardium.



FIG. 75.—Trachea partially occluded by pressure of aneurismal sac at *s*. Photograph by L. S. Brown. (DR. Oscar Richardson.)

These aortitis cases present a variety of pictures. The classical one is where the aortitis attacks the first portion of the aorta for varying distances above the aortic valve, and from that position extends down along the posterior walls of the sinuses of Valsalva and about the orifices of the coronary arteries, and involves the aortic

valve. The process may be extensive enough to close either one or the other of the arteries, and may cause sudden death. Less commonly, as in this case, an aneurism is produced and there may be rupture of the aneurism with sudden death due to hemorrhage. We have had cases of small and large aneurisms, but I have never seen a case with the development of an aneurism at this age and of this character with pressure on the trachea. We know syphilis is a septicemia, because Dr. Hartwell in this laboratory has taken rabbits, inoculated the testes with the blood of syphilitic patients, and later has found syphilis of the testes and still later syphilis of the eyes, keratitis, with recovery of the spirochetes from these lesions. That establishes the septicemia beyond all question. The portal of entrance of the infection is in some cases not known. For some reason in cases like this one the infection is located in the first portion of the aorta. Whether the spirochetes lurk in those minute vascular caverns, the vasa vasorum, or whether the attack is begun on the intimal side is uncertain. I am inclined to think that they locate in the vasa vasorum and that the tissue reaction begins there. This reaction is practically a gumma-like process ending in fibrosis. This accounts for the presence in the aortic wall of the variations between areas of great thickening of the coats and areas of excessive thinning. That makes in the wall points of weak resistance, all of which would favor the production of an aneurism. This is what happened in this case. These areas of thickening and fibrous thinning and longitudinal striation give to the aorta on its intimal aspect a gristly and scar-like aspect which is peculiar to syphilitic aortitis, and which no other form of sclerosis of the arteries seems to present.

Of the sudden deaths we have had in aortitis cases I think this is the first one at this age associated with an aneurism. We had one sudden death last year in a young man twenty-one years of age, who was supposed to have been poisoned; but that was a case of occlusion of the orifices of the coronary arteries. The only thing in the history of that case which was important as a single factor was chest pain. In this case the important thing was air hunger. You may, of course, have an aortitis with hypertrophy and dilatation of the heart, marked aortic regurgitation, with death probably due to heart exhaustion, but those cases do not present this picture of everything happening in a few minutes.

The production of the aortic regurgitation is interesting. The fibrosis extends down usually into the region of the contiguous margins of the cusps and forms a thick band, with more or less

extension on to the cusps themselves. This causes a narrowing of the width of the cusps and a pinning back of the margins so that you have more or less marked regurgitative conditions in the aortic valve without any great increase in its circumference. The cusps cannot come together and so form a base for the column of blood. In this way you get a drop of the column of blood equal to the size of the space due to the lack of closure.

I found great numbers of spirochetes in the sections of the aorta in the case last year, but so far few, if any, in the sections from this case. They have always interested us here, and it was in this laboratory that they were first found in the wall of the aorta. In some cases they are more or less numerous, but in others none are found. The older the process the less likely you are to find them.

A PHYSICIAN: Is it common for people suffering with angina to have attacks while lying perfectly still in bed?

DR. SMITH: Such attacks are uncommon. They do occur, however. Clinically one of the earliest manifestations of a beginning myocardial weakness may be angina pectoris. If you examine the patient you may be able to demonstrate no cardiac enlargement, you may have a pulse rate of 90, a soft systolic murmur, but you will always find a slight tendency to dyspnea on exertion which has come on in the last six months or year. Let us say you have a patient fifty years of age who begins to feel dyspneic on exertion. Now you must realize that that man's heart is becoming senile, in other words, there is arteriosclerosis and not sufficient blood supply to the muscle. That heart has got to hypertrophy in order to enable the man to go through the same exercises that he has gone through for years without discomfort. Take that patient and prohibit all exercise. Put him on digitalis in the appropriate amounts, not large doses but moderate ones, limit his exercise and watch his heart over a period of six months, and you will find increase in the left ventricle with disappearance of symptoms. If you do not do that, and do not put him on digitalis, but give him nitroglycerin, the next step will be that he will have more frequency of dyspnea on exertion and will get into the anginoid state. He will have attacks constantly. You can oftentimes relieve angina by establishing hypertrophy through rest and digitalis. After establishing hypertrophy I have known patients to go for years never having recurrent attacks of angina and with a perfectly competent heart unless forced by unusual exertion. They can do it because their heart has been made a 400-500 gram heart.

### Case LIV

A colored waiter of forty-two entered November 4, 1913, for the relief of pain in the left back and chest. He had had no illnesses except "grippe" at twenty-one. He smoked ten cigars a day and drank three glasses of whiskey until two years before admission. He had used practically none in two years. He had chancre five years before admission. His best and usual weight was 175, his present weight 151½. The loss had occurred during the past year.

Two years and a half before admission he began to have intermittent pains in the left back and flank, usually between one and seven in the morning. For the past two years the pain had become continuous in the left back, side and chest, never on the right. The pain was not sharp, but dull, like hard pressure, and "throbbing." His throat felt obstructed. Since September 20, when he caught "a cold" which had persisted, he had been having dyspnea accompanied by wheezing on inspiration and expiration. He had no palpitation. He coughed a great deal and raised considerable sputum, at first green and thick, during the past week streaked with blood three or four times, usually at night.

The Out-Patient Department record, November 19, 1912, notes: "Impulse 5th space, 1½ cm. outside the nipple line; right border 4 cm. outside the midline. Right pulse stronger than left. Lungs negative except that tactile and vocal fremitus were slightly greater on the right." January 7, 1913, he had been in a hospital two weeks. "Pain worse at discharge. Examination as above."

Examination showed a well-nourished negro breathing with slight distress and with an occasional coarse tracheal cough. The skin showed dark macules over the upper back and upper legs. The mucosae were slightly cyanotic. A definite pulsation was seen and felt in the left second and third spaces near the sternum. By very light percussion there was dullness over this area. The apex impulse

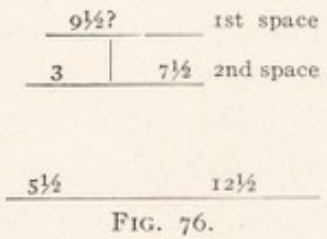



FIG. 76.

was faintly felt in the fifth space. The percussion measurements are shown in Fig. 76. The sounds were regular, of only fair quality. A very soft systolic murmur, loudest at the apex, was faintly heard

over the precordia, but not in the axilla or neck. No diastolic was heard. The pulmonic second sound was slightly increased. The pulses were unequal, otherwise normal. The artery walls were plainly palpable, but not tortuous. The blood pressure was, right, 150/70–120/45; left, 130/80–110/50. There was tracheal tug. The chest was somewhat barrel-shaped. Air did not enter the left lung as well as the right. The breathing was obscured throughout by coarse guttural sounds, probably from the trachea. Both inspiration and expiration were prolonged, but seemed of fairly normal quality. A few fine râles were heard at both bases. In the left back was an area of doubtful dullness near the midscapula. The abdomen was

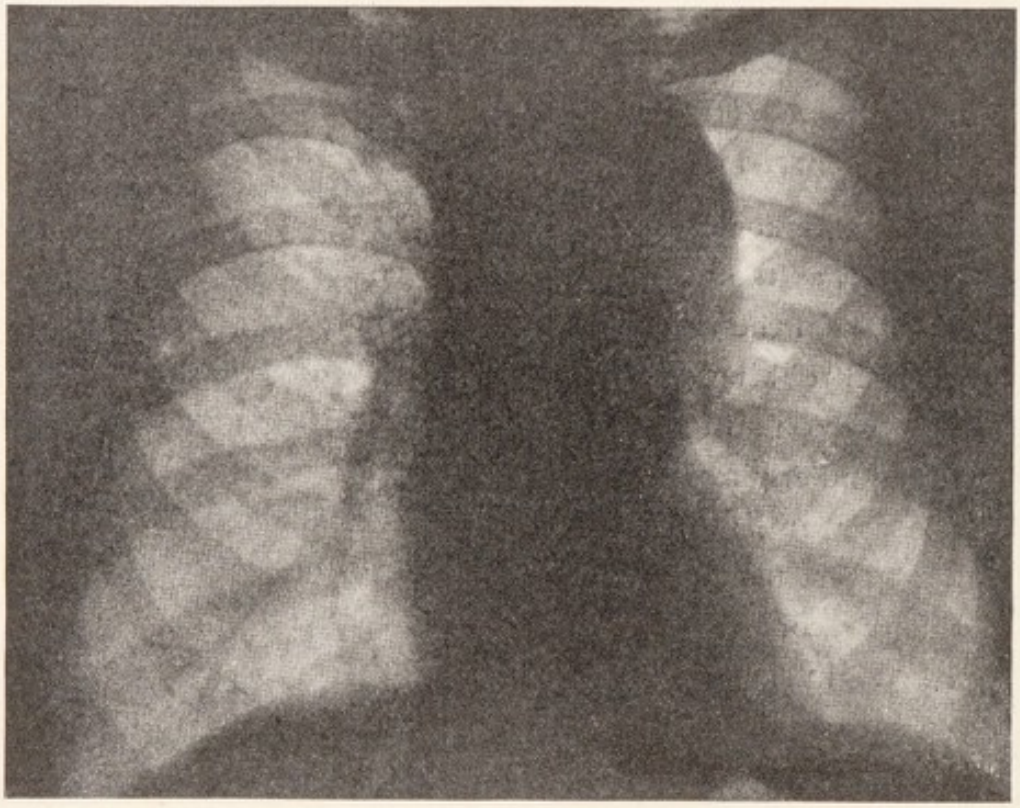


FIG. 77.—Aneurism of aortic arch.

held rigid. The liver dullness extended from the fifth rib to 3 cm. below the costal margin, where the edge was indefinitely felt. There was a slightly thickened scar on the penis shaft. The extremities and reflexes were normal. The pupils were slightly irregular, equal. Their reactions were questionable.

The temperature was 98° to 99.5°, rising to 100.4° November 24. The pulse was 65 to 93, the respiration normal. The urine was normal. The hemoglobin was 76%. There were 10,000 leucocytes, 84% polynuclears. The reds were normal. One Wassermann was negative, two later suspicious after provocative salvarsan. X-ray

(Fig. 77) showed a large mass in the upper mediastinum continuous with the aorta, more prominent posteriorly than anteriorly. No definite pulsation was made out.

The patient had at times a very deep, ringing cough, raising a little yellow-green sputum. When the head was tipped back the trachea could be seen to move to the right with each pulsation. 0.15 grams of neosalvarsan was given intravenously November 10. November 16 the breathing over the left lung was found to be distinctly less loud than over the right, but vesicular; air was thought possibly to enter slowly. He had less cough. November 17 0.3 grams of neosalvarsan was given intravenously. The next day the diminished breathing all over the left lung was more marked than before and a few scattered moist râles were to be heard over this side of the chest. He complained of a feeling of constriction in the throat, but had no pain during his entire stay. November 23 a visitor, Dr. Wilder Tileston, pointed out a fine, slight pulsation in the back near the spine of the scapula. The next day 0.45 grams of neosalvarsan was given, and a third Wassermann was suspicious. November 27 and 28 the cough was more troublesome. November 29 the heart measurements were as shown in Fig. 78. 0.6 grams of neosalvarsan was given. November 30 the patient was discharged slightly relieved.

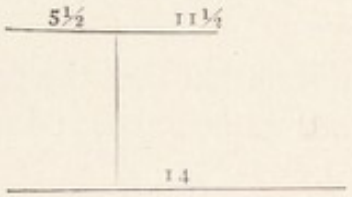



FIG. 78.

*Clinical Diagnosis.*—Aneurism of descending aorta.

*Later Notes from the Out-Patient Record.*—April 9, 1914.—Systolic blood pressure, right 160, left 150. Wassermann negative.

April 11.—Wheezing and shortness of breath on exertion, and cramping but not agonizing pain under the left clavicle over an area about 7 cm. in diameter. Is often troubled at night by similar pain in left shoulder and arm, extending even to fingers. This pain comes on gradually, and may last several hours. Thinks he is improving. *Examination:* Right clavicle more prominent than left. Visible bilateral subclavian pulsation, and strong epigastric pulsation over wide area. No pulsation or bulging in the back. Apex impulse not visible but barely palpable in 6th space 11.7 cm. to left

of mid-sternal line. Fairly loud, rough systolic murmur at apex, faintly transmitted to axilla. No basal murmurs heard. $A_2$ is greater than $P_2$. Lungs:—Back, dullness between and over scapulae seems more than normal.

April 13.—X-ray. Greatest transverse diameter of arch 9.5 cm.; oblique diameter of arch 9.5 cm.

July 14.—Has been working full time right along. Had a little trouble at first; now has no dyspnea. *Has had no pain at all!* Has taken no medicine. Comes for a "cold in the head" taken a week ago. Causes a wheezing cough. Present weight 174. *Examination:* Nose and throat clear, not injected: tonsils not enlarged. Vocal cords seem normal. Heart as in previous record. Lungs:—Tactile fremitus and voice decreased over the left back at angle of scapula.

Aug. 25.—Cough bothers. Stopped work a few days ago on account of it. No pain, edema or weakness. No diastolic murmur. Squeaking râles scattered in lungs, more on left.

*Outcome.*—Feb. 25, 1915, the patient writes, "I am glad to inform you that I have been working since May 15 up to the present time. The pains in chest and back are gone, and with the exception of a slight weakness in the left shoulder I am feeling better than I have for years."

March 2. 1916, he writes, "I am glad to say I am enjoying good health and have had no symptoms of past trouble, which seems to have left me altogether."

A friend adds the information that the patient says he never felt better in his life, and has gained sixty pounds since he was in the hospital.

### Necropsy 3908

A Canadian-American housewife of twenty-five entered August 29, 1891. She had always been delicate, with much weakness and stomach trouble. For nine months she had had debility and cough with sputum; of late increasing pain and tenderness in the stomach.

Examination was negative except for pregnancy of nine months. She weighed 90 pounds. The temperature was 98.6° to 101°, the urine and sputum negative. September 11 she was discharged much improved.

November 4, 1910, she returned complaining of substernal tightness and cough following a severe "cold" of three weeks' duration, with general malaise. In a few months her weight had fallen from 165 to 110.

Examination was negative except for pyorrhea and redness of the throat.

The temperature was 96.4° to 99.3°, the pulse 113 to 73, the respiration normal, the leucocyte count 10,000. No Wassermann was done. The urine, sputum and stools were negative. November 12 she was discharged.

December 7, 1918, she returned for relief of shortness of breath. She now gave a history of scarlet fever and measles in childhood. In 1906 she had a plastic operation on the uterus. She had had many sore throats for years, but none since a second attack of diphtheria in 1909. For many years she had been troubled by cough with at times a little tenacious sputum. She had palpitation on excitement and frequent dyspnea on exertion, sometimes at night. For a year she had urinated sixteen times by day and four at night, with marked polyuria. Her best weight was 165, her usual and present weight 110.

For six years she had been treated for asthmatic bronchitis manifested by dry cough with dyspnea. In March, 1918, she began to have attacks of acute dyspnea and "choking spasms." From that time her breathing became more difficult. She had continual unproductive cough, difficulty in swallowing, and several attacks of dyspnea daily, particularly likely to occur just after eating, and lasting five to twenty minutes. The "choking spasms" left her practically unconscious from asphyxia. They never occurred more than once daily until the night before admission, when she had two attacks of great severity. She lived in such continual dread of sudden death that her nights were practically sleepless.

Examination showed a poorly developed and nourished woman. The sclerae and throat were slightly injected. The thyroid was enlarged. The chest was poorly developed. The superficial veins were rather marked over the left chest in front. The breathing was nearly all diaphragmatic, with very little chest expansion. The throat and tracheal sounds were transmitted down both lungs. There was stridor of maximum intensity over the substernal part of the trachea. The apex impulse of the heart is not recorded. The left border of dullness was 10.5 cm. to the left of midsternum, 0.5 cm. outside the nipple line. The right border was 4.5 cm. to the right, the substernal dullness 7 cm. and slightly increased in degree. There was a soft diastolic murmur, loudest in the third left interspace next to the sternum. The pulses and artery walls were normal. The blood pressure was 120/80; right and left the same. The pupils

were regular. They reacted very sluggishly. The right was greater than the left. The abdomen, extremities and reflexes were normal. The uterus and ovaries had apparently been removed.

The temperature was 97.4° to 101.6°, the pulse 89 to 155, the respiration 14 to 36. The urine was normal except for a trace of albumin at the second of two examinations. The renal function was 50%. The hemoglobin was 80%. There were 10,800 leucocytes,

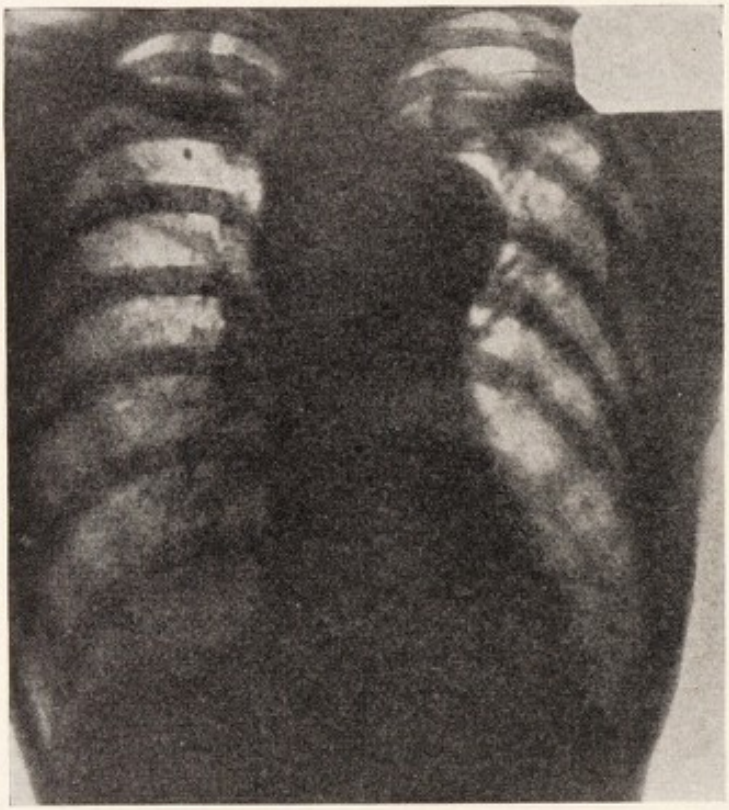


FIG. 79.—The diaphragm is low on both sides; the respiratory movements somewhat limited. General thickening of the markings throughout both chests and many calcified glands at the roots, also thickening of the larger bronchi. There is moderate scoliosis. The heart shadow appears considerably to the left of the spine. The aortic arch is prominent. There is also a dense round shadow lying in the region of or behind the upper segment of the sternum, possibly a mass of mediastinal glands. The heart is within normal limits. (Apparently the radiologist did not consider this aneurism.)

59% polynuclears. The platelets were increased. The reds showed slight variation in size, some achromia, and a rare stippled cell. A Wassermann was strongly positive. The sputum showed Gram-positive diplococci and long and short Gram-negative bacilli. X-ray showed the shadows pictured in Fig. 79.

A throat consultant reported, "Paralysis of the left cord. Obstruction to respiration is intrathoracic."

The patient had marked respiratory distress and a typical mediastinal cough. There was steady falling off in the character of

the pulse. December 11 she had a period of apnea in which for a few seconds she appeared to be dead. After this she coughed more and became completely aphonic. The periods of cyanosis became more frequent and severe. Oxygen had been used with benefit, but the patient was afraid to breathe it and lost the psychological advantage of it. Caffein seemed to relax the spasm. The distress was well controlled by morphia. An intubation set was kept at hand, but there seemed to be no one acute respiratory spasm. It was thought that the pressure was too low down for intubing to do any good. It was also thought any manipulation within the trachea would be dangerous. December 13 the patient died.

*Clinical Diagnosis (from Hospital Record).*—Cardiac weakness.

Aneurism of the aortic arch.

*Dr. Richard C. Cabot's Diagnosis.*—Syphilitic aortitis.

Aneurism of the arch of the aorta.

*Anatomical Diagnosis.*—Syphilitic aortitis.

Aneurism of the arch of the aorta with pressure on the trachea, the primary bronchi and the great vessels.

Purulent bronchitis.

Bronchopneumonia, lower lobe, left lung.

Fibrinopurulent pleuritis, left.

Congestion of the liver and kidneys.

Slight chronic pleuritis, right.

Slight chronic perisplenitis.

DR. RICHARDSON: This case, from an anatomical standpoint, is a very important one in regard to differential diagnosis. Practically all the questions I think can be answered.

The heart weighed 220 grams (normally 200–300) and there was nothing the matter with the valves. The coronary arteries were negative.

Beginning a short distance above the cusps there was a frank luetic process extending up the first portion of the aorta to the region of the arch. In the region of the arch there was a definite aneurism containing a thrombotic mass. This sac pressed upon the trachea and the left bronchus. That decreased their circumferences, their capacity, and in time bronchitis and bronchopneumonia developed. The bronchopneumonia and the bronchitis in the main were on the left side, where the greatest amount of pressure was.

In addition, and in line with the question, What did she die of? On the wall of the bronchus was a very thin area which had not quite

broken through. If she had lived a little longer it would have been broken through.

The lungs were out of the picture except for what we have said. The bronchial glands were negative.

A PHYSICIAN: What were the shadows the X-ray man told about?

DR. RICHARDSON: I think they were shadows.

The circulatory apparatus elsewhere was negative. The diagnosis of luetic aortitis was confirmed by microscopical examination, and the case is a typical one, showing irregularities of the surface of the intima presenting as fibrous plaques varying in size, with intermingling areas of depression, so that the intima of the aorta looked gristly and scarred. With such a condition scattered along the wall of a vessel which is under constant tension it is easy enough to understand how an aneurism would arise. I think the pressure on the great vessels by the aneurismal mass was the cause for the congestion of the liver and kidneys that was noted.

This brings out the point that when the luetic aortitis does not extend to the aortic valve the anatomical basis for an aortic regurgitation is not present, and regurgitation ought not to be found clinically.

The kidneys were negative.

In a word, this case anatomically was a distinct and clear-cut case of luetic aortitis with aneurism of the arch, pressure on the great vessels and the trachea and bronchi, no involvement of the heart at all, and with freedom of the coronary arteries. From the pressure resulted the bronchitis and bronchopneumonia.

A PHYSICIAN: Why was there no pain?

DR. RICHARDSON: That is a question. It may be from the peculiar situation of the aneurism. It came off postero-laterally to the left of the arch, and it might not in that particular place and for a time have involved the nerves. The laryngeal trouble came late.

A PHYSICIAN: How large was the aneurism?

DR. RICHARDSON: It was about $7 \times 6 \times 4$ cm.

A PHYSICIAN: Why do you think she had no chest pain when she was first admitted?

DR. CABOT: That was in 1891—a very long time ago.

A PHYSICIAN: Why the diastolic murmur?

DR. CABOT: I cannot explain it. I do not believe we should have heard it.

DR. RICHARDSON: The last thing was the infection, that is, the immediate cause of death was the terminal bronchitis and pneumonia.

This case is not so very unusual. I think at the same time we had two other cases just like it. When the process begins out of reach of the valve then the picture is entirely non-cardiac. It lacks all the things that give aortic regurgitation.

DR. CABOT: It should be noticed that although we can say that this all came down to one thing—the spirocheta pallida got into that person's blood and was deposited in the arch—still when we come to read the necropsy protocol what a number of things there are in it, what a number of things a person dies of! That is the experience of to the average post-mortem diagnosis. A complete diagnosis should contain many items, and I think one should aim at getting in all there is in a case.

For instance, the pneumonia was not recognized in this case, perhaps could not have been. They could not have treated it, but the length of life and the prognosis might have been affected by the pneumonia and their knowledge of it.

The arch of the aorta saddles the left primary bronchus. It is in very much closer relation to the left primary bronchus than to the right. It is for that reason that when we get pressure symptoms on the lung in aneurism they are almost always on the left lung, as it was here. By pressing on the bronchus the aneurism prevents the secretions from coming out, and so we have retention of the secretions and so pneumonia.

DR. RICHARDSON: The cutaneous vessels were notably enlarged on the left and not on the right.

A PHYSICIAN: If this patient had had pain where would it have been?

DR. CABOT: Usually pain comes when the aneurism is pressing outward toward the front with pressure upon the sternum. In the rarer cases where it goes backward toward the lungs it causes pain in the back radiating around the chest.

DR. HOLMES: In the X-ray examination a more careful localization of the tumor in the antero-posterior plane and an observation as to whether or not the tumor pulsated would have helped considerably in interpretation. The presence of the lateral scoliosis in the upper spine produced a shadow which was confusing.

### Necropsy 3656

An American police officer of forty-five entered October 8, 1915, for relief of pain in the chest and dyspnea. His mother died of "shock" at seventy-nine. His wife had had two miscarriages.

One child died of "indigestion" at seven months. The patient had scarlet fever at eight, typhoid fever at fourteen, gonorrhea and chancre at eighteen. For years he had had belching of gas after meals and constipated bowels, moving only every other day with laxatives. For years he had urinated once at night. Eight months before admission he had external hemorrhoids for three days. In 1912 he weighed 205 pounds, his best weight. His usual weight was 180, his present weight 172. He drank two or three glasses of whiskey and smoked four or five cigarettes a day.

Four years before admission he noticed a swelling at the right sterno-calvicular articulation. This gradually increased in size, but gave no other symptoms for two years. Then he began to have dyspnea and slight difficulty in swallowing, so that if he lay on his back he became choked up. At the same time he began to have dull burning pain in the region of the tumor and several daily attacks of sharp boring pain lasting a few seconds, like a "red hot poker being thrust through to his back," also radiating as a dull ache down the right arm as far as the elbow. The pain was worse when he lay on the right side, relieved by lying on the left side and by belching gas. It had been constant since the onset, but had not noticeably increased

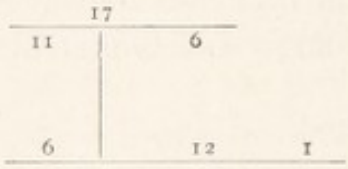



FIG. 80.

in severity. The tumor had increased in size since the onset, though at times he thought it got smaller. He often felt a beating over it. He was told by a physician he had pleurisy. Seven months ago he caught "grippe" and became "all choked up" and dyspneic. The chest pain was worse. His doctor now said that he had rheumatism.

Examination showed a well-nourished man with tracheal tug. There were dilated venules over the lower margin of the thorax in front. The apex impulse of the heart was in the sixth space 13 cm. to the left of midsternum, 1 cm. outside the nipple line. In the left lateral position the apex shifted 6 cm. The right border of dullness was 6 cm. to the right. The substernal dullness was 17 cm. at the second rib, 11 cm. to the right, 6 cm. to the left. (See Fig. 80.) X-ray showed the measurements as in Fig. 81. The sounds were of fair quality. There was an occasional premature beat. The aortic second sound was accentuated and palpable. At the apex in the

left lateral position was heard a blowing systolic and a long, harsh high-pitched diastolic. There was a systolic at the aortic area, not loud, transmitted upward, with a short diastolic blow. There was a rough low-pitched systolic over the pulsating tumor. The blood pressure was 140/80 left, 120/80 right. The tumor and lung signs

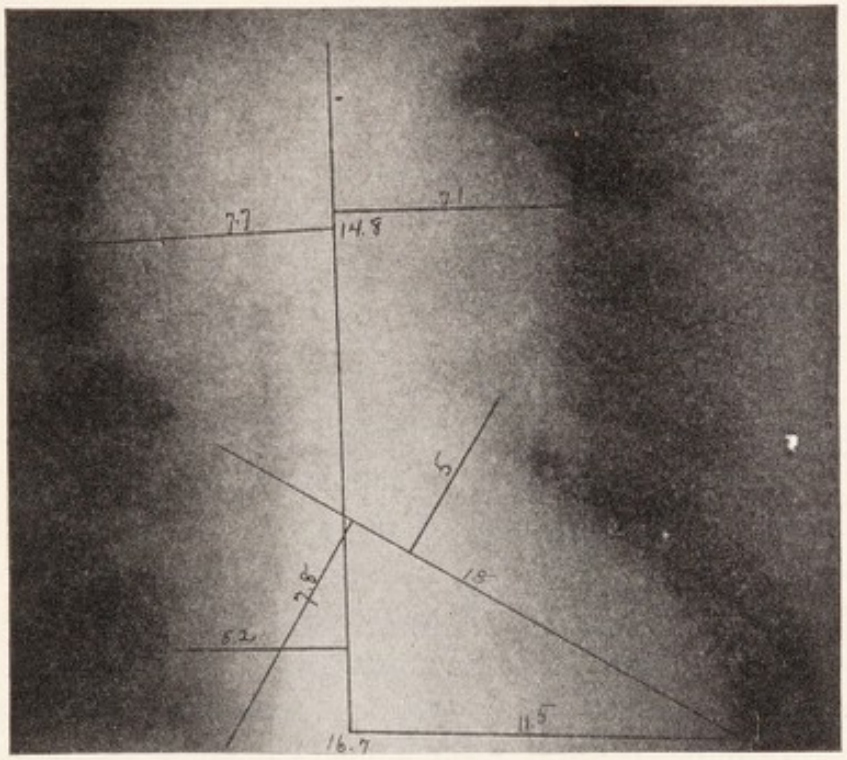



FIG. 81.—Seven-foot plate showing heart shadow below and aneurismal sac above. Syphilitic aortitis with aneurism filling the mediastinum, compressing esophagus, trachea, and bronchi.

were as shown in Fig. 82. There were many dilated venules over the shins. The abdomen, pupils and reflexes were normal.

The temperature was 96.4° to 99.3°, the pulse 66 to 102, the respiration normal. The output of urine was 32 to 70 ounces, the specific gravity 1.012 to 1.020. There was a slight trace of albumin

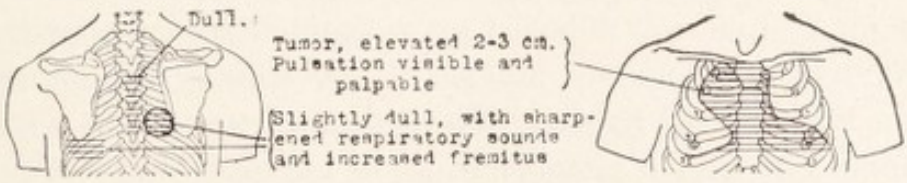



FIG. 82.

at one of three examinations. The hemoglobin was 80%. There were 12,800 to 7600 leucocytes, 79% polynuclears. A Wassermann was strongly positive. A throat consultant reported, "Slight chronic laryngitis. The vocal cords have normal excursion in phonation and respiration." A syphilis consultant reported, "Salvarsan is a dan-

gerous drug for this man. I should use potassium iodid in preference at first. If that does no good I think it proper to give salvarsan in repeated very small doses, not over 0.1 gram."

The patient was given antisyphilitic treatment. His condition was explained to him. He was discharged unrelieved October 19 with the understanding that he would get very light occupation.

The South Medical (Syphilis) Out-Patient Department record notes November 15 that the patient was doing desk work and was very much better. December 21 he reported that he had entered strenuous duty again and was having pain in the right chest. February 15 he was again at desk work, but was having dyspnea and increasing pain of greater frequency and longer duration.

A year later, November 13, 1916, he reported that he felt almost well, gained weight, and was able to do his work until July, when his wife was in the hospital ten days with her third miscarriage. During her absence he worked strenuously to keep up both home and office duties. As a result he had a "breakdown" and had not felt well since. One night in August he fell asleep in a chair near an open window and awoke a few hours later covered with "cold perspiration." Since that time he had had cough with a little sputum, a choking sensation due to the mucus which he could not raise, hoarseness, wheeziness, and increasing dyspnea, orthopnea, edema, palpitation and insomnia. Since August he had had frequency with incontinence every two hours day and night.

Examination showed him only fairly well nourished. He spoke only in a hoarse whisper. The mucous membranes and hands were cyanotic, especially the left hand, which was colder than the right. No tracheal tug was felt. The heart measurements by percussion were practically as before except that the right border was 7 cm. to the right. At the apex the sounds were regular, somewhat rapid, not forceful, and there was a low systolic murmur. In the aortic area was a faint systolic roll followed by an impure second sound. The pulmonic second sound was not heard. A faint systolic was heard along the right sternal border. The pulses were of poor volume and tension, not Corrigan or pistol-shot. The radials were not felt. The blood pressure was 115/80. The lung signs were as shown in Fig. 83. There was dullness at the right isthmus in front.

The temperature was 97.2° to 98.8°, the pulse 81 to 100, the respiration 31 to 17. The amount of urine was normal when recorded. The urine was cloudy at one of two examinations. The specific gravity was 1.000 to 1.010. There was a very slight trace of albumin

at one of two examinations. The hemoglobin was 80%. There were 10,400 to 10,600 leucocytes, 71% polynuclears. A Wassermann was moderately positive. X-ray showed a large area of diminished radiance in the region of the great vessels, more to the right than to the left. The heart shadow was enlarged and lay horizontally in the chest.

The night of November 14 the patient was very restless and complained of difficulty in getting his breath. Next morning he was more cyanotic. During the ward rounds he suddenly became very cyanotic, with labored breathing, and soon afterward became unconscious. He remained in this condition until his death at midnight.

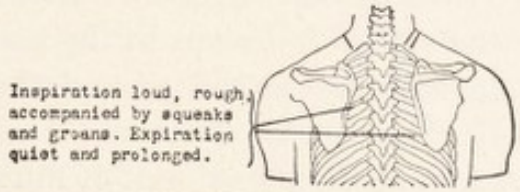



FIG. 83.

*Clinical Diagnosis* (*from Hospital Record*).—Tertiary lues.

Aneurism of the aortic arch.

Cardiac failure.

*Dr. William H. Smith's Diagnosis.*—Syphilitic aortitis.

Aneurism.

*Anatomical Diagnosis.*—Syphilitic aortitis.

Aneurism of the arch of the aorta with pressure on the neighboring structures.

Hypertrophy and dilatation of the heart.

Chronic passive congestion of the liver, spleen, stomach, kidneys, and intestines.

Focal necrosis of the pancreas with hemorrhage.

Emphysema of the lungs.

Chronic pleuritis.

Slight chronic perihepatitis, and splenitis.

DR. RICHARDSON: There was a large aneurism given off at the arch of the aorta filling the mediastinum, pressing slightly on the esophagus and markedly on the trachea, the bronchi, and the great vessels running to and from the heart. The chronic passive congestion mentioned in the anatomical diagnosis was largely due to the pressure inside the chest from the aneurismal sac. The coronary arteries were out of the picture, the aneurism springing from the

arch, although the first portion of the arch showed lesions many of which were syphilitic in appearance.

The kidneys showed some acute degeneration of the tubular epithelium, but were otherwise negative.

The microscopic appearances of the aortic wall and the region of the aneurism were those of syphilitic aortitis. The case anatomically was clear and formed a basis for the clinical picture.

DR. SMITH: There was not much regurgitation through the aortic valve, was there?

DR. RICHARDSON: None. The heart was moderately hypertrophied; the dilatation was the predominant characteristic. It is very common, of course, in syphilitic aortitis to have the orifices of the coronary arteries encroached upon. In this particular case, however, although the syphilitic lesions in the aorta extended along the first portion, they did not extend around the coronary orifices so as to produce any effect on the arteries. They were free, fairly capacious, and showed a moderate amount of fibrous sclerosis. The whole picture was due to marked pressure of the aneurismal sac on the great structures in the chest, trachea, bronchi, and the great vessels to and from the heart.

DR. SMITH: Dr. Richardson's discussion brings out the point I mentioned, that angina pectoris may be due to a stretching of the aortic plexus, in view of the fact that here the coronary arteries were not involved.

### Necropsy 4388

An Irish laborer of fifty-three entered August 11, 1922, complaining of cough and pain in the back and chest. He had measles, chickenpox, whooping cough in childhood, and malaria for two winters in Georgia. He once had "rheumatism" for a year and a half—swelling, lameness and redness of the knees and hands, with no fever. Six years before admission he had a violent cough like the present one lasting two months. In the Philippines he had dhobie itch—white blisters around the genitals and armpits. His wife had one miscarriage after the birth of two healthy children. For four years his left eye had been more prominent than the right and the pupil larger. His bowels required physic often. He took two or three glasses of whiskey on Saturday nights.

Six months before admission he began to have spasms of violent dry cough for five to twenty-four hours, each attack leaving him feeling short of breath and stifled. For five months he had had dyspnea.

He was more comfortable lying on the left side, but if he lay on it too long he had pain from his left shoulder to his ear. He thought he had had a swelling off and on in the left side of the neck for four months, and thought it was associated with the pain. Four months ago he lost consciousness in church for about five minutes. Three months ago the attacks of cough became more frequent, and he began to raise a good deal of watery frothy sputum. For three months he had had pain under the sternum and in the back between the shoulders. Sometimes, for instance when he moved quickly, it was like a knife stabbing from front to back. If he lay still fifteen or twenty minutes he had dull pain under the sternum and in the back. When he kept moving he did not notice this. Two months ago he began to be hoarse, and the cough became wheezing. Twice he lost his voice altogether for half an hour, and at the same time lost power in his right hand, with complete recovery. A week ago he coughed up half a teaspoonful of bright blood. He now slept very little. At admission he could climb a flight of stairs only with difficulty. His best weight was 180 pounds, ten years ago. For five months he had been losing weight, in all thirty-five pounds. His present weight was 140.

Examination showed a well nourished man with suffused cyanotic face and neck. The external jugulars were distended. The teeth were very carious, several missing. There was marked pyorrhea. The apex impulse of the heart was not seen or felt. The sounds were not heard. The supracardiac dullness was 9.5 cm., the right border 6 cm., the left border 10½ cm., the midclavicular line 11 cm. The left pulse was not felt. The blood pressure in the right arm was 128/60, in the left arm 65/55. There was questionable tracheal tug, palpable episternal pulsation. The lungs were clear except for questionable dullness at the right isthmus. Palpation of the abdomen was rendered unsatisfactory by what appeared to be voluntary spasm. The left pupil was greater than the right. Both were regular. Their reactions and the other reflexes were normal.

The temperature was 98.8° to 101.5°, the pulse 112 to 88, the respirations not remarkable. The output of urine was 38 ounces when recorded, the specific gravity 1.018. The urine was cloudy and alkaline at the single examination. There were three to four leucocytes to a high power field. The hemoglobin was 70%, the leucocytes 39,300 to 12,300, the polynuclears 80 to 64%, the reds 5,220,000; slight achromia August 11, reds normal August 12. No Wassermann was done. The stools were strongly positive to guaiac August 12. X-ray August 14 showed a large fusiform shadow in the upper and

medial portions of the chest obliterating the posterior mediastinal space. (See Fig. 84.) No pulsation was visible in the shadow.

The day after admission the right apex was dull, possibly the left apex also. The breath sounds at the apices were almost amphoric, more so on the right.

At five o'clock the evening of August 13 the patient suddenly fainted, became very pale and pulseless, and broke into a profuse cold sweat. He regained consciousness in a few minutes, although he remained pulseless, pale and very weak. He coughed up about an

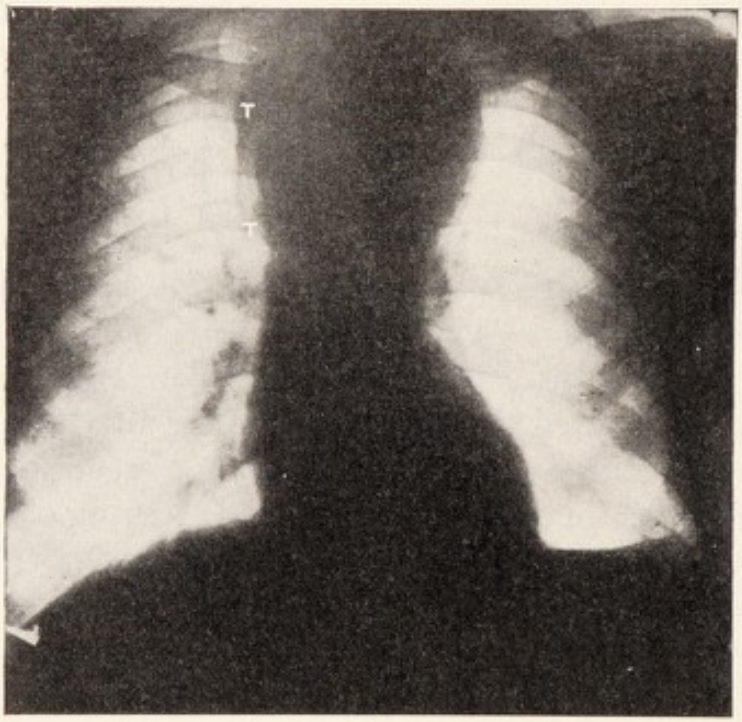



FIG. 84.—Necropsy 4388. Aneurism of the aorta. Syphilitic aortitis. A large fusiform shadow occupies the medial and upper portions of the chest, its borders curved and sharply defined. It is about the size of the heart shadow and extends from above the sternal notch down over the upper portion of the aortic arch. The shadow *T*, *T* on the right may very well be a much displaced trachea. The left lung is held up by adhesions. Its diminished radiability is no doubt due to limitation of the diaphragm excursion. (Roentgenological Department, Massachusetts General Hospital.)

ounce of bright frothy blood, and a few minutes later had a stool that was mostly fecal material mixed with bright blood. He remained conscious for about an hour, then went into coma which continued to his death at four o'clock the next morning.

*Clinical Diagnosis.*—Aneurism of the aorta.

*Dr. Richard C. Cabot's Diagnosis.*—Aneurism of the aorta, with rupture.

*Anatomical Diagnosis.*—Aneurism of the aorta with rupture into the esophagus.

Luetic aortitis.
Hemorrhage into the gastrointestinal tract.
Anemia.
Soft spleen.
Chronic pleuritis, left.
Chronic perisplenitis.
Slightly defective closure of the foramen ovale.

DR. OSCAR RICHARDSON: We were not permitted to examine the head. The skin and mucous membranes were very pale. The peritoneal cavity and appendix were out of the picture. The glands were negative. There was no fluid in the pleural cavities, and no adhesions on the right, but many on the left. There was a little blood and blood clot in the trachea and bronchi; they were otherwise out of the picture except for a point to be mentioned later.

The pericardium was negative. The heart weighed 333 grams. It was a good looking heart, with good valves, a little dilatation on the right, but otherwise out of the picture.

The liver, gall-bladder, bile ducts, pancreas, duct of Wirsung, and the spleen, the adrenals, kidneys and genito-urinary apparatus were out of the picture, except that the spleen was a little soft.

The esophagus contained a small amount of bloody fluid and presented an opening in its wall which we shall speak about in a moment. The stomach contained a blood clot weighing 1260 grams —that is nearly as large as a liver—and besides that 200 c.c. of bloody fluid. The mucosa and the pylorus were frankly negative. The intestines contained a large amount of fluid blood and many blood clots, but their walls were negative.

The only thing left is the aorta. The aorta showed an aneurism and this aneurism was luetic in its nature. The luetic process began above the aortic valve and the coronary orifices were free, so the heart was out of the picture anatomically. But from that point up there was syphilitic aortitis, and in the situation of the arch there was a large sac, nine cm. in diameter,—a frank aneurism. This of course contained the usual concentric layers of thrombotic material, and on the posterior wall where it was against the anterior surface of the vertebra the wall was very thin and there was erosion of the surfaces of the vertebrae. On the left lateral aspect the wall of the aneurism was adherent to the apex of the lung on its mesial aspect. We note however that there was nothing the matter with the lungs; the apices were negative. This aneurism had pushed the trachea away over to the right but had perforated, not the trachea, but the

esophagus. So the death was due to hemorrhage from rupture of the aneurism through the wall of the esophagus, and the man bled to death into his stomach.

DR. CABOT: Isn't that in your experience an unusual way?

DR. RICHARDSON: Yes, it is very unusual. Somewhere recently I saw an article reporting such a case because the writer thought perforation of the esophagus was so rare.

DR. CABOT: Looking at the X-ray in connection with your report of the adhesions, would those match up with the appearances at the left base?

DR. RICHARDSON: It might be. The left lung was bound down by adhesions of years, on the right none.

DR. CABOT: When we know a fact like these adhesions on the left it is always good to go back to the X-ray to see if there is any more density of the left lung, any less radiance on the left than in the right. Dr. Richardson has just told us there are adhesions all through the left chest.

DR. MEANS: I do not think I should be willing to say that the plate is abnormal.

DR. CABOT: No; but there is a question whether the chest does not look on the whole a little less radiant on the left and is not on the whole a little smaller.

DR. MEANS: The diaphragm is higher.

DR. CABOT: It seems as if the rays went through more easily on the right. Of course, I am not pretending that I made the diagnosis before I knew the adhesions were there.

DR. RICHARDSON: This aneurism instead of going laterally in its main trend went directly back against the vertebra; the picture shows that.

DR. CABOT: Yes, it is right in the middle.

DR. MEANS: We have another man in the wards who has a trachea pushed to one side, who has amphoric breathing, breathing that would make one think of a cavity. But there was no cavity. I think that where there is tracheal stenosis we get extraordinary breathing of all kinds.

A PHYSICIAN: How big was the opening?

DR. RICHARDSON: The area of perforation was three cm. above the bifurcation of the trachea and measured three by two cm.

DR. CABOT: With even a large eroded area outside, a bulge on the chest as large as one's fist, the probability is that it will erode through some internal structure before it does through the skin.

I think that is a very important fact. If we can say to the friends especially, and perhaps to the patient himself if he is the right type, "You won't have this terrible tragedy of an external rupture," I think that is of some reassurance. I cannot remember ever having seen an aneurism which ruptured externally, and I have seen cases where the skin looked like paper, where one was afraid to touch it.

A PHYSICIAN: I had an instructor once who said he had seen one rupture on the outside. That is the only one I ever heard of.

A PHYSICIAN: I cannot understand why he lived so long if he had that opening into the gullet.

DR. RICHARDSON: The pressure around the opening did not allow the blood to run out very fast.

DR. CABOT: Also, the bleeding lowered his blood pressure so much that the heart did not pump out more blood.

*Note by Dr. A. S. Merrill.*—In Fig. 84 the left diaphragm is held up, evidently by the adhesions. The left lung is a little less radiable than the right, no doubt because of diminished expansion due to the limitation of the diaphragm excursion, not because of the condition of the lung itself.

The shadow T, T on the right may very well be a much displaced trachea.

**Necropsy 1280**

An Irish iron worker of forty-three entered November 25, 1904. His past history was negative except for measles in childhood and erysipelas at twenty-one. He drank one to two dozen bottles of ale a week and an occasional glass of whiskey.

Two months and a half before admission he began to have sharp pain in the right breast, constant, worse at times, and increased by exertion. Once it was so severe that he almost fainted. It extended across the breast and sometimes down the left arm. He had a good deal of dyspnea and some palpitation. For four weeks he had had some cough, especially in the morning, and considerable yellow sputum. He had occasional headache and rare nosebleed. His appetite was poor. He had distress and gas after meals. Sometimes his bowels were constipated. He sometimes urinated three or four times at night, but usually after drinking the evening before. Except for advice he would have gone to work instead of coming to the hospital.

Examination showed a well nourished man. The mucous membranes were cyanotic, the conjunctivae slightly injected, the throat reddened. The apex impulse of the heart was in the sixth space

seven inches to the left of the median line. The borders of dullness were 8¼ inches to the left, 1½ inches to the right of the median line. The action was regular, the sounds of fair quality, the pulmonic second sound accentuated. Systolic and presystolic murmurs were heard at the apex, the systolic transmitted into the left axilla, nearly replacing the first sound. A harsh systolic murmur, loudest in the pulmonic area, was transmitted into the neck. There was a diastolic murmur, loudest at the fourth left interspace, heard over the precordia. (See Fig. 85.) There was marked tenderness in the fourth left interspace near the sternum. The pulse was Corrigan. The artery walls were palpable. There was a suggestion of capillary pulse in the lips. The lungs showed dullness, diminished breath sounds and numerous fine moist râles below the angle of the scapula in the right back and two inches below the angle of the scapula on the left. The fremitus was slightly diminished on the right. The expiration was somewhat prolonged throughout both backs. The liver dullness extended from the sixth rib to the costal margin. The edge was felt. There were a few medium sized glands in the groins and axillae. There was very slight edema of the ankles. The pupils and plantars were normal, the knee-jerks slight.

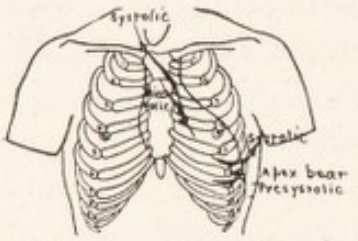



FIG. 85.—Heart signs in Case 1280.

The temperature at entrance was 99.6°, afterwards normal, the pulse 105 to 92, the respirations 28 to 21. The output of urine was 22 to 111 ounces, neutral, specific gravity, 1.025, the slightest possible trace of albumin, no sugar, rare hyalin and finely granular casts, occasional blood corpuscles. The hemoglobin was 95%, the leucocyte count 16,200.

The patient was very comfortable except for a dry harsh cough which distressed him a good deal, especially at night. He also had thoracic pain. The heart's action was good. Fluoroscopic examination of the chest failed to disclose any enlargement of the aorta. A throat consultant found no paralysis of the cords or signs of obstruction in the trachea, but the bifurcation was not seen. November 30 another X-ray examination showed increase of the shadow in the position of the thoracic aorta with a pulsating shadow just above and to the left of the heart.

The morning of December 1 while he was dressing the patient fell to the floor in a slight convulsion, became very pale, and in a few moments died. There was no marked cyanosis.

*Clinical Diagnosis (from Hospital Record).*—Acute cardiac dilatation.

Coronary obstruction.

Thoracic aneurism with relative aortic and mitral regurgitation.

*Dr Richard C. Cabot's Diagnosis.*—Syphilitic aortitis with aortic regurgitation, and blocking of the coronary arteries.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

*Anatomical Diagnosis.*—Aneurism of the ascending portion of the aorta with rupture into the pericardium.

Syphilitic aortitis.

Arteriosclerosis of the aorta.

Anthracosis of the lungs.

DR. RICHARDSON: This case occurred before the man who made the post-mortem knew definitely that he was presented with syphilitic aortitis. In going back over it years afterwards I find that the case was syphilitic aortitis. The death was due to a ruptured aneurism of the aorta.

The peritoneal cavity, including the liver, spleen, kidneys, etc., was out of the picture. There was some congestion of the kidneys.

The heart was not especially hypertrophied. The reason is apparent when we see that the main lesion was an aneurism. The amount of sclerosis in the aorta was slight, not sufficient to produce much hypertrophy and dilatation. There was slight fibrosis of the aortic cusps. Just above the aortic cusps was a frank aneurism which had ruptured into the pericardium. The aneurism, as stated in my record, was somewhere between the size of a hen's egg and a goose egg.

We made the diagnosis here in after years from the description written at the time, which shows good work on the part of the man who did it. It is perfectly apparent from the gross description, whether we have the microscopic or not, that we are dealing with an aneurism of the aorta in that particular spot where syphilitic aortitis is usually located. Aneurism itself produces no definite hypertrophy and dilatation of the heart, but syphilitic aortitis may extend down and involve the aortic cusps and produce aortic regurgitation. The cusps were slightly affected in this case.

Another point of interest in this case is arteriosclerosis. At forty-three there is usually some arteriosclerosis, and we cannot always dissociate aortitis and arteriosclerosis even after death. But the aortitis was well marked, and farther down in the aorta

there was some typical arteriosclerosis, but not in any great amount.

This man was an iron worker, and he had what is anatomically called anthracosis of the lungs. There was increase of interstitial tissue, and there was no tuberculosis. The other day we had a very marked case of pneumoconiosis in which again we were unable to find any evidence of tuberculosis. All of which goes to show that there is some argument for the group of men who feel that excessive irritation from dusts of various sorts can produce chronic pneumonitis without the aid of tuberculosis.

DR. CABOT: This was all coal dust, no iron or stone in it?

DR. RICHARDSON: There would probably be a mixture. The lungs generally were blackish in color. The posterior and inferior lobes showed sulcus-like furrows more or less outlining the lobules at the pleural surface. This was most marked in the inferior lobe of the right lung. These portions of the lung were tough and leathery to the touch. What better description do we want of chronic pneumonitis? On section the lung tissue generally showed an excessive amount of blackish pigmentation. In the posterior portions of the inferior lobes the lung tissue on section was tough and showed ill defined streaks and areas of a homogeneous smooth somewhat shining opaque black appearance, the typical picture of anthracosis, and extensive enough so that it was perfectly apparent macroscopically.

DR. CABOT: We got the main points in this case right, and I think if I had not switched off we should have put in the aneurism. I forgot about the question of aneurism. But it seems to me that second X-ray report, if we made the diagnosis of syphilis, as we did, should naturally be taken as aneurism. In the present series we find that every aneurismal tumor is syphilitic. There is only one cause. So if we can made a diagnosis of aneurism we have to make a diagnosis of syphilis. We do not confuse that with the compensatory harmless aortic enlargement which comes on in arteriosclerosis. But aneurism as such, a definite pocket-like dilatation, means syphilis and nothing else. We have had no cases of any other type, and I have seen no competent account of any other type.

The other point is the one that Dr. Richardson brought out in the condition of the lungs. In Barre, Vermont, where there is a great deal of granite cutting, careful study has been made in recent years of this question of stone dust in the lungs,—not coal dust. I think, on the basis of what they have done there, and what we have

seen here, studying it partly with the stimulus of their interest, that the question is clearly settled now. We can perfectly well have anthracosis without tuberculosis, and pneumoconiosis, the more general word for dust in the lung, without tuberculosis. We had here a very marked case in which the man died from the results of his stone-cutter's lung disease with no other important trouble. Mr. Frederick L. Hoffman, a statistician formerly with the Metropolitan Life Insurance Company, who studied the disease from a statistical point of view at Barre, gave me the other day a very interesting piece of information which has also been published. He went at the question of tuberculosis or non-tuberculosis in these stone-cutters who died of lung trouble in a very interesting way. We believe of course that tuberculosis is contagious. If then we had a lot of men dying of tuberculosis, we should have a lot of evidence that their wives and families also caught tuberculosis. He went through at great length the cases of stone-cutters who had died of lung trouble, to find out what the wives died of, and he found no evidence that the wives died of tuberculosis any more than anybody else's wives. That was a very important piece of evidence, covering a great number of cases. And he is very definite in his belief that stone-cutters' lung is not tuberculosis.

This last case shows the effects, which I did not realize at all, of another dust, coal dust. Coal dust, so far as I know, does not ever bring about death, and Dr. Richardson did not suggest in any way that the injury to this man's lungs from the coal dust shortened his life. It is a pneumonitis with interstitial increase. But so far as we can see it did the patient no harm. That is the rule. Coal-miners seem to have tuberculosis less than other people. Perhaps they get injury from the dust, but it is not proved. On the other hand people who get steel into their lungs, from scissor grinding and quartz from granite cutting, certainly do often die of the effects of this dust in the lung. Do you remember any other cases in which anthracosis caused a real pneumonitis?

DR. RICHARDSON: I do not remember. They are not very many. We had some in which we found what we believed to be tuberculosis.

DR. CABOT: This is a debated question. I am on the side of those who believe that pneumoconiosis is not tuberculosis though it may be complicated in the end by tuberculosis.

A PHYSICIAN: I wonder if the dust is protective in some way.

DR. CABOT: That is what has been often claimed about coal-dust, that if there were a few tubercle bacilli in the lung they were sealed

in by this connective tissue reaction to the coal dust, so that they became harmless.

### Necropsy 4539

A Swedish engineer of thirty-nine entered June 11, 1923. No past history was obtained except a positive one of luetic infection. The history of the present illness was given in part by the patient's physician. A year and a half before admission the patient first complained of precordial pain radiating into his back with very slight dyspnea. He went to the South in the winter for two years. He felt much more comfortable when warm, and even slept in the furnace room when working in New Hampshire. He had two stays in a hospital in Boston, one in 1922, another in 1923, about two months each time. A year before admission his left side became suddenly paralyzed. This wore off gradually in three months. About the same time he began to have pain under the upper sternum, slightly more on the right, not throbbing but steady, sharp and fairly severe, coming in attacks lasting two hours or so, induced by lifting, exertion

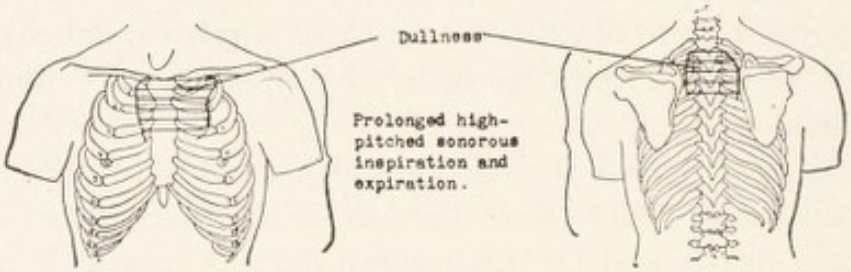



FIG. 86.

and excitement. The attacks of this pain continued until eight or nine months ago, when he began to have thumping pain under the upper sternum. For three months he had not been able to walk across the floor because of dyspnea. He also had severe pain over the precordia, brassy cough, and hoarseness. During the past two months he had had difficulty in breathing and swallowing.

Examination showed a fairly well developed and nourished man with a very distressed expression, breathing with slow stertorous inspirations and expirations. The face had a reddish cyanotic tinge. The skin was moist; during periods of extreme respiratory difficulty it was dripping with perspiraton. The cervical, epitrochlear, axillary and inguinal glands were slightly enlarged. The throat was slightly reddened. There was definite tracheal tug. The chest expansion was slightly greater on the left. The lung signs were as shown in Fig. 86. The apex impulse of the heart was in the fifth space 10 cm. to the left, 1 cm. outside the midclavicular line. The

right border of dullness was 4.5 cm., the supraclavicular dullness 13.5 cm. The sounds and action were normal. There was a very soft short systolic murmur heard to the right of the sternum in the first interspace. No thrill was felt, although there was a palpable pulsation in the first and second right and left interspaces and above the left clavicle. The pulses were somewhat prolonged in tension. There was slight sclerosis of the radials, temporals and brachials. The blood pressure was 135/85. The liver was palpable, the tip of the spleen barely palpable. Both external inguinal rings admitted the tip of the little finger. There was a small depressed penile scar. The shins were slightly roughened. The pupils and reflexes were normal.

The temperature was 98.1° to 100°, the pulse 80 to 127, the respirations 9 to 29. The urine was normal in amount, the specific gravity 1.022. There was a slight trace of albumin at the single examination, a very slight trace of bile and leucocytes. The hemoglobin was 70% to 85%, the leucocytes 16,000 to 24,000, the polynuclears 80%, the reds and platelets normal. A Wassermann was moderately positive. The non-protein nitrogen was 37.5 mgm.

The patient complained of very severe pain running through the upper chest and particularly localized in the back in the region of the third and fifth thoracic vertebrae. His respiratory distress was constant and alleviated only by frequent doses of codeia. It was thought that the general problem was to keep him comfortable, therefore antiluetic therapy was not pushed. Late in the afternoon June 13 the character of the heart sounds was poor. In the early evening he lapsed into unconsciousness. The blood pressure dropped and the heart sounds became more rapid and forcible. That evening he died.

*Clinical Diagnosis (from Hospital Record).*—Late syphilis.

Aortic aneurism.

Syphilitic heart disease.

*Dr. Richard C. Cabot's Diagnosis.*—Syphilitic aortitis.

Aneurism of the aortic arch with compression of the trachea and possibly erosion of the vertebrae.

General arteriosclerosis.

*Anatomical Diagnosis.*—Luetic aortitis.

Aneurisms of the arch of the aorta with pressure on the trachea, bronchi, and the great vessels running to and from the heart, with extensive erosion of the thoracic vertebrae.

Slight hemorrhage into the periesophageal tissues.

Gumma or old infarct of lung.

Congestion of the liver, spleen and kidneys.

Chronic pleuritis.

DR. RICHARDSON: The examination of the brain was negative. The pleural cavities showed only a few c.c. of thin pale clear fluid. There were only a few old pleural adhesions on the left. In the region of the lower part of the trachea an aneurism, to be described later, pressed upon the left lateral wall, bulging it inward rather markedly, with considerable decrease of its lumen. The pressure of the aneurism shut off to a great extent the left primary bronchus. The right primary bronchus was not definitely pressed upon. There was a small collection of reddish semi-fluid mucous material in the region of the distal end of the left primary bronchus and its branches. The bronchial glands were negative.

The tissue of the right lung was generally spongy pale red and yielded a small amount of reddish frothy fluid. In the region of the apex of the left lung the aneurism was bound by old adhesions to the upper lobe. The tissue of the upper lobe was dark reddish, a little leathery, and yielded a small amount of dark reddish fluid. The tissue of the lower lobe was pale red, a little leathery, and yielded a small amount of thin reddish frothy fluid. In the region of the upper part of the lower lobe just beneath the pleura there was a rather discrete mass about 3 cm. by 2 cm. On section it showed dirty brownish boggy to resistant tissue, a little disintegrated in its central portions. Along the region of the lower margin of this lobe there was a round firm rim about 9 cm. long and 7 mm. across. Its sections showed tissue like that in the mass just mentioned. The question that arises of course is whether it was a syphilitic lesion or not. Further examination of the tissue under the microscope showed necrotic material in the midst of chronic inflammatory tissue, but there was no good evidence of plasma cells or lymphocytic infiltration. Altogether the process suggested a syphilitic one, but we were not able definitely to state that it was.

The heart weighed 321 grams. The myocardium, valves and cavities were negative. The coronary arteries were free and negative. The circumference of the aorta in the region of the sinus was 10 cm., in the region of the junction of the ascending thoracic and the arch 11½ cm. The ascending thoracic showed only a few slight areas of fibrous sclerosis in its first portion, but beginning at a point about 5 cm. above the aortic cusps, the ascending thoracic all along up to the arch showed much fibrous change, consisting of intermingling plaques and areas of depression and thinning, but with little thickening of

the aortic wall in general. In the region of the middle portion of the arch the process ended in a fibrous rounded ridge margining the opening of a sac about 11 cm. in length and 20 cm. in circumference. In the region of the junction of the arch and descending thoracic the sac ended in a fibrous rounded ridge margining the junction. The wall of the aneurism was generally fibrous and from very thin to 2½ mm. in thickness. On its inner aspect the wall of the aneurism was lined with laminated pinkish gray-brown thrombotic material to which in turn more recent thrombotic material was adherent, and to this in turn blood clot was adherent. The wall of the aneurism rested on the spine from the second to the seventh thoracic vertebrae and eroded their bodies. In places the wall of the sac was very thin, slightly disintegrated, and through it there was infiltration of blood in moderate amount along and in the periesophageal tissues for a distance of 12 cm. The esophageal wall was intact. The pulmonary artery, veins and venae cavae were negative except that from its situation the aneurism pressed on the trachea and the great vessels running to and from the heart. The process described in the aorta faded out as the abdominal region was reached. The great branches were negative. The portal vein and radicles were negative.

You will note that this syphilitic process in the aorta began some distance above the aortic valve, so that there was no anatomical basis for an aortic regurgitation.

### Case 4494

An unmarried American girl of nineteen entered March 14, 1923, complaining of difficult respiration with loss of weight and appetite.

She had measles, whooping cough and scarlet fever (?) when young. Since these illnesses she had always been well. During the past winter she had occasional sore throats. Recently she had had occasional sharp pains on the right side in the breast region, and considerable gas. Two months before admission she lost appetite.

Fourteen months before admission she began to have "neuritic" pain in the right scapular region and later in the right arm, coming on especially at night. It was relieved by electrical treatment. At the same time she noticed a gland in the right supraclavicular region. A physician had X-rays taken which showed a mass in the right chest. She was sent to a hospital, where X-rays of the chest January 19, 1922, showed a large mass in the region of the right hilus nearly four inches up and three inches out into the lung. It had the appearance of an encapsulated gland. There was some

fibrosis throughout the lung adjacent to this. The gland involved the mediastinum so that the heart was pushed slightly to the left. The diaphragm was high on this side and the lung not well aerated. (See Fig. 87.) There was a cervical rib on the left side. She was given five radium treatments averaging 3000 millicurie hours each. August 9 the plates still showed the definite mass in the right lung without much change from the previous observation. Since the radium treatment she had coughed, feeling something in her throat. Three sputum examinations were negative. She could no longer raise sputum. November 1 the small nodule had disappeared, but there was no other apparent change. She was given an appointment

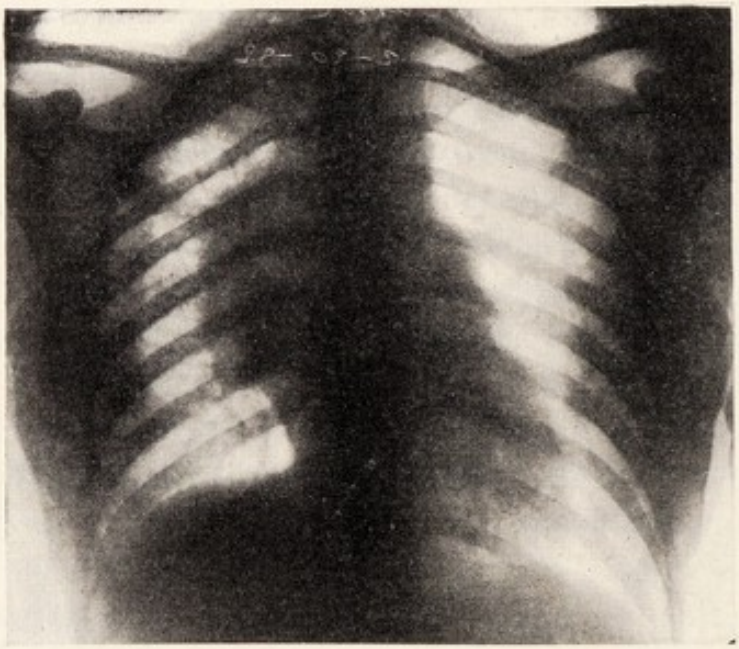


FIG. 87.—Malignant lymphoma involving the right lung, the right pleura, the pericardium, the wall of the right auricle and the superior vena cava. February 20, 1922. Shows essentially the same findings described January 19. The mass in the right hilus region has the appearance of an encapsulated gland, and involves the mediastinum so that the heart is pushed slightly to the left. There is some fibrosis throughout the lung adjacent to this. The diaphragm is high on the right and the lung not well aerated.

for deep X-ray therapy, but did not keep it. During her stay at the hospital her leucocyte count varied from 9000 to 15,000. The differential count averaged polynuclears 70%, small lymphocytes 22%, mononuclears 6%; red cells showed considerable anisocytosis and achromia. The patient entered another hospital, where she was tapped. She had some chills and moderate cough. During nine weeks she was given one X-ray treatment. She was discharged four weeks before she came to the Massachusetts General Hospital. She had been "all right" until March 4, when she began having difficulty

with breathing beginning by her losing her breath completely and becoming semiconscious. At first she was thought to be dying. After this she continued to have difficulty in breathing, especially at night. She had lost thirty-five or forty pounds.

Physical examination showed a poorly developed and nourished girl, breathing rapidly. A few small supraclavicular nodes on both sides, more marked on the right. Heart: Apex impulse and measurements not recorded. No apparent displacement. No murmurs or thrills. Beats forceful. Sounds loud. Rate rapid. Blood pres-



FIG. 88.—The same, March 16, 1923. Right side of the chest completely dull. Outline of the heart and diaphragm obscured and heart shadow considerably displaced in the opposite direction. Findings probably represent a large quantity of fluid in the right pleural cavity, obscuring the condition of the lung.

sure 124/83. Chest: Left lung expanded a little more than the right, which lagged a little. Entire right lung flat to percussion. Left hyperresonant. Tactile fremitus decreased on the right. Bronchial breathing in right chest anteriorly and above angle of scapula posteriorly. Breathing exaggerated at right base posteriorly. Egophony over root of right lung posteriorly. A metallic note to percussion in this area. No râles heard anywhere. Vocal fremitus increased over entire right lung.

Temperature 98.1°–103°. Pulse 84–150. Respiration 21–41. Amount of urine not recorded. Sp. gr. 1.030. A slight trace of

albumin, a few pus cells and occasional red cells. Blood. Hgb. 85%, leucocytes 21,900–26,500, polynuclears 80%. reds 4,056,000. Wassermann negative. Medical consultation March 16. Signs of fluid at the right base with possible cavity or localized pneumothorax. Extensive pathology in the right lung . . . Possibly fluid; may be purulent. X-ray March 16. See Fig. 88.

The patient had a great deal of respiratory difficulty, particularly at night, relieved by morphia and atropin. March 16 she felt worse than at any other time. Lying on the right side seemed to increase the dyspnea and to cause engorgement of vessels in the neck. March 19 the attacks were more severe. The next morning there was a very severe attack of dyspnea lasting twenty minutes with considerable cyanosis and difficulty of expiration. March 21 in a very severe attack of dyspnea the patient became cyanotic and died.

*Clinical Diagnosis.*—Tumor of right chest.

Empyema?

*Dr. Richard C. Cabot's Diagnosis.*—Tumors of right lung, probably lymphoma.

Empyema.

*Anatomical Diagnosis.*—Malignant lymphoma (scirrhous type) involving at the right lung, right pleura, pericardium, wall of right auricle, superior vena cava, and tracheal lymph glands.

Occlusion of superior vena cava.

Compression of trachea, bronchi and great vessels.

Bronchiectasis, right.

Empyema, right.

Congestion of liver, spleen and kidneys.

Dr. Richardson: There was a large mass involving the upper two-thirds of the right lung, also a mass between the upper portion of the lung and the trachea and bronchi. Of course the whole thing pressed on the trachea and bronchi and on the great vessels running to and from the heart. The mass pushed the heart over so that the bulk of the heart rested in the left pleural cavity. The pericardium was distended with fluid, 250 c.c. or more. Whether that had anything to do with the X-ray picture or not I do not know.

Dr. A. S. Merrill: An observation of pulsation in the mass would have been of some value.

Dr. Richardson: The left lung was normal, and there was only a little fluid in the left pleural cavity. On the right side however there was much fibrinopurulent material. There was pus here, empyema.

The greater curvature of the stomach was within 5 cm. of the pubes. The cecum rested over in the region of the left lower quadrant, bringing the appendix there. The congestion of the liver, spleen and kidneys was probably due to the pressure on the great vessels to and from the heart.

We were not permitted to examine the head.

# APPENDIX

## 1. DISSECTING ANEURISM

Utterly different from syphilitic aneurism both in etiology and in clinical development is the so-called dissecting aneurism, a complication of arteriosclerosis in the aorta, whereby a split occurs in the wall of the aorta allowing the blood to seep in and gradually to dissect off or peel off an inner layer, until finally the aorta may be transformed into a double tube.

This is to be regarded as a rare, and from a clinical point of view quite unimportant complication of arteriosclerosis in the aorta. In our series there were five cases of this lesion, which I have not included in the 1846 cases studied in detail for this book.

Clinically the lesion produces few recognizable symptoms or physical signs, though in case 3681, which follows hereafter, the X-ray picture shows a widening of the aortic shadow presumably due to the dissecting aneurism which was found at necropsy in this case.

## 2. SEPTIC OR MYCOTIC ANEURISM

I have not studied in detail and have not included in this series of cases, the three examples of septic or mycotic aneurism complicating an acute or subacute endocarditis on the aortic valves. This also has little or no clinical importance as it cannot be recognized in life. It is of course entirely separate from syphilitic aneurism both in etiology and course. None of our cases went so far as to rupture the aorta.

### Necropsy 3681. Dissecting Aneurism

A Cape Breton cook of sixty-two entered in 1911 for a fracture of the right external malleolus. With firm bandaging she made an uneventful recovery. The heart at this time was normal.

October 4, 1916, she returned, very ill, complaining of sharp pains in the back coming through to the chest. Because of her condition and a very poor memory the history is unreliable. One brother died of cancer of the stomach, one sister of tuberculosis. She had measles at twelve years, pneumonia at an unknown age. At thirty-two she had a child stillborn. At this time she had an operation, and developed a "milk leg" on the right. The leg had

remained swollen. She had had no other children. For forty years she had had attacks of bronchitis with cough every winter lasting from a week to a month. With these she had sore throat of three to four days' duration. She passed the menopause at fifty-five. In 1906 her memory began to be poor, and in 1911 it "left her entirely." At that time she had dull precordial pain localized over the heart, with palpitation, dyspnea on exertion, and orthopnea. For four years she had urinated at night. In 1911 she weighed 200 pounds, her best weight. Her usual weight was 130–170.

The evening before admission she was seized with sharp stabbing pains in the midscapular region radiating straight through to the lower part of the sternum, not to the arm, and lasting for five minutes at intervals of an hour. Dull constant aching followed these attacks. During the night the attacks became more frequent and severe, forcing her to sit up to get her breath and leaving her very dyspneic. They kept her awake all night. The morning of admission she vomited all the food eaten the day before.

Examination showed an obese woman. The apex impulse of the heart was not found. The dimensions by percussion and by X-ray are shown in Figs. 89 and 90. The action was slow and irregular. The sounds were of fair quality. The aortic second sound was accentuated. There was a presystolic roll at the apex. The

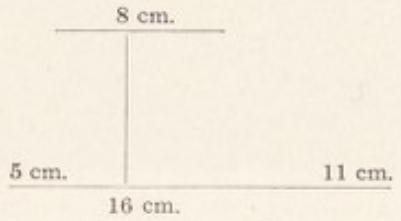



FIG. 89.—Measurements by percussion.

arterial walls were palpable. The blood pressure was 260/140 to 150/100. There was a few coarse moist râles at the base of the left lung posteriorly, and a possible pleural rub. The abdomen was negative. There was slight edema over the shins. The pupils were irregular and did not react to light. The other reflexes were normal.

The temperature was 97.1° to 100.8°, the pulse 50 to 120, the respirations 20 to 40. There was a normal amount of urine. The specific gravity was 1.016 to 1.006 There were slight traces of albumin at five of nine examinations, leucocytes at eight, granular or hyalin casts at four. Catheter specimens October 23: left ureter, sediment and culture negative; right ureter, sediment shows a rare polynuclear; no organisms; culture negative. Renal function tests

October 4 and 9 gave no specimens. October 5 in one hour a single specimen showed 20%. October 13 one specimen at the end of two hours showed 10%. A urine culture October 20 was negative. The hemoglobin was 80%. There were 15,400 to 9600 leucocytes, 85% polynuclears. A Wassermann was negative. The blood nitrogen was 62 mgm. per 100 gm. blood. The stools were negative to guaiac and the microscope at two tests. The fundi were normal. A genito-urinary consultant reported after cystoscopy, "The bladder is essentially normal; slight redness near the base may be the evidence of a slight cystitis which would cause a mild pyuria. The ureters look normal and have no obstruction. Both kidneys are active. Kidney X-ray might help; if negative there is certainly nothing to justify treatment."

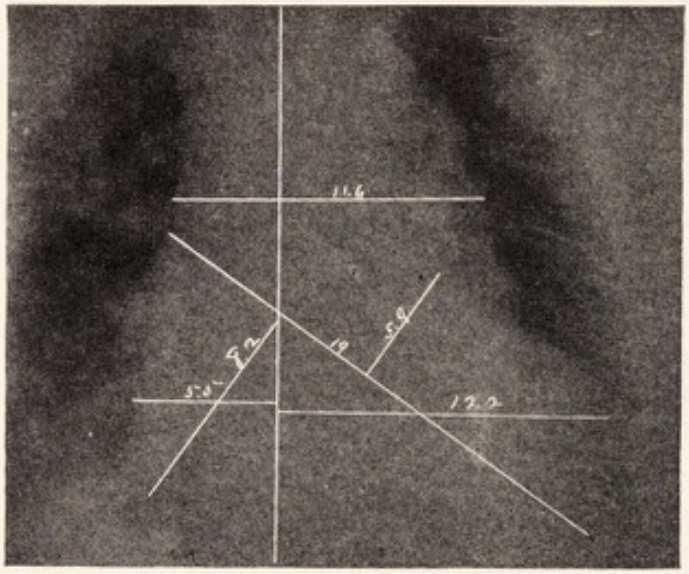


FIG. 90.—Arteriosclerosis with dissecting aneurism.

The pulse was slowed by the digitalis and the heart sounds were louder. The patient had no more severe attacks of precordial pain, but the cardiac condition improved very slowly. There was almost constant precordial ache, with radiation down the left arm. Following the cystoscopy there was slight cystitis. November 9, 1916 she was discharged relieved.

January 13, 1917, she began to vomit after meals and to have increased distress on movement. January 17 she was suddenly seized with agonizing pain over the sternum and through to the back at the scapular level. This persisted without relief. She reentered the hospital five hours after its onset, too ill for examination. The temperature was 98°, the pulse 80, the respiration 48, the hemoglobin 80%. There were 12,000 leucocytes, 88% polynuclears. The

evening of admission she had a sudden very sharp pain in the region of the heart, and died in about ten minutes.

*Interpretation of X-ray.*—Aneurism of the descending aorta.

*Clinical Diagnosis (from Hospital Record).*—Arteriosclerosis.

Cardiac decompensation.

Angina pectoris.

*Anatomical Diagnosis.*—Arteriosclerosis.

Dissecting aneurism of the aorta with great hemorrhage into the peri-aortic and retropleural tissues.

Subacute pericarditis.

Arteriosclerotic nephritis with amyloid infiltration.

Hypertrophy and dilatation of the heart.

Organized fibro-calcareous thrombosis of the right external iliac vein.

Slight hemothorax.

Slight hemopericardium.

Chronic pleuritis.

Defective closure of the foramen ovale.

Emphysema of the lungs.

Dr. Richardson: The heart weighed 606 grams (normally 200–300), and the kidneys 107 (normally 200–400). There was some arteriosclerosis of the coronary arteries. The miscroscopical examination showed the condition to be one of arteriosclerosis and not of syphilis.

## CHAPTER IV

## HYPERTENSIVE HEART DISEASE

### I. DEFINITION

A group of cases believed to be characterized during life by a more or less permanent hypertension and showing a hypertrophied and dilated heart, without valvular disease or chronic pericarditis. Nephritis and arteriosclerosis may be present or absent.

This definition brings together all the hearts in this series which are enlarged but which show no mechanically obstructing lesions in valves or pericardium.*

**The Relation of Nephritis to Cardiac Hypertrophy.**—The nephritic and the non-nephritic hypertrophies are here lumped together: (1) because it is in some cases impossible to distinguish them in life, and (2) because at necropsy the state of things in every part of the body, except the kidney, may be identical in the nephritic and in the non-nephritic cases. There may be the same degree and type of cardiac enlargement, the same dropsy (or lack of it), the same hypertension as in life. "Uremic" or infectious phenomena may be present or absent in both groups; in age and sex they are not conspicuously different.

This is not to assert that the presence of nephritis makes no difference, but merely to acknowledge ignorance of *what* difference it makes. On the average, as will be shown presently, the hearts are larger in the nephritic than in the non-nephritic group, but to this rule there are many exceptions. For example, 18 of our 267 cases of chronic nephritis showed no cardiac hypertrophy at all. In 23 others the heart weighed under 450 grams, and in 17 of these 23, under 400 grams. On the other hand, in the non-nephritic group there were hearts weighing 870, 793, 775, 750 grams, and many others of large size.

**What Has Arteriosclerosis to Do with It?**—The place of arteriosclerosis, like that of nephritis, in relation to hypertensive heart disease, is one that I make no attempt to settle on the basis of this series of observations. But though I think that few of us can doubt

* In this definition other rare causes of hypertrophy and dilatation, such as pernicious anemia and hypoplastic aorta are also assumed to be absent.

that nephritis (with hypertension) is closely linked with the development of cardiac hypertrophy, one cannot say the same of arteriosclerosis. Taking first the simple facts of association or correlation: in 267 cases of nephritis there was hypertrophy and dilatation in 249, or 93%. No other so-called "cause" for hypertrophy and dilatation has so high a correlation. Valvular disease, though not causing hypertension, shows 184 cases or 83% with hypertrophy and dilatation, and 36 without it. Chronic pericarditis shows 89 with hypertrophy and dilatation and 25 without it, a 78% correlation.

Arteriosclerosis, on the other hand, occurred 668 times with hypertrophy and dilatation, and 283 times without hypertrophy and dilatation, a correlation of only 64%.

TABLE 81.—CORRELATION OF CARDIAC HYPERTROPHY AND DILATATION WITH 1552 CASES OF VARIOUS DISEASES

| | | |
|---|---|---|
| Nephritis | 93% correlation in | 267 cases. |
| Valve Lesions | 83% correlation in | 220 cases. |
| Chronic pericarditis | 78% correlation in | 114 cases. |
| Arteriosclerosis | 64% correlation in | 951 cases. |

This is in close accord with Ophuls' figures.* He found 35% of cases with marked arteriosclerosis but without cardiac enlargement. Our own percentage is 36. Our figures, however, refer to varying grades of generalized arteriosclerosis, including a few cases of slight degree. In 132 cases of especially marked and extensive arteriosclerosis (with this word mentioned first in the anatomical diagnosis) 106 or 80% showed cardiac hypertrophy and dilatation, and 20% did not.

*Arteriosclerosis by itself*, uncomplicated by nephritis, by valve lesions or pericarditis, occurred 248 times with hypertrophy and dilatation, a correlation of only 39%, and 383 times without it. Whereas nephritis alone occurred 67 times with hypertrophy and dilatation and only 18 times without it, a correlation of nearly 79%, or double that of arteriosclerosis. (See also pages 424 and 436.)

In this chapter I have merged all types of nephritis: first because many of those whose nephritis was called "acute" or "subacute" at necropsy gave every *clinical* evidence of having had the nephritis for months or years; and secondly, because the terms acute and subacute

* Archives of Internal Medicine, Vol. 9, page 156 (1912).

are used in our pathological records to denote certain histological pictures and not with any definite *time* limits in mind. In the 66 cases of subacute nephritis occurring in 4000 necropsies, 21 were associated with cardiac enlargement, and 45 were not associated with cardiac enlargement. In 113 cases of acute nephritis 53 had hypertrophy and 60 none.

The only type of nephritis that seems to belong in a class by itself, so far as the connection with cardiac enlargement is concerned, is the *amyloid* variety, long recognized to be connected loosely, if at all, with heart changes. There are but 21 cases designated as amyloid nephritis in our necropsy records. Three of these had also cardiac hypertrophy, 18 did not. It is well known that this type of nephritis is not ordinarily associated with hypertension.

The best working hypothesis then seems to be that whatever influence produces a chronic hypertension will produce as a result cardiac enlargement. We have then, beside the local mechanical obstacles—valvular disease and adherent pericardium—only one other common cause for cardiac enlargement, namely, *hypertension*, however produced. Nephritic hypertension seems a clearly established subtype. Whether arteriosclerotic hypertension is a reality or not seems quite open to question, on the basis of these figures and in accordance with the observations of others. Certainly there are many cases of hypertension without arteriosclerosis or any other supposed "cause" (Allbutt's "Hyperpiesia"). All of these, so far as I know, are associated with cardiac hypertrophy. Perhaps whatever causes this hypertension may also result, if long continued, in arteriosclerosis, in nephritis, or both. The term "hypertensive heart disease" then, expresses the belief that almost all the enlarged hearts not due to valvular disease or pericardial adhesions, develop as an organic response to vascular hypertension, however produced. I have never known a case of long-standing hypertension* without enlarged heart. If such cases can be proved to exist, the hypothesis and the terms here used must be modified. Until then they seem to express best the present state of our knowledge.

Discussion of certain rarer causes of cardiac enlargement follows in the next section.

* Temporary hypertension due to muscular work, to emotion or to intracranial disease (e.g. cerebral hemorrhage) certainly does not enlarge the heart. H. Batty Shaw (*Hyperpiesia and Hyperpiesis*, Oxford Press, 1922) finds enlargement of the heart in all cases of hypertension lasting more than four days.

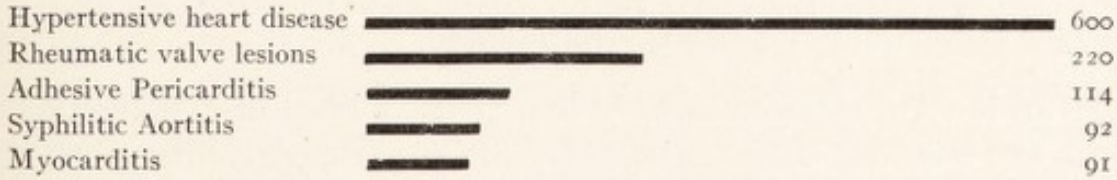
## II. HYPERTENSIVE CARDIOVASCULAR DISEASE

*Hypertrophy and Dilatation of the Heart is the Commonest of All Diseases of the Heart.*

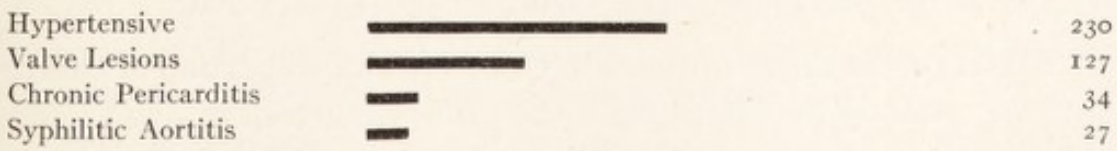
Although the boundaries of the group here described are not accurately determined, it is reasonable, I think, to include in it all the necropsied cases showing a hypertrophied and dilated heart associated with nephritis, with arteriosclerosis, with both, *or with neither*, but without valvular disease, chronic pericarditis, and any other recognized cause of cardiac enlargement. Thus limited *the group includes* 599 *cases*. There are about 50 other cases, possibly of this type but here excluded, because other more prominent lesions carry them into other groups. For instance, among the 80 cases of *uncomplicated acute pericarditis** there were 11 with enlarged hearts. Similarly there are 19 cases of *uncomplicated acute endocarditis* with cardiac hypertrophy and dilatation not accounted for; 17 of chronic nondeforming endocarditis, 4 of myocarditis. All these *may* have been in fact cases of chronic hypertension with a harmless local thickening of a valve, a trifling myocardial scar, or with terminal infection of endocardium or pericardium. If they were all added, the number of cases here analyzed would be 649. Roughly, then, we deal in this chapter with from 600 *to* 650 *cases* as compared with 220 cases with valve lesions, 114 with chronic pericarditis, 94 of syphilitic aortitis, 91 of myocarditis.

This overwhelming excess of hypertensive cases is entirely in accord with clinical experience.

TABLE 82.—HEART DISEASE MANIFEST OR LATENT




MANIFEST HEART DISEASE




* 185 cases of acute pericarditis in all, but 105 of these are complicated by other lesions, some of which are often linked with hypertrophy, and so are not considered here.

### III. LATENT VS. MANIFEST CASES OF HYPERTENSIVE HEART DISEASE

About 230* of these 600 cases were of the *manifest* type, i.e., associated with dropsy and chronic passive congestion at necropsy and without any obstruction, such as mediastinal or peritoneal neoplasm, to account for the dropsy. The remaining 369 were symptomless and represent the cases of enlarged heart discovered at necropsy after a death due chiefly to neoplasm, infection, violence, toxemia, or angina pectoris.

Compare now these 230 manifest or dropsical cases with the figures in other types of heart disease known to produce dropsy.†

| | |
|---|---|
| Among the 220 cases with valve lesions | 127 were dropsical |
| Among the 114 cases with chronic pericarditis | 34 were dropsical |
| Among the 92 cases with syphilitic aortitis | 27 were dropsical |
| Total | 188 |

It appears then that among 418 cases (188 plus the 230 mentioned above ) of manifest dropsical heart disease, 230, or 55%, are of the hypertensive type, while only 188, or 45%, are of any other type.

These figures justify our calling cardiac hypertrophy and dilatation a disease rather than, like fibrous myocarditis, a mere post-mortem finding.

Cardiac hypertrophy and dilatation is often latent, but so are all the other types of heart disease, as the following table shows.

TABLE 83.—LATENCY OF HEART DISEASE

| | | |
|---|---|---|
| Hypertrophy and dilatation latent in | 61% | Congestive death in 39% |
| Valvular disease latent in | 38% | Congestive death in 62% |
| Syphilitic Aortitis latent in | 73% | Congestive death in 27% |
| Chronic Pericarditis latent in | 70% | Congestive death in 30% |

### AGE INCIDENCE

In the 230 *manifest* cases of hypertensive heart disease the average age was forty-nine years; Batty Shaw's average is fifty years; in our inactive or latent cases, fifty-three years.

### SEX

Males predominate in the active cases in the proportion of twelve to five. Among the latent cases the ratio is fourteen to five.

* There are probably twenty or thirty more cases belonging with these, but so much entangled in other groups that I cannot definitely distinguish them.

† Myocarditis is not here included since I am not convinced that by itself it is a cause of dropsy.

### HEART WEIGHT

The average heart weight in active cases is 474 grams, in the latent cases 433 grams.

## IV. THE ETIOLOGY OF CARDIAC ENLARGEMENT

(a) *Work-hypertrophy.*—The data of this study are consistent with the belief that all cardiac enlargement is a *work-hypertrophy*, if we mean by this the heart's response to the need to do more work so as to maintain the circulation despite the burden of valvular lesions tight pericardial adhesions or vascular hypertension. To keep up the circulation in the face of such obstacles demands more work, more muscle, i.e. a hypertrophy of the heart. In this sense the causes of cardiac hypertrophy are, so far as we know, the causes which throw continuous extra work upon the heart over a period of months or years.

But the term "work-hypertrophy" has been used also to mean an enlargement supposedly caused by the individual's *intermittent* muscular exertions in work or sport. Of such a cause this series gives no evidence although some evidence has been adduced for it by others. The hearts of "Marathon runners" repeatedly examined, year by year, after the annual 25 miles run ending at Boston, Mass., have never shown evidence of hypertrophy or of dilatation. Indeed the X-ray measurements have shown apparently a slight diminution in the size of the heart shadow after the race. But C. Ward Crampton in his book on *Physical Exercise for Daily Use* refers to cardiac hypertrophy in six-day bicycle racers. Possibly the more *continuous* nature of this exercise gives it a special effect on the heart.

In the Massachusetts General Hospital series there was no evidence that the hearts of athletes or of men who had done very heavy work during life were any larger than the hearts of sedentary people. Men of large frame have large hearts and men of small frame have small hearts but without any discernible relation to their daily occupations.

(b) *Alcohol* also played no discernible part in the cardiac hypertrophies of this series. Heavy drinkers, whether of beer or whiskey, showed no larger hearts than temperate people.

(c) *Emphysema and chronic pneumonitis* have often been supposed to produce a predominantly right-sided cardiac hypertrophy and dilatation. But we could find evidence of this in only one case (3191) in which a right-sided hypertrophy and dilatation was

associated with emphysema. Even then the heart weighed only 294 grams and there was no chronic passive congestion.

(d) *Syphilis* has, so far as our observations go, no effect on the size of the heart unless syphilitic aortitis with an incompetent aortic valve is present.

There remain the long-familiar causes or associates of cardiac hypertrophy, such as valvular disease, chronic nephritis and pericardial adhesions. The frequency of these associations has been carefully studied in this series of cases, with the following results:

TABLE 84.—CARDIAC ENLARGEMENT AND THE ASSOCIATED (POSSIBLY CAUSAL) LESIONS

| Lesions | Total Sufferers with this Lesion | No. showing Cardiac Hypertrophy at Necropsy | Percent showing Hypertrophy |
|---|---|---|---|
| Rheumatic Valvular Disease | 220 | 184 | 83 |
| Chronic Pericarditis | 114 | 89 | 78 |
| Nephritis* | 377 | 275 | 73 |
| Syphilitic Aortitis | 92 | 63 | 68 |
| Pernicious Anemia | 23 | 22 | 95 |
| Leukemia | 9 | 9 | 100 |
| Hypoplastic Aorta | 19 | 8 | 47 |
| Goiter† | 10 | 6 | 60 |
| Arteriosclerosis, with arteriosclerotic degeneration of kidneys | 142 | 93 | 65 |
| Chronic non-deforming Endocarditis | 235 | 144 | 61 |
| Myocarditis‡ | 99 | 83 | 83 |
| Acute Endocarditis | 180 | 88 | 49 |
| Acute Pericarditis | 185 | 102 | 54 |
| Arteriosclerosis | 1051 | 668 | 63 |

* 198 chronic, 66 subacute, 113 acute.
† Of these, 6 were exophthalmic, 3 toxic adenoma, 1 doubtful.
‡ Including myocardial abscess (15 cases) and myocardial infarct (20 cases).

1. Among the 1846 individuals harboring the 4037 cardiovascular lesions which this book studies, 1209 were recognized by the pathologist as having some *enlargement of the heart.**

But there is so much overlapping among some of these items that without explanation they are quite misleading. For instance, out of the 668 cases of arteriosclerosis, only 248 were uncomplicated by other lesions very possibly capable of producing hypertrophy and dilatation. Out of 83 cases of myocarditis only 4 were uncomplicated!

Let us therefore separate out the uncomplicated cases of each lesion, i.e., those *in which hypertrophy and dilatation is associated with only one other recognized cardiovascular lesion.* We then find in 508 cases the following figures:

TABLE 85.—CARDIAC HYPERTROPHY IN UNCOMPLICATED ("SINGLE") CARDIOVASCULAR LESIONS

| | | | | |
|---|---|---|---|---|
| Group I. | With Hypertension | Hypertrophy in | 12 out of 12 | or 100%. |
| | With Leucaemia | Hypertrophy in | 7 out of 7 | or 100%. |
| | With Pernicious Anemia | Hypertrophy in | 12 out of 13 | or 92%. |
| | With (a) Rheumatic Valvular Disease | Hypertrophy in | 69 out of 86 | or 80%. |
| | With (b) Syphilitic Valvular Disease | Hypertrophy in | 17 out of 20 | or 85%. |
| | With Nephritis | Hypertrophy in | 67 out of 78 | or 85%. |
| | With Goitre | Hypertrophy in | 4 out of 6 | or 66%. |
| | With Chronic Pericarditis | Hypertrophy in | 16 out of 28 | or 57%. |
| Group II. | With Arteriosclerosis* | Hypertrophy in | 248 out of 513 | or 46%. |
| | With Myocarditis | Hypertrophy in | 4 out of 10 | or 40%. |
| | With Acute Endocarditis | Hypertrophy in | 19 out of 54 | or 35%. |
| | With Chronic non-deforming Endocarditis | Hypertrophy in | 17 out of 56 | or 30%. |
| | With Hypoplastic Aorta | Hypertrophy in | 5 out of 15 | or 33%. |
| | With Acute Pericarditis | Hypertrophy in | 11 out of 80 | or 13%. |
| | | | 508 | |
| | Add the cases in which Hypertrophy and dilatation is present alone and without any of these "causes"..... | | 154 | |
| | | | 662 | |

* In 46 the arteriosclerosis was very marked and was written first in the anatomical diagnosis. 40 of these 46 cases, or 8%, 9 had hypertrophy and dilatation. (See below.)

* Of these 1088 are recorded as showing hypertrophy and dilatation and 121 as showing hypertrophy alone. This distinction, however, is of very little importance here, since the single word "hypertrophy" has often been used in our records to cover the same facts as the longer phrase "hypertrophy and dilatation."

Among 662 cases of hypertrophy and dilatation, then, there were (1) 154 with no anatomical "cause" discovered and (2) 248 with no "cause" except arteriosclerosis, which (if it is a "cause" at all) is active in only 46% of cases.

**Cardiac Hypertrophy and Arteriosclerosis.**—No one, I take it, would suppose that uncomplicated acute pericarditis, though associated in 13% of cases with hypertrophy and dilatation, is a real 'cause" for hypertrophy and dilatation, except possibly in a few of the prolonged, (i.e., nearly or quite subacute,) cases, in which a tough, though plastic exudate might conceivably bring increased work upon the heart. Similar speculations are justified regarding all the members of group II in Table 85. In a few cases of "acute" endocarditis, the lesions may have lasted long enough and interfered with valve-function sufficiently to increase the heart's work and so to bring about hypertrophy. A few cases of chronic endocarditis believed by the pathologist to involve *no* valvular deformity, may in fact have produced *some* deformity and so have acted as valve lesions to burden the heart. Most myocardial fibrous scars are so small that (as the figures show) they do not embarrass the heart's action enough to produce hypertrophy and dilatation. But sometimes the damage might conceivably be so extensive (e.g. near the apex) as to call out hypertrophy in the rest of the organ.

We know that hypertension can by itself cause cardiac hypertrophy, even in the absence of nephritis and arteriosclerosis. It is therefore entirely possible that in most of the cases of group II (or in many of them) the hypertrophy and dilatation may be due to a chronic hypertension, and that therefore the association with arteriosclerosis or with any other of the lesions set down in this group is one of coincidence and not of cause and effect.

**What Degree of Arteriosclerosis Is Concerned Here?**—But if we confine ourselves to arteriosclerosis of the extremest grade, that actually *was* associated with hypertrophy and dilatation in 40 out of the 46 uncomplicated cases. These 46 cases are selected from the whole group of 1202 arteriosclerotics by two marks (a) because they show so marked and widespread a type of the disease that the words "arteriosclerosis" was written first in the pathologist's summary of his anatomical diagnosis and (b) because no other known cause for hypertrophy and dilatation (such as nephritis, valvular disease and pericarditis) was present. They present, therefore, a type of *pure* yet extreme generalized arteriosclerosis and, as has been said, almost 90% of them showed hypertrophy and dilatation. The

average age of these individuals was 58 years and three-fourths of them were males. The degree of hypertrophy, however, was not marked, the average cardiac weight being 480 grams, which for men of 58 is not an extreme grade of enlargement. Moreover there remain 467 cases of general though not extreme arteriosclerosis in only 208 of which, or 44%, was there any hypertrophy at all. Exactly where these cases of arteriosclerosis belong between the "slight" and the "extreme," we have no way to determine. They are not confined to a few spots, such as the aortic arch; they all represent a generalized process. But precisely *how* generalized they were could not be ascertained without a minute dissection of many peripheral arteries, which was not undertaken.

In the 208 cases of arteriosclerosis with hypertrophy and dilatation, (the 44% above referred to) *the average heart weight was 429 grams.* In contrast with this, take the 154 cases of "pure" hypertrophy and dilatation without arteriosclerosis or any other known "cause." The average weight is 384 grams, i.e., 45 grams difference. This is a considerable difference in the average, although there were in this group hearts weighing 694, 618, 564, 559, 534, 518. But the contrast is here greater than it would be but for leaving out the cases dying with hypertrophy and dilatation and various terminal lesions, such as acute pericarditis, essentially the same condition with a different end. In these (9 cases) the heart averaged 471 grams in weight.

In a further attempt to estimate how much effect, if any, is produced upon the heart by arteriosclerosis alone, I have separated out the cases of hypertrophy and dilatation *plus* nephritis alone, and compared them with cases of hypertrophy and dilatation *plus* arteriosclerosis *plus* nephritis. In 53 cases of hypertrophy and dilatation with nephritis alone, the heart weight averaged 453 grams. In 65 cases of hypertrophy and dilatation *with nephritis and arteriosclerosis*, the average heart weight was 487 *grams*, a difference of 34 grams.

Further graphic representation of the nephritic group of cases is as follows:

TABLE 86.—HYPERTROPHY AND DILATATION PLUS NEPHRITIS ALONE

| Heart weights | Number and type of cases |
|---|---|
| 250–300 grams | = 1 (subacute) |
| 301–350 grams | = 9 (3 = acute, 6 chronic) |
| 351–400 grams | = 12 (1 = acute)<br>(2 = subacute)<br>(9 = chronic) |
| 401–450 grams | = 7 (chronic) |
| 451–500 grams | = 6 (1 = subacute, 5 chronic) |
| 501–550 grams | = 8 (1 = subacute, 7 chronic) |
| 551–600 grams | = 4 (1 = subacute, 3 chronic) |
| 601–650 grams | = 2 (chronic) |
| 651–700 grams | = 1 (chronic) |
| 740 grams | = 1 (chronic) |
| 750 grams | = 2 (1 = subacute 1 chronic) |
| | 53 |

4 = acute nephritis
7 = subacute
42 = chronic

53

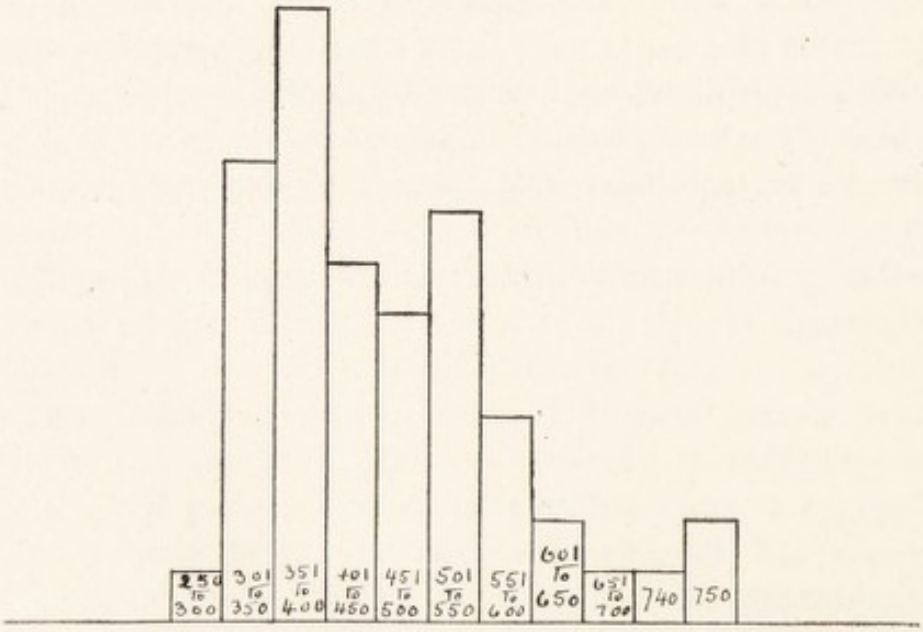



TABLE 87.—HEART WEIGHTS IN UNCOMPLICATED NEPHRITIS

TABLE 88.—HYPERTROPHY AND DILATATION PLUS NEPHRITIS PLUS ARTERIOSCLEROSIS

| Heart weights | Number and type of cases |
|---|---|
| 250–300 grams | = 1 chronic |
| 301–350 grams | = 6 chronic |
| 351–400 grams | = 11 (1 = subacute) (1 = acute) |
| 401–450 grams | = 9 (1 = acute) (1 = amyloid) |
| 451–500 grams | = 8 chronic |
| 501–550 grams | = 11 chronic |
| 551–600 grams | = 10 chronic |
| 601–650 grams | = 3 (1 = acute) |
| 651–700 grams | = 2 chronic |
| 701–750 grams | = 3 chronic |
| 751–800 grams | = 1 chronic |
| | 65 |

| | |
|---|---|
| Acute | 3 |
| Subacute | 1 |
| Chronic | 60 |
| Amyloid | 1 |
| | 65 |

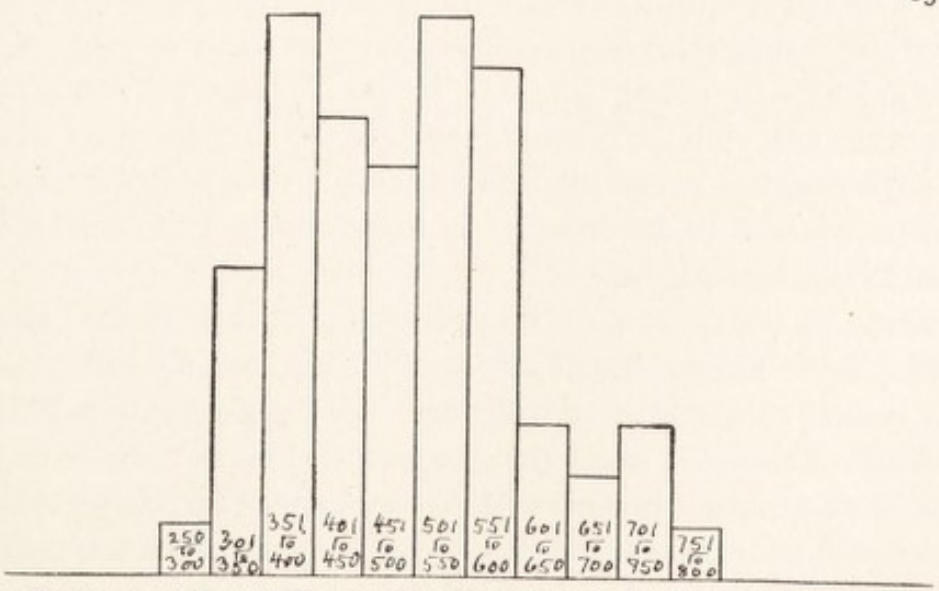



TABLE 89.—HEART WEIGHTS IN NEPHRITIS PLUS ARTERIOSCLEROSIS

It appears then on the evidence of pure association that *arteriosclerosis is only a 34 gram factor in the total sum of the factors, linked with cardiac hypertrophy.* Of course it is possible to read the whole matter in the opposite direction, and to suppose that arteriosclerosis is not a cause of cardiac hypertrophy at all. We may say that it is *one result* of hypertension and that cardiac hypertrophy is *another* result. And we can suppose that hypertension, arteriosclerosis, and enlarged heart are all three the results of some unknown (perhaps toxic) cause.

**"Arteriosclerotic Degeneration of the Kidney" in Its Relation to Hypertrophy and Dilatation of the Heart.**—By this term is meant foci of arteriosclerosis in the kidney, perhaps sufficient to diminish its function to some degree but not sufficient to constitute a nephritis. The kidney has its share of the general arteriosclerosis of the body, but no more.

Among 142 cases so diagnosed at necropsy, in our series 93 (or about two-thirds) were associated with enlargement of the heart. In these 93 there were other "causes" for hypertrophy in 34, leaving 59 (or 41%) in which there was no obvious cause for the enlargement except the general arteriosclerosis, of which the renal lesion formed part. But general arteriosclerosis alone (without renal degeneration) occurred in 248 cases (or 48%) with hypertrophy and dilatation, and in 265 cases without it, in a total of 513 cases, so that there is no evidence that the renal degeneration alone added anything to the cardiac hypertrophy.

### V. HYPERTROPHIED AND DILATED HEARTS OCCURRING WITHOUT NEPHRITIS OR ARTERIOSCLEROSIS, IN YOUNG PERSONS

We are apt to think that hypertension, without nephritis, is confined to persons at or past middle life. Certainly we do not often see it clinically in young people. Hence if hypertension is (as I believe) the cause of most of the cases of cardiac hypertrophy without obvious anatomical cause, we should expect the cases of hypertensive heart disease to be mostly in persons past the fortieth year. This turns out to be true.

A study of our 402 cases of hypertrophy and dilatation without nephritis, valve lesions or chronic pericarditis, reveals but ten individuals under 40 with any considerable degree of hypertrophy. The details of these 10 cases are shown in Table 90.

*One* of these cases, however, (No. 2086) is enough to prove (if the observations are accurate, as I believe they are) that even at 30

TABLE 90

| Case No. | Necropsy number | Age & sex | Heart's weight grams | Blood pressure | Cause of death | Remarks |
|---|---|---|---|---|---|---|
| 1 | 606 | 37-M | 618 | ..... | Pneumonia | No arteriosclerosis or nephritis |
| 2 | 2890 | 30-M | 564 | 120 systolic two days before death | Chronic passive congestion | No arteriosclerosis or nephritis |
| 3 | 650 | 36-M | 535 | ..... | Sepsis | No arteriosclerosis or nephritis |
| 4 | 2086 | 30-M | 520 | 200 systolic two mos. before death | Chronic passive congestion | No arteriosclerosis or nephritis |
| 5 | 3000 | 39-M | 518 | 105/80 one day before death | Sepsis | Chronic pneumonitis |
| 6 | 2201 | 34-M | 501 | ..... | Crushed | |
| 7 | 1972 | 36-M | 489 | ..... | Poisoning | |
| 8 | 3940 | 32-M | 480 | 138/78 three days before death from pneumonia | | |
| 9 | 506 | 35-M | 479 | ..... | Cirrhosis | No. artsclerosis or nephritis |
| 10 | 1924 | 35-M | 465 | ..... | Pneumonia | |

a man may have a hypertension of 200 mmHg. of systolic blood pressure without nephritis, arteriosclerosis or any other known cause. The other cases *may* have been of the same type, for although three of them showed no hypertension, these measurements were taken so near the time of death as to be without great significance.

## VI. THE LARGEST HEARTS

1. In Table 91 are shown the measurements of fifteen of the largest hearts in this whole series of 1906 cases. As we study this table, certain facts become evident. The first of these is that *chronic pericarditis with mediastinitis produces the largest hearts that are to be found.* In the largest of all the hearts of this series, and in four others very close to it in size, chronic pericarditis was the *only* recognized causative factor. In one other case, (No. 2 in this table) valvular disease and subacute glomerulonephritis were also present and may well have contributed to enlarge the heart. But the outstanding fact remains that *in the five largest hearts of this entire series*

*chronic pericarditis was the sole cause in four and one of the causes in the fifth.* In a sixth case chronic pericarditis was a factor along with valvular disease. Although this heart is set down as the sixth in our series it should by right belong much higher up, since it occurred in a boy of eleven, while all the others in this group of fifteen occurred in persons who had reached the limit of their growth. For a boy of eleven a heart weighing 799 grams represents probably as great an enlargement as those at the top of this table.

Besides the cases thus far mentioned there was one more, No. 7 in this series, in which chronic pericarditis was also an important factor. Hence chronic pericarditis apparently played a part in enlarging *seven out of fifteen* of the largest hearts in this total series and was the *only known factor in four out of the five largest.*

2. In old times one used to say that the *cor bovinum*, standing for the largest type of heart known, was due primarily to *pure aortic regurgitation.* This cause *appeared as the second in our list.* If we take the *average* weight of the hearts with syphilitic aortitis and aortic regurgitation, this cause for cardiac hypertrophy stands first and rheumatic stenosis second as compared with the average weight of the hearts enlarged by other causes. But here we deal with individuals not averages (see Table 92). The 10th, 11th and 12th hearts among the 15 largest of our series were due to syphilitic aortitis with aortic regurgitation and aneurism, but without aortic stenosis.

3. Two very large hearts—the 13th and 14th of this series—showed no anatomical "cause" for this enlargement unless general arteriosclerosis is such a cause. But according to the interpretation which seems to me most plausible this heart represents the result of "primary" hypertension, the arteriosclerosis being an associated but not in all probability a causative factor. Arteriosclerosis was also present in association with two other of the cases of very great cardiac enlargement already mentioned (Nos. 8 and 9).

4. *Mitral stenosis uncomplicated* by a lesion at any other valve does not appear in this series of very large hearts. For comparison I add at the bottom of it, the largest heart of this type; weight 750 grams. The *multiple* valvular lesions of rheumatic origin in this series produced 6 hearts weighing over 750. But in general one may say that in rheumatic heart disease the heart does not reach an extreme degree of enlargement unless the aortic valve is involved.

. . .

It has repeatedly been suggested by writers on cardiac disease that *youth* is an important adjuvant to disease in producing cardiac

hypertrophy. From our table this observation seems to have only a very limited amount of truth. Thus among the possessors of the 15 largest hearts that I have studied, only six were under the fortieth year at the time of death, four were between forty and fifty, three were between fifty and sixty, while one was 78. Youth does not appear to be much of a factor. If we examine the ages of those at the very top of the list it appears that five out of the first six were below the age of thirty-two. But all these patients were suffering from *chronic pericarditis*. It appears therefore to be true that *when chronic pericarditis (the most powerful of all the hypertrophy-producing causes that are known) is in evidence, it acts most strongly in young people*. But this may merely mean that the severe cases die young. Case 4 is obviously an exception to this remark. The *average* age in the series of fifteen patients with huge hearts is forty years.

TABLE 91.—THE LARGEST HEARTS

| No. | Necropsy number | Heart's weight | Age and sex | Remarks |
|---|---|---|---|---|
| | | | | **Chronic Pericarditis** |
| 1 | 266 | 1328 | 31-M | Chronic pericarditis with mediastinitis. Rheumatism 4, 6 and 8 years ago. Double murmur over precordia. Aortic valve 7 cm. Chronic passive congestion. |
| 2 | 2550 | 1273 | 30-M | Chronic pericarditis with mediastinitis. Rheumatism at 7 and 15. Frequent tonsillitis. Aortic orifices enlarged. Acute endocarditis (aortic). Subacute glomerulonephritis. Chronic deforming aortic and mitral. Blood pressure 205/almost zero. Double murmur everywhere. Chronic passive congestion. |
| 3 | 3345 | 1158 with pericardium | 19-M | Chronic pericarditis with mediastinitis. Rheumatism 2x. Valve orifices enlarged. Chronic non-deforming endocarditis aortic. Double murmur. Corrigan and pistol shot. |
| 4 | 1975 | 1150 | 52-M | Chronic pericarditis. Rheumatism 2x. Valve orifices enlarged. Systolic at apex (loud). Acute endocarditis, aortic, mitral and tricuspid. Chronic passive congestion. |
| 5 | 3290 | 1140 (1205 with pericardium | 31-M | Chronic pericarditis with mediastinitis. Chronic passive congestion. Valve orifices enlarged. Chronic non-deforming endocarditis. Double murmur, Aortic second loud and ringing. Blood pressure 180/120. Diagnosis made in life. |
| 6 | 1063 | 799 | 11-M | Pick's syndrome. Rheumatism at 5. Double murmur. Chronic non-deforming endocarditis, mitral, aortic and tricuspid. |
| | | | | **Chronic Pericarditis with Aortic Stenosis** |
| 7 | 3345 | 1000 | 19-M | Dyspnoea 4 years. Chronic pericarditis, mediastinitis and pleuritis. Valoular disease. |
| | | | | **Aortic Stenosis Alone** |
| 8 | 2603 | 900 | 49-M | Rheumatism 4x. Angina pectoris; sudden death without stasis. |
| 9 | 3610 | 872 | 55-F | Orifice admits only scissor blades. Slight arteriosclerosis. Blood pressure 152/115. |

TABLE 91.—THE LARGEST HEARTS.—(*Continued*)

Aortic Regurgitation with Syphilitic Aortitis

| | | | | |
|---|---|---|---|---|
| 10 | 2936 | 1000 | 50-M | Aneurism also. |
| 11 | 3930 | 950 | 49-M | Aneurism also. |
| 12 | 2734 | 940 | 47-M | Aneurism also. |

General Arteriosclerosis

| | | | | |
|---|---|---|---|---|
| 13 | 1468 | 910 | 45-M | |
| 14 | 1833 | 870 | 78-F | |
| 15 | 706 | 844 | 39-M | Valves M A T 12, 7, 13, cm. Presystolic thrill at apex. Three murmurs. Angina pectoris. A2 very faint. Heart and lung clots. Arteriosclerosis of aorta only. |

Rheumatic Heart Disease

| | | | | |
|---|---|---|---|---|
| | 341 | 750 | 39-M | Mitral stenosis. Acute glomerulo nephritis. |

## VII. DEGREES OF CARDIAC HYPERTROPHY IN DIFFERENT LESIONS

TABLE 92.—AVERAGE CARDIAC WEIGHTS

1. Syphilitic aortic regurgitation..... 613 grams
2. Aortic stenosis (rheumatic) 580 grams
3. Chronic pericarditis—uncomplicated cases*..... 567 grams
4. All valve lesion except those given above..... 512 grams
5. Chronic pericarditis (all cases)..... 500 grams
6. Mitral stenosis alone..... 426 grams
7. Arteriosclerosis and nephritis..... 487 grams
8. Nephritis alone..... 453 grams
9. Arteriosclerosis alone..... 429 grams
10. Hypertrophy and dilatation without any "cause"..... 384 grams

With these may be compared

11. Chronic endocarditis (alone)..... 423 grams
12. Acute endocarditis (alone)†..... 420 grams
13. Acute pericarditis (alone) (8 cases)..... 470 grams

*See Table 91. †See Table 85.

These figures show (1) that the *cor bovinum*, the classical extreme of enlargement, is most often a result of aortic regurgitation, as has been so frequently observed before.

(2) That although chronic pericarditis *with mediastinitis* produces the largest of all hearts met with in this series, the *average* case of chronic pericarditis (*without* mediastinitis) has less effect on the heart than aortic disease.

(3) That when several valves are involved in a rheumatic lesion it comes next to pure aortic disease and to uncomplicated chronic pericarditis as a stimulant to cardiac overgrowth, while

(4) Pure mitral stenosis produces on the average but slight enlargement and

(5) Is exceeded by nephritis (with or without arteriosclerosis) in the effect on the heart muscle.

(6) That the "uncaused" and uncomplicated cases of hypertrophy and dilatation are mostly of slight degree—slighter indeed than any group except those associated with uncomplicated arteriosclerosis.

TABLE 93.—ELEVEN CASES OF CARDIAC HYPERTROPHY ASSOCIATED WITH CHRONIC PERICARDITIS ALONE

| Case No. | Necropsy No. | Heart weight | Age and sex | Remarks |
|---|---|---|---|---|
| 1 | 266 | 1328 | 31. M | Chronic non-deforming mitral and aortic endocarditis. Mediastinitis. Rheumatism 4, 6 and 8, years ago. |
| 2 | 1535 | 337 | 31. F | |
| 3 | 1673 | 528 | 53. M | "Orthopnea since 16." No rheumatism. Chronic passive congestion. Tb. of a rib. Diagnosis "Myocarditis and mitral regurgitation." Mitral valve = 10 cm. |
| 4 | 1700 | 625 | 69. M | Died of appendicitis and general peritonitis. |
| 5 | 2314 | 297 | 36. M | Hemiplegia and epilepsy. Pericarditis very slight. |
| 6 | 2504 | 400 | 56. M | Calcified pericarditis. Died of pneumonia. |
| 7 | 2737 | 305 | 40. M | Tuberculosis of lungs and peritonitis. |
| 8 | 3009 | 448 | 20. F | Pick's syndrome. Diagnosis correct in life. |
| 9 | 3290 | 1140 | 31. M | Rheumatism 4 times. Diagnosis correct in life. Valve orifices enlarged. Mediastinitis. Double murmur. |
| 10 | 3520 | 425 | 40. M | Valve orifices enlarged. Mediastinitis. Pneumonia. |
| 11 | 3714 | 308 (slight hypertrophy) | 22. F | Cancer of stomach with metastases. |

(7) Arteriosclerosis alone is associated with a slight though definite cardiac hypertrophy, but may, as already said, be itself a concomitant result of a common cause (hypertension) producing the hyper-

TABLE 94.—ARTERIOSCLEROSIS OF AORTA

| No. | Age and sex | Duration | Chronic passive congestion | Weight heart | Blood pressure | Murmurs | Circumference of valves in cm. | | | Clinical diagnosis | Necropy |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Mitral | Aortic | Tricuspid | | |
| 554 | 46. M | 2 wks. | + | 514 | ..... | 0 | 12 | 8 | 15.5 | Phthisis | Phthisis. Iliac sclerosis too. No cardiac symptoms. |
| 622 | 38. M | + | + | 500 | ..... | 0?<br>Syst.? | ....<br>12 | 8.5 | ....<br>13 | Mitral regurgitation<br>Arteriosclerosis | Hypertrophy and dilatation. Arteriosclerosis. Chronic passive congertion. Heart clot (aorta). |
| 706 | 39. M | 2 yrs. | ++ | 844 | ..... | Systolic & presystolic | 12.5 | 7.5 | 13.5 | Mitral stenosis<br>Mitral regurgitation | Same diagnosis with clot in right auricular appendix and lung. Angina. Presystolic thrill. A2 very faint. Chronic passive congestion. (Flint type?) |
| 720 | 40. M | 2 yrs. | ++ | 711 | ..... | Presystolic | 10 | 6 | .... | ..... | Same diagnosis. Same clots. Hemiplegia twice, 10 weeks ahd 4 weeks ago. Chronic passive congestion |
| 800 | 43. F | 0 | 0 | 385<br>H & D | ..... | ..... | .... | 6.5 | .... | Chronic nephritis | Cerebral hemorrhage. Arteriosclerosis, cardiac hypertrophy and dilatation. No cardiac symptoms. |
| 908 | 61. M | 0 | 0 | 420<br>H & D | ..... | ..... | ....<br>11 | ...<br>8 | ....<br>14 | Cancer stomach | No passive congestion. |
| 972 | 75. M | ? | + | 556 | ..... | 0 | 11 | 9 | 14.5 | Apoplexy | Extensive cerebral softening. No *known* cardiac symptoms but no history. |
| 1019 | 70. F | ? | + | "very large" | ..... | 0 | .... | 8 | .... | Chronic nephritis | Hypertrophy and dilatation first item in anatomical diagnosis. Chronic passive congestion. Clots both ventricles and lung and kidney. Senile degeneration of kidneys. |
| 1077 | 55. F | 0 | 0 | 397<br>H & D | ..... | ..... | .... | ... | .... | Cirrhosis | Cirrhosis of the liver. |

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1200 | 56. F | 0 | 0 | 360 H & D | ..... | ..... | 10 | 7 | | Erosion of cervix | Post operative death with hemorrhage after amputation of cervix for erosion. |
| 1441 | 50. F | 0 | 0 | 398 H & D | ..... | ..... | 11.5 | 7.5 | 13.5 | ..... | Post operative death. Sarcoma of jaw. Embolism of brain after operation. |
| 1446 | 54. M | 0 | 0 | 345 H & D | ..... | 0 | 16 | 8.5 | 13 | ..... | Appendix. General peritonitis. Clot in branch of right pulmonary artery. |
| 1469 | 59. M | 0 | 0 | 361 H & D | ..... | ..... | 10.5 | 8.8 | 12 | ..... | Cancer of stomach. Perforation. General peritonitis. |
| 1481 | 47. M | 0 | 0 | 412 H & D | ..... | ..... | 11 | 6 | 12 | ..... | Slight arteriosclerosis of aorta. None of vessels of Willis. Cerebral hemorrhage. |
| 1484 | 55. M | 0 | 0 | 502 | ..... | ..... | 11 | 8 | 14 | ..... | Smash. |
| 1605 | 70. M | 0 | 0 | 380 H & D | ..... | ..... | 11 | 8.3 | 13.5 | Pneumonia | *Slight* arteriosclerosis of aorta. Pneumonia. |
| 1630 | 51. F | 0 | 0 | 390 H & D | ..... | ..... | 9 | 7 | 11 | ..... | Hernia operation. General peritonitis. *Slight* arteriosclerosis of aorta. |
| 1750 | 40. F | + | + | 525 | ..... | ..... | 9.5 | 6.5 | 12 | Acute endocarditis Acute peritonitis | Puerperal case. Pulmonary embolism one month after delivery. Chronic passive congestion. Cardiac signs and symptoms. |
| 3100 | 60. F | 0 | + | 444 H & D | 150/? | ..... | 10 | 7.2 | 13 | Syphilis | Arteriosclerosis of aorta; arteriosclerotic occlusion of circle of Willis and renals. Scars. |
| 3198 | 47. M | 0 | 0 | 362 H & D | 98/60 | ..... | 11 | 7.5 | 14.5 | ..... | Cancer of the ampulla of Vater. *Slight* arteriosclerosis of aorta. |

trophy of the heart and the arterial changes as parallel compensatory lesions.

It is noteworthy that in *twenty cases of arteriosclerosis* (see Table 94) *confined to the aorta* and slight even there in four cases, the average heart weight was 463 grams, or greater than in the "generalized" cases with sclerosis extending beyond the aorta.* Among these twenty cases were hearts weighing 844, 711, 556, and 500 grams, and another called "very large" but not weighed. How can anyone suppose that the arteriosclerosis was an important cause of cardiac enlargement in these cases? But if not in these, then why should we pay attention to it when it is associated with smaller hearts in its generalized form? All these patients may have had hypertension before we saw them in their terminal hospital weeks. The cardiac enlargement may perfectly well have been due to that, the arteriosclerosis being due also to the hypertension or to some unknown cause.

It would be natural that slight arteriosclerosis should go with slight cardiac hypertrophy and extensive arteriosclerosis with extensive hypertrophy if the possibly causative hypertension had lasted but a short time in the first group and a longer time in the second.

## VIII. CARDIAC FAILURE IN HYPERTROPHY AND DILATATION

For unknown reasons a hypertrophied and dilated heart of the type that I have been describing is prone sooner or later to fail—more prone, that is, than hearts of normal size. Just why this failure occurs I do not know. It used to be said that the hypertrophied heart was as strong as the normal heart or stronger, but that when *dilatation* supervened, the stretched, thinned organ naturally became inefficient. Dilatation used to be regarded as a late untoward mechanical complication of the benign process which began with simple hypertrophy. But there is in most cases, so far as I know, no reason to believe this. Hypertrophy and dilatation are ordinarily associated together, and this not merely in the late and incompetent states of the disease, but in the very early and mild cases. Nor has myocarditis any demonstrable part in producing the failure of enlarged hearts. Most of them show no myocarditis.

### The Association of Hypertrophy with Dilatation

In 4000 necropsies hypertrophy and dilatation together occurred 1088 times as the records stand.

* And in a few extending to the cerebral or iliac arteries.

In 4000 necropsies hypertrophy alone occurred 121 times.*

In 4000 necropsies dilatation alone occurred 118 times.

As soon as we can recognize the one we usually find the other. Moreover there is just as much reason for supposing that dilatation is a compensatory and helpful change as there is for such a belief regarding hypertrophy. Probably without dilatation compensation never could be established or maintained.

All that we know is that very large hearts (without valve lesions or pericarditis) are apt to fail, perhaps from chronic effort against hypertension. *Circulatory failure in the absence of considerable cardiac enlargement is not common* except in connection with some quite obvious cause such as surgical operation, hemorrhage, infection, or poisoning. But *the greatly enlarged heart may become incompetent without any special "cause" and without our having any considerable warning that it is failing.* In many cases it seems clear that infection, intoxication, or operative insult is the exciting cause of circulatory heart failure (see statistics in Table 95). But there seems no doubt that these causes act much more strongly on hearts already hypertrophied and dilated as a result of some previous agency, known or unknown. It is true that a heart of normal size, wholly free from anatomical defect or disease, may in a few days become seriously or fatally weakened as the result of an infection such as pneumonia or typhoid fever, or of a toxemia such as acidosis. Much less common are the instances of circulatory failure following operation upon a patient whose heart was sound at the beginning of the operation. But although these facts are well established *there is no question that these three dangerous influences—infection, intoxication, and operative insult—are more often followed by death when the heart is already hypertrophied and dilated.*

*Mode of Death in 230 Cases with Congestive Heart Failure among 599 Cases of Cardiac Hypertrophy and Dilation with and without Nephritis*

1. Of these the death seemed due *wholly* to chronic passive congestion in 95 cases.

2. In the other 135 cases, although there *was* chronic passive congestion post-mortem, it was not obvious in life or of exclusive influence in producing death as judged post-mortem, the picture being overshadowed by

*This is the number as it stands in the pathological records. But it is too large for Dr Richardson tells me that the record often *says* "hypertrophy" when it means "hypertrophy and dilatation."

TABLE 95

| | Cases | |
|---|---|---|
| Uremia | 24 | 63 |
| Uremia plus Sepsis | 21 | |
| Sepsis | 18* | |
| Apoplexy | 7 | |
| Operative insult | 3 | |
| "Cancer" | 7 | |
| Pneumonia | 7 | |
| Pernicious Anemia | 6 | |
| Leucemia | 4 | |
| Phthisis | 4 | |
| Cirrhosis | 3 | |
| Cirrhosis plus Sepsis | 3 | |
| "Smash-ups" | 3 | |
| Diabetes and Sepsis | 3 | |
| Angina | 2 | |
| Eclampsia, Status lymphaticus, Stokes-Adams syndrome, each 1 | 3 | |
| Doubtful | 17 | |
| Total | 135 | |

*Sepsis in all combinations totals 47 cases excluding the consumptives, whose condition may have been essentially sepsis also.

In many of these latter cases the finding of an enlarged heart and chronic passive congestion at necropsy was a total surprise. The other lesions seemed in life the only ones of importance. Yet the presence of the chronic passive congestion is sufficient, I take it, to show that the cardiac hypertrophy and dilatation played *some* part in the patient's death.

There is little if any evidence for believing that sepsis or any infectious disease can in and of itself produce *chronic* passive congestion in the absence of hypertrophy and dilatation or any other heart lesion. Among the twenty-five cases of extreme general septicemia with secondary acute endocarditis studied in Chapter VII there was not one which showed any chronic passive congestion post-mortem. The same is true of the eighteen cases of chronic nephritis without cardiac hypertrophy. But doubtless *the infections* (especially streptococcus sepsis, pneumonia and tuberculosis), *the toxemias* (uremic, diabetic, neoplastic, hepatic and eclamptic), and the *mechanical injuries* (operative, traumatic, apoplectic), as well as the anemias and angina pectoris, may join in the attack upon the body's resources and cooperate with chronic passive congestion to cause death.

3. The heart weight in the 95 "pure" cases* averaged 548 grams. In 24 cases dying with a chiefly uremic clinical picture (though showing chronic passive congestion and hypertrophy and dilatation of

*I.e. cases dying with enlarged heart and general dropsy but without other known cause for death.

the heart at necropsy) the average heart weight was 501 grams; in the 18 showing chiefly sepsis the average heart weight was 487 grams. *Apparently then the hearts of about 550 grams weight have reached the danger point even if no infection or kidney trouble complicates the situation.*

4. The *ages* in 93* of the "pure cases averaged fifty-two, and the *sex* (in 95) 64 men to 31 women, i.e. two to one.

5. The *duration* of known symptoms or complaints *averages nine months* in 87 "pure" cases in which it could be fairly well estimated. This is calculated from the duration of dyspnea and edema, which were the standard complaints. If, from this 87 cases, 16 long ones are subtracted (averaging two and a half years' duration) the 71 remaining cases averaged five months; so that we can say that *in four-fifths of the cases the average duration of complaints is five months.*

6. In a group of 192 cases showing no passive congestion after death, 136 had no circulatory symptoms at any time; 56 had some complaints which *might* have some relation to the circulation, though in many of these the complicating uremia or pneumonia which finally produced death may well have been the cause of *all* the symptoms. *There was, however, a small but definite percentage in which the ante-mortem symptoms led us to expect that chronic passive congestion would be found after death, though in fact no such congestion was found. These cases number nineteen.* This figure, which is 5% of our cases, may be taken as the error inherent in the principle of division which I have here adopted between active and latent cases of heart disease. In 95% of cases of cardiac enlargement, inactive yet with some symptoms apparently of circulatory origin during life, we may consider that uremia, pneumonia or some other factor not primarily circulatory caused these symptoms and the patient's death. These complications, however, in the inactive cases extended over more than a year in half the cases; in the rest they lasted three months or less.

Hence it appears that about 5% of cases of cardiac enlargement may be associated with chronic circulatory symptoms not demonstrable as chronic passive congestion after death. In this degree the classification here adopted ("Active" or "Inactive") is incorrect.

*Age and Sex in Relation to Latency or Congestion.*—Allowing however for this error, it appears that while men suffer two or three times as much as women from cardiac disease of the hypertensive type, the disease when it *does* affect women is oftener of a more serious type than when it affects men. For women made up 32% of the serious

* Age not recorded in two cases.

or active cases and only 21% of the inactive cases. The discrepancy is not striking but probably has some significance.

*The age difference*, as one contrasts the two groups of cardiac cases, latent and manifest, has no interest. The difference between fifty-three years, the average age of the latent cases, and fifty-two, the average for those dying of passive congestion as shown at necropsy, has no significance.

7. *Arteriosclerosis* (post-mortem) was present in 78, absent in 17 patients dying of hypertensive heart disease without complications, i.e., arteriosclerosis is the rule. The heart-weight in sixteen of the seventeen cases without arteriosclerosis averaged 455 (in one the weight was not recorded). The general average of the 95 cases was 548 grams. But the ages of these sixteen non-sclerotic cases averaged forty-one years as against fifty in the whole group. So that these figures prove nothing so far as I see.

8. *Blood pressure* was recorded in 82 cases.

TABLE 96

| | | |
|---|---|---|
| Systolic blood pressure 120 or less | in 15 cases | 30 |
| Systolic blood pressure 130–139 | in 5 cases | |
| Systolic blood pressure 140–159 | in 10 cases | |
| Systolic blood pressure 160–180 | in 13 cases | 52 |
| Systolic blood pressure 181–200 | in 13 cases | |
| Systolic blood pressure 201–220 | in 13 cases | |
| Systolic blood pressure 221–240 | in 6 cases | |
| Systolic blood pressure 241–280 | in 6 cases | |
| Systolic blood pressure 300 | in 1 case | |

i.e., hypertension was usually present even at the end of life. In my belief it had *always* been present earlier in life and was the cause of the cardiac enlargement and weakening.

9. *Diastolic pressure* was recorded in 49 cases.

TABLE 97

| | | |
|---|---|---|
| Diastolic blood pressure 48 | in 1 case | 18 |
| Diastolic blood pressure 60–75 | in 6 cases | |
| Diastolic blood pressure 80–95 | in 11 cases | |
| Diastolic blood pressure 100–105 | in 4 cases | 31 |
| Diastolic blood pressure 110–125 | in 11 cases | |
| Diastolic blood pressure 130–150 | in 10 cases | |
| Diastolic blood pressure 165–188 | in 5 cases | |
| Diastolic blood pressure 220 | in 1 case | |

10. *Pulse Pressures* in 49 cases.

TABLE 98

| | | |
|---|---|---|
| Pulse pressure 15–25 in 5 cases | | |
| Pulse pressure 30–40 in 6 cases | 22 | |
| Pulse pressure 45–55 in 11 cases | | |
| Pulse pressure 58–60 in 4 cases | | |
| Pulse pressure 70–75 in 7 cases | | |
| Pulse pressure 80–95 in 10 cases | 27 | |
| Pulse pressure 100 in 4 cases | | |
| Pulse pressure 120 in 2 cases | | |

11. *Nephritis* (subacute or chronic), was present in 33 of 95 "pure" cases dying a cardiac death with chronic passive congestion. In the whole 230 cases-with-some-chronic-passive-congestion there were 112 cases of nephritis subacute or chronic, and three of acute, = 115 or just one half.

That this nephritis itself chemically contributed to the chronic passive congestion, aside from any mechanical hypertrophy and dilatation with weakening, is of course possible, but of this there is, as I have said above, no evidence.

12. The *largest hearts* among the 230 cases of manifest or dropsical hypertensive heart disease are listed in Table 99.

TABLE 99

| | |
|---|---|
| (a) Chronic Nephritis with Arteriosclerosis | 780 grams |
| | 750 grams |
| | 740 grams |
| | 720 grams |
| | 705 grams |
| | 700 grams |
| | 700 grams |
| (b) Chronic Nephritis without Arteriosclerosis | 750 grams |
| (c) Arteriosclerosis alone | 870 grams |
| | 710 grams |
| (d) No "cause" whatever | 910 grams |
| | 793 grams |
| | 775 grams |
| | 750 grams |
| | 711 grams |

13. *Murmurs.*—Systolic murmurs, usually loudest at the apex, were heard in most cases, if carefully looked for.* They were definitely noted as absent in only 20%. Diastolic murmurs were also noted in 24% of the cases despite normal aortic valves.

* Some of the patients died in surgical wards, where little attention was paid to the minutiae of cardiac examination.

*Relation of Systolic Murmurs to the Size of Valve Orifices.*—Selecting five cases characterized by especially loud apical systolic murmurs widely transmitted (axilla and back) we find the valve measurements post-mortem as follows:

TABLE 100

| Necropsy No. | Mitral valve | Tricuspid valve |
|---|---|---|
| 212 | 10. cm. | 12.5 cm. |
| 311 | 10.5 cm. | 14. cm. |
| 500 | 10. cm. | |
| 849 | 12.5 cm. | 13. cm. |
| 2891 | 10.5 cm. | 13. cm. |
| Average..... | 10.7 cm. | 13.1 cm. |

Compare with these a set of six cases in which the *absence* of murmurs is definitely recorded.

TABLE 101

| Necropsy No. | Mitral valve | Tricuspid valve |
|---|---|---|
| 129 | 12. cm. | 12.5 cm. |
| 1811 | 10. cm. | 12. cm. |
| 1948 | 11.5 cm. | 14. cm. |
| 2711 | 12.5 cm. | 15. cm. |
| 2718 | 10.5 cm. | 12. cm. |
| 3701 | 11. cm. | 15. cm. |
| Average..... | 11.2 cm. | 13.4 cm. |

Obviously one can make no prediction of an enlarged valve orifice ("relative insufficiency") on the basis of loud systolic murmurs in life. The orifices are no larger (actually a trifle smaller) in the cases with systolic murmurs than in those free from murmurs.

With *diastolic* murmurs the case is not so clear. The following table lists seven cases of hypertensive heart disease with diastolic murmur loudest along the left sternal margin but without valve lesions at necropsy.

TABLE 102

| Necropsy No. | Aortic valve |
|---|---|
| 181 | 7. cm. |
| 462 | 8.5 cm. |
| 876 | 6.5 cm. |
| 1317 | 8.5 cm. |
| 2318 | 9.5 cm. |
| 2376 | 7. cm. |
| 2590 | 8.3 cm. |
| Average..... | 7.9 cm. |

This shows a slight increase in the aortic ring circumference, whether enough to be significant I cannot say.

**Cerebral Symptoms.**—In contrast with rheumatic and syphilitic types of heart disease the patient of the hypertensive group (mostly elderly) presented as a rule some cerebral disturbances—usually delirium in the last weeks of life. Apoplexy is of course the terminal event in many cases largely similar to the 230 here analyzed. But these apoplectic cases are not here considered as I am isolating the cases with death by congestive heart failure.

**False Pericardial "Friction."**—Eight of these cases were supposed to have an acute pericarditis because of "friction sounds" heard along the left sternal edge; but no pericarditis was found at necropsy in these cases.* Possibly the dryness of the pericardial surfaces in some dying patients of this type may account for these sounds.

**Diagnosis.**—Most of these cases of Hypertensive Heart Disease were called "Mitral Regurgitation" or "Myocarditis" in life.

* Though in 102 *other* cases acute pericarditis *was* associated with hypertrophy (see *Acute Pericarditis*).

"Aortic Regurgitaion" is also a not infrequent diagnosis and "Cardio-renal" appears occasionally. In some heart disease was not suspected at all. The true diagnosis was seldom made.

## IX. PHYSICAL SIGNS

The only reliable evidences of this type of heart disease during life are, hypertension and demonstrable cardiac enlargement.

Only in the minority of the cases here analyzed was blood pressure measured, as a large number of them date from a period when blood pressure measurements were not a routine of our records. The results of the measurements recorded as shown in Tables 103*k*, 104*b*, and 106.

In some of these cases doubtless the pressure was low because the patients of this series were seen only near the close of life. But experience in office practice and out-patient work with patients seemingly of this type leads me to believe that in cases of this type hypertension is almost invariably present for months or years before compensation fails. That is to say, *we almost never see enlarged hearts in clinical work without evidence of valve lesions or pericarditis, unless there is a definite chronic hypertension.*

In a minority of cases there is elevation of systolic blood pressure without any considerable change in the diastolic. But many of these I believe represent the later stages of cases which (if seen earlier) would have shown a higher diastolic pressure as well. When *nephritis* was present the diastolic as well as the systolic was usually elevated. When no nephritis could be found elevation of systolic blood pressure alone was not uncommon.

As regards the *evidence of cardiac enlargement* there is little to be added to what any textbook on physical diagnosis teaches, but a good deal to be subtracted. X-ray is of course the most reliable agent for proving whether the heart is or is not enlarged. This was available in only a small minority of our cases, but often brought to light enlargements which would have escaped us otherwise. Indeed in this group of cases the usual methods of physical diagnosis yield little evidence of value as to size of the heart, because in elderly persons (such as form the great bulk of those in this type) the costal cartilages are often ossified and the whole thorax enlarged into the shape designated as the "barrel chest." With this change in the shape of the chest there comes the general hyperresonance on percussion (long falsely supposed to point to pulmonary emphysema)

which makes it difficult or impossible to outline the cardiac dullness by percussion. The same change in the shape of the chest often makes it impossible for us to identify the position of the cardiac apex by palpation. In many, possibly the majority of these cases no cardiac impulse whatever can be found.

Hence previous to the introduction of X-ray plates and blood pressure measurements we were helpless in the attempt to diagnose most of these cases, unable to decide whether a cardiac hypertrophy was or was not present. But now *the presence of hypertension allows us to conclude with only a negligible percentage of error that the heart is enlarged,* even when by percussion and palpation we get no such evidence. As for the *degree* of enlargement we must rely on the X-ray or guess at it.

None of the *auscultatory* signs of cardiac enlargement proved of much value in patients of this series, probably because they were seen as bed patients, shortly before death rather than in the early stages of their malady. Accentuation of the aortic second sound, for instance, was present in only ¼ of the cases. As a rule both second sounds were feeble, the aortic especially so. Change in the quality of the sound (the so-called "metallic" or "ringing" second) was occasionally noted but was usually absent.

Arrhythmia, alternation, defects of conduction may be absent until near the close of life in these cases. Murmurs and heart sounds give us no information of value. Hence the discovery of the disease prior to the terminal stage rests wholly on our ability to recognize hypertension and cardiac enlargement. Without X-ray the latter is often impossible to be sure of. Thus the essential diagnostic data are reduced to one,—blood pressure measurement—the most important fact to know about the majority of all heart cases, since hypertensive heart disease is commoner than any other type.

## X. BLOOD-PRESSURE AND OTHER DATA IN THE GROUP OF 179 CASES OF CHRONIC, 15 OF SUBACUTE AND 13 OF ACUTE NEPHRITIS ASSOCIATED WITH HYPERTROPHY AND DILATATION AND WITHOUT VALVE LESIONS OR PERICARDIAL ADHESIONS

In chronic nephritis, 179 cases, we have 112 with arteriosclerosis and 67 without it. The statistics of blood pressure and other data are shown in the following tables.

TABLE 103.—CARDIAC HYPERTROPHY AND DILATATION WITH NEPHRITIS AND ARTERIOSCLEROSIS—112 CASES

(*a*) *Age*

| | |
|---|---|
| 21–30 | 2 |
| 31–40 | 19 |
| 41–50 | 21 |
| 51–60 | 30 |
| 61–70 | 24 |
| 71–80 | 12 |
| 81–90 | 4 |
| | 112 |

(*b*) *Duration*

| | | |
|---|---|---|
| 25 years | 1 | |
| Some years | 1 | |
| 10 years | 2 | 5–10 years, 12 cases |
| 9 years | 1 | |
| 8 years | 4 | |
| 5–6 years | 5 | |
| 3 years | 2 | 1–3 years, 20 cases |
| 2–3 years | 7 | |
| 1–2 years | 11 | |
| 11 months | 1 | 4–12 mos. 10 cases |
| 10 months | 2 | |
| 9 months | 1 | |
| 8 months | 1 | |
| 4–5 months | 5 | |
| 3 months | 2 | 2–12 weeks, 17 cases |
| 2 months | 4 | |
| 1–2 months | 8 | |
| 3 weeks | 1 | |
| 2 weeks | 2 | |
| 1 week | 4 | Under a week, 7 cases |
| 1 day | 3 | |
| Unknown | 44 | |
| | 112 | |

(*c*) *Sex*

| | |
|---|---|
| Male | 81 |
| Female | 31 |

(*d*) *Type of Nephritis*

| | |
|---|---|
| Chronic (unspecified) | 10 |
| Chronic diffuse | 5 |
| Chronic glomerular | 20 |
| Chronic interstitial | 31 |
| Chronic arteriosclerotic | 45 |
| Subacute glomerular | 1 |
| | 112 |

(*e*) *Heart Weight*

| | | |
|---|---|---|
| 250–300 | 1 | |
| 301–350 | 8 | |
| 351–400 | 15 | 51 |
| 401–450 | 13 | |
| 451–500 | 14 | |
| 501–550 | 22 | |
| 551–600 | 12 | |
| 601–650 | 9 | |
| 651–700 | 8 | 61 |
| 701–750 | 6 | |
| 751–800 | 1 | |
| 801–850 | 1 | |
| 851–900 | 2 | |
| | 112 | |

Average heart weight—508 grams.

(*f*) Analysis of 42 Necropsies

| With Chronic Passive Congestion | Without Chronic Passive Congestion |
|---|---|
| 2 | 1 |
| 3 | 4 |
| 2 | 5 |
| 4 | 2 |
| 1 | 3 |
| 1 | 1 |
| 4 | 1 |
| 5 | 0 |
| 2 | 0 |
| 0 | 0 |
| 1? | 0 |
| 25 | 17 |

(*g*) *Chronic Passive Congestion*

| | |
|---|---|
| Ante and post mortem | 48 or 42% |
| Ante alone | 77 or 6% |
| Post alone | 14 or 12% |
| None | 43 or 39% |
| | 112 |

(*h*) *Murmurs*

| | |
|---|---|
| Systolic | 15 |
| Diastolic | 5* |
| Double | 3 |

(*i*) *Type of Arteriosclerosis*

| | |
|---|---|
| General | 83 |
| "Slight" | 9 |
| Aorta and great branches | 13 |
| Aorta and Vessels of Willis | 3 |
| Aorta and Coronaries | 6 |
| Aorta | 2 |
| Coronary | 1 |
| Slight except coronaries | 1 |
| Iliacs only | 1 |
| Vessels of Willis | 1 |
| Aorta, Coronaries, vessels of Willis, branches | 1 |
| | 112 |

* 821 (aortic 8), 2256 (aortic 8), 2270 (aortic 7), 2689 (aortic 7.5), 3364 (aortic 7).

*(j) Type of Death*

| | |
|---|---|
| Uremia | 52 |
| Sepsis | 18 |
| Passive congestion | 11 |
| Post-operative | 8 |
| Cerebral lesions (apoplexy) | 7 |
| Pneumonia | 3 |
| Coma of doubtful origin | 2 |
| Angina pectoris | 1 |
| Heart block | 1 |
| Hemorrhage | 1 |
| Cancer | 1 |
| Eclampsia | 1 |
| Cirrhosis | 1 |
| Coronary thrombus | 1 |
| Fracture of femur | 2 |
| ? | 2 |
| | 112 |

*(k) Systolic Blood Pressure*

Recorded in 63 of 112 cases

| | |
|---|---|
| 250–300 | 10 |
| 200–249 | 33 |
| 170–199 | 14 |
| 150–169 | 3* |
| Under 150 | 3 |
| | 63 |

1 had Blood Pressure 300/220 with heart of 384 grams
1 had Blood Pressure 220/160 with heart of 303 grams
1 had Blood Pressure 220/188 with heart of 401 grams
1 had Blood Pressure 160/155 with heart of 664 grams
1 had Blood Pressure 115/100 with heart of 780 grams
1 had Blood Pressure 98/68 with heart of 390 grams

TABLE 104.—CARDIAC HYPERTROPHY AND DILATATION WITH CHRONIC NEPHRITIS, BUT WITHOUT ARTERIOSCLEROSIS—67 CASES

Average age 36 years

*(a) Sex*

| | |
|---|---|
| Male | 43 |
| Female | 24 |

*(b) Systolic Blood Pressure*

| | |
|---|---|
| Increased | 3 |
| 250–300 | 1 |
| 200–249 | 11 |
| 150–199 | 8 |
| 100–149 | 5 |
| No record | 39 |
| | 67 |

* 1 had blood pressure 160/130.

*(c) Heart Sounds*

| | | |
|---|---|---|
| No change | | 7 |
| $A_2$ | Accentuated | 31 |
| " | Absent | 4 |
| " | No record | 14 |
| $P_2$ | Accentuated | 11 |
| | | 67 |

*(d) Chronic Passive Congestion*

| | | |
|---|---|---|
| Ante and Post-Mortem | 39 | 45 |
| Post-Mortem | 6 | |
| None | 14 | |
| Ante-Mortem | 4 | |
| No record | 4 | |
| | 67 | |

*(e) Heart Weight*

Average ..... 470 grams

*(f) Heart Enlarged in Life*

| | | |
|---|---|---|
| Not enlarged | 23 | |
| Slightly | 23 | 40 |
| Moderately | 13 | |
| Greatly | 4 | |
| No record | 4 | |
| | 67 | |

*(g) Type of Nephritis*

| | |
|---|---|
| Chronic Interstitial | 12 |
| Chronic Glomerular | 10 |
| Chronic (unspecified) | 36 |
| Arteriosclerotic Nephritis | 3 |
| Diffuse | 4 |
| (Pyelonephrosis and pyelonephritis) | 1 |
| Amyloid | 1 |
| | 67 |

*(h) Murmurs*

| | |
|---|---|
| Systolic | 37 (1 musical and loud.)* |
| Systolic and diastolic | 6 (2 with friction) |
| None | 16 |
| No record | 6 |
| Presystolic | 2 |
| | 67 |
| Thrill (Systolic) | 3 |

*(i) Arrhythmia*

| | |
|---|---|
| Present | 7 |
| Absent | 44 |
| No record | 16 |
| | 67 |

*(j) Palpitation*

| | |
|---|---|
| Present | 6 |
| Absent or no record | 61 |
| | 67 |

* M. 10.5 T. 11.5 Heart o.k. B.P. 220/165.

TABLE 105.—HYPERTROPHY AND DILATATION WITH SUBACUTE NEPHRITIS,* 15 CASES

| | WITHOUT ARTERIOSCLEROSIS | WITH GENERAL ARTERIOSCLEROSIS |
|---|---|---|
| *(a) Age* | | |
| 20–29 | 2 | 1 |
| 30–39 | 5 | 1 |
| 40–49 | 1 | 1 |
| 50–59 | 1 | 1 |
| 60–69 | 0 | 1 |
| 70–79 | 0 | 1 |
| | 9 | 6 |
| *(b) Sex* | | |
| Males | 6 | 3 |
| Females | 3 | 3 |
| *(c) Duration* | | |
| 9 mos. | 1 | 0 |
| 4 mos. | 1 | 2 |
| 3 mos. | 0 | 2 |
| 2 mos. | 0 | 1 |
| 6 weeks | 1 | 0 |
| 4 weeks | 1 | 0 |
| 3 weeks | 1 | 0 |
| 1–2 weeks | 0 | 1 |
| 1 week | 1 | 0 |
| Unknown | 3 | 0 |
| | 9 | 6 |
| *(d) Chronic Passive Congestion* | | |
| Ante and Post-Mortem | 6 | 5 |
| Post-Mortem | 2 | 1 |
| No record | 1 | 0 |
| | 9 | 6 |
| *(e) Murmurs* | | |
| Systolic | 3 | 4 |
| Diastolic | 1 | 0 |
| Double | 1 | 1 |
| None | 0 | 1 |
| No record | 4 | 0 |
| | 9 | 6 |
| *(f) Heart Weight* | | |
| 700–750 | 1 | 0 |
| 551–600 | 2 | 2 |
| 451–500 | 2 | 0 |
| 401–450 | 1 | 2 |
| 351–400 | 2 | 1 |
| 250–300 | 1 | 1 |
| | 9 | 6 |

* All subacute glomerulonephritis, (1 capsular, 1 diffuse, 1 intracapillary, 1 intracapillary and capsular.)

TABLE 106.—HYPERTROPHY AND DILATATION WITH ACUTE NEPHRITIS, 13 CASES

| | WITHOUT ARTERIO-SCLEROSIS | WITH ARTERIO-SCLEROSIS |
|---|---|---|
| *(a) Age* | | |
| 0–9 | 1 | 0 |
| 10–19 | 1 | 0 |
| 20–29 | 4 | 0 |
| 30–39 | 2 | 0 |
| 50–59 | 2 | 1 |
| 60–69 | 0 | 1 |
| 70–79 | 0 | 1 |
| | 10 | 3 |
| *(b) Sex* | | |
| Male | 4 | 2 |
| Female | 6 | 1 |
| *(c) Duration* (Four records only) | | |
| | 1 yr. | 4 weeks |
| | 17 days | |
| | 4 days | |
| | No further data. | |
| *(d) Chronic Passive Congestion* | | |
| Ante and Post-Mortem | 1 | 1 |
| None | 7 | 2 |
| No record | 2 | 0 |
| | 10 | 3 |
| *(e) Murmurs* | | |
| Systolic | 4 | 0 |
| Double | 0 | 1 |
| None | 4 | 2 |
| No record | 2 | 0 |
| | 10 | 3 |
| *(f) Heart Weight* | | |
| 551–600 | 0 | 1 |
| 451–500 | 1 | 0 |
| 351–400 | 0 | 1 |
| 301–350 | 3 | 1 |
| 251–300 | 4 | 0 |
| 201–250 | 1 | 0 |
| Under 200 | 1 | 0 |
| | 10 | 3 |

| | WITHOUT ARTERIO-SCLEROSIS | WITH ARTERIO-SCLEROSIS |
|---|---|---|
| *(g) Type of Nephritis* | | |
| Chronic and acute | 2 | 0 |
| Acute glomerular | 6 | 3 |
| Acute intersterial | 1 | 0 |
| Papillary mycotic | 1 | 0 |
| | 10 | 3 |
| *(h) Blood Pressure* | | |
| 5 records only. | | |
| 180/100, 150/?, 125/70, 100/70, 100/60 | | |

## XI. DIAGNOSIS ACTUALLY MADE IN THESE CASES

In the first ten or fifteen years of the period covered by the cases of this group the diagnosis standing on the hospital record was usually "myocarditis" or "mitral regurgitation." Only within the last

ten years has it been recognized at the Massachusetts General Hospital that *neither of these diagnoses is ever justified before death as a statement of the main cause of death.* Facing the same facts as those once called myocarditis or mitral regurgitation, we now call these cases "hypertrophy and dilatation of the heart" or "hypertensive cardiovascular disease," *in case we are able to recognize cardiac disease at all.* 369 out of 599 had no suggestion of a cardiac diagnosis. The old diagnosis of mitral regurgitation rested on the presence of a systolic murmur, often loudest at the apex but not infrequently loudest at the base of the heart. Even this murmur was not audible in most of the cases of our series, though 62 cases showed it. The mistaken use of the term "myocarditis" in our records rests on the evidence of arrhythmia in an elderly patient without clear signs of valvular disease.

Of most interest is the *occasional* occurrence of *diastolic or presystolic* murmurs without any lesions of the aortic or mitral valve. The interpretation of these murmurs is wholly a matter of speculation. Some prefer to believe that they are due to a relative insufficiency of the aortic or of the pulmonary valve due to high blood pressure above the valve, or to dilatation of its ring. Others are inclined to suppose, as an explanation, some disturbed function at the mitral orifice or in the mitral leaflets. All this however is mere uncontrolled speculation. The solid fact is that in any markedly enlarged heart, diastolic or presystolic murmurs may be heard in the region of the cardiac apex, without there being also any reason to believe that a valvular lesion is present. I have long been accustomed to teach that when the heart is much enlarged, especially in an elderly man, we can rarely draw any conclusions from murmurs heard in the vicinity of the apex. Neither systolic, diastolic nor presystolic murmurs under these conditions have any particular significance.

Out of 599 cases of this group examined we have found only 230, or 39%, to be of the active or manifest type. This may be contrasted with 62% of the rheumatic type, 27% of the syphilitic, and 30% of chronic pericarditis, which are the figures for the proportion of active or manifest disease in those lesions (see above, Table 2).

## XII. SUMMARY AND CONCLUSIONS

1. Hypertensive heart disease means an enlarged and often incompetent heart without valve lesions or pericarditis. The cause, though not established in all the cases of this series, is probably hypertension however produced.

2. The relation of nephritis and of arteriosclerosis to hypertensive heart disease is not clear. Certain it is however that the disease often exists for years without evidence of either renal or arterial disease. But the longer it exists the more apt are we to find nephritis or arteriosclerosis at the necropsy, whether as results or complications or concomitant symptoms of some cause underlying both them and the hypertension, we do not know.

3. The disease is much commoner than any other cardiac malady. In this series it is commoner than all the other types of heart disease put together, whether we consider all cases or only those showing decompensation before and after death. It is often wrongly diagnosed as myocarditis, mitral regurgitation, "cardiorenal disease," "senile heart" etc.

4. It is more than twice as common in men and appears usually about the fiftieth year, though cases occur in every decade except the first.

5. There is little or no evidence that muscular work or alcohol can produce any cardiac hypertrophy.

6. Hypertensive heart disease is to be distinguished from the cardiac enlargement associated with pernicious anemia and with leukemia, in both of which blood pressure is normal or low.

7. The degree of cardiac hypertrophy in hypertensive heart disease is, in early cases, slight; and even in long-standing and decompensated cases is never of the extreme grade caused by chronic pericarditis (with or without valve lesions). No hearts in this series are enlarged beyond 910 grams as a result of hypertensive heart disease. Most of them are much smaller. Comparing averages they are smaller than those associated with syphilitic aortic regurgitation, rheumatic aortic stenosis (with regurgitation), and the multiple valve lesions. But hypertensive heart disease enlarges the heart more on the average than uncomplicated mitral stenosis does.

8. Hypertensive heart disease in its earlier and milder stages lasts for years without producing any considerable discomforts and limitations of the individual's activity. The patient is not aware of it unless he chances to undergo, while in full health, a physical examination, e.g. for life insurance or as a protective hygienic measure. Increased blood pressure is then found and perhaps a slight cardiac enlargement detected, though without X-ray measurements this is often impossible.

9. As the disease progresses its effects show themselves in a slight dyspnea on exertion, in a "pounding" in the arteries of the neck,

or an awareness of the cardiac impulse. Or it may contribute its influence to prostrate the patient when infection, toxemia, accident, or surgical operation bring an unusual strain upon him.

10. Less often—in only about one-third of our cases—are cardiac decompensation and stasis the cause of the patient's death, without cerebral hemorrhage, surgical operation, infection or toxemia. In ⅘ of the cases the duration of the patient's complaints is extraordinarily brief, averaging 5 months in our series.

11. In such cases the physical signs are those of a failing heart—arrhythmia, tachycardia, general passive congestion—usually with hypertension persisting, less often with low or normal blood pressure. Systolic (and occasionally diastolic) murmurs are present but are of no diagnostic value. The same is true of an accented aortic second sound.

Diagnosis rests on the hypertension, the cardiac enlargement, and the lack of evidence pointing to valvular disease or pericarditis.

## HYPERTENSIVE HEART DISEASE—ILLUSTRATIVE CASES

### Necropsy 4769

A Finnish grocer of fifty-six came to the Emergency Ward December 17 for relief of generalized edema of a week's duration. A fragmentary history was obtained from his wife. He had been married over twenty years. His wife had had no children and no miscarriages. She knew of no illnesses of his before the present one. Twenty years ago he weighed 160 pounds. He had gradually gained weight until four months ago he weighed 260 pounds and two weeks ago 280 pounds. He became disinclined to work, but maintained an appetite. For three years he had not seemed well, had been dyspneic, had seemed sleepy day and night, and frequently dozed off during a conversation. During the naps he talked to himself a good deal. In the past few years he had developed frequent nosebleeds which grew progressively worse until the past summer, but had decreased in frequency and severity during the past few weeks. For four months his feet had been swollen. Four months ago he became noticeably dyspneic, and during the past month still more so. For six weeks he had been unable to sleep in a reclining position because of orthopnea and cyanosis; he sat up in a chair or walked about most of the night. On some days during the past week he had raised a tablespoonful of blood. For two weeks he had had generalized edema. He had complained but little of pain, and that over the precordium.

Examination showed an obese, water-logged, cyanotic man with Cheyne-Stokes respiration. The skin showed diffuse erythema over the lower abdomen and legs, many excoriations on the legs, and two ulcers on the right knee. The mucous membranes were cyanotic. There was moderate pyorrhea. The heart did not seem enlarged. The sounds were faint. (The chest wall was very thick.) A soft systolic murmur was heard over the whole precordia. The blood pressure was 170/100. The lungs were clear except for a few coarse bronchial râles in the left upper back and the right upper chest in front. The abdomen was enormously distended, tense, dull to percussion except high in the midline. There was fluid wave. Over the lower portion was induration. Palpation of masses was impossible. There was pitting and massive edema of the genitals and legs and moderate edema of the hands, arms, and back. The left pupil was greater than the right and irregular; both reacted normally. The reflexes could not be obtained because of edema.

The temperature was 98°–100.8°, rectal, the pulse 112–58, the respirations 23–32. The amount of urine is not recorded. The specific gravity was 1.016. There was the slightest possible trace of albumin at one of two examinations. The sediment was loaded with pus and showed five to ten red blood cells at both examinations, two trichomonas were seen in motion at one. The hemoglobin was 90%, the leucocytes 8000, the polynuclears 71%, the reds 6,496,000, with moderate stippling and polychromatophilia. The non-protein nitrogen was 46 mgm. A Wassermann was negative. An abdominal tap gave 2500 c.c. of opaque yellow fluid, specific gravity 1.012, leucocytes 1700, polynuclears 13%, lymphocytes 79%, large mononuclears 8%, 2500 red blood cells, no organisms. Culture was negative.

The orders were as follows. December 17, salt free low protein diet, magnesium sulphate ℥ i in the morning. December 18, digifolin ampules vi intramuscularly at once, digitalis gr. vi by mouth at once and gr. iii 4 i.d., morphia gr. ⅙ s.c. at once and gr. ⅙ s.c. every three hours p.r.n., limit fluids to ℥ 50. December 18, caffein sodium salicylate gr. x intramuscularly at once and gr. xv intramuscularly p.r.n. for marked cyanosis or collapse; digifolin ampules iv. December 19, caffein sodium salicylate gr. xv.

December 18 the patient became very cyanotic and the Cheyne-Stokes respiration was marked. Caffein and oxygen were tried. He had in all thirty-three grains of digitalis. At three o'clock the morning of December 19 he died.

*Clinical Diagnosis.*—Hypertensive heart disease with congestive failure.

Hypertension.
Anasarca.
Obesity.

*Dr. Richard C. Cabot's Diagnosis.*—Hypertension, "primary."
Hypertrophy and dilatation of the heart.
Arteriosclerosis.
Chronic passive congestion.
Terminal infection?

*Anatomical Diagnosis.*—(Hypertension.)
Great hypertrophy and dilatation of the heart.
(No Arteriosclerosis)
General chronic passive congestion.
Hydropericardium.
Beginning hydrothorax.
Ascites.
Anasarca.
Chronic pleuritis.
Lymphoma of the mesenteric and retroperitoneal lymph glands.
Slightly defective closure of the foramen ovale. (Not abnormal).

DR. RICHARDSON: An enormously stout man. We were not permitted to examine the head. There was massive edema of the lower extremities and genitals. The abdomen was distended, but the wall was not rigid. There was also edema of the upper extremities and the dependent portions of the trunk. The face and ears were purplish. I go into this a little more in detail because it is a classical picture of hypertension.

The muscles were large. The subcutaneous tissues were wet. The peritoneal cavity contained at least 2000 c.c. of thin pale clear fluid. The peritoneum and appendix were negative. The mucosa of the esophagus was pale bluish red, otherwise negative. The stomach and intestines showed a very striking picture of chronic passive congestion,—the purplish red velvety mucosa oozing thin bloody fluid. This was even better marked in certain portions of the small intestine than in the stomach, but was extraordinarily well marked all along the tract.

The right pleural cavity contained a small amount of fluid, the left 300 c.c.—beginnings of hydrothorax.

The diaphragm on the right was at the fifth rib, on the left at the seventh. There were no pleural adhesions at the apex, but some

posteriorly to the diaphragm and some to the pericardium on the right. On the left side there were scattered adhesions to the diaphragm and the pericardium.

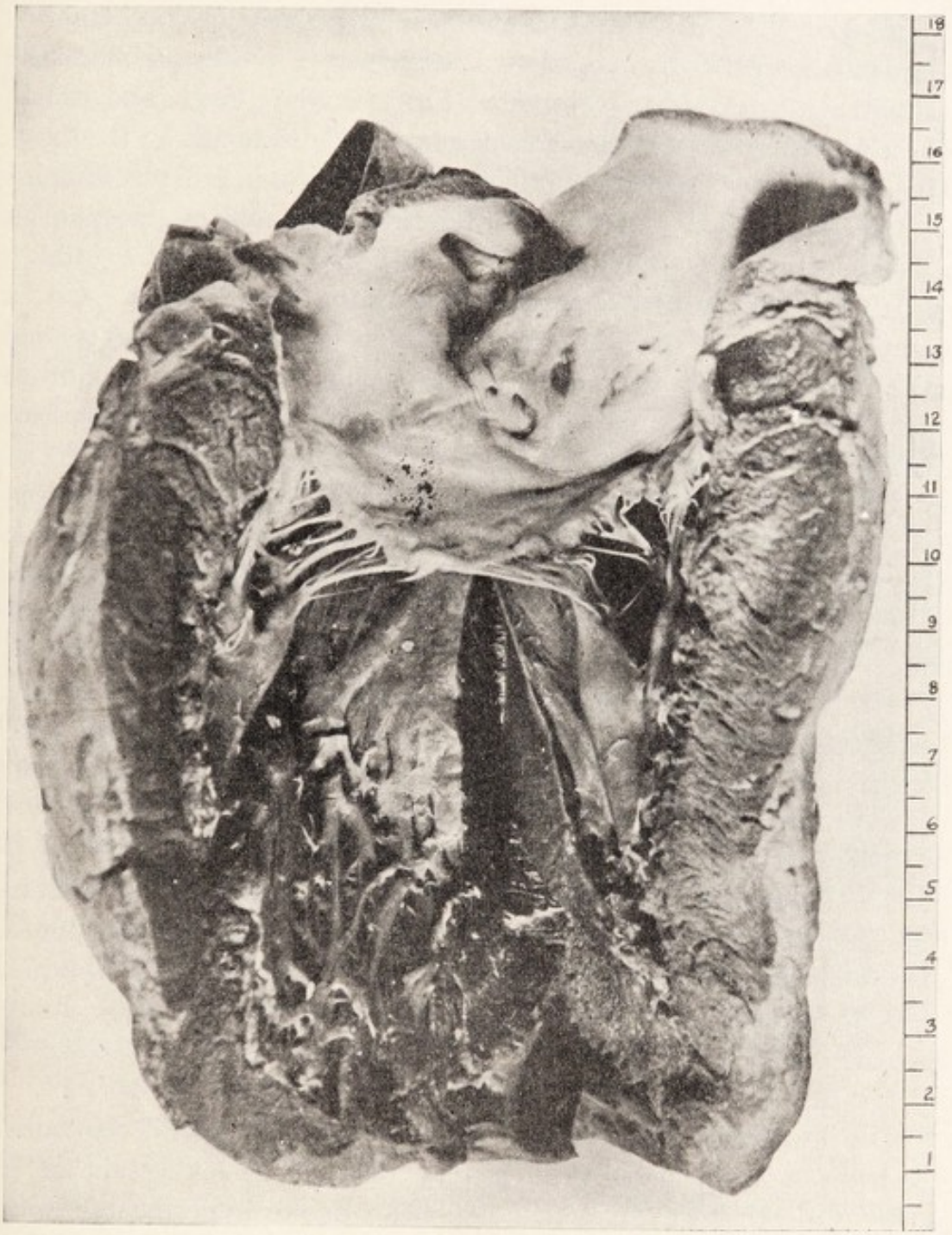


FIG. 91.—Necropsy 4769.—Hypertensive heart disease with great hypertrophy and dilatation of the heart. Heart weighed 960 grams. No nephritis. No arteriosclerosis. Valves negative. (Photograph by Lewis M. Adams. Dr. Oscar Richardson.)

The mucosa of the trachea and bronchi was brownish red. They contained much thin bloody frothy fluid. The bronchial glands were plump, brown-red and juicy. The lung tissue was spongy to slightly

leathery, brown-red, and yielded a moderate amount of brownish-red frothy fluid,—chronic passive congestion.

The pericardium contained 300 c.c. of thin pale fluid,—hydropericardium. The heart weighed 960 grams,—greatly enlarged. (See Figure 91.) The myocardium was generally thick, of good consistence, pale brown-red. The right ventricle was five to six mm., the left sixteen mm. The columnae carneae were large and thick. There was slight dilatation on the left, marked dilatation on the right. The cavities contained much blood and blood clot, in greatest amount on the right. The valve circumferences were: mitral 11.5, aortic 7.5, tricuspid 13.5, pulmonary 8.5 cm. The valves were frankly negative. The auricular appendices were free. There was a slightly defective closure of the foramen ovale. The aorta and great branches showed only a very slight amount of fibrous sclerosis. Coronaries, capacious, free, and the circulatory apparatus elsewhere negative.

The liver weighed 2307 grams, and showed chronic passive congestion. The gall-bladder, except for a little edema of the wall, was negative. The spleen weighed 430 grams,—moderately enlarged, the tissue plump, elastic, brown-red, bloody,—chronic passive congestion.

The kidneys weighed 427 grams; the capsules stripped readily, the surfaces were bluish-brown-red and smooth, the tissue of good consistence, plump, wet. The vessels were engorged, the pelves and ureters negative,—chronic passive congestion.

There was a slight hydrocele on each side.

The inguinal rings were large but free and there was no definite pouching of the peritoneum. The mesenteric and retroperitoneal glands generally were markedly enlarged up to 4½ cm.,—lymphoma.

A PHYSICIAN: You did not find any reason why the urine should be loaded with pus?

DR. RICHARDSON: No.

DR. CABOT: I think everybody here present should remember the looks of this heart and this history, because it fixes certain things once for all: that we can have a huge heart due to hypertension so far as we know, with no nephritis, no arteriosclerosis, no valve lesion,—nothing. Is it rare? No. I suppose it is the commonest of all types of heart disease. It is the commonest of all heart disease. In my opinion the nephritis or arteriosclerosis which often goes with it is not the important thing, and no one knows what causes the hypertension which, therefore, is called "essential."

A PHYSICIAN: I think that is a fact that is very little appreciated by the general profession. What details would you require added to the picture to make a diagnosis of arteriosclerosis?

DR. CABOT: None. As I said, I expected to find some arteriosclerosis. If we looked in the retina and saw it, or if we felt it in the brachial, we should say he had arteriosclerosis there but we should not know that it was present elsewhere or in any important degree. We have no diagnostic signs of generalized arteriosclerosis.

A PHYSICIAN: Does that mean that the diagnosis of arteriosclerosis is apt to be in fact this condition?

DR. CABOT: Yes.

### Necropsy 960

A railway engineer of forty-eight was brought to the Accident Room unconscious. A policeman said the patient suddenly dropped while at work in the dynamo room of the railway and was picked up in the same condition as at entrance. Later a friend said that the patient had been working all the time for twenty years and had always been well. He drank a little ale and beer. After dinner the day of admission he had some discomfort.

Examination showed a very obese man with cyanotic face and noisy, labored, jerky respiration. There was some blood on his lips and mouth. His tongue was bitten in two places. His breath smelt alcoholic. A few coarse râles were heard in the lungs in front. The back was not examined. The apex impulse of the heart was not recorded. The left border of dullness was about at the nipple line. There was no enlargement to the right. A systolic murmur was heard all over the precordia. The aortic second sound was accentuated. The pulses were regular, very full volume, very high tension. The artery walls were sclerosed. The pupils were small, equal, and did not react. The other reflexes were normal. Supraorbital pressure was negative. There was no edema. The temperature, pulse and respirations were not recorded.

While the examination was being made the breathing stopped. It was soon restored by artificial respiration and oxygen. About fifteen minutes later it again ceased. The patient vomited several times, brownish material with the odor of beer. He was catheterized, bled about a pint, and infused with normal saline while the bleeding was being done. His condition grew steadily worse, he became very cyanotic, and died in less than two hours after his admission.

*Clinical Diagnosis.*—Chronic diffuse nephritis.
Uremia.
Hypertrophied heart.
*Anatomical Diagnosis.*—Hypertrophy and dilatation of the heart.
Chronic passive congestion of the lungs.
Soft spleen.
Hypernephroma of the right kidney.
Cholelithiasis.
Foci of fat necrosis in pancreas.

The heart weighed 694 grams. The cavities generally were large. The wall of the left ventricle opposite the base of a papillary muscle was about 17 mm. thick, the wall of the right ventricle 6 mm. thick. There was *no arteriosclerosis or nephritis.* The hypernephroma did not diminish the amount of secreting renal tissue.

### Necropsy 1854

An American weaver of thirty-nine entered December 31. Two of his brothers died of consumption. He had measles in childhood and frequent sick headaches in his youth. At twenty-five he had gonorrhea and at thirty-one typhoid fever. Since that illness he had never been strong, and his headaches had been more severe. For eight years his eyes had been prominent. At thirty-six he had "malaria,"—fever without chills for six weeks. He took a glass of whiskey a week and an occasional glass of beer.

Three months before admission he began to have severe constant generalized headache with some nausea. He vomited every other day and felt weak and played out. He gave up work and was in bed off and on for a day or two at a time. At the end of a month the headache left him, but the vomiting continued. He lost weight and strength. Four weeks before admission the vomiting became more severe. Since that time he had vomited nearly everything taken. His bowels were constipated, not moving for four days. He urinated three or four times at night. Two weeks before admission he began to have dyspnea, which had grown steadily worse. Six days before admission he began to have edema of the feet and legs which increased rapidly for three days, then subsided somewhat. For two weeks he had had a considerable nosebleed every two or three days.

Examination showed a very round shouldered, fairly well nourished man looking haggard and sick. His skin was dark, his mucous membranes pale. The teeth were poor, many missing. The throat

showed three small ecchymoses on the uvula and the posterior pharyngeal wall. The apex impulse of the heart was seen and felt in the fifth space 4½ inches to the left of the midsternum, half an inch outside the nipple line, coinciding with the left border of dullness. The right border of dullness was an inch and a quarter to the right of midsternum. The action was regular, the sounds rather loud, the aortic second sound sharp and ringing. A rather hissing early systolic murmur was heard slightly over the whole precordia, best at the apex, and transmitted a short distance into the axilla. The pulses were normal. The systolic blood pressure was 210. The lungs showed a few fine moist râles at both bases behind. The liver dullness extended to the iliac crest. An indistinct edge was felt. There was much tenderness over the liver. There was marked soft edema of the feet. The pupils reacted very slightly. The other reflexes were normal.

The temperature was 97° to 98.3° with a terminal rise to 102°. The pulse was 97 to 110. The respirations were 9 to 31. The output of urine was normal, the specific gravity 1.007–1.800. There was a slight trace of albumin at both of two examinations. The sediment showed pus at one. The hemoglobin was 55%, the leucocytes 8600, the polynuclears 90%, the reds 2,972,000, with no marked achromia, poikilocytosis or polychromatophilia and no stippling.

The patient was unable to lie down, slept very poorly even with opiates, and grew rapidly worse. He vomited several times. The edema diminished. His respirations slowed down to six or eight a minute with no opiates. Unless roused he lay with his eyes rolled up and lids half closed. When roused, however, the respiration improved and he looked bright. The night of January 3 he became very delirious and sank rapidly. The morning of January 4 he quietly died.

*Clinical Diagnosis.*—Chronic nephritis.
Uremia.
Myocarditis.
Arteriosclerosis.

*Dr. Cabot's Diagnosis.*—Chronic nephritis.
Uremia.
Hypertrophy and dilatation of the heart.
Chronic passive congestion.
Terminal infection.

*Anatomical Diagnosis.*—Chronic arteriosclerotic nephritis.
Hypertrophy and dilatation of the heart.

Septicemia, streptococcus.

Pneumonia.

Soft hyperplastic spleen.

Foci of obsolete tuberculosis of the lungs.

DR. RICHARDSON: The head was not examined in this case. There was a slight amount of fluid in the peritoneal cavity. The liver at the time of necropsy was only three fingers down in the right mammillary line. It makes a difference where the line is that is taken when the liver is measured. The distance between the lower margin of the right lobe of the liver in the anterior axillary line and the crest of the ilium is pretty short. But if taken in the right mammillary line the liver might have been much farther up.

DR. CABOT: We have noticed over a long period of time a great many observations in which clinically we got a very big liver and post mortem you got only passive congestion, no enlargement. Are the anatomical conditions such that you can easily imagine a liver greatly engorged in life and collapsing after death?

DR. RICHARDSON: Yes, to a certain extent, but not so marked as in this case.

DR. CABOT: Have you ever tried the experiment of injecting the liver to see if it can be made to reach this size?

DR. RICHARDSON: No.

DR. CABOT: It is always in this type of case that these discrepancies come. They do not come in leukemia, where we have a tough firm liver. It is always in the circulatory cases that we differ from the post-mortem findings.

DR. RICHARDSON: The diaphragm on the right was at the fifth rib, on the left at the fifth interspace. That is about right, although with these levels the pleural cavities were full of fluid,—hydrothorax. The pleura was coated with fibrinous exudate in places, and the lungs showed here and there small focal areas of frank pneumonia, otherwise some congestion.

There was moderate hydropericardium. The heart weighed 740 grams. That is a pretty large heart. The myocardium was pale, opaque. Some question was raised at the time about the myocardium. We made a histological examination. There was no increase of interstitial tissue, but a note was made that the individual fibers seemed to be enlarged. The cavities were increased in size. The mitral valve measured twelve cm., the aortic eight, the tricuspid thirteen and a half. But there were no lesions of the valves, nothing within the heart to account for its size.

The aorta was fairly smooth; no definite amount of arteriosclerosis except that in the renal arteries there was some, not very marked. I mean by that, the artery that runs from the aorta to the kidney and the first branches where it breaks up into kidney substance. As a matter of fact we found later that there was considerable sclerosis of the arteries in the kidney substance.

The liver weighed 2190 grams. That is a large liver, but still only three fingers down. The tissue was slightly doughy and cinnamon brown in color.

The spleen weighed 240 grams, slightly soft, the follicles and trabeculae visible; some congestion and some softness from the infection present, a streptococcus septicemia.

The combined weight of the kidneys was 200 grams. That is 740 grams of heart against 200 grams of kidney. Even with small kidneys that is a markedly hypertrophied heart. The question from the anatomical standpoint is raised whether there may have been an earlier "essential" hypertension. The capsules came off fairly easily, leaving granular surfaces generally. The tissue was tough, the markings indistinct, the cortex varying from two to four mm., the cut ends of the vessels prominent. On the whole, macroscopically an arteriosclerotic nephritis, and the histological examination bore that out. There was no evidence of any glomerulonephritis.

The gastro-intestinal tract showed a velvety mucosa, dull reddish, juicy,—passive congestion.

### Case 3971

An Irish stationary engineer of sixty-four entered July 28. Two days before admission he was seized with acute pain in the epigastrium. He was nauseated, and after a while began to vomit a green watery fluid. He continued to vomit frequently all the next day. The pain became localized in the right lower quadrant. He had no appetite, and no movement of the bowels in spite of five soap-suds enemata. During the day his abdomen became slightly distended and "sore all over." The morning of admission he awoke feeling nauseated, but did not vomit. He had never had a similar attack.

Upon examination he was well nourished. The lungs were normal. The heart was moderately enlarged to percussion. The aortic second sound was accentuated. A systolic murmur was heard at the apex, a systolic and a diastolic at the base. The radial arteries were palpable, the brachials tortuous. The systolic blood pressure was 174, the diastolic 122. The abdomen was somewhat distended,

very tympanitic, with questionable shifting dullness; no liver tympany. The right side of the abdomen was spastic. There was slight tenderness throughout, decidedly more marked in the right lower quadrant. The genitals and extremities were negative. The rectum was ballooned, the prostate slightly enlarged. The pupillary reactions and the other reflexes were normal.

Before operation the temperature was 99.6°, the pulse 85; the respirations are not recorded. The leucocyte count was 18,000. No specimen of urine could be obtained.

Operation was done at once. The abdomen was found filled with a large amount of dark, old blood, mostly liquid. The appendix was normal. Stones were felt in the gall-bladder, but the gall-bladder felt normal. The condition of the pancreas could not be definitely determined, but was thought to be the cause of the trouble. There was no evidence of obstruction, malignant disease, or active hemorrhage.

The patient returned to the ward apparently little worse for the operation. Considerable staining persisted; also draining of bile. July 30 there was more distension. No peristalsis was heard with the stethoscope. The patient grew rapidly worse and died that day.

*Clinical Diagnosis (from Hospital Record).*—Acute pancreatitis.

*Dr. Hugh Cabot's Diagnosis.*—Mesenteric thrombosis?
Acute hemorrhagic pancreatitis?
Intestinal obstruction?
Arteriosclerosis.

| | |
|---|---|
| *Anatomical Diagnosis.*—1. Primary fatal lesions. | Cholelithiasis.<br>Cholecystitis with necrosis and perforation of the bladder wall.<br>Hemorrhages into the gall-bladder and the peritoneal cavity. |
| 2. Secondary or terminal lesions. | Acute peritonitis.<br>Arteriosclerosis.<br>Hypertrophy and dilatation of the heart.<br>Hemorrhagic edema of the lungs. |
| 3. Historical landmarks. | Slight scoliosis.<br>Operation wound, exploratory laparotomy. |

The heart weighed 520 grams. The myocardium was pale brown-red and of fair consistence. The right ventricle wall was 3 mm. thick, the left ventricle wall 11 mm.

Here is a case of purely latent or inactive hypertrophy and dilatation of the heart, with hypertension probably chronic. We have no evidence that the heart was even a minor contributary factor in his death.

### Autopsy 3034

An English blacksmith of fifty-four entered March 18. Except for the diseases of childhood the patient had been well until the present illness. His wife had had one miscarriage and lost one child an hour after birth. For a year he had noticed occasional dyspnea on heavy exertion. The attacks seemed to be related to cold, disagreeable weather. For two months he had had increase of dyspnea on exertion and excitement. Walking up hill or in the face of the wind seemed to affect him more than heavy work. He had some palpitation and slight dry cough, occasionally at night raising white phlegm. Within two weeks he had had to increase to two pillows. He stopped work May 8 on account of dyspnea. He had made little or no improvement, had lost appetite, and had epigastric distress relieved by considerable eructations of gas.

Examination showed a well nourished, strong man. The mucosae were slightly cyanotic. The teeth were decayed. There was marked pyorrhea. The apex impulse of the heart was faintly felt in the fifth space 11 cm. to the left of midsternum and a centimeter and a half outside the nipple line, coinciding with the left border of dullness. The right border was 3 cm. to the right of midsternum. The action was occasionally irregular. There was a strong suggestion of gallop rhythm, and occasional reduplication of the second sounds at the base. The sounds were distant and varied in intensity. The pulmonic second sound was accentuated. A soft systolic murmur was heard over the lower precordia, transmitted to the axilla. The pulses were of poor volume and fair tension. The vessel walls were slightly thickened. The blood pressure was 115/95; 130(?)/95. There was prominence of the second ribs and upper sternum, with moderate lower sternal retraction. There was dull tympanitic resonance throughout, except at the right base posteriorly and in the right axilla, where it shaded off into dullness. Expiration throughout was prolonged and roughened. There were occasional râles throughout at the end of inspiration, a few moist sticky râles at the

right base with both inspiration and expiration. The abdomen was held slightly rigid, especially in the epigastrium, where there was some protuberance, with dull tympany. The liver dullness extended from the fifth rib to 1.5 cm. below the costal margin. The edge was not felt. The genitals were normal except for a small right varicocele. There was moderate edema of both upper and lower legs, very little at the ankles or over the feet. The pupils were irregular, otherwise normal, as were the reflexes.

The temperature was 95.3° to 98° until March 3, then rising rapidly to 105.8°. The pulse was 88 to 120. The respirations were 20 to 48; until March 3 not remarkable. The output of urine was 3 to 28 ounces, the specific gravity 1.026–1.030. There was a slight trace of albumin at the last of five examinations, the slightest possible trace of bile, leucocytes and hyalin and granular casts at the last two. The hemoglobin was 75%, the leucocyte count 19,600–8000–27,600, the polynuclears 74%. A Wassermann was negative. The fundi were normal.

March 21 the edema was gone, but the patient still felt very weak and ill. Digipuratum was not well borne; the heart became more irregular, the stomach upset. After three days the digitalis was omitted. A surgical consultant declined to make a diagnosis, but agreed to operate if the condition warranted it later. March 30 the left border of dullness was four centimeters outside the nipple. The patient had occasional attacks of great weakness, precordial distress, belching of gas and labored breathing lasting five to thirty minutes, relieved by morphia or nitroglycerin. By April 3 these attacks were occurring twice to four times during the twenty-four hours, chiefly at night. He became jaundiced, the liver enlarged and very tender. Nothing was found in the lungs. April 9 he died.

*Clinical Diagnosis (from Hospital Record).*—Arteriosclerosis.
Coronary sclerosis.
Myocarditis.
Decompensation.
Terminal pneumonia.

*Dr. Richard C. Cabot's Diagnosis.*—Arteriosclerosis.
Coronary sclerosis.
Hypertrophy and dilatation of the heart.
Chronic passive congestion.
Cholangitis.
Cholecystitis.

*Anatomical Diagnosis.*

| | |
|---|---|
| 1. Main cause of death. | Streptococcus sepsis. |
| 2. Secondary or terminal lesions. | Slight arteriosclerosis.<br>Hypertrophy and dilatation of the heart.<br>Chronic passive congestion, general. |
| 3. Historical landmarks. | Slight icterus.<br>Slight chronic perihepatitis and splenitis.<br>Chronic pleuritis.<br>Meckel's diverticulum.<br>Horseshoe kidney. |

The heart weighed 699 grams. The organ was considerably enlarged and the chambers were distended with blood clot. On section the myocardium generally was rather lax, pale brownish red and slightly cloudy. In the region of the upper part of the posterior wall of the left ventricle there were two areas of myocarditis, one of which measured 3½ by 1½ cm. and the other 4 by 2½ cm., which showed through the epicardium. In the posterior wall of the right ventricle there was an area about 2½ by 1½ cm. similar to the others. These areas showed in their peripheral portions a narrow irregular dirty pinkish brownish yellow margin with pale brownish yellow central portions.

The kidney structure appeared normal.

*Note by Dr. Cabot.*—In view of the anatomical findings, his chronic dyspnea probably depends on hypertension and its results. The amount of arteriosclerosis present does not account for so much hypertrophy and weakening of the heart. The icterus is probably to be explained as a manifestation of septicemia. The surgical consultant showed good judgment in refusing to operate.

### Necropsy 3865

[The following case though not one of hypertensive heart disease is included here because I do not know where else to put it. The enlarged heart certainly seemed a factor in bringing about the patient's death. It was not enlarged by mechanical obstruction or leak. The blood pressure was low throughout his illness. The case presents a challenging problem.]

An American teamster of thirty-two entered June 14, 1913, five years before his final admission, for relief of vomiting, anorexia, weakness and dyspnea. He had had gonorrhea at fourteen. At twenty-eight he was ill two weeks with "typhoid malaria." For

the past two or three years he had had stomach trouble with occasional vomiting and diarrhea. He became pale and yellowish. His stools were sometimes black. Six weeks before admission he began to have stomach trouble, vomited nearly every day, and had sore gums, dizziness and palpitation. He grew pale and weak, lost about forty pounds in weight, and at entrance was unable to walk fifty feet.

Examination showed a very pale man. The heart sounds were irregular and rapid. There was a loud blowing systolic murmur over the pulmonic area, not transmitted.

The temperature was 99° to 102.2° for the first four weeks, then normal. The pulse was 80 to 120. The respirations are not recorded. The hemoglobin was 10% to 65%, the leucocytes 4500 to 8800, the polynuclears 49% to 60%, the reds 700,000 to 1,700,000. The smear showed great variation in size, shape and staining, much stippling and polychromatophilia. The platelets were rare.

The patient made marked improvement while taking Fowler's solution, gained thirty-two pounds in weight, and was discharged relieved August 3.

After leaving the hospital he felt much better for four weeks. From September to December and from March to July he had had attacks similar to the previous ones. The following autumn he grew progressively weaker and more dyspneic.

December 21, 1914, he returned. During his stay in the hospital he grew progressively worse. January 5 he was transfused with 600 c.c. of blood. Splenectomy was done, with uneventful recovery. The red count was 2,300,000 at entrance, 1,700,000 at discharge.

After leaving the hospital he felt well until May; then he was weak and vomited until August, when he began to improve. In December he went to work in a shop, weighing 206 pounds, his best weight. After six weeks he again began to lose weight and strength. May 28 he had fainting attacks, with a burning sensation in his chest, lasting about fifteen minutes. June 1, 1916, he reentered the hospital.

June 11 he received 600 c.c. of blood by transfusion. For the next three days he suffered from hallucinations, and while demented tried to injure a neighbor and to kill himself.

The hemoglobin rose from 12% at entrance to 56 % at discharge. The leucocyte count was 4000 to 16,200. The reds were 792,000 to 2,128,000. The smear at entrance showed marked achromia, polychromatophilia, 3 normoblasts and one megaloblast in 100 cells. The platelets were much decreased. At discharge, June 18, there was tendency to macrocytosis of reds. No blasts were seen.

After leaving the hospital he gained a pound a day for twenty days. He worked out-cf-doors during the summer. Four weeks before readmission he began to grow weaker and had a great deal of gas from the stomach. At readmission, October 16, 1916, he could barely walk.

During his stay in the ward he continued to fail until October 11, when transfusion was done. October 29 a second transfusion was done. The second seemed to have a little effect, but the bone marrow did not respond as on previous occasions. The reds rose from 1,228,000 to 1,628,000 at discharge, the hemoglobin from 34% to 35% at discharge, the leucocytes from 7400 to 6600 at discharge. The smear showed seven blasts per 100 cells. November 5 he was discharged.

For two weeks after leaving the hospital he felt well. Then he had severe gas pain for four or five hours every day without much relation to meals. In the late autumn he felt very weak and had increasing numbness of the hands and feet. December 8 he was hysterical. December 9 he entered the hospital for the fifth time.

At entrance the hemoglobin was 20%, the leucocytes 3600, the reds 1,200,000. Transfusion of 600 c.c. was done December 21. At discharge December 24 the hemoglobin was 33%, leucocytes 8400, reds 1,500,000. The smear at entrance showed marked variation in size and shape, moderate numbers of Jolly bodies, two blasts; at discharge there was more stippling, Jolly bodies increased, polychromatophilia, five blasts.

March 6, 1917, he entered a sixth time, having felt fairly well and gained ten pounds since his discharge. Ten days before readmission he began to fail rapidly, to have much flatulence and dyspnea and to be very weak.

March 3 transfusion of 600 c.c. of blood was done. He was discharged relieved March 11.

September 11, 1918, he came to the Emergency Ward to be transfused, very pale and in a state of collapse. He died before he reached the ward.

*Discussion by Richard C. Cabot.*—The history of the first entry gives undoubtedly a diagnosis of pernicious anemia. It is a clear and typical history. The remarkable thing about it is that the signs of pernicious anemia appeared about seven years before his death. That is a longer course than most of the cases run. The intermission between the first and second entries is a little more than usual. There is no question, however, about the diagnosis. The post-

mortem examination ought to show fatty degeneration of the organs, a slightly enlarged heart, very possibly fatty degeneration in it, and hyperplastic marrow.

*Clinical Diagnosis (from Hospital Record).*—Pernicious anemia.

*Dr. Richard C. Cabot's Diagnosis.*—Pernicious anemia.

Fatty degeneration of the organs.

Slight hypertrophy and dilatation of the heart.

Possibly fatty degeneration of the heart.

Hyperplastic bone marrow.

*Anatomical Diagnosis.*—

| | |
|---|---|
| 1. Primary fatal lesion. | Pernicious anemia. |
| 2. Secondary or terminal lesions. | Hyperplasia of the bone marrow. |
| | Fatty degeneration of the myocardium. |
| | Hypertrophy and dilatation of the heart. |
| | Edema of the lungs. |
| | Slight hydropericardium. |
| | Very slight hydrothorax. |
| | Very slight anasarca. |
| 3. Historical landmarks. | Scar of old operation wound, splenectomy. |
| | Slight chronic pleuritis, left. |
| | Hemosiderosis of the liver. |

DR. RICHARDSON: A point which has been brought out at this necropsy table for some years is the association of hypertrophy and dilatation of the heart with pernicious anemia. Here we have a very remarkable instance. The heart weighed 710 grams. The aorta, its great branches and the valves were negative, and there was nothing in the kidneys to account for the marked hypertrophy of the heart. The myocardium showed well marked fatty degeneration, but no evidence of myocarditis. This was the largest heart I have ever seen associated with pernicious anemia, and with no other basis of hypertrophy.

The kidneys were large, weighing 532 grams (normally 200–400), with a wide cortex; but microscopically they showed nothing remarkable.

The hyperplastic marrow was typical of this disease,—meaty, dark raspberry red, and the microscopical examination showed numerous megaloblasts.

The hydropericardium, hydrothorax and anasarca indicate that the heart had become a little decompensated.

The lesions as tabulated in the anatomical diagnosis present a typical anatomical picture of pernicious anemia.

DR. CABOT: We had more hope in the combination of splenectomy and transfusion four or five years ago than we have now. After splenectomy the transfusion holds them a little longer than if transfusion were done without splenectomy. If there is some one at hand who is accustomed to doing splenectomy, it does not add any considerable risk to have it done. My feeling is that if I had pernicious anemia I should have my spleen taken out, although it would not be brilliant therapeutics. I do not believe this man would have lived so long if he had not had splenectomy.

This man's age is a notable fact. The average age for pernicious anemia is between forty and fifty; rarely anywhere near thirty.

It is a fact of some interest that we have almost never made a wrong diagnosis of this disease in this hospital. Some years ago I figured up the percentage of failure in diagnosis here,* from 16 % of right diagnoses in acute nephritis up to practically 100% of right diagnoses in this disease, in typhoid fever (92%) and diabetes (95%). That is of importance. We ought to realize just where we are strong and where we are weak.

A PHYSICIAN: How about splenectomy in cases of leukemia?

DR. CABOT: I do not see the benefit of splenectomy in leukemia; at least up to date I do not know of any. I regret that the operation has been popularized by the example of the Mayo clinic.

### Necropsy 3845

An American tanner of fifty-eight entered May 17 for relief of dyspnea and palpitation. His father died of heart trouble, his mother of kidney trouble. He had all the diseases of childhood, including scarlet fever and diphtheria. He had occasional attacks of sick headache when a boy. He had gonorrhea at twenty-one and again at twenty-two. He denied syphilis. His wife had four or five miscarriages before the birth of two children who were living and well. His general health had been excellent until the present illness. He had been on a good many drinking parties, but never was a steady heavy drinker. He smoked a great deal until the present illness, and chewed one plug a day. He drank more than a gallon of coffee

*R. C. Cabot, Diagnostic Pitfalls Identified During A Study of 3000 Autopsies. Jour. Am. Med. Assoc., Dec. 28, 1912, Vol. LIX, pp. 2295–2298.

a day until a year before admission. For a year he had taken none. Until the present illness he used a great deal of salt.

A year and a half before admission he began to feel his heart beating, and was short of breath on any exertion. These symptoms gradually grew worse, and forced him a year ago to give up his work. For a year he had urinated two or three times at night. For the same period he had been taking digitalis in various amounts. Exertion brought on occasional attacks of shortness of breath with some dizziness. With them was some pain in the stomach region, not severe. These attacks became more frequent until they sometimes came on without exertion. In the intervals the dyspnea and palpitation grew gradually worse. At times he noticed some puffiness about the eyes. Six months before admission he suddenly began to have blurring in front of the right eye. Since that time he had been on a low-protein salt-free diet and had taken an occasional purge. His condition however grew gradually worse. His weight two years ago was 240 pounds; six months ago, 170 pounds.

Examination showed a fairly well nourished man with slight exophthalmos. The skin was dry. There was much pyorrhea with sordes. The apex impulse of the heart was felt in the sixth space 15 cm. to the left of midsternum and 6.5 cm. outside the nipple line, coinciding with the left border of dullness. The right border was one cm. to the right, the supracardiac dullness 5 cm. There was no shift in the left lateral position. The action was irregular, with occasional premature beats. The sounds were of good quality. Both the pulmonic and the aortic second sounds were loud and ringing, the pulmonic accentuated. There was a blowing systolic murmur at the apex and a systolic at the base. The artery walls were markedly palpable, the brachials very tortuous. The lungs, abdomen genitals, extremities, pupils and reflexes were normal.

The temperature was 96.8° to 99.6° until June 19. The pulse was 100 to 59, the respiration 15 to 26. The systolic blood pressure was 200 to 230, the diastolic 100 to 140. The output of urine was 23 to 63 ℥ till June 17, the specific gravity 1.006 to 1.014. The urine was cloudy, alkaline at one of three examinations, with a trace to a slight trace of albumin at all, rare granular casts at one, leucocytes at two. The renal function on May 20 was zero; May 22, it was 5%. The hemoglobin was 75%, the leucocyte count 5200 to 11,000, the polynuclears 88%. The urea nitrogen was 57 to 153 mgm. per 100 c.c. of blood. A Wassermann test was negative. The fundi showed well marked retinitis of the right eye with a few retinal hemor-

rhages, and star-shaped arrangement in the macular region. The left eye showed a few hemorrhages.

The dyspnea became much less. May 25 the patient was up in a chair. After this however he was troubled with nausea and vomiting and a "bad taste." There was considerable improvement after a sweat bath. Then he became weak and tired and had occasional nausea and vomiting. His mentality became cloudy. He was delirious at night. His eyes looked staring. His breath was ammoniacal. June 19 the temperature dropped to 93.3°, the pulse to 60. He became practically comatose and bled profusely from the nose and mouth and beneath the finger nails. June 21 he died.

*Clinical Diagnosis (from Hospital Record).*—Chronic nephritis.
Arteriosclerosis.
Uremia.

*Dr. W. H. Smith's Diagnosis.*—Chronic nephritis.
Uremia.

*Anatomical Diagnosis.*—Arteriosclerosis of the aorta and its great branches.
Arteriosclerosis of the vessels of Willis.
Arteriosclerotic nephritis.
Hypertrophy and dilatation of the heart.
Edema piae.
Slight chronic pleuritis, right.
Obsolete tuberculosis of a bronchial lymph node.
Papillary adenoma of the right kidney.

Dr. Oscar Richardson: The background was one of arteriosclerosis. The heart weighed 625 grams (normally 200–300). The myocardium was generally thick, and at the time of necropsy the cavities showed considerable dilatation. The valves were negative. The coronary arteries showed a rather peculiarly arranged sclerosis. At first they seemed to be fairly capacious and free, but rather tortuous. On laying them open fibrous plaques were found scattered along the walls, then a piece which was fairly good, then plaques again. This peculiar arrangement caused tortuosity of the vessel without marked decrease in its lumen. The aorta and the great branches were capacious and showed marked fibrous, fibrocalcareous and atheromatous sclerosis. The vessels of Willis and their remote branches showed marked arteriosclerosis.

The kidneys weighed 160 grams. Those, of course, were very small organs. (Normally 200-400 grams.) The capsules were slightly adherent in places and the surfaces were finely granular and

red. The kidney tissue was tough, the markings more or less indistinct, and there was decrease in the width of the cortex. Here and there in the section surfaces the cut ends of the arteries showed sclerosis. In the pelvis of the kidney the mucosa in a few places showed small dark red hemorrhagic areas;—a small arteriosclerotic kidney. It is in this type of nephritis that you are likely to get blood in the urine.

**Case 4327**

An American carpenter of forty-two entered March 27, 1922, complaining of difficulty in breathing of two months' duration.

He had measles, mumps and chicken-pox in childhood, "typhoid fever and pneumonia" at sixteen, prolonged attacks of rheumatic fever at eleven years, nineteen and thirty-one. He had always urinated twice at night. He had sick headaches until he was twenty-two. A years before admission he had a severe electric shock and was in bed ten days with symptoms like those of the present illness. Best weight 220 pounds, weight last September 195, present weight, 176.

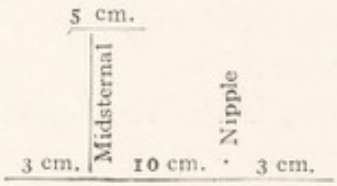



FIG. 92.

Two months before admission, after excessive work in cranking an automobile followed by a supper of oysters, he had dyspnea, somewhat relieved by lying on the right side and using two pillows. He also had palpitation and eructations of gas. During the following week he had dyspnea on exertion and at night, with gas, distress and distension after eating. When he milked his two cows he had sudden attacks of sharp pain with a sense of a heavy pressure across the front of the chest radiating down the right arm, and a choking sensation lasting half an hour and associated with dyspnea. After ten days of complete remission he again ate oysters and had the same trouble as before. The symptoms had persisted except for periods of two or three days once a week. Three times he had had nausea and vomiting at night. He had had hacking cough at night. For five weeks he had been taking twelve digitalis pills a week. He had also taken an average of three nitroglycerin tablets a day swallowed with water for attacks of sharp pain in the chest and right arm. The pain was relieved within half an hour. For three weeks he had been in bed.

For two weeks he had had an external hemorrhoid. Three days before admission he had a sudden onset of dull aching in the right side of the head. The left side of the head and neck and the left arm and leg became numb. His speech was mumbling, his jaw sagged, and his pupils were dilated. He was able to move all his muscles, though his limbs felt very heavy. In six hours this passed off entirely except for the numbness under the right arm, which lasted several hours longer. He had no edema.

Physical examination showed him to be well nourished. Cheeks very high colored. Skin dry. Mucosae red. Tongue showed two serpiginous white lines on the left edge. Apex impulse of the heart

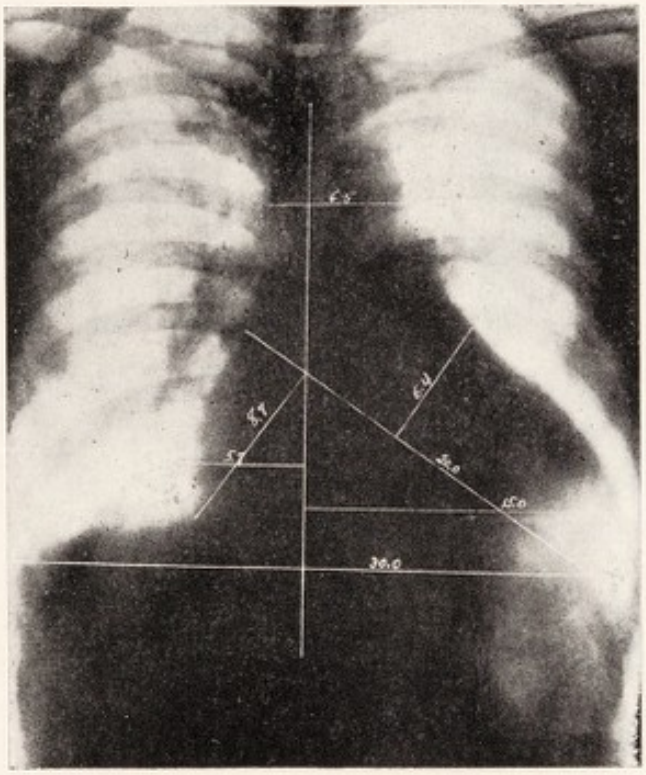


FIG. 93.—Necropsy 4327. Hypertrophy and dilatation of the heart, primary. Weight 703 grams. Mural thrombi in the right auricular appendix and the left ventricle. X-ray shows the heart shadow very much enlarged, especially to the left. Region of the right auricle also somewhat prominent. Enlargement appears somewhat symmetrical and is more suggetive of dilatation than of hypertrophy. Supracardiac shadow not abnormal. (Roentgenological Department, Massachusetts General Hospital.)

felt in the 5th space 9 cm. to the left. Percussion measurements as shown in the diagram. Action regular, with very rare extra systoles. $P_2$ slightly accentuated. No murmurs made out after slight exercise. Pulses and arteries normal. B.P. 100/70–110/80–105/85. Electrocardiogram showed sinoauricular tachycardia. Rate 110. Small complexes in all leads. P wave bifurcated and wide. Diphasic T. Abdomen and genitals negative. Inguinal rings enlarged. Rectal

examination: Dilated thrombosed external hemorrhoid. Extremites, pupils and reflexes showed nothing of significance.

Temperature. 96.4°–98.9° until March 30, then 98.9°,– 101.9° rectal to April 6; afterwards 97.4°–100.9. Pulse, 89–120. Respiration, 20–36. Urine: ℥12–28–6 in 24 hours. Sp. gr. 1034–1030. Cloudy at one of three examinations, alkaline at two, the slightest possible trace to a slight trace of albumin at all. Renal function 30%–50%. Blood: Hemoglobin 80%. Leucocytes 23,400–12,000. Polynuclears 66%. Reds and platelets normal. Wassermann negative. Non-protein nitrogen 33–40.1 mgm. Sputum blood-streaked. Few organisms. X-ray, see Fig. 93.

When the patient was asleep the breathing was Cheyne-Stokes with apneic periods of from five to fifteen seconds. At midnight the night of admission he complained of very severe pain in the left leg from the knee down. It was instantly relieved by sodium bicarbonate. Dr. Paul D. White found proto-diastolic gallop rhythm, mitral facies, prominence óf the left auricle by percussion, occasional premature beats. "In the presence of the weak heart action there is no mitral diastolic murmur evident to-day. The first sound has a rumbling character."

By the 31st the patient had difficulty in breathing during the day and looked very ill. The morning of April 1 he fainted on slight exertion. He was put on the "dangerous" list. The high leucocyte count was unexplained. April 3 Dr. W. H. Smith found a suggestive Broadbent. The right leg was markedly swollen, hot and painful, the tenderness over the deep veins extending up to midthigh. April 6 the temperature was normal, but the next day rose again to 99.9°. The heart sounds were weaker. The mental factor was very large. He had prompt relief from bicarbonate of soda, which he thought was morphia; no benefit from codein dissolved in milk. April 10 he was very ill. There was gallop rhythm with a probable diastolic rumble. That day he fell over, striking his head. He became extremely cyanotic and the left arm seemed weak. His accounts showed that he did not lose consciousness, and it was believed that he complained largely to obtain sympathy. April 12 he had to be catheterized because of edema interfering with micturition. April 14 he suddenly died.

*Clinical Diagnosis.*—Mitral stenosis.

Myocarditis.

Phlebitis of right popliteal.

Edema of the foreskin.

Acute endocarditis.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic adhesive pericarditis.

Hypertrophy and dilatation of the heart.

Acute endocarditis?

Passive congestion, general.

*Anatomical Diagnosis.*—Hypertrophy and dilatation of the heart.

Mural thrombi in the right auricular appendix and the left ventricle.

Infarcts of the lungs.

Infarct of the left kidney.

Chronic passive congestion, general.

Hydrothorax, right.

Slight ascites.

Anasarca.

Chronic pleuritis, left.

Small mass of persistent thymic tissue.

DR. OSCAR RICHARDSON: We were not permitted to examine the head. The face and ears were dusky to purplish, the skin generally sallow with a faint yellowish tinge,—an appearance suggesting the end conditions of heart cases. The dependent portions of the trunk pitted, and the lower extremities were swollen and pitted on pressure.

There were 500 c.c. of fluid in the peritoneal cavity—slight ascites. The gastro-intestinal tract was negative except for an astonishingly good picture of chronic passive congestion. That is, all the mucous membranes were dark red, velvety, oozing bloody fluid. The glands were negative. The liver was 15 cm. below the costal border. The diaphragm was at the sixth rib on the right and the sixth interspace on the left. That means something in the pleural cavities. We found 2000 c.c. of fluid in the right cavity, and on the left about 50 c.c. There was a little fibrous pleuritis on the right, on the left a few old adhesions. There was a bit of the thymus gland remaining. There was nothing in the lungs except chronic passive congestion and infarcts. That means of course that although there was chronic passive congestion we should still look to see if we could find any vessels plugged.

The pericardium contained 50 c.c. of fluid, and was smooth and shining. The heart weighed 703 grams,— a very large heart. The myocardium was of pretty good consistence, fair color, and I could make out no definite myocarditis whatsoever. The cavities were all markedly dilated and all contained a large amount of blood. If that indicates anything, he died in diastole. In the right auricular

appendix there were small thrombotic masses which with the condition of chronic passive congestion and emboli from them produced the infarcts in the lungs. In the apical region of the left ventricle there was a small adhering thrombotic mass.

The mitral valve measured 15 cm. There were no definite lesions. The aortic was 7 cm., the cusps 8 cm. That means the ring where it is attached to the lower end of the aorta,—what is called the aortic ring. A peculiarity was that there were only two large cusps to the aortic valve; there are usually three. In the regions of the conjoined margins these two cusps presented a little roll of fibrous tissue which might be a little fibrous endocarditis, small, chronic, only one cm. long, two or three mm. in the other dimensions. The tricuspid valve measured 16 cm. The mitral circumference was nearly as large as the tricuspid. The pulmonic was 9 cm. The coronaries were fairly capacious, free, negative. The foramen ovale was closed. The mitral and tricuspid valves except for great increase in their circumferences, were negative.

The ascending thoracic portion of the aorta showed a few small fibrous plaques, the arch and descending portion a slight amount of fibrosis. The aorta and great branches on the whole were rather small for the size of the man. That was the only thing about this apparatus worth noting.

The liver weighed 2110 grams and showed typical nutmeg markings. The gall-bladder, pancreas and ducts were negative. The spleen showed chronic passive congestion. The kidneys weighed 390 grams, were bluish-brown-red, large, and showed here and there one or two infarcts. These were due of course to emboli from the thrombotic mass in the left ventricle.

We put hypertrophy and dilatation of the heart first this time. Presumably the condition in his leg was due to a bit split off from the thrombus in the left ventricle passing into the vessels of the leg.

This heart was one of those to which we give the name of idiopathic hypertrophy. It is an extraordinarily good case,—a heart of seven hundred odd grams giving out with no definite cause.

DR. CABOT: I was obviously wrong in supposing pericarditis as the cause of heart hypertrophy. We found no cause. The valves were all big but normal. They were part of the hypertrophy and dilatation. We have no idea why this heart gave out. It does not come into any of the classifications. Of course he may have had a hypertension. It does not show in the arteries, does not show anywhere. There was no evidence of it while he was here.

DR. RICHARDSON: He had a low blood pressure.

DR. CABOT: While here. We should have to say it was high before he came here.

A PHYSICIAN: What was the cause of the liver?

DR. RICHARDSON: It was due to the chronic passive congestion, which was very great all the way through. [The assumption that hypertension had existed before this patient was examined may be false. If so the cause of cardiac enlargement and failure is a mystery.]

### Case 3742

First entry. An American schoolboy of sixteen entered March 5, two years before his final admission.

His mother died of Bright's disease.

He had had mumps and a fracture of the left thigh at the age of six.

A week ago he began to have involuntary purposeless movements of all his muscles, growing rapidly more violent.

He was well nourished. Restless, moving his head about occasionally and making frequent grimaces and purposeless movements of the body, arms, and hands; only slight movements of the legs and feet. Breathing irregular. Small reddish scaling area on left side of the lower jaw. Small palpable gland on right lower jaw. Glands the size of peas in axillae. Palpable glands in groins. Apex impulse of heart seen and felt in 5th space 10 cm. to the left of midsternal line, 2 cm. outside the nipple line, corresponding with the left border of dullness. Right border of dullness 3 cm. to the right of midsternal line in 4th space. Slight palpable thrill at the apex. Action slightly rapid. At apex first sound loud, sharp and reduplicated. With it was heard a harsh low-pitched systolic murmur faintly transmitted to the axilla. Much of the murmur was between the two first sounds. At the base was heard a loud harsh systolic murmur slightly louder in the pulmonic and transmitted to the neck. Second pulmonic sound greater than the aortic and accentuated. Pulses slightly rapid. Artery walls palpable. Lungs normal. Abdomen: Liver dullness from 6th rib to costal margin. Edge not felt. Genitals: Meatus reddened and swollen. Pupils normal. Reflexes not recorded.

Temperature 97.4°–100.8°. Pulse 69–95. Respirations 18–28. Urine normal amount. Sp. gr. 1013–1023. A large trace of albumin to the slightest possible trace at all of six examinations. A very rare red corpuscle at two. Blood: Hgb. 80%. Leucocytes 12,800–4200.

The patient threw himself about in bed, chafing his skin so that it was necessary to pin his legs into a set of pillows and use a pneu-

monia swathe on his chest and bandages on his arms. He was given about 40 minims of Fowler's solution daily. March 18 the loudest murmur at the apex was a presystolic and the first sound was no longer reduplicated. A soft systolic murmur was faintly heard at the apex, where a slight thrill was felt. There was a loud systolic murmur at the base, loudest in the pulmonic area, transmitted to the neck. He had a few boils. The chafing of the skin was now only slight. By March 29 there were very few involuntary movements. The excretion of urine was greater. The apex was 1¼ inches outside the nipple line. There were thrills and systolic and presystolic murmurs at the apex, a loud systolic murmur at the base, slightly louder in the aortic area than in the pulmonic area, heard in the neck. March 31 beside the murmurs of the apex a harsh systolic murmur was heard at the aortic area and to a less extent at the pulmonic, transmitted to the neck. The boils had healed, except one an inch and a half in diameter on the back of the neck. Under staphylococcus vaccine treatment this was practically healed by April 4. The urine was almost normal, and he was quite free from twitching. April 6 the apex impulse was 3¾ inches to the left of midsternum. There were definite double murmurs at the apex. That day he was discharged.

Second entry. April 15, two years later, he returned.

Since leaving the hospital he had felt absolutely well until six weeks ago, when he began to vomit his breakfast. Five weeks ago his face became bloated. This passed off in a week. Three weeks ago he woke up so bloated that he could hardly see. In a few days his legs swelled. His abdomen became distended, and at night the edema came up out of his legs into his back and chest. He had gained considerable weight. He had been living on milk, water and other liquids. His eyes blurred at times.

Physical examination as before except as noted. No glands palpable in axillae or groins. Apex impulse of heart in 5th space 12.5 cm. from midsternum, 2.5 cm. outside nipple line. Right border 4 cm. from midsternum. First sound at apex sharp and double. At the apex and transmitted toward the base with increasing intensity was a purring rough systolic murmur, loudest over the aortic area, heard in the neck and axilla. $A_2$ markedly accentuated. Pulses normal volume and increased tension. Abdomen full. Dullness in flanks and over pubes shifting with change of position. Fluid wave. Liver dullness 5th space to costal border. Edge not felt. Genitals: Penis and scrotum greatly swollen. Prepuce could not be retracted.

Very marked swelling of face, arms, hands, and very great soft edema of thighs and legs. Considerable edema of back and over abdominal and chest walls.

Temperature 96.5°–100°, with one rise to 101° May 24. Pulse 60–101. Respirations 11–31. Systolic blood pressure 135–210, diastolic 100. Urine: ℥ 5–48. Sp. gr. 1014–1027. Smoky, cloudy or turbid at all of thirty examinations. Albumin at all. Guaiac questionably positive at five, red blood corpuscles at fifteen others. Hyalin, granular and fatty casts. Stool: Guaiac strongly positive (estimated ¼–½ blood). Fundi apparently normal.

The patient sweat profusely in hot air baths and seemed to enjoy them. The amount of albumin in the urine fell from 1% to .4% by April 22. The edema slowly became less, though by May 4 the face was still puffy and there was a thick pad of edema over the lumbar region. The circumference of the abdomen was 78 cm., 7 cm. less than at entrance. May 6 the temperature jumped to 100° and continued 99.8° for the next two days. The patient was very active and delirious. Digipuratum was given. The amount of urine rose from 23 to 35 ounces. A little free blood now appeared in the urine for the first time. It was very hard to keep the bowels moving. He vomited a good deal. May 12 he was considerably better. The chest showed less edema than ever before, but the amount of urine was lower. The abdomen now measured 72.5 cm. He was somnolent and mentally confused. May 16 the amount of urine was increased and he was much brighter. His face was more puffy, perhaps because of the forcing of fluids. The twenty-four-hour amount of urine remained about ℥ xxx. Nevertheless he was gradually getting more and more edematous. Hot air baths were resumed. May 24 the temperature rose to 101° in the morning, but was 99° by night. The abdomen measured 78 cm. He gradually became more somnolent. Four days later the twenty-four-hour amount had fallen to ℥ 23. There was a marked systolic murmur at the base. On the 29th the abdomen was 71 cm. That day be became semicomatose and quite noisy. His blood pressure rose to 210 and he seemed to be going downhill. Venesection was done, ℥ xvi of blood removed and 400 c.c. of salt solution given intravenously. He began to rally slowly. As his breathing became labored and he wheezed a good deal, a tracheotomy layout was kept by his bed. The urinary output now averaged ℥ 20. June 5 the hot air baths were omitted because they exhausted him. June 11 he vomited. June 13 the amount of urine had jumped from ℥ 15, where it had been for a week, to ℥ 30.

The blood pressure was now 145. June 14 he vomited again. By June 17 he had gained a pound and a half in ten days and seemed much stronger. The blood pressure had risen to 155. The specific gravity of the urine was 1016. The patient was brighter than he had been for weeks, but looked bloated and pasty. During the next week he gained weight rapidly and his face and tongue became greatly swollen. After a hot air bath June 25 he had a convulsion which was followed by greatly increased respiration accompanied by loud stertorous dyspnea. He was unconscious and the dyspnea rapidly increased. Tracheotomy was done, relieving the dyspnea. In a short time he recovered consciousness. Later he breathed well through his mouth and no air came through the tube. He seemed very uncomfortable at night and at midnight began suddenly to be very dyspneic. He was resuscitated with difficulty, and the dyspnea was again relieved, but only for a few moments. Then he became very dyspneic and the wound leaked blood into the tube and trachea. The tube was removed but he could not breathe even with the trachea and wound wide open. A larger tube was inserted and through it an attempt was made to swab out the trachea, but without avail. He ceased to attempt to breathe. After fruitless attempts at artificial respiration he died of asphyxiation.

*Clinical Diagnosis.*—Chronic glomerulo-nephritis.
Hypertrophy and dilatation of the heart.
Acute mitral and aortic endocarditis.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic glomerulo-nephritis with acute exacerbation.
Hypertrophy and dilatation of the heart.
Mitral stenosis.
Possibly aortic stenosis.
Passive congestion, general.
Ascites.
Hydrothorax.
Hydropericardium.
Possibly terminal infection, with acute endocarditis.
Terminal acidosis.

*Anatomical Diagnosis.*—Chronic glomerulo-nephritis.
Chronic fibrous endocarditis of the aortic valve.
Verrucose endocarditis of the aortic valve.
Slight verrucose endocarditis of the mitral valve.
Hypertrophy and dilatation of the heart.
Chronic passive congestion, general.

Anasarca.

Septicemia, staphylococcus, terminal.

Chronic pleuritis.

DR. CABOT: Was there any acute endocarditis?

DR. RICHARDSON: It does not seem to me so. The process was a chronic one, involving the aortic valve mainly.

The heart weighed 305 grams, showing considerable hypertrophy and dilatation for him. The lungs showed frank passive congestion.

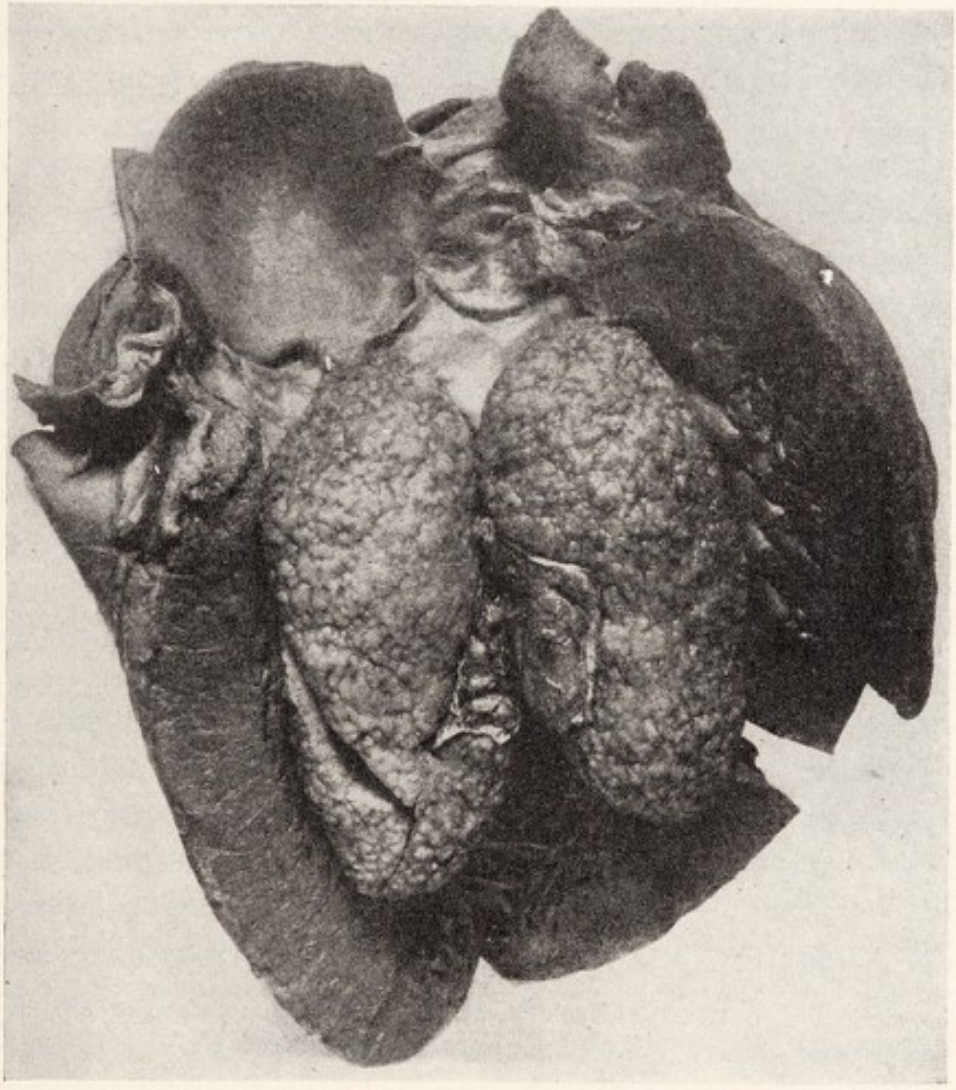


FIG. 94.—Chronic glomerulo-nephritis with hypertrophy and dilatation of the heart. Both kidneys are placed in the left ventricle. (Photograph by Lewis S. Brown. Dr. Oscar Richardson.)

There was no fluid in the plural cavities, but considerable in the lungs.

Our best picture of chronic glomerulo-nephritis and hypertrophied heart is this one (Fig. 94), showing the contracted kidneys easily resting within the left ventricle of the hypertrophied heart.

The stomach mucosa was thick, red and velvety, exuding thin bloody fluid—a good example of chronic passive congestion.

DR. CABOT: Was the blood shown by the guaiac test from that?
DR. RICHARDSON: I think so.

### Case 4312

A Canadian barber of sixty-five entered February 18, 1922, complaining of pain and blueness of the right leg of eight weeks' duration.

His father and one brother died of "shock."

His general health had been good. At fourteen he had a severe fever of unknown cause. The same year a stone weighing six or seven hundred pounds fell on his right leg and foot, leaving a bruise which persisted for six months and pain lasting four months. He had a recurrent abscess in the right ear for several years in his youth. For many years he had had an attack of bronchitis of two or three weeks' duration nearly every winter. His teeth were soft and poor. His bowels were slightly constipated for many years; now moved regularly with cascara. Five years ago he had pneumonia. He had once had piles, which had now disappeared. Occasionally he urinated once at night. He denied venereal disease, though he admitted frequent exposure. Average weight for the past ten years 170 pounds, before that 140 pounds. He thought he had lost a little weight on account of insomnia during the past three weeks.

He formerly took whiskey to excess at long intervals. He had been an excessive pipe smoker all his life.

Eight weeks ago on stepping out of bed he felt a dull pain in the lower right leg and noticed the foot was white. He felt no more pain for three days. Ever since then he had had intermittent dull aching pain in the lower two-thirds of the leg and severe shooting pain in the region of the great toe. The right leg and foot had been purplish since a few days after the onset. The pain had been getting worse week by week, although some days he was entirely free from it. It was increased by cold and alleviated by warmth. The foot was cold, numb and tingling all the time. During the past three weeks the symptoms had grown very severe, so that he had been in bed, unable to set the foot on the floor or to sleep at night, and had had to take tablets to induce sleep and deaden the pain, during the past three weeks as many as twenty-five tablets a day. During this time his hands, feet and face had grown increasingly bluish. He had had no treatment except that he had rubbed the leg and foot with a liniment and later with mustard oil with only temporary relief. The skin had been inflamed ever since this treatment.

A poorly nourished man, dyspneic. Face and extremities slaty gray. Mucous membranes reddish-gray. Sclerae slightly injected. Teeth all missing. Chest slightly "emphysematous." Lung signs as shown in the diagram. Motility of the spine limited. Apex impulse of the heart not found. Measurements by percussion: midclavicular line 9 cm., left border of dullness 8 cm., right border 4 cm., supracardiac dullness 5 cm. Action normal. Sounds distant. Slight systolic murmur at the apex in the left lateral position. Pulses poor volume and tension. Artery walls palpable, brachials tortuous.

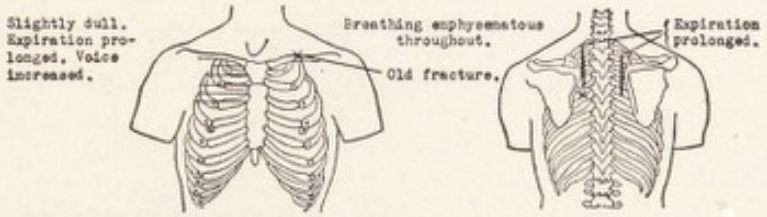



FIG. 95.

Blood pressure 150/90. Abdomen distended, tympanitic. Genital. and rectal examination negative. Extremities. Slight fine tremors Finger markedly clubbed. Right foot blue gray and cold up to six inches below the knee. Particulary intense blue in the foot, which was very cold. Right dorsalis pedis not palpable. Skin scaling, with several reddish-blue spots in the lower part of the shins. Veins of both feet distended. Pupils and reflexes normal.

Before operation temperature 96.6°–100°, pulse 80–111, respiration 20–31. Urine—normal amount, sp. gr. 1028–1022, cloudy at one of three examinations. Renal function 30%. Blood: Hgb. by Tallqvist method chocolate colored. (See below.) No estimate made. Leucocytes 13,400–22,800. Polynuclears 84%. Reds and platelets normal. Wassermann negative. Non-protein nitrogen 61.8 mgm. Bleeding time 2½ minutes. Clotting time 4 minutes. Two throat cultures negative.

February 22 the patient felt considerably better. The foot was kept warm and was much less painful. His physician reported that he had given him not more than twenty-four tablets of "sel codeia" which may have contained acetanilid. Tests for methemoglobin and spectroscopic examination negative. The 24th and 25th the pains in the foot were very severe. The foot became increasingly blue, and by the 27th was very blue from toes to midshin.

February 28 amputation was done. The next day the heart action was somewhat irregular. March 4 there was some slight distension. The wound was surprisingly clean considering that there was practically no bleeding at the time of operation.

*Dr. Hugh Cabot's Pre-operative Diagnosis.*—Thrombosis of popliteal artery from arteriosclerosis.

*Pre-operative Diagnosis.*—Gangrene of the foot.

*Operation.*—Spinal anesthesia. Amputation of the right leg through mid-thigh for thrombosis. Anterior and posterior flaps with muscle flaps. No bleeding from stump after tourniquet was removed. Femoral artery thrombosed as high as it could be traced. Wound closed loosely with continuous catgut.

*Pathological Report.*—The popliteal artery contains a solid thrombus. There is no necrosis of the toes or foot.

Microscopic examination shows degeneration of the wall of the artery with small round cell infiltration and thrombus formation.

Arteriosclerosis.

Thrombosis.

March 9 the patient had sudden right-sided paralysis involving the face on the same side. He was semiconscious, but unable to speak. He had clonic convulsions involving the whole right side and left arm in very slow, deliberate movements. Any stimulation of the right limbs resulted in slow contraction of the whole musculature, usually flexion. The pulse ranged from 92 to 120. March 12 he seemed improved. He was still unable to talk, but smiled and seemed to understand what was said. The facial involvement was less, though the right-sided paralysis was still complete. March 16 the temperature rose to 101.2°. He grew gradually worse, showed evidence of pain, March 18 became comatose, and March 19 died.

*Clinical Diagnosis.*—Arteriosclerosis.

Embolisms of right leg.

Amputation of right leg, mid-thigh.

*Dr. Hugh Cabot's Diagnosis.*—Arteriosclerotic gangrene (leg).

Arteriosclerosis.

Hypertrophy and dilatation of the heart.

Cerebral embolism.

*Anatomical Diagnosis.*—Arteriosclerosis.

Mural thrombus of the aorta.

Thrombosis of right femoral artery.

Thrombosis of the right iliac and femoral veins.

Embolism of splenic, hepatic and renal arteries.

Infarcts of brain.

Infarction of spleen and left kidney.

Infarcts of right kidney.

Focal pneumonia.

Wet brain.
Recent amputation of left thigh.
Chronic pleuritis.
Obsolete tuberculosis, apices of lungs.
Emphysema of left lung.

The autopsy is interesting as showing the extent to which thrombi and emboli may go and yet life continue. There was nothing about




FIG. 96.—Heart and aorta from case 4312. P, point of attachment of a pennant-shaped mural thrombus of the aorta.

the condition of the urine to lead one to suspect the extent of the interference with the circulation.

I do not see that they have anywhere accounted for the clubbed fingers, and wonder whether it was a faulty observation. Clearly he had enough things to die of, and it is surprising that he had not done so before.

### Necropsy 4251

First entry. An unmarried Irish laundress of twenty-seven entered February 27, 1914, for relief of pain in the back.

Her past history was negative except for frequent sore throats and a run down condition in 1905.

Record of the Out-Patient Department, December, 1905. Complaint, abdominal pain for two months, with headache and water brash. Cheeks puffy in the morning with swelling of hands and legs. Physical examination showed the eyelids puffy, but no edema of the legs. Heart not enlarged. No murmurs. Slight dullness at the right apex front and back. Urine negative. She did not stop work, and was better after a few weeks.

Eight years later, a year before admission she began to have occipital headache radiating to the orbital region, coming on early in the morning and lasting all day, accompanied by frequent vomiting and confining her to bed. The attacks came about once a month, without relation to catamenia. About this time she had swelling of the feet and puffiness of the eyelids, which had increased. Four months ago she was so much worse that she gave up her cooking because of weakness, but chiefly because of pain in the left sacroiliac region on walking. A week ago she gave up work altogether. She had had dyspnea on much exertion for a long time, but worse recently. For a few days she had had cough without sputum. Her bowels moved only with medicine. She passed less urine than usual. She had lost an unknown amount of weight.

She was well nourished, slightly pale. Palatal arch rather high and narrowed. Teeth much decayed. Some pyorrhea. Tonsils slightly enlarged. Heart: no enlargement made out. Apex impulse forceful. Sounds of good quality. $P_2$ accentuated. A soft systolic murmur accompanied the first sound, heard best at the pulmonic area, slightly at the apex. Pulses fair volume, good tension. Right slightly greater than left. Walls not felt. Blood pressure 125/90. Abdomen normal. Edema of the sacrum and marked edema of the lower legs and dependent portions of the thighs. Pupils normal except that the right was slightly irregular. Reflexes. Left knee-jerk somewhat sluggish. Right obtained only on reenforcement. Plantars normal.

Temperature 97.2°–99°. Pulse 61–80. Respiration 17–29. Urine: ℥ 17–32. Sp. gr. 1014–1024. Cloudy at one of six examinations, a very slight trace to a trace of albumin at all, red blood cells and many leucocytes at all. Renal function 40%. Blood: Hgb. 80%. Leucocytes 5200, polynuclears 79%. Wassermann negative. X-ray of kidneys negative. Left antrum looked opaque, but there was a good deal of distortion.

By March 14 there was no edema. The other symptoms had improved markedly, and she was up and felt strong. The urine remained unchanged. She was discharged that day with advice as to diet.

Records of the Out-Patient Department show a visit in March, 1915, to the Orthopedic Room, where her feet were strapped, and one to the Throat Room in April 1918, for acute tonsillitis, streptococcus. March 5, 1920, she came for headache, vomiting, and edema of the face, the legs and perhaps also the abdomen. The urine was cloudy, sp. gr. 1010, a large trace of albumin. Heart slightly enlarged. Blood pressure 180/134.

Second entry, March 25, 1920.

Her father died of kidney trouble.

She had sore throats all her life until fourteen years ago; rarely since then. Her bowels were always costive. Six years ago she had considerable bilateral greenish discharge from the nose. Five years ago she had a right earache and discharging ear. The ear had been slightly deaf ever since. A year ago she had influenza. That year she had occasional metrorrhagia for one or two days, never more than twice a month. A few months ago while costive she noticed a few specks of bright red blood in the stool. For five weeks she had had frequency of urination. Occasional scalding. Her weight gradually increased from 122 to 130 pounds, her present and best weight.

Habits. Good.

She now gave a history of five months' treatment at another Boston hospital in 1913 and three weeks in 1916 for the same complaint treated here in 1914. In 1915 she was well. Three years ago she came to the Out-Patient Department of this hospital with the same complaint and was given a diet, but had never followed it. Since that time she had been in the other hospital once and in the Out-Patient Department several times. She had been fairly well for a year until five weeks before admission, when she began to see specks everywhere before her eyes. Her face, abdomen and legs began to swell. She had been nauseated much of the time, and had vomited once. She was getting a little dyspneic, had some palpitation on exertion, and had used two pillows at night. Five weeks ago and again at admission her urine was red. She had some scalding and frequent urination in the day and dull ache in the lower abdomen after eating, relieved by hot drinks and by passing gas by rectum. She was occasionally dizzy and had a flushing sensation over the

TABLE 107

| Schlayer test, March 27 | | | | | | | |
|---|---|---|---|---|---|---|---|
| Time | Volume of urine in c.c. | Sp. gr. | Nitrogen | | Sodium gm. per liter | Chloride total | Meals |
| | | | Gm. per liter | Total | | | |
| 10 | 60 | 1010 | 5.98 | .36 | 8.3 | .50 | Breakfast 8 a.m. |
| 12 | 85 | 1009 | 4.73 | .40 | 8.5 | .72 | |
| 2 | 65 | 1008 | 5.86 | .38 | 8.8 | .57 | Dinner 12 m. |
| 4 | 50 | 1006 | 6.30 | .31 | 7.6 | .38 | |
| 6 | 65 | 1010 | 6.50 | .42 | 8.2 | .53 | |
| 8 | 75 | 1005 | 6.25 | .47 | 8.5 | .64 | Supper 6 p.m. |
| Night.... | 350 | 1006 | 6.02 | .21 | 6.8 | 2.38 | |
| Total.... | 815 | .... | .... | 2.55 | ... | 5.72 | |
| Intake... | 1700 | .... | .... | 11.00 | ... | 8.50 | |

head and neck. She had taken $MgSO_4$ at home every morning for five weeks, during which period she had had practically constant occipital headache.

She was well nourished. Slight edema of the face. Mucosae slightly pale. Slight deafness of both ears. Pus pocket at roots of lower incisors. No pyorrhea. Tonsils large, crypts prominent. Lungs clear. Heart not enlarged.* $A_2$ accentuated. $P_2$ double. A soft blowing systolic murmur heard all over the precordia, loudest at the base. Blood pressure 190/120–150/100. Abdomen. Shifting dullness in the flanks. Slight tenderness in the right upper quadrant. Slight right costovertebral tenderness. Right kidney questionably palpable. Extremities. Slight edema of the feet, legs, thighs and lower back. Pupils and fundi normal. Knee-jerks sluggish.

Temperature and respiration not remarkable. Pulse 65–95. Urine: ward record ℥ 11–72. Cloudy at all of four examinations. Neutral at one. Sp. gr. 1008–1024. A trace to a slight trace of albumin at all. Loaded with red blood corpuscles at three examinations, rare at the fourth, leucocytes at all. (No catheter specimen.) Note on Schlayer test by Dr. Reginald Fitz. "Test apparently shows inability of kidney to excrete water and nitrogen. Chloride reactions show good concentrative powers of kidney and that

*The measurements were: midclavicular line 8 cm., left border of dullness 9.5 cm., right border of dullness 3.5 cm., supracardiac dullness 5.5 cm.

total output approaches intake." Renal function. March 25 25%, April 5 15%. X-ray. Pus pocket at root of an incisor. Left antrum hazy, also both frontal sinuses. Heart as shown in the illustration.

Orders. Salt free low protein diet. Limit fluids to 1200 c.c. $MgSO_2$ ℥ iss.

By April 4 the patient was much better, the edema was gone, and there was no headache or visual disturbance. The urine and

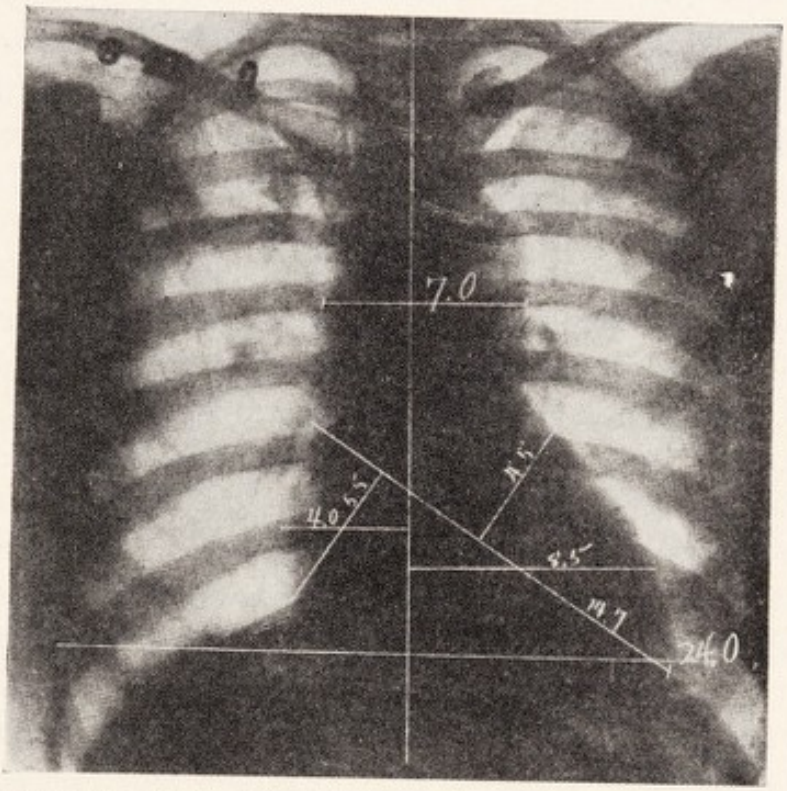



FIG. 97.—Necropsy 4251. Chronic glomerulo-nephritis with hypertrophy of the heart and subacute pericarditis. X-ray shows the heart apparently slightly enlarged. There is unusual angulation in the border of the left ventricle; otherwise the shape of the heart is not abnormal. Supracardiac dullness abnormally wide, and aortic area shows generalized increase in size, suggesting a diffuse dilatation. (Roentgenological Department, Massachusetts General Hospital.)

blood chemistry, however, were worse than at admission. She was put upon the regular hospital diet and began to gain two pounds a day. In four days edema appeared over the shins. Nevertheless she insisted upon going home, and April 9 did so.

Third entry, September 13, 1921.

She had been married two years. Since leaving the hospital she had had slight edema of the ankles nearly every day, disappearing at night, and headaches as often as once a week. She had not kept to any diet or taken any medicine A year ago she had blurring of vision, equal in both eyes, with gradual recovery. She considered

them nearly normal at present. Five weeks ago she began to have smothering sensations, worse at night and on exertion, preventing her from lying flat, and gradually increasing in severity and frequency. The nocturnal frequency increased to every hour or half hour until her doctor gave her some green liquid for her heart two weeks ago, since when it had decreased to four times a night. The dyspnea had remained the same. A week ago she had a slight nosebleed. About this time she caught cold and had had slight cough ever since. For four nights she had had very severe attacks of breathlessness accompanied by a sensation of great constriction in the chest and some shooting pains over the heart. During the attacks she thought she was going to die. At the end she found that her hands and feet were moving in an uncontrollable way. In two months she had lost twenty-two pounds.

Physical examination. A poorly nourished, pale, thin woman sitting upright in some respiratory distress. Skin dry, icteric. Mucous membranes pale. Sclerae injected. Chest expansion and diaphragm excursion slightly greater on the left. Slight dullness at both bases, more on the left. A few moist crackling râles at both bases. Marked pulsation above the clavicle. Spine tender over lumbo-sacral region. Heart action rapid (100). Sounds of good quality. $A_2$ greater than $P_2$, both accentuated. $A_2$ sharp and ringing. Soft systolic murmur at the apex and over the precordia. Pulses of poor volume and tension. Artery walls normal. Blood pressure 220/140–170/110. Abdomen. Liver dullness from sixth rib to costal margin. Edge not felt. Extremities. Slight edema of shins and sacrum. Pupils slightly irregular, otherwise normal. Fundi not abnormal except for greatly dilated veins. Reflexes. Knee-jerks not obtained.

Temperature 98.5°–96.3° (by mouth); after September 26 96.5°–99.6° (rectal.) Pulse 101–60. Respirations 31–9. Urine: ℥ 18–6 (?). Sp. gr. 1006–1012. Cloudy at four of six examinations, pink at one, alkaline at all, a trace to a large trace of albumin at all. Red blood corpuscles at one, leucocytes at one. Renal function O (two tests). Blood: Hgb. 50%–40%. Leucocytes 24,000–11,800. Polynuclears 71%. Reds 2,224,000–2,640,000. Smear and platelets normal. Two Wassermanns negative. Non-protein nitrogen September 13 95.1 mgm., September 20 133.2, and September 26 333 mgm. Bleeding time 2:10 minutes. Clotting time 6:45–8:45.

By September 15 the patient was vomiting practically all the food ingested. She was too ill for a Schlayer test. Two days later she

was very weak and not clear mentally. Rectal taps were not well retained. The night of the 17th there was pain in the upper half of the left chest in front on deep breathing, and a definite pleural friction rub heard just to the left of the arch of the aorta. She grew steadily more stuporous. By the 23rd there was edema of the left hand and both feet. Morphia in ⅙ gr. doses was used to control the vomiting. There was some blood in the vomitus. Another fundus examination showed a small hemorrhage in the left eye. September 26 the heart sounds were exaggerated. There was a friction rub over the apex, which the following day was unusually loud and grating all over the precordia, obscuring the heart sounds. Electrocardiogram Sept. 29, showed arrhythmia, brady cardia, rate 55–80; probably due to variation in site of pace maker; sometimes auricular with upright P wave; sometimes auricular-ventricular or ventricular escape with absence of auricular activity. Left ventricular preponderance. High T wave.

Against all expectations the patient lived in stupor until October 1, suffering some pain.

*Clinical Diagnosis.*—Chronic glomerulonephritis.
Uremia.
Hypertension.
Pericarditis.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic glomerulonephritis.
Hypertrophy and dilatation of the heart.
Acute pericarditis.
Secondary anemia.
Chronic passive congestion.

*Anatomical Diagnosis.*—Chronic glomerulonephritis.
Hypertrophy of the heart.
Chronic passive congestion of the spleen.
Subacute pericarditis.
Ascites.
Edema of the ankles.

Dr. Oscar Richardson: The ankles pitted slightly on pressure. Unfortunately the incision in this case was restricted to a six-inch cut in the midline, but they found that the kidneys were small,—215 grams—and that the condition was chronic glomerulonephritis macroscopically and microscopically. Evidently there was some trouble in getting the heart, but enough was seen to establish the hypertrophy, —a hypertrophy of the left side. The wall of the left ventricle was fifteen mm. thick, indicating hypertrophy of the wall of the left ventricle. There was fibrinous pericarditis.

# CHAPTER V

## MYOCARDITIS

### 1. Chronic Fibrous Myocarditis

Few terms have undergone so many vicissitudes or have been used in more different senses within a century. In the years 1890–1910 we frequently heard the term used as a diagnosis to explain cardiac weakness and arrhythmia in elderly people without evidence of valvular disease. When post-mortem examinations became more frequent and their results were more often correlated with the clinical findings, the term began to be abandoned, since, as I showed many years ago,* most clinical diagnoses of fibrous myocarditis made at that time were demonstrably wrong post-mortem, and most of the actual fibrous myocarditis discovered post-mortem had not been suspected during life. I have therefore maintained for many years that the diagnosis of myocarditis is impossible during life. Against this it was for years contended, partly on the authority of Krehl, that one could find myocarditis in most failing hearts if one searched all parts of the heart muscle by microscopic examination. Since this is rarely done the contention is difficult to refute. But even if it were true it would not prove that such microscopic foci of fibrous tissue caused any functional weakness of the heart. As a matter of fact it can be demonstrated, as I shall show below, that even gross, easily recognized patches of fibrous myocarditis in its extremest form, may exist unassociated with any chronic passive congestion demonstrable after death.

In view of these facts, the use by H. A. Christian† and others, of the term "myocarditis" when there is clinical evidence simply of an enlarged and weakened heart, seems to me unjustifiable. Any other muscle in the body may become hypertrophied and then if over-

* A Study of Mistaken Diagnoses, Journal of the American Medical Assn., Oct. 15, 1910, Vol. LV.

† Chronic Myocarditis (Clinic). Med. Clinics of North America, January, 1918, Vol. I, 813.

Chronic Myocarditis: A Clinical Study. Jour. Am. Med. Assoc., 1918, Vol. LXX, 1909.

Chronic Myocarditis and Its Management. Southern Med. Jour., 1921, Vol. XIV, 587.

worked may give out and cease to function. We call this giving out "fatigue," although its physics and chemistry are not yet clear. Why should we not suppose that the muscle of an overworked heart gives out from chronic and excessive "fatigue?" In such a heart there may or may not be patches of fibrous myocarditis. But neither their presence nor their absence is necessarily of any importance so far as the work of the heart is concerned.

Of recent years, since the electrocardiograph has added so much to our knowledge of cardiac physiology, there has been a tendency to return to the practice of making clinical diagnoses of myocarditis, now supposedly based on electrocardiographic findings. But it has never been proved to my satisfaction that the presence of alternation, or of heart block in any of its varieties, or of any other defect demonstrable by the electrocardiograph, gives us no good grounds for predicting that fibrous myocarditis will be found post-mortem. Exception may perhaps be made of certain cases of complete heart block. Nevertheless it still seems to me true that myocarditis has little if any standing as a recognizable clinical syndrome. It remains, I think, essentially an item of post-mortem anatomy.

## THE MATERIAL HERE ANALYZED

Among the 91 necropsies which form the basis of this chapter, there are two distinguishable groups, (a) those of acute cardiac infarction, making up 25 cases out of the total of 91, and (b) those of chronic fibrous myocarditis, which in its pure, unmixed form, is exemplified in 66 cases. Infarction and fibrous myocarditis were present together in 18 cases. It is because of this latter association, and also because of their anatomy that I have discussed the two seemingly different entities together. It certainly appears to be the fact that, in some cases, chronic fibrous myocarditis is an end result of acute or subacute infarction. A coronary artery, usually the descending branch of the left coronary, becomes narrowed or altogether occluded, either by arteriosclerosis alone, by a thrombus, or by a combination of both. The area of heart wall supplied by this vessel becomes anemic, necrotic, and then is gradually replaced by scar tissue. In some hearts different phases or stages of this process can be found side by side. In others the acute infarction maims the heart so seriously that death follows at once before any fibrous change takes place.

I am well aware that this series of events will not account for all cases of fibrous myocarditis. In 38 cases out of my series of 91

there was no actual coronary obstruction or marked narrowing, either at the mouth of the vessel (syphilitic aortitis) or in its course (arteriosclerosis or thrombus). Most of these 38 cases have some coronary *sclerosis but no* considerable *narrowing* of the vessels. The origin of the fibrous myocarditis in these cases is obscure. They appear, however, to be relatively common. Warthin's belief that most of them are due to syphilis may well be correct.

The cases of fibrous myocarditis may be further subdivided into those which are diffuse and those which are limited to a small portion of the heart, most often the left ventricle near its apex (18 cases out of 91). These cases have no clinical peculiarities to distinguish them from the rest of the series. The same may be said of our six cases of *cardiac aneurism.*

One case in this series may be described separately under the heading.

### SYPHILITIC MYOCARDITIS

An ill-nourished male infant of four weeks lived one day in our wards, with dyspnea, a bloody nasal discharge and a normal temperature. Post-mortem examination showed the lesions of congenital syphilis (Necropsy 3346). On the posterior wall of the left ventricle and the anterior wall of the right ventricle there were areas of increased resistance and marked pallor. In these areas the heart's wall was about one and a half times as thick as in other parts of the ventricle. The tissue was firm, homogeneous, and glistening, yellowish-gray in color, like the uncooked white meat of chicken. The coronary arteries appeared smaller than normal. The heart weighed twelve grams.

In one other case of our series a single isolated area of necrosis in the heart wall suggested gumma to the pathologist; and it may well be that among the cases of fibrous myocarditis unassociated with coronary disease, syphilitic lesions may have preceded the scar formation. The work of Warthin suggests this. (See also p. 424.)

### ETIOLOGY OF FIBROUS MYOCARDITIS

Fibrous myocarditis is a disease of old men. 70 men out of 89 cases in which the sex is known were above the fiftieth year at the time of death, and only two were under thirty. In these eighty-nine cases there were only nineteen females. It may be maintained with some plausibility that the sex incidence argues in favor of a syphilitic etiology. On the other hand, the duration of life argues somewhat against this, unless we are to maintain that all coronary disease is syphilitic in origin, whether we find any lesions of syphilis or not.

The average age in this group is notably greater than in the cases of syphilitic aortitis, or indeed in any other group of heart lesions known to me. Certainly in the majority of cases, those associated with *coronary sclerosis*, it seems safer to say that the cause of most cases of chronic myocarditis, and of many if not most acute cardiac infarctions, is arteriosclerosis, whatever further etiological mysteries may be hidden behind this familiar term.

That *infectious disease* has any particular connection with fibrous myocarditis, except in so far as infection may be supposed to favor the development of arteriosclerosis, we have no evidence. There was no special frequency of typhoid fever or of any other infection, or of all infections taken together, in the histories of these patients. They were not notably alcoholic or in any other respect different from the old men dying of other causes at the Massachusetts General Hospital. The *infarct group* contains acute infectious cases with coronary thrombosis, due to acute endocardial vegetations.

*Cardiac hypertrophy* was present in almost every case. Indeed there were but five cases without some demonstrable enlargement of the heart post-mortem. But since, as shown in Chapter I, myocarditis is usually associated with other lesions producing cardiac enlargement, we cannot reasonably say that cardiac hypertrophy is itself the cause or the result of myocarditis.

Only in six cases was hypertrophy linked with myocarditis alone in the absence of other lesions suspected at least of being causes of hypertrophy. The figures follow:

Myocarditis without cardiac hypertrophy..... 5
Myocarditis with cardiac hypertrophy.....86

Among these 86 cases we find but six in which myocarditis was the only lesion. In the remaining eighty the associated lesions were as follows:

TABLE 108

| | |
|---|---|
| Chronic non-deforming endocarditis in | 12 cases |
| Arteriosclerosis | 24 cases |
| Nephritis | 15 cases |
| Arteriosclerotic degeneration of kidney | 4 cases |
| Chronic pericarditis | 6 cases |
| Valve lesions | 8 cases |
| Acute pericarditis | 6 cases |
| Goitre | 1 case |
| Acute endocarditis | 1 case |
| Syphilitic aortitis | 1 case |
| Others | 2 cases |
| | 80 cases |

The five largest hearts of the series show the following data:

TABLE 109

| Case No. | Age Sex | Necropsy No. | Heart weight | Remarks |
|---|---|---|---|---|
| 1 | 49 M | 2603 | 900 | No congestion. Anginoid death. Aortic stenosis. Narrowed coronaries. |
| 2 | 49 M | 251 | 830 | Mitral stenosis. Chronic nephritis, uremic. Cardiac infarct. Left coronary *descendens* blocked. Passive congestion. |
| 3 | 20 M | 2291 | 807 | Chronic passive congestion. Acute dilatation at end. |
| 4 | 67 M | 2746 | 780 | Prostatism. Chronic nephritis. B. P. 200/105. Chronic passive congestion. |
| 5 | 55 M | 2877 | 775 | Chronic passive congestion. Left coronary blocked. B.P. 230/175. |

In all these cases and in most of those in Table 108 we may reasonably suppose that the hypertrophy is due to hypertension. There is no reason to believe that myocarditis causes cardiac hypertrophy. A truer statement seems to be that a small percentage of enlarged hearts (86 out of 1209) get myocarditis.

It seems to me therefore that we may sum up the etiology of myocarditis, so far as this series is concerned, as: coronary sclerosis in most cases (five-ninths), syphilis (demonstrable) *in a few*. The cases of suppuration in the heart wall, and of acute embolic myocardial inflammation as a part of malignant endocarditis, are dealt with on pages 512, 527.

## DIAGNOSIS OF MYOCARDITIS

Only in the infarctive subgroup of cases can the diagnosis be often reasonably suspected during life. Some such suspicion may arise in any case of angina pectoris, expecially when it occurs in elderly men. This suspicion is justified, as has already been shown, because we know that angina pectoris may be associated with obstruction of the coronary arteries, either at their orifices or in their further course, and that such obstruction often leads to myocarditis. But on the other hand angina is often *not* connected with coronary disease

and this often fails to produce myocarditis; so that the surmise is ill founded at best.

*Angina* was present in twenty-seven of our cases, and in this group, therefore, some slight suspicion of myocarditis was justified. But one can hardly say that our diagnostic right extends beyond a vague suspicion. For, in the first place, there is the group of cases on which great stress has been laid by Sir T. Clifford Allbutt, in which angina pectoris is not associated with any form of coronary obstruction. Eleven necropsies in this series illustrate this. (See Chapter VI.) Moreover, very marked narrowing of the coronary arteries, amounting sometimes to a practical obstruction, may exist without any myocarditis. Nevertheless the association of angina pectoris, coronary obstruction, and fibrous myocarditis is sufficiently frequent to justify some degree of diagnostic suspicion, under the conditions mentioned, especially if there are signs of peripheral embolism, of cardiac infarct or of acute pericarditis (see below).

Aside from the presence of angina and the favoring conditions of age and sex, we have almost no clues to the diagnosis of fibrous myocarditis. *Arrhythmia*, which used to lead straight to a diagnosis of myocarditis when the irregularity was present in an old man suffering from decompensated heart disease and without evidence of valve lesion, was noted in only 23 out of 91 cases. *Cardiac hypertrophy*, as has been already said, was present in 86 out of 91 cases. The *blood pressure* was measured in only 43 cases. It was high in 17 and notably low in six, the remainder being approximately normal. (See Tables 110, 111, and 112.)

TABLE 110.—SYSTOLIC BLOOD PRESSURE

| | | |
|---|---|---|
| Not high | 2 | |
| 85–100 | 4 | |
| 101–120 | 9 | 26 |
| 121–140 | 7 | |
| 141–160 | 4 | |
| 161–180 | 7 | |
| 181–200 | 3 | |
| 201–220 | 3 | 17 |
| 221–240 | 2 | |
| 245 | 1 | |
| High | 1 | |
| | 43 | |

TABLE 111.—PULSE PRESSURE

| | |
|---|---|
| 20– 29 | 1 |
| 30– 50 | 15 |
| 51– 75 | 6 |
| 76– 89 | 1 |
| 90–110 | 4 |

TABLE 112.—DIASTOLIC BLOOD PRESSURE

| | | |
|---|---|---|
| 60– 80 | 12 | 17 |
| 81– 99 | 5 | |
| 100–119 | 3 | 10 |
| 120–140 | 4 | |
| 141–160 | 1 | |
| 161–180 | 2 | |

TABLE 113.—AGE AND SEX

| | | | |
|---|---|---|---|
| 6 weeks (syphilitic) | 1 | | |
| 20–30 years | 2 | | |
| 31–40 years | 6 | | |
| 41–50 years | 17 | | |
| 51–60 years | 22 | Males | 70 |
| 61–70 years | 29 | Females | 19 |
| 71–80 years | 10 | Sex unknown | 2 |
| 81–90 years | 2 | | |
| "Elderly" | 1 | | |
| Unknown | 1 | | |

*Thrombi* adherent to the ventricular endocardium were present in thirty-two or about one-third of the cases. They are formed presumably on a patch of ill-nourished endocardium, this malnutrition being due to the underlying myocarditis, especially when this is of the acute or subacute type, closely related to infarct. Hence, if during life we get evidences of embolism or peripheral infarct, we may suspect that these are due to detached portions of an intracardiac thrombus, and so if we are rash we may argue from this the presence of a myocarditis beneath that thrombus. Occasionally such a guess may come out right. But (1) the other sources of peripheral embolism or infarct and (2) the cases of intracardiac thrombus without myocarditis, are so many that unless there were associated evidence of angina, cardiac infarct or pericarditis (due to the infarct) the guess at a diagnosis of myocarditis would be largely unfounded.

TABLE 114.—10 CASES OF EXTREME DIFFUSE FIBROUS MYOCARDITIS

| Age—sex | Necropsy No. | Heart weight | Chronic passive congestion | Blood pressure | Remarks |
|---|---|---|---|---|---|
| 20 M | 2291 | 807 | + | ? | 1 yr. ago dysp. and cyan. 3 wks. Last 2 mos. dysp. and cyan. Died with acute dilatation. |
| 39 F | 2413 | 426 | + | ? | Palpit. 1 yr. ago. Signs as of mitral stenosis. 3 weeks dyspnea now. |
| 50 M | 2488 | 585 | ++ | ? | Syph. aortitis obliterating one coronary orifice. Died in attack of angina. Aortic regurgitation. |
| 70 M | 2787 | 605 | o | 140/? | 5 yrs. gradual decompensation. Infarction at end. |
| 88 M | 2795 | 450 | o | 120/? | Senile gangrene. Chronic nephritis. No cardiac symptoms. |
| 65 M | 2871 | 600 | + | ? | 2 weeks epigastric pain (infarct?) 2 weeks orthopnea. |
| 46 M | 3226 | 548 | + | 160/120 | 3 yrs. dyspnea. Renal stone. 2 mos. orthopnea. Died on way to cystoscope room. |
| 66 M | 3259 | 446 | o | 100/70 | 3 yrs. angina. Actinomycosis of lung. No decompensation. |
| 65 M | 3278 | 375 | o | ? | Cancer of stomach. No cardiac symptoms. |
| 56 M | 3563 | 703 | + | 200/120 | Dyspnea 4 mos. (since pneumonia). Angina. |

In Table 114 I have separated out ten cases in which the fibrous myocarditis was of the extremest grade, diffused throughout the whole heart. The table shows that this group does not differ in any important essential from the remaining 81 cases. One of the most remarkable features about it is that in four of the ten cases there was no discoverable passive congestion post-mortem, which means that *relatively good cardiac function must have been carried on to the end of life, despite a very extensive replacement of muscle tissue by fibrous*

*tissue* in the walls of the heart. Evidently it is not this replacement alone or in itself that makes the disease serious to life. Patients, even with extreme grades of fibrous myocarditis may have no cardiac symptoms and may die (as in this group) of cancer or nephritis.

With this I have contrasted, in Table 115, a small group of seven cases in which the amount of fibrous myocarditis was very notably slight, only a few patches here and there. In this group one might expect that the passive congestion would have been very slight, and indeed this seems to be somewhere near the truth, since in four out of seven cases no such congestion could be demonstrated. This gives us some contrast with the whole group, in which passive congestion was demonstrated post-mortem in 41 out of 66, or two-thirds of the cases of uncomplicated fibrous myocarditis. Nevertheless the difference is not very striking. The weight of these seven hearts averaged distinctly less than that of the ten serious cases of Table 114, averaging 477 grams in the slight cases as against 554 in the extreme cases.

TABLE 115.—7 CASES OF NOTABLY SLIGHT FIBROUS MYOCARDITIS

| Age—sex | Necropsy No. | Heart weight | Chronic passive congestion | Blood pressure |
|---|---|---|---|---|
| 57 M | 1386 | 343 | o | -- |
| 45 M | 1394 | 557 | + | -- |
| 74 M | 1876 | 480 | o | -- |
| 67 M | 2667 | 620 | + | 165/135 |
| 53 M | 2992 | 465 | + | -- |
| 65 M | 2686 | 510 | o | -- |
| 82 M | 2777 | 365 | o | -- |

## MODE OF DEATH

In only a few cases, mostly of the infarct group, was the weakening of the heart wall itself apparently the cause of death. If we separate out the cases in which neither angina pectoris nor the infarct syndrome was present during life, we find (see Table 116) that death resulted from chronic passive congestion in only three cases, in other words, that *death usually has very little to do with the myocarditis.*

Chronic nephritis was apparently the main factor in the fatal result in twelve cases, streptococcus sepsis in eight, pneumonia in four, valvular disease in five. Arteriosclerotic gangrene and its results brought about the fatal issue in at least three cases, gastro-intestinal cancer in two, prostatic disease, pernicious anemia, cerebral hemorrhage, delirium tremens and cirrhosis of the liver were apparently the chief factors in other cases. *Only in three out of forty-eight cases could one say that the myocarditis may well have been an important cause of death.* In these three cases the heart weights were 413, 545, and 807 grams respectively, and as the lightest of these belonged to a wizened old woman it, like the rest, was considered to be hypertrophied. But all these cases may well belong to the hypertensive group and their deaths may be attributed primarily to hypertension and its results, one of which, for all I know, may be myocarditis.

I have already referred above to the very considerable hypertrophy which was present in almost all the hearts of this group. In the five largest hearts, nephritis was apparently a factor in four. The remaining case presumably represented one of those examples of hypertrophy associated with a hypertension of unknown cause. From these as from the whole group one gets the impression that these patients have acquired an enlargement of the heart from the usual causes which lead to arteriosclerosis as well as from chronic nephritis or from hypertension of unknown origin; that in the course of this disease they have acquired also a certain amount of fibrous myocarditis; but that, in the absence of coronary disease and especially of coronary infarct, this myocarditis has been of very little clinical importance.

TABLE 116.—MAIN FACTOR IN DEATH IN 48 CASES WITHOUT ANGINA OR CARDIAC INFARCT

| | |
|---|---|
| Chronic Nephritis | 12 |
| Septicemia or acute endocarditis | 8 |
| Valvular disease | 5 |
| Pneumonia | 4 |
| Gangrene of extremity | 3 |
| Passive Congestion | 3 |
| Apoplexy | 2 |
| Neoplasm | 2 |
| Alcoholism | 2 |
| Others | 7 |

Necropsy No. 2291 is unique in our series in that it showed an extreme degree of fibrous myocarditis in a boy of twenty years,

without important previous illness. A year before his death, he had his first attack of dyspnea and cyanosis and went to bed for three weeks. Two months before death dyspnea, cyanosis and edema of the face reappeared and continued. They were severe for the last three days, preventing sleep and accompanied by vomiting. There was also edema of the legs and ascites with intense cyanosis of the face, hands, and feet.

The heart's impulse was in the fifth interspace, two and a half inches outside the nipple. The right border of dullness extended three inches to the right of midsternum, and there was a moderate increase of the supracardiac dullness. The heart was irregular and rapid, its sounds often obscured by noisy respiration. No murmurs. The pulses were equal, synchronous, irregular, of fair volume and low tension. The pulmonic second was sharply accentuated.

He collapsed on his way to the ward and was bled three ounces, apparently with good effect, but attacks of tremendous cyanosis and extreme dyspnea continued. The outlines of cardiac percussion suggested pericardial effusion but the action of the heart was forcible and the dullness did not extend beyond the impulse to the left. He died in one and a half hours.

Necropsy showed a huge, gristly, fibrous heart weighing 807 grams. On cutting through the myocardium of the left ventricle great resistance was offered by the tissues which were everywhere tough and gristly to the touch, though more so in some places than in others. Section surfaces showed replacement of the muscle by gray-white homogeneous, apparently fibrous tissue in larger or smaller confluent areas. Microscopic examination confirmed these impressions.

The coronaries were free and smooth. The left ventricle was twelve mm. thick, the right eight. The valves were normal. The other organs were normal save for chronic passive congestion. No evidences of nephritis or syphilis.

Here was a tremendous hypertrophy and dilatation quite unexplained and in a boy of twenty without any history to throw light on it. Was the myocarditis here a cause for hypertrophy? What caused the myocarditis? I cannot answer. In most of our cases myocarditis was apparently only an incident in the hypertensive cardiovascular disease of arteriosclerotic old people. But here the conditions are wholly different. One suspects syphilis but no evidence of it was obtained in the necropsy.

## SUMMARY AND CONCLUSIONS

1. Fibrous myocarditis is an item of post-mortem anatomy, occurring especially in elderly men with arteriosclerosis, nephritis, and hypertension.

2. It is not clinically recognizable, though its presence may be vaguely suspected when angina pectoris is present and especially when this symptom is associated with evidence of cardiac infarction, of peripheral embolism, and of acute pericarditis.

3. It is rarely the cause of congestive heart failure.

4. Only in one case had we adequate evidence of syphilis.

5. Coronary narrowing was present in about five-ninths of our cases and is the most clearly recognizable factor in etiology. Syphilis may also play a part.

6. Fibrous myocarditis is rarely the only cardiovascular lesion present.

7. It is usually associated with cardiac hypertrophy (present in 86 out of 91 cases) but since such lesions as chronic nephritis, valve lesions, chronic pericarditis are also usually present, we have no evidence that the myocarditis is the cause of the hypertrophy.

8. Evidence of chronic passive congestion was found in 51 of 91 cases, but when present need rarely be attributed to the myocarditis.

9. Since fibrous scars in the myocardium—even when numerous and extensive, are not always associated with evidence of heart failure in life or of passive congestion after death, there seems no sufficient reason to believe that they interfere with the heart's action in any material way. There is all the more reason to suspect that fibrous myocarditis is usually harmless, because in the cases which *are* associated with heart failure and passive congestion causes other than the myocarditis are almost always present.

10. Myocarditis is an obvious danger to life only when it leads to cardiac aneurism or rupture (see below) or when it causes intracardiac thrombosis and acute pericarditis.

## FIBROUS MYOCARDITIS FOLLOWING INFARCT—ILLUSTRATIVE CASES

### Necropsy 2871

A Scotch engineer of sixty-five entered June 20. His father and mother both died of "heart disease," one brother of brain trouble. The patient had all the diseases of childhood, then vigorous health until he was fifty-seven. He had taken no alcohol for five years. Before that he had whiskey before breakfast and half a pint during

the day. He drank to excess three or four times a year. A year before admission he was advised in the Out-Patient Department to enter the wards because of obstructing prostate.

For eight years he had had occasional nausea and vomiting, usually attributed to excesses in food and drink. He had occasional epigastric distress an hour or two after meals, relieved by food. On a restricted diet without meat he did well. His vomitus was never bloody or coffee-colored. *Two weeks before admission he was shovelling coal as well as tending his engine.* He felt severe general abdominal pain, vomited, and fainted. Distress persisted for four days. Since this attack he had had increasing dyspnea and orthopnea. For four days his feet had been slightly swollen. His urine was scanty. He had no nycturia at entrance, though formerly he had had frequency by day and several times at night. His bowels required catharsis.

Examination showed a fairly well nourished, orthopneic man with many dilated venules on the face. The mucosae were cyanotic. The apex impulse of the heart was not found. The left border was not made out. The right border was 1 cm. to the right, the upper border at the third rib. The sounds were of very poor quality, almost inaudible in the mitral and tricuspid areas, apparently normal at the base. The pulses were of fair volume and tension. The artery walls were not felt. The systolic blood pressure was 115. Both lungs showed dullness increasing toward the bases, from the fourth rib in front to a little below the scapula in the back. Breathing and vocal fremitus were much diminished at the bases. Many medium and coarse moist râles were heard, most numerous in the axillae and the angles of the scapulae. The abdomen, extremities, genitals, pupils, and reflexes were normal.

The temperature was normal, the pulse 80 to 90, the respirations 30 to 34. The urine is not recorded. The hemoglobin was 90%, the leucocytes 16,000, the smear normal.

The patient continued to have dyspnea and sat up almost all the time. June 21 just after midnight he died.

*Clinical Diagnosis (from Hospital Record).*—Myocardial weakness.

*Dr. William H. Smith's Diagnosis.*—Arteriosclerosis.

Possibly angina pectoris with coronary occlusion and myomalacia cordis.

Much less probably rupture of the aortic arch.

*Anatomical Diagnosis.*—Arteriosclerosis.

Arteriosclerotic occlusion of the coronary arteries.

Myomalacia cordis.
Chronic interstitial myocarditis.
Thrombi of the left ventricle.
Hypertrophy and dilatation of the heart.
Infarcts of the kidneys.
Chronic passive congestion, general.
Hydrothorax, double.
Chronic perisplenitis.
Diverticulum of the duodenum.
Hypertrophy of the prostate.
Hypertrophy of the trabeculae of the bladder with small diverticula.
Cholelithiasis.

DR. RICHARDSON: There was arteriosclerois of the aorta and great branches and marked arteriosclerosis of the coronary arteries. The sclerosis was more pronounced in the left artery than in the right, so pronounced, in fact, that it presented points of occlusion with corresponding areas of degeneration and softening in the myocardium. There was only a small strip of myocardium left that was fairly normal in appearance, but the greater portion of the wall of the left ventricle showed marked changes due to the sclerosis of the coronary artery and presented, on the whole, a typical picture of degenerative changes in the myocardium. That is, it showed degeneration of the muscle tissue and replacement by areas of necrosis and fibrosis. The muscle of the right ventricle was in fairly good condition, because the lumen of the right artery was sufficient to give a fair blood supply. The thrombi in the left ventricle were erected, of course, on an endocardium which had become degenerated. Beneath it the wall showed chronic interstitial myocarditis.

The heart weighed 600 grams (normally 200–300), showing considerable enlargement. The cavities showed considerable dilatation. The valves, with the exception of a slight increase in their circumferences, were negative. From middle to old age the valves of the heart usually show a certain amount of fibrosis, because they are lined with endocardium, which plays the same rôle on the valve that the endothelium intima plays in the blood vessels.

The kidneys weighed 427 grams (normally 200–400). When a pair of kidneys weighs more than 400 grams you are suspicious of glomerulo-nephritis. There are other conditions, however, which cause increase in size of these organs. In this case the man probably

had fairly large organs to start with. Then there is a certain amount of chronic passive congestion which has increased the size, mainly due to edema. Sometimes edema is the only thing found in kidneys of 400 or even 500 grams. As we have pointed out before, the increase occurring in glomerulo-nephritis is due to the obstruction of the glomeruli and the capillaries. These kidneys were plump, were of the so-called "hog-back" type, and were pale to dark brown-red in color, in some places cyanotic-looking. The infarcts to the kidneys were due to small pieces from the thrombi in the left ventricle which were brought by the blood stream. With the exception of the conditions described, the kidneys were very good for a man of sixty-five. It is rather striking in persons over fifty years of age that the kidneys stand the wear and tear of life remarkably well, as a rule. The kidney is a very resistant organ, and works to the best of its ability whether there be much or little of it left. The kidneys usually show at this man's age some areas of arteriosclerotic atrophy. When the condition present is sufficient to be called "arteriosclerotic nephritis" the kidneys, unless the process is very extensive, are of extra good size, in marked contrast to those affected with glomerulo-nephritis. The development of the classification of kidney diseases into these two groups has come about, within the last ten or fifteen years.

There was some hypertrophy of the prostate. With this there was, of course, back pressure. The bladder worked to expel the urine, with more or less consequent hypertrophy of the trabeculae.

### Necropsy 2787

A Prussian lithographer of sixty-six entered December 16, 1904, He had never been ill in bed. He had taken no tobacco or alcohol for the past six months. Formerly he used both in great excess—"used to be a high liver." He drank ten to fifteen cups of coffee a day.

For twenty years he had had "indigestion,"—gas, sour stomach and pain two to three hours after meals. The attacks at first came on only after indiscretions of diet or drinking, but for the past two weeks he had had dull pain all of the time which at intervals became much worse and crampy in character. He had never felt nauseated and had never vomited. He had lost twenty-five to thirty pounds within the past three months. His bowels, usually regular, had been constipated for four weeks. His appetite had always been good until the past month.

Examination showed a poorly nourished man with normal heart, lungs, and abdomen except for liver dullness from the sixth rib to an inch and a half below the costal margin where the edge was felt. The reflexes were normal. There was no edema.

The temperature was 98° to 99°, the pulse 64, the respirations 22, the leucocytes 5900. A test meal showed free HCl 0.06%, total acidity 0.14%. At a second test there were no fasting contents and no residue of the test meal after an hour.

The patient was discharged on January 5, 1905, with a diagnosis of neurasthenia.

He was well until the middle of January, 1906, when he "caught cold riding horseback." Cough kept him awake, and he raised considerable white or yellowish sputum, sometimes blood-stained. February 3 he reentered the hospital.

Examination showed a systolic murmur all over the precordia, loudest at the apex. The lungs were slightly hyperresonant, with occasional moist râles. Expiration was faint, especially in the backs.

The temperature was 98° to 99°, the pulse 66, the respirations 25, the leucocytes 8800. The sputum showed no tubercle bacilli.

By February 7 his cough was much better and he was discharged with a diagnosis of subacute bronchitis.

After leaving the hopsital he "never felt better in his life." But early in June, 1906, he found that he could not walk uphill or rapidly without dyspnea and a sense of oppression in the chest. June 5 he drank a glass of ale when tired and shortly afterwards had considerable discomfort rather than pain in the epigastrium and much nausea. He vomited for the first time in his life. He had some pain in the epigastrium relieved by sodium phosphate and sodium carbonate. He slept very little the next three nights. He got up five or six times a night to urinate. He had lost no weight. June 8 at 4 p.m. he was suddenly seized with sharp pain in the epigastrium spreading into the chest, through to the back, down both arms, especially the left, and very marked in the precordia. At its onset "he burst into a cold sweat," panted for breath, and had "terrible nausea" without vomiting. Gastric lavage did not relieve the pain.

Examination at his third admission, June 8, 1906, showed the cardiac impulse and dullness in the fourth space just inside the nipple line. There was no enlargement to the right. The action was regular, the sounds distant, the aortic second sound greater than the pulmonic second. A soft systolic murmur was heard in the aortic area transmitted upward but not heard in the neck, a soft systolic in the mitral

area and the axilla. The arteries were sclerosed and tortuous. The lungs showed a few râles at the bases, hyperresonance throughout, prolonged, low pitched expiration. The liver dullness extended from the fourth space to the costal margin. The edge was felt one inch below the costal margin. The temperature was 98° to 101.5°, the pulse 70, the respirations 20, the leucocytes 10,900. The sputum showed no tubercle bacilli.

About 10 p.m. June 9 the patient had a sudden attack of cardiac weakness following a small dose of trional and sulphonal. Subsequently his pain did not return, his appetite returned to normal and the heart sounds improved in quality under treatment with digitalis. June 21 he was discharged relieved.

After leaving the hospital he was comparatively well. He walked seven to eight miles daily, but did nothing more violent. He had practically no "stomach trouble." Taking care not to overdo, he had no cardiac symptoms. December 29, 1906, he caught cold in the head and thought he spat up a quart of yellow sputum. He was unable to sleep, worried considerably over financial difficulties, and lost ten pounds in ten days. January 8, 1907, he entered the hospital for the fourth time.

Examination showed the impulse of the heart forceful, corresponding with the dullness in the fifth space just inside the nipple line. There was a soft systolic murmur at the apex, not transmitted. The arteries were palpable, slightly tortuous. The lungs showed scattered coarse râles. The temperature was 98.4° to 99°, the pulse 80, the respirations 24, the leucocytes 13,400.

On January 14 the patient was discharged with a diagnosis of bronchitis.

After leaving the hospital he had only slight dyspnea and cough. He had however some gastric distress. On January 8, 1911, he cought cold; his dyspnea and cough became worse and soon his sputum became bloody. He had no definite thoracic pain. January 19 he was seized with severe gastric pain and vomited once. After this he felt very weak and helpless and had a good deal of abdominal pain, chiefly in the region of the stomach and the right costal margin. He had some pain in the region of the bladder, some frequency, and burning on micturition. January 20 he entered the hospital for the fifth time.

Examination showed a well nourished man with Cheyne-Stokes respiration lying propped up in bed, The mucous membranes were pale. The apex impulse of the heart was seen and felt in the fifth

space just outside the nipple. The retrosternal dullness was slightly increased. The upper border of flatness was at the third rib, the lower at the fifth space. The sounds were practically inaudible. There were no murmurs. The systolic blood pressure was 140. The pulses were of very poor quality. The artery walls were tortuous and showed fibrous thickening. The lungs posteriorly showed many coarse and fine moist râles. The patient was coughing considerably and raised blood-tinged sputum. The reflexes were normal except for a double Babinski. There was no stiffness of the neck, no edema, and no paralysis.

The temperature was 99° to 101°, the pulse 80, the respirations 28, the leucocytes 15,000, the polynuclears 93%. The sputum showed thin mucus with fresh blood and a few pus cells. The specific gravity of the urine was 1.026 to 1.032. There was the slightest possible trace of albumin at two of four examinations. The sediment showed a few coarsely and finely granular casts and an occasional red blood corpuscle.

Following admission the pulse was very weak and the patient's condition extremely grave. January 24 the apex of the heart was just outside the nipple line in the sixth space. The action was heaving and forceful. The sounds were very distant. There was gallop rhythm. No murmurs were heard. The pulmonic second sound was greater than the aortic second, not accentuated. The aortic second sound was sharp and ringing. January 29 the patient appeared somewhat improved. A systolic murmur was heard all over the precordia, loud over the aortic area, but loudest at the apex and transmitted a short distance to the axilla. The aortic second sound was greater than the pulmonic second. February 3 he became pulseless and suddenly died.

*Clinical Diagnoses (from Hospital Records).*

First entry. Dec. 1904. Neurasthenia?

Second entry. Jan. 1906. Subacute bronchitis.

Third entry. June 1906. Myocarditis with dilatation.

Fourth entry. Jan. 1907. Arteriosclerosis.

Fifth entry. Jan. 1911. Arteriosclerosis.

Coronary sclerosis with thrombosis. Myomalacia cordis.

*Dr. Richard C. Cabot's Diagnosis.*—Arteriosclerosis.

Hypertrophy and dilatation of the heart.

Narrowing of the coronary arteries, very possibly with Myocarditis.

Terminal infection?

*Anatomical Diagnosis.*—Arteriosclerosis.

Arteriosclerotic occlusion and thrombosis of the coronary arteries.

Infarct of the myocardium.

Chronic interstitial myocarditis with slight aneurismal dilatation of the wall of the left ventricle.

Mural thrombi, left ventricle.

Fibrous and fibrocalcareous endocarditis of the mitral and aortic valves.

Hypertrophy and dilatation of the heart.

Chronic interstitial hepatitis.

Hyperplasia of the spleen.

Chronic pleuritis.

Obsolete tuberculosis of the lungs.

Emphysema of the lungs.

DR. RICHARDSON: The chronic interstitial hepatitis was not very marked.

DR. CABOT: A point of great interest in this last case is that the symptoms pointing to coronary occlusion and cardiac infarction apparently showed themselves as much as seven years before the patient's death. They are more definite and recognizable in the later years of his illness, but still are spaced by long intervals of very fair health e.g. between 1907 and 1911. Perhaps this case exemplifies a group intermediate between some of those ordinarily classed as *chronic fibrous* myocarditis (without acute seizures) and the more *acute infarction* next to be described.

## II. MYOCARDIAL INFARCTION

Since the studies of Herrick, Wearn*, and others the clinical picture corresponding to the blocking of a coronary artery with necrosis and infarction of the heart wall has been emerging more and more clearly.

The present series contains twenty cases of acute cardiac infarction, fifteen of them with blocked coronary. There were *nineteen males and one female.* The ages varied from thirty-five to seventy-nine but only three were under fifty, and *the average age was sixty years.* Considering that it is the arteriosclerotic coronary lesions which produce the infarction in most cases, this age is what one would expect. The extraordinary excess of males in this as in all the various series of cases either of infarct or of angina pectoris so far reported, has

* Wearn: Amer. Jour. Med. Sciences, February, 1923.

not so far as I know been satisfactorily explained. In Wearn's nineteen autopsied cases there were but nine women.

*Chronic passive congestion* was present in life and after death in twelve of our twenty and in eight of Wearn's nineteen. There had been previous anginoid attacks in six of Wearn's nineteen cases and in seven of our twenty cases. The interval between the first attack and the fatal seizure varied from two weeks to six months in five of our seven recurrent cases. But in one, the anginoid attacks began two years before death and in two others (men of seventy) there had been numerous anginoid attacks scattered through the seven years preceding death. One of these men described his sufferings as beginning in the left upper chest sometimes with "rheumatic" (dull?) pain, somtimes with "twisting pain." With this was a sense of tremendous pressure in the precordia and a feeling "as if his left arm was being torn off from his body." These seizures lasted three or four hours and had no definite relation to exertion, but seemed to him connected with "catching cold" (i.e. perhaps with cough due to passive congestion?). The attacks had been increasing in intensity, so that the patient said he knew he was "playing with death" (often near death?).

In the hospital ward December 11, 1902, he had prolonged precordial distress not relieved by nitroglycerin. December 17 an acute conjunctivitis appeared. The patient had been up and about the ward until the 25th, when slight dyspnea appeared. On the 27th there came a sudden sense of suffocation without pain but with great pallor and dyspnea. The heart was slow and regular but the chest rapidly filled with râles and he died in fifteen minutes from the onset of the attack.

At necropsy there was arteriosclerosis of the aorta and coronaries with thrombosis of the left *descendens* and some of its branches, fibrous myocarditis and an area of acute infarction in the left ventricle. Syphilitic hepatitis. Chronic passive congestion.

The occlusion of the coronaries was in places due to calcareous material, in other places to blood clot more or less organized.

The other long case which lasted from 1904 to 1911 has been described above (p. 508).

Histories like this and the frequent association of chronic fibrous scars with the acute infarction, as in twelve of our twenty cases, make it reasonable to assume that some at least of the older fibrous patches are due to previous infarcts which have accompanied earlier attacks and have healed so as to make recovery possible.

## SITE OF PAIN

A point of great importance is the *epigastric localization* of the initial pain in ten of our twenty cases. Such pain is rarely thought of by physicians as having any possible relation to coronary occlusion, but is often attributed to "acute indigestion," especially when it begins after a meal and is associated with vomiting. Probably this explains many of the sudden deaths reported in daily papers under the diagnosis of "acute indigestion" or "ptomaine poisoning," especially when the seizure occurs at a banquet, perhaps at the end of an exhausting address.

In Wearn's series the acute epigastric pain caused several patients to be operated on with diagnosis of perforated peptic ulcer, acute pancreatitis or acute cholecystitis. Others were mistaken for pneumonia or pleurisy.

Epigastric pain may be the beginning of attacks of ordinary angina pectoris without infarction. Moreover seven of the infarcted hearts in this group were associated with precordial and never with epigastric pain. Three were free from pain altogether, one dying after an operation for cancer of the sigmoid, the other two of diabetic coma with gangrene. In four cases the pain began in the epigastrium but was later felt in the precordia and in the left arm.

In distinguishing the epigastric pain due to cardiac infarct from the pain of acute peritonitis, it is of value to note that the pain of infarction is often accompanied by sudden pulse failure and fainting which are not signs of an early acute peritonitis. *Pulse failure*, i.e. a sudden rapidity, irregularity, then partial or complete disappearance of the radial pulse, was noted in nine of our twenty cases. *Sudden faintness* with or without nausea and vomiting was recorded in four cases. There was arrhythmia, usually auricular fibrillation, noted in nine cases.

*The duration of the attacks of pain* was for *hours or for days* in all our 20 cases. This point together with the lack of any relation to exertion or of any considerable relief from nitrites, give us the most important clues to the exclusion of ordinary angina pectoris.

*The coronary arteries* were arteriosclerotic in all our cases and one or more were occluded in fourteen of twenty cases. In the remaining six cases the coronaries are described as:

1. Sclerotic but not occluded (one case).
2. Sclerotic but, free (one case)
3. Sclerotic and narrowed but free (three cases).
4. Sclerotic and capacious (one case).

In four cases the occlusion was due to thrombosis (as well as to arteriosclerosis); in sixteen there was arterioslerosis alone.

*The systolic blood pressure* was over 165 in three cases (highest 250), 160/100 in one, normal in seven, not recorded in nine. *Hypertrophy and dilatation* of the heart was present in nineteen out of twenty cases, as would be expected from the age and the arteriosclerosis. Five of the hearts weighed 600–680 grams, eight from 500–600, two from 400 to 500, and five less than 400 grams.

*Intracardiac thrombi* were recorded in eleven of twenty cases.

*Fever* was present in eight cases, slight *leucocytosis* in thirteen; but neither of these was sufficiently constant or marked to be of diagnostic value in patients so acutely and desperately ill.

*Syphilitic aortitis* was found in three out of 20 cases (1816, 2488, 3759) and in two of these the mouths of the coronaries were obstructed by the syphilitis process though there was some sclerosis in the coronaries as well. In addition to these three there was evidence of syphilis in the liver of a fourth patient (992). The Wassermann reaction was tried for in 2 cases and was positive in neither.

As to *physical signs of cardiac infarction* we may say that there are none. No characteristic electrocardiographic changes are present, though peculiar complexes have been noticed in some cases. Arrhythmia may be present especially near the end of life but is not constant or characteristic. Systolic murmurs are often to be heard and never significant. In one of our cases (667) during the last seven hours of life (tortured with dyspnea and pain) there was audible over the precordia a series of short sharp sounds spaced at equal intervals and all exactly alike—apparently all first sounds. In the pulmonary area a faint ticklike suggestion of a second sound was to be heard after each of the first sounds. The latter corresponded in rhythm with these sounds. There was no arrhythmia. At the end the heart stopped suddenly.

Necropsy showed a thrombosed right coronary (sclerotic) and an area of acute infarction in the right ventricle. The thrombus extended down from a large clot in the sinus of Valsalva. Corresponding with the infarcted area there was a mural thrombus inside the heart and an acute pericarditis outside.

The patient's symptoms had begun six weeks ago with a sudden vertigo and a "feeling that his heart had stopped," together with severe precordial pain unrelated to exertion. These attacks recurred several times in the next six weeks.

This case is the only one in our series in which the right coronary alone was affected. In the other cases either the left coronary alone (9 cases) or both coronaries were affected (5 cases), or there was no actual occlusion (5 cases).

Clearly the *diagnosis* of coronary occlusion with infarction of the heart must depend chiefly on the history and course of the disease, not on physical examination. Hence it may be well to quote a few more of the patients' stories.

1. "A week ago I got a terrible pain at the pit of my stomach and an awful *pressing in* along with it. A while after I felt it in my heart and inside my left arm."

Two days after that on which he told us this, the patient (a man of sixty-three) woke at 2:50 a.m., suddenly sat up, gave a cry and fell back dead. Necropsy showed that the apical part of the left ventricular wall and about one-third of the ventricle above this was thinned to five or six mm. and showed much fibrous change in the midst of which there were, here and there, small dirty-yellowish homogeneous soft necrotic areas. The right coronary was free but showed much fibrous or fibro-calcareous change. The left was like a beaded pipe-stem or a solid cord, its lumen gone. In places this was owing to the sclerosis, but the vessel contained also a recent thrombus. There was an intraventricular thrombus over the site of the fresh infarction.

During the last two days of his life his cardiac impulse was regular and forceful though the heart sounds were weak. No cardiac murmurs were detected. His blood pressure was 120/85.

As he had dyspnea and palpitation for seven years, we may conjecture that the coronary thrombus originated from the intraventricular thrombus, though the sequence may have been in the opposite direction.

. . .

2. For seven or eight years a man of sixty-five had had occasional epigastric distress one to two hours after meals. Two weeks ago he had severe abdominal pain lasting most of four days, with dyspnea, orthopnea and edema. He lived forty hours in the hospital with orthopnea and double hydrothorax. I could hear almost no heart sounds at the apex. At the base they were normal though faint. No murmurs. Pulse regular, fair volume and tension. From his usual position leaning forward over his knees he suddenly fell back against the bed-rest. The interne went to him immediately, found

him pulseless and could hear no heart sounds. Apparently his sudden death was not preceded by pain.

Necropsy showed that the left coronary was practically occluded by arteriosclerosis. The right showed a moderate amount of fibrous and fibrocalcareous change and a reduction of its lumen at one point. The left ventricle was everywhere reduced in thickness except for a strip about 2.5 cm. wide where its wall measured fifteen mm. In the thinned portions it often came down to one mm. The wall (except for the strip just described) showed opaque discrete or confluent dull grayish-yellow areas. Also grayish-white streaks and patches of fibrous tissue. Towards the apex the wall was like thin leather. The changes extended into the region of His's bundle.

. . .

3. A man of sixty-eight had suffered for five years with attacks of epigastric pain and faintness. For a year he had felt soreness in the precordia, worse on exertion. Ten days ago he suddenly began to have pain limited to the epigastrium, severe, non-radiating (duration?). For four days he has had dull pain and "weakness" in the middorsal region.

During his first two days under observation he had "three distinct attacks of angina, with dyspnea and phlegm in his throat." The heart showed no enlargement clinically, was regular and not rapid. No murmurs. For most of the time during the last days of his life his pulse was strong and regular. In some of his anginal attacks his heart continued to be regular though the sounds became weak. In other attacks it was absolutely irregular with a large pulse deficit. Blood pressure 130/90. He died in a week.

*Necropsy:* Coronary sclerosis without occlusion. The myocardium of the left ventricle was reduced to one to five mm. in thickness with streaks and patches of fibrous tissue and indefinitely bounded, dull, dark brownish red, homogeneous areas. A mural thrombus in the left ventricle could only with difficulty be distinguished from the softened myocardium beneath it. The wall of the left ventricle was generally boggy and yielding. It tore easily and in places bulged (cardiac aneurism). There was subacute and also chronic pericarditis. The heart weighed 600 grams, though no enlargement was detected in life. Valves normal.

. . .

4. A woman of sixty-one was seized six days before she entered the hospital with very severe epigastric pain, faintness and vomiting. These symptoms recurred twenty-four hours before her entrance with

vomiting, and did not afterward abate. The heart was moderately enlarged, rapid and regular, with a rough apical systolic murmur replacing the first sound. The pulse was barely perceptible and disappeared soon after she reached the ward. Seen by Dr. Maurice H. Richardson who wrote: "There is a very strong indication for operation—probably to relieve pancreatic hemorrhage—but her general condition is too bad to warrant any interference." Dr. Hugh Cabot concurred in this diagnosis. A definite mass palpable in the epigastrium lent support to the idea of pancreatitis. The leucocytes were 17,000, the temperature normal. She died on the day of her arrival at the hospital.

At necropsy there was arteriosclerotic occlusion of the left coronary with softening and necrosis of the wall of the left ventricle. The heart weighed 307 grams, its valves normal. The epigastric mass was a congested liver. The thymus was persistent.

. . .

6. A medical army officer of fifty-two had been in excellent health and had done very strenuous work at Camp Devens in the spring of 1917. From May 3 to 13 he was prostrated with an obscure type of fever, thought to be typhoid or paratyphoid but without cardiac symptoms. (Wassermann negative, syphilis denied.) Recovery seemed complete, but June 3d and 4th he had several attacks of palpitation and irregular pulse, with belching but no pain. The attacks came soon after eating. Electrocardiogram June 14th showed a flat T-wave in Lead II; otherwise normal. June 24th at 8:10 he suddenly cried, "I can't get my breath," clutched the region of his heart, became very cyanotic, and died in a few minutes.

At necropsy the left coronary was practically occluded by arteriosclerosis, the right considerably narrowed. The left ventricle showed chronic fibrous myocarditis and fresh infarcts.

. . .

I have allowed a good deal of space for the description of myocardial infarct because it is certainly not uncommon as a cause of sudden death yet is not often considered at all in the differential diagnoses attempted in life. A skillful and conscientious medical examiner tells me that about 10% of all the medico-legal cases of sudden death examined by him in the last few years have proved to be cases of coronary occlusion with infarct.

We may distinguish four types:

(1) Sometimes death follows the occlusion so rapidly that there is no time for the formation of an infarct. This was true in four cases of

TABLE 117.—INFARCT GROUP, 20 CASES (INCLUDING 3 OF SYPHILITIC AORTITIS)

| Symptoms of stasis | Necropsy No. | Age, sex | Angina too | Clinical infarct symptoms | Necropsy fibrous myocarditis | Heart weight | Necropsy infarct | Cardiac aneurism | Remarks |
|---|---|---|---|---|---|---|---|---|---|
| 0 | 667 | 65 M | + | ? | 0 | 481 | + | 0 | Blocked right coronary. Cardiac infarction with mural thrombus. |
| + | 992 | 70 M | + | + | + | 400 | + | 0 | Syphilitic hepatitis. Left *descendens* blocked. |
| + | 1313 | 60 M | + | + | + | 566 | + | + | |
| 0 | 1537 | 79 M | + | 0 | 0 | 425 | 0 | 0 | Thrombus left coronary. Sudden death. |
| 0 | 1635 | 60 M | 0 | 0 | 0 | 340 | 0 | 0 | Sclerotic and thrombotic block left coronary. Sudden post-operative death. |
| + | 1816 | 36 M | + | + | + | 660 | 0 | 0 | Syphilitic aortitis. |
| 0 | 1840 | 68 M | 0 | 0 | 0 | 545 | + | 0 | Thrombus, left *descendens* with mural thrombus. Strep. sepsis. diabetic gangrene. |
| + | 2094 | 74 M | 0 | + | + | 555 | + | 0 | |
| + | 2134 | 69 M | 0 | + | 0 | 516 | + | 0 | |
| + | 2371 | 42 F | + | + | 0 | 332 | + | 0 | Syphilitic aortitis. |
| + | 2488 | 58 M | + | + | 0 | 585 | + | 0 | Syphilitic aortitis. |
| + | 2694 | 35 M | 0 | + | 0 | 555 | + | 0 | |
| P.M. | 2735 | 61 F | + | + | 0 | 307 | + | 0 | Left *descendens* blocked. Pancreatitis diagnosed in life. |
| 0 | 2787 | 70 M | 0 | + | + | 605 | + | + | Symptoms probably due to infarctions occurred at intervals for 7 years |
| + | 2820 | 65 M | 0 | + | + | 553 | + | | |
| + | 2871 | 65 M | 0 | + | + | 600 | + | | Did hard muscular work untill 2 weeks before his death |
| 0 | 3025 | 75 M | ? | + | + | 365 | 0 | | |
| 0 | 3177 | 69 M | 0 | + | 0 | 477 | + | 0 | Post-operative thrombosis of coronaries. |
| 0 | 3466 | 60 M | 0 | 0 | 0 | 390 | + | 0 | Chr. interstitial orchitis. Coronaries free. Diabetic gangrene, post-operative infarct. |
| 0 | 3843 | 66 M | + | + | + | 600 | + | + | |

our series (1537, 2371, 2566, 3177). Here the clinical picture may be exactly the same as in those with infarct. For example a woman of forty-two, with syphilitic aortitis which at necropsy occluded both coronary orifices, had suffered from typical attacks of angina pectoris for six months before her death. In the first of these she fainted and was unconscious for half an hour. Her second and fatal attack of faintness came on suddenly with clutching pain in the precordia. The heart beat feebly a few times and stopped. Post-mortem there was no infarct.

A second case of the same type but without evidence of syphilis occurred in a woman of sixty who had had one severe attack of angina pectoris three years previously and been dyspneic ever since then, with orthopnea for the past three weeks. Nevertheless a plastic operation on the abdominal wall was undertaken for the relief of enteroptosis then (1902) just beginning to become a fashionable diagnosis. She was cyanotic at the beginning of the operation and after it had a weak but regular pulse of 120. The heart was negative. On the second day after the operation vomiting and pulse failure suddenly supervened at 5:45 a.m. and she died in twenty minutes.

Necropsy showed that the left ventricle was only four mm. in thickness; its muscle largely replaced by fibrous tissue. The branch of the coronary running toward this thinned portion was more or less occluded by a reddish-gray translucent thrombus. The wall of the vessel showed a yellowish discoloration. The right coronary was slightly sclerosed. There was an acute pericarditis and infarcts of the liver and spleen. The heart weighed 435 grams, valves normal. No cardiac infarct.

*Aortic Stenosis with Acute Vegetations Blocking the Coronary Orifices.*—There was extensive fibrous myocarditis but no infarct and in life no pain or other symptoms beyond those of aortic stenosis, until one hour before her death when she suddenly began to have extreme dyspnea and became pulseless. (Necropsy 2238.)

(2) The second type, represented by seven cases of this series, shows acute infarction without chronic fibrous myocarditis and death follows the *first* coronary blocking.

(3) The third or *recurrent type* shows post-mortem both acute infarction and chronic fibrous myocarditis. The clinical picture and the history of these cases allows us to suppose that the fibrous myocarditis (or part of it) corresponds to previous occlusion of coronary branches, the infarct having become organized. Later a larger

branch is blocked and this results in an infarction which leads to immediate death.

(4) There are also occasional infarctions without demonstrable occlusion of an artery and without clinical symptoms. For example:

*Typical Cardiac Infarct without Any Symptoms in Life* (Necropsy 2183).—The patient died after an operation for diabetic gangrene and sepsis, without any particular heart symptoms and without any noticeable suddenness.

### SUMMARY AND CONCLUSIONS

1. Cardiac infarction is a frequent cause of sudden death in elderly men. As the patient may vomit in his attack, such deaths are often explained as due to "acute indigestion."

2. When death does not follow so swiftly, the severe epigastric pain often complained of may lead to an operation for pancreatitis, perforated peptic ulcer, etc.

3. An infarction may heal and another may occur months or years afterwards.

4. Sudden, severe, prolonged epigastric pain (often lasting for hours without relief by nitrites), especially if associated with faintness and pulse failure, should always lead us to suspect cardiac infarction. If in an elderly man there are prior, concomitant or subsequent "gastric" or anginoid attacks of the ordinary type, the diagnosis of infarct is probable.

5. Only about half the cases show any evidence of passive congestion before or after death.

6. Four types may be distinguished:
   (a) Potential infarction (blocked coronary and sudden death before an infarct has time to form.)
   (b) Rapidly fatal infarct.
   (c) Recurrent infarct with an interval of weeks months or years between the first attack and death.
   (d) Symptomless acute infarct.

### MYOCARDIAL INFARCTION—ILLUSTRATIVE CASES

#### Case 4138

First entry. An American factory girl of twenty-two entered November 7, 1895, for treatment of an abscess of the thigh which had discharged for seven years. Four operations had already been done.

At the fifth two discharging sinuses were enlarged and the abscess cavity curetted out. After a somewhat troublesome convalescence

she was discharged January 22, with a diagnosis of osteomyelitis May 20, 1896, she wrote that she had gained a great deal and was able to walk about the house without crutches.

Second entry. January 29, 1920. Age: 47.

Both parents died of heart disease. Her mother had several miscarriages. One living brother was very ill during childhood, had black skin, which peeled off, and was said to have inherited "bad blood." Several relatives died of cancer.

Additional past history. Measles and scarlet fever in childhood. Bronchitis for about six weeks every winter. Typhoid fever thirty-one years ago. Injured the right leg just above the knee thirty years ago. The disease described in the first entry followed. The knee was opened thirteen times and discharged constantly for nine years. Twenty-five years ago she had a sudden eruption of erysipelas over the legs several times. Twelve years ago she had tonsillitis. Nine

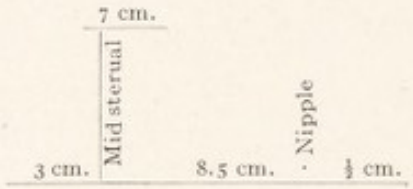



FIG. 98.—Measurements by percussion.

years ago the right leg was amputated. For a number of years she had throbbing headache every two weeks, better for the last twelve years, when she had worn glasses. For ten years, with the attacks of bronchitis, she had slight cough, palpitation, sharp localized precordial pain, and occasional attacks of orthopnea. She urinated five or six times at night. For the past few years there was occasional burning. Catamenia began at fifteen, but after one period ceased until eighteen. Since then it had been very regular, at times rather profuse. Slight brownish yellow discharge, especially after the periods. Four years ago she weighed 160 pounds, her best weight. Usual weight 150.

Three days ago, during the ten minutes' walk to the office where she was now a telephone operator, she had a feeling of depression and intense pain under the upper part of the sternum and suprasternal region. After sitting down for a short time she felt normal, but on resuming her walk had the same feeling until she sat down in her office. For the next two days she had the same pain on the way to the office. The day before admission it was more severe. In the late afternoon she had another attack, this time very severe, and for the first time radiating to the left shoulder and down the whole

left arm to the finger-tips. This lasted more than ten minutes, relieved by small white pills given by a physician. In the evening she had morphia, which kept her awake the greater part of the night. She vomited several times.

The patient was well nourished. Head and throat negative. Apex impulse of the heart not found. Action slow. Sounds of good quality. $P_2$ greater than $A_2$. Electrocardiogram showed normal rhythm, inverted T wave in Lead II. Pulses and arteries normal. Systolic blood pressure 100–125, diastolic 50–80. Abdo-

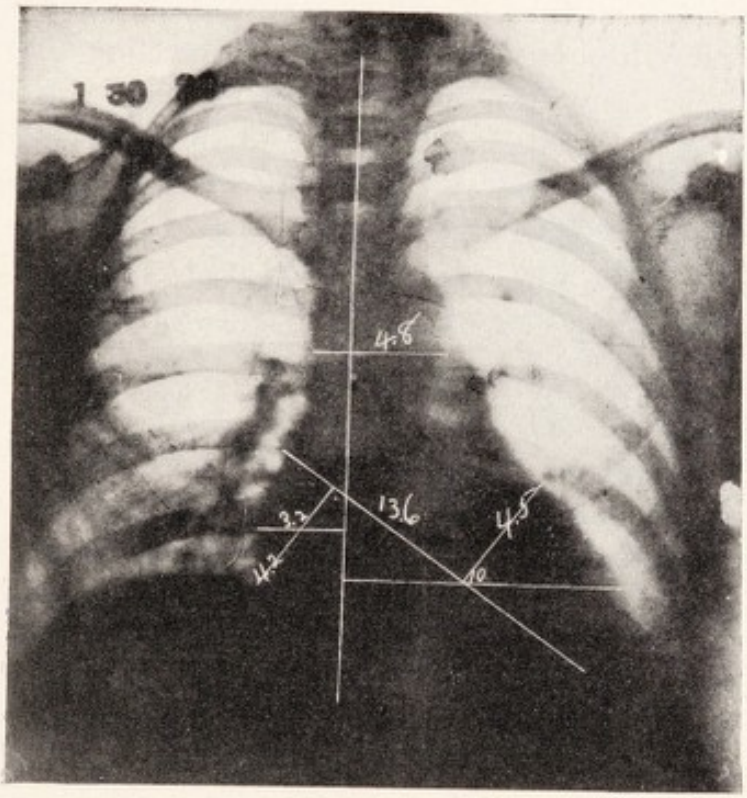



FIG. 99.—Necropsy 4138. Arteriosclerosis of the aorta with coronary occlusion. Thrombus of the left coronary artery. Mural thrombus of the left ventricle. Hypertrophy and dilatation of the heart. Measurements by X-ray the day before death. (Roentgenological Department, Massachusetts General Hospital.)

men: Liver dullness 5th rib to costal margin. Edge just felt on inspiration. Genital, rectal and pelvic examinations not made. Extremities and reflexes normal except for amputation of right leg above the knee. Pupils normal except for sluggish reactions to light and distance.

Temperature normal except for rise to 100° the day after entrance. Pulse 68–100. Respirations 15–28. Urine: Normal amount, except one record of ℥ 10. Sp. gr. 1020–1030. Cloudy at one of three examinations, a very slight trace to the slightest possible trace

of albumin at two. Renal function 60%. Blood: Hgb. 75%, leucocytes 8600–6400, polynuclears 79%. Wassermann negative. X-ray. Heart shadow enlarged downward and to the left. Prominence also in region of left auricle and pulmonary artery. Diameter across base of heart not particularly increased. Aortic knob prominent. (See Fig. 99.)

The patient was given the usual hospital diet and codeia gr. ½. Shortly after entrance she had an attack of pain in the chest referred down the left arm, relieved by nitroglycerin gr. 1/100. The same dose was given on ten out of fifteen days until February 13. Aspirin gr. v was ordered on four occasions for headache; also phenacetin gr. v once, and pantopon gr. ⅓ in four doses at one-hour intervals. February 21 and 22 pyramidon gr. v. was given. She had had no recent attacks at that time, had been up and about in a chair and able to walk a few steps. February 24 she was discharged to stay for a week in the country before returning to work.

Third entry, November 14, 1920. Since her discharge she had been very comfortable until two weeks ago, except for occasional precordial pains, easily and immediately controlled by sodium nitrite tablets. Two weeks ago she had a sudden sharp pain in the epigastric region, thought at first to be acute indigestion, somewhat relieved by eating toast and taking soda. Two days later the painful attacks were higher in the precordial region and occasionally radiated to both arms. The nitrite tablets had less effect, the attacks lasting five minutes or more and occurring on any exertion. She had taken




FIG. 100.

as many as twelve tablets a day. The morning of entrance a severe attack began at five o'clock and lasted until after eight without relief from codeia or morphia. She brought a note from her physician saying that she had had no treatment until two days ago. The morning of entrance she had had ¾ of a grain of morphia. Amyl nitrite made her vomit and increased her pain. She had been on sodium nitrite gr. i 4 i.d. for months and digitalis leaves gr. i.b.i.d. for two days. The present attack came on without any unusual exertion.

Physical examination. Obese. Complained of precordial pain after every movement. Grayish pallor with slight cyanosis of skin and mucosae. Lungs: Many fine and coarse bubbling râles at both bases behind, on the left rising to the angle of the scapula. Apex impulse of heart not found. Action slightly rapid. Faint tic-tac rhythm. Sounds of very poor quality. $P_2$ doubled and accentuated. Pulses of poor volume and tension. Arteries normal. Abdomen: Liver dullness from 5th rib to 3 cm. below costal margin, where a smooth non-tender edge was felt. Very slight edema of left ankle. Pupillary reactions very slight.

Temperature 97°–100.5°. Pulse 100–109. Respiration 20–27. Blood pressure 85/60–80/65. Urine not recorded. Blood: Hgb. 90%. Leucocytes 9600. Polynuclears 90%. Wassermann negative.

The patient was given soft solids. Fluids were limited to 1500 c.c. Morphia in ⅙ grain doses, once with atropin gr. $\frac{1}{120}$, was given p.r.n. every three hours, whiskey ℥ 2 b.i.d., digitalis leaves gr. iss t.i.d. She had a rather poor night and complained of soreness over the lower sternal region. Next day she suddenly became cyanotic, complained of sharp pain in the precordial region, had Cheyne-Stokes breathing, and died.

*Clinical Diagnosis.*—Angina pectoris.
Myocardial insufficiency.
Coronary occlusion?
Amputation of right leg.

*Dr. Richard C. Cabot's Diagnosis.*—Arteriosclerosis.
Arteriosclerosis of the coronary arteries with occlusion.
Myocardial infarction (probably).
Hypertrophy and dilatation of the heart.
Chronic passive congestion.

*Anatomical Diagnosis.*—Slight arteriosclerosis of the aorta.
Arteriosclerosis of the coronary arteries with occlusion.
Thrombus of the left coronary artery.
Infarct of myocardium.
Mural thrombus, left ventricle.
Hypertrophy and dilatation of the heart.
Edema of the lungs.
Fatty metamorphosis of the liver.
Soft hyperplastic spleen.
Fibromyomata of the uterus.
Edema piae.
Old amputation right thigh.

DR. OSCAR RICHARDSON: The brain was frankly negative, the vessels of Willis negative. The abdominal organs were out of the picture.

The heart weighed 415 grams. She was a strongly built, muscular woman, and the hypertrophy is moderate in amount. If she had a heart that weighed 325 grams we should not think anything about it. The cavities showed slight dilatation and the valves were negative. The left ventricle wall was 13 mm. and the right 3. That is about right. The myocardium generally showed no chronic myocarditis. But in the region of the left apex there was an area about two inches across where there was some thinning. In places it was somewhat fibrous and in other places rather soft. The right myocardium microscopically was negative. There was possibly a little fatty infiltration.

The left coronary showed considerable sclerosis, and about two cm. from the region where the coronary breaks up into branches one of the branches was occluded by a small thrombus. Of course the area in the left ventricle was connected with that occluding thrombus. Whether that was the last thing that occurred I do not know.

The vessel generally showed considerable sclerosis, and although the first portion of the right coronary artery was in pretty good condition, still it showed some sclerosis. Looking on a little further we found a marked pipe-stem artery. These coronaries had been diseased for a long time, and the left had been occluded by the small thrombus, which was the source of the infarct in the myocardium in the region of the apex.

The remarkable thing is that we should not have found more frank myocarditis. But every once in a while for some reason the tissues maintain their condition under the decreased supply of the blood. In the left ventricle, over the area of thinning and infarction, there were several frank thrombotic plaques, thrombi arising on the endocardium over that area of degeneration, in this case a perfectly frank cause for them. Somtimes we find them and really cannot state anatomically why they are there. There was no pericarditis and no growth from the blood, so that if there was any temperature it must have been due to the thrombi.

There was only a very slight amount of arteriosclerosis in the aorta. In this case it seemed to be particularly of the coronaries and no other vessels in the body. We took some tissue to be sure there was no syphilitic taint, and there was none.

The lungs showed some edema, otherwise nothing. The lymphatic apparatus was negative.

The anatomical diagnosis is essentially the basis for the clinical picture in this case.

A PHYSICIAN: Do you often find arteriosclerosis so localized as that?

DR. RICHARDSON: Yes; that is one of the strange things about arteriosclerosis.

DR. CABOT: That is one of the common things and one of the tragic things. We may have arteriosclerosis somewhere else and it will do no harm, but if we have two or three inches of it in this little place in the heart, that sclerosis will kill. This was a typical case of its kind. I think everyone sees cases of its exact counterpart. They run very much alike.

A PHYSICIAN: How common is it at that age?

DR. CABOT: Not very uncommon. We expect it later.

A PHYSICIAN: Are the coronaries more prone than other arteries to arteriosclerosis?

DR. CABOT: I don't think I could say that. I should say the abdominal arteries get it most, and then the big arteries like the aorta. I should say the brain arteries suffer as often as the coronaries.

DR. RICHARDSON: Yes, it seems to hit the coronaries or the vessels of Willis.

## III. MYOCARDIAL ABSCESS

In our 4000 necropsies there were fifteen cases in which genuine abscess of the myocardium was found. Cases of myomalacia (infarction) without true pus are here excluded. Only five of these fifteen cases were over the twenty-fourth year. One case occurred in a baby only eighteen months old, and excluding this case there were six others at or under the fifteenth year. There were thirteen males (seven boys) and two females in this group.

In all cases the cardiac abscess was only a minor item in a general septicemia. This septicemia originated in osteomyelitis in four cases. The septic focus in two cases was the heart valves, in one case a carbuncle, in one case the middle ear, and in two cases a small incised wound of the forearm. In four cases no primary focus could be distinguished.

In one case an actinomycosis of the chest wall was the beginning of the disease which in this case ran a much more chronic course than any of the others, corresponding to the difference in etiology.

The micro-organisms present were staphylococcus aureus in nine cases, streptococcus in three cases, both of these organisms in one case, actinomycosis in one case, while the remaining case remains in doubt.

Ten acute septic cases ran a rapid course lasting from two to eighteen days from the first symptom to death. One case of actinomycotic abscess lasted four months and really belongs to a different group from the rest.

*The symptoms are those of a general septicemia without anything to call attention to the heart.* Chills, "picket-fence temperature," marked leucocytosis (90,000 in one case), and increasing stupor ("typhoidal state") are the main features. One of our cases was mistaken for typhoid fever. In the rest the diagnosis of a general septicemia was made, but there was no suspicion of any cardiac involvement.

*Nor were there any physical signs of importance.* The heart was not enlarged, clinically or at necropsy, in twelve of the fifteen cases; in three cases (aet. 28, 40, and 46) there was slight enlargement (345, 429 and 450 grams). No murmurs were heard in thirteen cases, systolic apical murmurs in two. There was no arrhythmia.

Suggestions of *embolism* were noted in one case in which one radial pulse disappeared and the spleen increased rapidly in size during observation—an event pointing always (in my experience) to an infarct, whether bland or septic. In two other cases a double septic parotitis gave evidence of the diffuseness of the septic processes, if not of embolism.

At necropsy the following metastatic infectious processes were present:

TABLE 118.—LOCAL SEPTIC PROCESSES ACCOMPANYING CARDIAC ABSCESS

| | |
|---|---|
| Renal abscess | 12 |
| Pericarditis | 9 |
| Lung abscess | 8 |
| Empyema | 5 |
| Splenic abscess | 4 |
| Subcutaneous abscess | 3 |
| Multiple synovitis | 3 |
| Liver abscess | 2 |
| Brain abscess | 2 |
| Abscess of the prostate | 1 |
| Abscess of the thymus | 1 |
| Abscess of the rectus muscle | 1 |
| Retroperitoneal abscess | 1 |

During life there were, as has been said, identifiable foci of sepsis, most often in the bones, in peripheral wounds, or on the heart valves.

The coronary arteries showed nothing of note in thirteen cases. In one case (Necropsy 59) the branches of the coronary artery in the left ventricle contained grayish-red plugs. In another, one of the arteries was disorganized. One of the abscesses extended to the region just beneath the epicardium. They were usually covered with patches of fibrous exudate. Outside the well marked abscesses, there was often invasion of the spaces between the muscle fibers by polynuclear leucocytes, and masses of cocci.

## SUMMARY

Our general conclusion, from these cases, is that it is hopeless to make any attempt to diagnose or to treat myocardial abscess. It may be suspected, like renal abscess and pericarditis, as a frequent complication of staphylococcus sepsis, and especially of osteomyelitis, in boys.

One wonders whether healing and recovery ever take place in such cases. I see no reason why this is not possible. Perhaps some of the patches of scar tissue—which we find in the myocardium without any close relation to the main branches of the coronary—are the remains of healed myocardial abscesses.

## MYOCARDIAL ABSCESS—ILLUSTRATIVE CASES

### Necropsy 3167

A nine-year-old school boy came to the Accident Room March 2. His father said that except for measles and whooping cough the boy had been perfectly well until a week before admission, when he bruised his shin. That evening when his shoe was taken off he complained of pain just above the ankle. The next morning he was unable to stand on the foot and complained of pain in the lower leg. This had persisted. February 24 the leg was red and swollen. February 25 the left arm became sore, and the following day the lower arm and elbow were red and swollen. The day of admission the right arm began to be sore. The boy had complained chiefly of pain, most severe in the leg. He had eaten and slept very little.

Examination showed a fairly well nourished, rather pale boy. The throat was somewhat reddened. The tonsils were slightly enlarged. The heart and abdomen were normal. There were râles throughout both lungs. The right leg from the knee, including the

ankle, was much swollen and red. About the lower part of the foot and ankle, there were many blebs filled with serum. There was exquisite tenderness on pressure over the tibia. The left arm from the elbow to the wrist was swollen, very tender, and in the lower half somewhat reddened. There was an indefinite sense of fluctuation in the soft parts. The right elbow was somewhat swollen and very tender to pressure and passive motion. X-rays showed nothing definite. The temperature at admission was 104°, the pulse 120, the leucocytes 22,000.

Operation was done the day of admission. The periosteum of the right tibia was dissected entirely free the length of the bone by a large abscess. There were pockets of pus in the popliteal space and about the ankle. Other abscess cavities were found on the right ulna and the right humerus. Trephine holes into all these bones showed pus. The boy was in poor condition after the operation. The next day the pulse and general condition seemed a little better. March 5 there was tender swelling in the region of the right parotid and the boy seemed worse. The following day this swelling was very marked and was extending upward over the scalp. There was also some swelling in the left parotid region. March 7 both sides were opened and pus was obtained from inside both parotid sheaths. March 9 the boy died.

*Clinical Diagnosis.*—Osteomyelitis, right tibia, right radius, left ulna.

Double septic parotitis.

Sepsis.

Exhaustion.

*Dr. Young's Diagnosis.*—Septicemia, staphylococcus.

Osteomyelitis.

Multiple abscesses.

*Anatomical Diagnosis.*—Septicemia, staphylococcus pyogenes aureus.

Osteomyelitis.

Pyemia, abscesses of the lungs, myocardium, thymus, kidneys, and anterior thoracic wall.

Serofibrinous pericarditis.

Fibrinopurulent pleuritis, double.

Septic spleen.

Operation wounds.

DR. RICHARDSON: We were not permitted to examine the head. In these cases abscesses and meningitis are found at times.

DR. YOUNG: There were no mental symptoms mentioned.

DR. RICHARDSON: One each side of the neck just below the angle of the jaw an open wound extended into the deep tissues. In the region of the left forearm an open wound extended down to the ulna, in which there was a small surgical opening. In the region of the inner aspect in the lower part of the right upper arm an open wound extended down to the humerus, in which there was a small surgical opening. In the region of the right leg an open wound extended down to the tibia, in which there was a small surgical opening.

There was a small amount of subcutaneous fat. The muscles were pale and soft. At one place in the subcutaneous tissues of the anterior thoracic wall there was a small collection of pus.

The peritoneal cavity and gastro-intestinal tract showed nothing to record. Once in a while in these septicemias we do find lesions in the mucosa of the gastro-intestinal tract. They vary from hemorrhagic areas to areas of hemorrhagic necrosis, and may go on to perforation. In one case along the mucosa of the small intestine there were small collections of pus,—abscesses. In this case although there was a marked infection the gastro-intestinal tract escaped.

The lungs were weakly bound to the parietal pleura by yellowish fibrinopurulent material,—abscesses of the lungs which had extended through and involved the pleura. The bronchial lymphatic glands, the trachea and bronchi were negative.

The pericardium showed a frank acute pericarditis. The heart weighed 125 grams. In the myocardium here and there were small collections of pus,—abscesses. The circulatory apparatus otherwise was negative. The kidneys were dotted over with minute abscesses. The culture from the spleen, which was soft, gave a good growth of the staphylococcus aureus. There were some small abscesses in the thymus. All told a frank case of pyemia, the portal of infection debatable.

DR. YOUNG: He hit his shin and it started immediately after that.

DR. RICHARDSON: That is the usual story.

DR. CABOT: I should like to say a word on the modern hope of finding some sort of chemical treatment for these cases. He applied that only to the very serious cases like this. I venture to prophesy that if we develop valuable drugs of the gentian violet type, we shall apply them not only to the apparently hopeless case but to the early case which, if left alone, becomes appendicitis, becomes cholecystitis. We do not often take our histories from the point of view of the hypothesis that those supposed local lesions are septicemias

first. We know that typhoid, pneumonia, meningitis are first septicemias and later local, because those have all been proved. I believe that appendicitis and cholecystitis are the same. I believe that with careful history-taking and especially with this point of view behind that history-taking, we are going to be able to recognize these cases in the septicemic stage before they become appendicitis and cholecystitis, and, if we get any sterilizing drug, to put it in early as we do with tetanus now when it has much more chance of doing good.

I feel that we are going to have a rearrangement of our ideas about the so-called local lesions, which I believe are not first local and then general, septicemia coming from the local, but general septicemia first and then the local lesion.

A PHYSICIAN: Do you think that principle might apply to furunculosis?

DR. CABOT: I think so. I do not believe furunculosis is a lesion that comes in from the skin in most cases; it comes from a weakening of the powers of resistance against the bacteria which are constantly in the skin or in the blood. Adami and others have shown how common it is to have bacteria circulating in the blood, how common to have mild septicemias which we never complain of at all, the bacteria being excreted by the kidney and never making any localizations. That sort of work and the work of Dr. Frank Kidd* seem to me to show how mild septicemias often are. I think, we often do not find them out. Whereas we have thought of septicemia as a terrible disease almost invariably fatal, I think it is a common disease often mild and in most cases not recognized at all.

DR. YOUNG: The trouble is that up to the present the drugs are themselves very upsetting. There is no question but that there is a considerable risk of mercurial poisoning in using mercurochrome. I think the drug has to be of guaranteed harmlessness to have it used in the majority of cases. But I do believe what Dr. Cabot has said about the large number of these cases at least starting as septicemias.

### Necropsy 3575

An American reporter of twenty-two entered March 26. Two years before admission he stayed out of work for two months because he was run down and had lost weight. Otherwise he had always been well until six weeks before admission, when he was ill in bed for two weeks with "grippe." He was feverish, coughed a little, and lost fifteen pounds. A week before admission he had a swelling on

* Common Infections of the Kidneys—London, 1920.

the back of the neck. This became increasingly large, painful and tender. He had hardly been able to move his neck, had had a temperature of about 103°, and had felt very ill. For the past two days the pain had prevented him from swallowing.

Examination showed a fairly well developed, poorly nourished man looking ill and holding his head turned stiffly to one side. He was unable to open his mouth more than half an inch. The skin was moist and pale, the tongue thickly coated. On the back of the neck was a sloughing area the size of a nickel, surrounded by brawny induration extending half-way around the neck. The edema completely encircled the neck. The heart was normal except for a systolic murmur at the apex. The rest of the physical examination is not recorded.

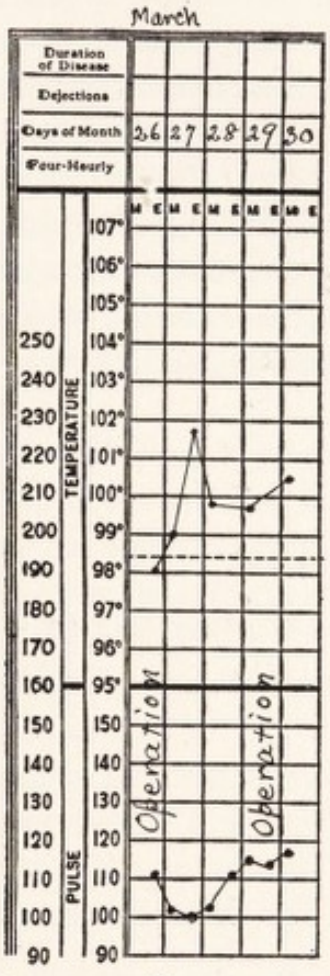



FIG. 101.—Temperature and pulse in Case 3575.

The temperature and pulse are shown in Fig. 101. The respirations were 23–28. The urine was normal in amount, the specific gravity 1.030. There was a slight trace of albumin. The blood was not examined until after operation. A throat consultant reported, "I cannot see the glottis, but as far as I can see there is no edema. The symptoms of inspiratory without expiratory dyspnea, esophageal obstruction, and good phonation point to an obstruction between the larynx and the sternal notch affecting both trachea and esophagus. The only possible pharyngeal neurosis would be a bilateral abductor paralysis, which is very improbable except in cases of tabes."

Operation was done the day of entrance. A T-incision was made through the sloughing area deep into the muscles of the neck. No pus or necrotic tissue was obtained. A culture showed staphylococcus.

The patient made a good post-operative recovery and was somewhat relieved, being able to open his jaws about an inch. There was a fair amount of staining. He complained of some difficulty in breathing. The edema and induration on the back and sides of the neck were somewhat increased. The leucocyte count was 23,400. March 28 he was distinctly worse, unable to separate his jaws more than a quarter of an inch.

March 29 a two-inch incision was made in the right post-cervical region. Finger dissection revealed no free pus. The tissue was brawny, red, and indurated. A similar incision was made on the other side of the neck, but no free pus was obtained. The wounds were left wide open and packed with iodoform gauze. He was very comfortable during the afternoon, but early in the evening began to grow rapidly worse, with difficulty in breathing, principally inspiratory stridor. March 30 he suddenly died.

*Clinical Diagnosis (from Hospital Record).*—General septicemia.

Carbuncle of neck.

Cardiac failure.

Incision and drainage, March 26.

Wider incision and drainage of neck, March 29.

*Dr. Hugh Cabot's Diagnosis.*—Deep cervical abscesses not satisfactorily drained.

General septicemia, staphylococcus.

*Anatomical Diagnosis.*—1. Primary fatal lesion. Septicemia, staphylococcus.

2. Secondary or terminal lesions.
- Abscesses of the lungs, myocardium, kidneys, and rectus muscle.
- Small mural thrombus of the right ventricle.
- Acute pericarditis.
- Acute peritonitis.
- Subacute interstitial heptatitis.
- Soft hyperplastic spleen.

3. Historical landmarks.
- Operation wounds.
- Slight chronic pleuritis.

Dr. Richardson: I would not be sure that there was no free pus in the neck. So far as I could dissect there was not any. There was no edema of the larynx.

Typical staphylococcus infection with abscesses everywhere, pericarditis, and peritonitis,—a profound infection.

## IV. Cardiac Aneurism

In twenty-five out of 91 cases of myocarditis there was a thinning of the left ventricular wall near the apex. *Cardiac aneurism occurred in seven* of these, associated with fibrous myocarditis or cardiac infarcts. The lesion caused no recognizable signs in life

and is of no practical importance to clinical medicine. Ordinarily the weakened tissues bulge outward but in one peculiar case (No. 2413) the myocardium was split and bulged inward so as to produce a narrowing of the mitral orifice.

The patient, a woman of thirty-nine, had suffered with cough and palpitation for a year but had had no dyspnea till three weeks before we saw her, when it led rapidly to orthopnea and prevented sleep. Edema and other evidence of stasis were obvious at the time of her admission to the hospital. The heart showed moderate enlargement with a weak diffuse impulse over which was heard a loud harsh systolic murmur replacing the first sound and ending in a feeble second sound. There was no arrhythmia. The pulmonic second sound was accented, the aortic second not remarkable. Edema persisted despite rest and digitalis. The pulse rose steadily in rate, reaching 160 in ten days. After that there were occasional extra systoles at regular intervals but no fibrillation. The patient in most respects seemed to be holding her own during the month of her stay, but on the twenty-eighth day she suddenly screamed, turned blue, and died in a few minutes.

Necropsy showed a heart weighing 426 grams, the right ventricle four mm. in thickness, the left eleven mm. The valves were normal. With the heart laid open and held apex down, the anterior cusp of the mitral valve was continuous with a projection from the left ventricular wall forming a sort of shelf which roofed over a cavity in the ventricular wall itself. The floor of this cavity consisted of the myocardium of the left ventricle and papillary muscle. Its roof was formed by the elevated endocardium, the mitral valve, and the left auricle which skirted over and slightly around it. The cavity measured six cm. in greatest diameter and during life, when full of blood, must have caused quite a little obstruction of the mitral orifice, though the valve was itself normal.

The myocardium showed fibrous scars. The coronaries were normal.

Among the other cases of this series, four showed no signs or symptoms during life by which they could be differentiated from the ordinary senile failing heart of the hypertensive-arteriosclerotic type. Two cases showed no passive congestion post-mortem, one being clinically pneumonia without heart symptoms, the other senile dementia. Either angina pectoris or symptoms suggesting cardiac infarction or both were present in five out of seven cases. There was coronary occlusion in three.

TABLE 119.—CARDIAC ANEURISM

| Case No. | Necropsy No. | Age, sex | Heart weight | Condition of heart wall | Angina | Infarct | Passive congest. | Arrhythmia | Mural thrombi | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 1060 | 59 M | 545 | Left ventricle thinned, fibrous (4 mm.) bulging at apex. Left *descendens* blocked. | 0 | 0 | 0 | 0 | 0 | Clinically pneumonia and nothing else. Chronic lepto-meningitis. |
| 2 | 1313 | 60 M | 566 | Apex thinned with an angurismal bulge. Coronaries thickened but free. | + | + | + | 0 | + | 3 attacks suggesting infarction of the heart. B.P. 170 syst. Strep. sepsis post-mortem |
| 3 | 1431 | 58 F | 410 | Apical aneurism, left ventricle; left *descendens* blocked. | + | ? | + | + | 0 | Cardiac infarct suggested by symptoms 7 years previously. |
| 4 | 2413 | 39 F | 426 | Aneurism bulged *inward* impinging on mitral valve so as to narrow it. Coronaries free. | 0 | 0 | + | 0 | 0 | Clinically decompensated heart. |
| 5 | 2787 | 70 M | 605 | Coronaries sclerosed but free. Apex showed marked thinning and yields to the touch (aneurism left ventricular apex). | 0 | + | 0 | 0 | + | B.P. 140 systolic. Decompensation. Sudden, painless death. |
| 6 | 3365 | 76 F | 565 | Left ventricle thin (1–2 mm.) membranous, baggy. Left coronary occluded. | + | 0 | + | + | + | B.P. 105/70. Clinically, senile dementia. |
| 7 | 3843 | 66 M | 600 | Left ventricle thinned, fibrous, tears easily. Shows aneurism and infarction. Coronaries sclerosed. | + | + | 0 | 0 | + | B.P. 130/90. Clinically, decompensated heart. |

On the whole it seems probable that the myocarditis and *thinning of the apex (left ventricle) with or without an aneurismal bulge, represents the healing of an old cardiac infarct.*

There are no clinical manifestations corresponding to such a thinning, whether it bulges (aneurism) or not.

## ILLUSTRATIVE CASES—CARDIAC ANEURISM

### Necropsy 3842

An unoccupied American of sixty-eight entered May 27 for relief of weakness and abdominal pain. His father died of enlarged liver, possibly cancer, one sister of cancer of the stomach. One child died of throat trouble at ten days. The patient had the usual diseases of childhood, including scarlet fever. In youth he often had tonsillitis. Fifteen or twenty years before entrance he was operated upon for hemorrhoids, and since that time had had them two or three times for short periods. He had had no gastric symptoms except very occasional burning indigestion easily relieved by mint tablets. For a year he had had uncomfortable soreness over the precordia, worse on exertion. He rarely used tobacco, and had taken very little alcohol for six years. Before that he was for years a steady drinker, three or four times a day, and was occasionally slightly intoxicated. Eight years before admission he weighed 180 pounds, his best weight; a year ago, 140. He had not lost during the six months preceding admission.

Except for the soreness over the precordia he had had no cardiac symptoms until May 15, when after no unusual exertion but rather indigestible food he had sudden severe non-radiating pain limited to the epigastrium, with marked dyspnea and orthopnea. He described the pain as a sense of "a ball in his stomach acting as a shut-off to his breath." He became markedly constipated and during ten days had not had a normal movement, though he had passed flatus freely and had belched some gas. One enema gave a partial result, two others very meager results. The stools were straw-colored, like his former stools. For three or four days at the onset he had marked dull constant pain and weakness in the middorsal region and lower in his back near the midline, relieved by massage and catharsis, which also somewhat relieved the epigastric pain. For three days after the onset he had flashes before his eyes and dizziness. Since the onset he had had slight nausea and marked repugnance to food.

Examination showed a fairly well nourished man, cyanotic, with moderate inspiratory distress. The eyelids were puffy, the skin dry, the mucosae slightly cyanotic. The few remaining teeth were poor. The heart was normal in size and position. The action was regular, not rapid. The sounds were of poor quality. The aortic second sound was accentuated and was heard in the neck. The artery walls were palpable, the brachials tortuous. The lung signs were as shown in Fig. 102. There was slight tenderness over the liver. The liver dullness extended 5 to 7 cm. below the costal margin, lower on deep inspiration. The edge went deep into the flank. The extremities, genitals and reflexes were normal. The pupils were equal, irregular, especially the right, and reacted to light and distance.

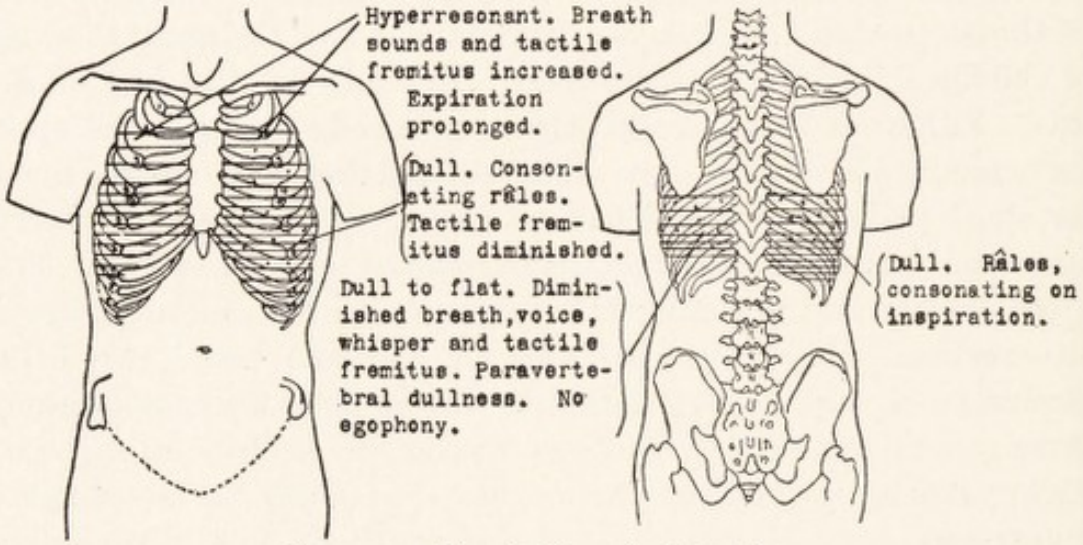



FIG. 102.—Physical signs in Case 3842.

The temperature was 96.6° to 99.2° until May 30, then 98.1° to 102.8° until June 2, falling to 95.4°. The pulse was 88 to 130, the respiration 78 to 36. The systolic blood pressure was 135, the diastolic 90. The urine was normal in amount, the specific gravity 1.028 to 1.014. There was the slightest possible trace of albumin at two of three exminations, pus at two, rare red blood corpuscles, hyalin and granular casts at the last. The hemoglobin was 80%. There were 17000 to 9200 leucocytes, 80% polynuclears. The smear showed achromia and slight variation in size. A Wassermann was negative.

The patient had three distinct attacks of anginoid pain accompanied by difficulty in breathing and in the pharynx and the trachea a sense of phlegm which he felt would cause suffocation if he did not raise it. He coughed hard, producing nothing. The effort precipitated a real attack with great sense of constriction of the chest and

a feeling of suffocation, horror and air-hunger. During the attacks his pulse became absolutely irregular. There was marked pulse deficit with real delirium cordis. The heart sounds were weak, the face ashen-gray. The heart became regular several hours after the attacks, and remained regular during the last one. It was doubted if amyl nitrite helped him. June 1 he became very much worse, his chest was full of râles, and he was expected to die. The therapy was continued, however, digitalis by mouth, digifolin, morphia, and also large doses of caffein, gr. v, at frequent intervals. He at once showed improvement, which continued until the night of June 3. Then his pulse became very weak and rapid, his temperature subnormal. He coughed considerably. His breathing was very difficult, though there were fewer râles than previously. June 4 he died.

*Clinical Diagnosis (from Hospital Record).*—Chronic myocarditis.
Arteriosclerosis.
Angina pectoris.
Chronic nephritis.
Chronic bronchitis.
Chronic decompensation.

*Dr. William H. Smith's Diagnosis.*—Arteriosclerosis.
Arteriosclerotic heart.
Probably coronary occlusion.
Passive congestion, pneumonia, or infarction of the lungs.
Arteriosclerosis and infarctions of the kidneys.

*Anatomical Diagnosis.*—Arteriosclerosis.
Arteriosclerotic occlusion of the left coronary artery.
Chronic myocarditis with aneurism and infarction.
Mural thrombus, left ventricle.
Chronic pericarditis.
Subacute pericarditis.
Hypertrophy and dilatation of the heart.
Purulent bronchitis.
Edema of the lungs.
Hydrothorax, right.
Arteriosclerotic degeneration of the kidneys, very slight.
Slight chronic interstitial hepatitis with hyaline degeneration of branches of the hepatic artery.
Chronic pleuritis.
Obsolete tuberculosis of bronchial lymph nodes.
Focus of obsolete tuberculosis, apex of the right lung.
Chronic perihepatitis and splenitis.

Cholelithiasis.

Cysts of the right kidney.

Slightly defective closure of the foramen ovale.

DR. RICHARDSON: The background in this case was arteriosclerosis, and the various lesions in the organs were the end-result of it. The aorta and great branches were rather capacious, but in the first portion of the aorta there was only a slight amount of fibrous sclerosis. When we came to the arch, the descending portion of the thoracic aorta and the abdominal portion of the aorta, we saw that the great artery and its branches were the seat of marked fibrous, fibrocalcareous and atheromatous sclerosis, a good anatomical basis for the clinical condition.

The heart weighed 600 grams (normally 200–300). The valves showed nothing worthy of discussion. The right coronary artery showed a moderate amount of fibrous sclerosis. It was rather capacious, and the areas of sclerosis were scattered. The left one, however, showed an entirely different picture. Along a length of three or four cm. of the first portion, the wall showed marked fibrous thickening with much fibrocalcareous change which occluded the artery at one point. From this point on the sclerosed artery and branches are lost in an area of myocarditis. As a result of these changes in the coronary artery, which had of course gone on for years, there was present in the wall of the left ventricle a well established chronic myocarditis in a large area supplied by the branches of the left coronary artery. There were also numerous dirty brownish-red areas which resembled areas of infarction, and the probability is that some of the branches running to that area of myocarditis had become occluded, with resulting softening and necrosis in the areas of chronic myocarditis. On the endocardial surface which rested along the area of myocarditis just described, there was erected within the ventricle a large thrombus, so intimately associated with the wall, which was thinned out and bulging, that in the necessary manipulation in the examination, the heart wall tore off with pieces of the clinging thrombus attached to it. There we have the whole process, a slow-growing one due to the gradual decrease in the supply of blood and later more or less sudden occlusion of some of the branches with areas of infarction and the production of a mural thrombus. It is interesting to note that while ordinarily pieces of thrombus sweep off into the blood stream with the erection of infarcts in different organs, in this particular case there were no infarcts anywhere. A possible reason is that the thrombus developed beneath the smooth

membrane which rested over the exposed surface in the ventricular cavity and prevented it from fracturing.

The pericardium showed at one place a broad band of fibrous adhesions, and in addition a general coating of grayish-red granular material which weakly bound the two layers of the pericardium together; that is, a band of chronic pericarditis and a more or less extensive subacute pericarditis.

There was a moderate amount of chronic pleuritis and some few areas of obsolete tuberculosis both in the bronchial lymph nodes and in the apex of the right lung. These pleural adhesions plus the adhesions of the pericardium, together with the arteriosclerosis, are sufficient in themselves to cause the extensive hypertrophy of the heart.

About the liver and spleen there were numerous old fibrous adhesions, chronic perihepatitis and splenitis. The liver showed slight cirrhosis along the portal canals with some hyalin degeneration of branches of the hepatic artery.

The gall-bladder contained about seventy small stones, but the mucosa was negative. The bile ducts and pancreatic ducts were also negative.

The kidneys weighed 340 grams (normally 200–400) and were in very good condition.

**Necropsy 4283**

An Irish laborer of sixty entered January 7, 1922, for relief of dyspnea and cough of twenty-five years' duration. He was in considerable respiratory distress and not cooperative. He had measles, mumps and scarlet fever in childhood. His teeth had always been bad. In general his health had been very good until he was thirty-five. He was ill in bed at forty with pneumonia. He had typhoid fever at forty-five. For a year he had had some difficulty in starting the urinary stream and some burning on urination. He formerly drank a pint of whiskey a week. For the past few years he had taken none.

For twenty-five years he had had "asthma," "bronchitis," and frequent palpitation accompanied by slight dizziness and slight cough with thick brownish sputum. He had slight dyspnea on moderate exertion with the asthma until 1917. During the past twenty years he had had attacks of precordial pain, numbness and soreness over the precordial region, and sharp pain in the pit of the stomach and the left arm. These attacks had no relation to meals,

were accompanied by gaseous eructations, considerable belching of gas, and marked nausea. *Soda had always relieved the pain!* For five years his dyspnea had occurred more easily and he had urinated fifteen times at night and frequently by day. During the past year he had had to rest for several periods of half an hour each during the day to relieve the dyspnea, and to use five pillows at night. Six months ago he visited a Boston hospital for relief of "indigestion and cough," and was given pills and powders which relieved the "indigestion" and made him bring up a "quart of sputum at a time." He worked until two weeks before admission taking care of horses. His last attack of pain occurred a few days before admission and lasted two hours and a half.

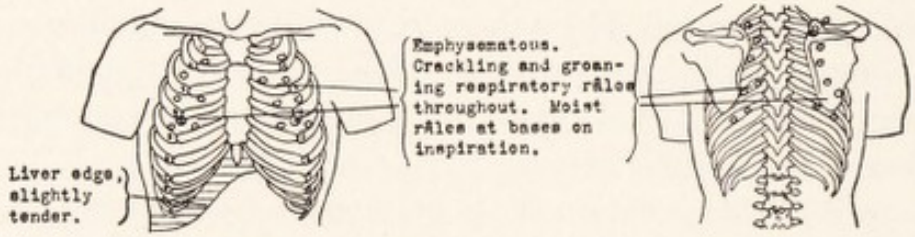



FIG. 103.—Physical signs in Case 4283.

Examination showed a well nourished man with marked respiratory distress. The skin was dry. The mucous membranes were slightly cyanotic, the sclerae injected. There were many broken tooth roots and marked pyorrhea. The lung signs were as shown in Fig. 103. The chest expansion was small, the diaphragm excursion limited. The apex impulse of the heart was seen and felt in the sixth space 11 cm. to the left, 2.5 cm. outside the midclavicle. The right border of dullness was 5.5 cm. to the right of the midsternum, the substernal dullness 8 cm. The action was rapid, regular, with tic-tac quality. The pulmonic second sound was equal to the aortic second. The artery walls were negative. The pulses were of fair volume and tension. The systolic blood pressure was 120, the diastolic 90. The abdomen was distended. The liver dullness extended from the fifth rib to two centimeters below the costal margin. There was reducible right inguinal hernia. The fingers and toes were clubbed. The rectal examination showed the prostate enlarged. The pupils and reflexes were normal.

The temperature was 99° to 101°, the pulse 81 to 111, the respirations 20 to 45. The amount of urine is not recorded. The specific gravity was 1.030. The urine was alkaline at the single

examination and showed the slightest possible trace of albumin; no sugar. The hemoglobin was 75%. There were 22,000 to 29,600 leucocytes, 82% polynuclears, 4,568,000 reds, slight achromia. The platelets were increased. A Wassermann was negative.

The morning after admission the patient was evidently much worse. He had Cheyne-Stokes respiration with apneic periods as long as fifteen seconds. During these periods he would not respond to questions, though he was rational during the times of active breathing. That afternoon when apparently in about the same condition he suddenly died.

*Clinical Diagnosis (from Hospital Record).*—Emphysema.
Chronic bronchitis.
Myocarditis.
Pericarditis.
Arteriosclerosis.

*Dr. Richard C. Cabot's Diagnosis.*—Arteriosclerosis of the coronary arteries, with obstruction.
Myocarditis.
Hypertrophy and dilatation of the heart.
General arteriosclerosis.
Chronic bronchitis?
Bronchiectasis?
Bronchopneumonia?
Chronic passive congestion.

*Anatomical Diagnosis.*—Arteriosclerosis of the coronary arteries.
Arteriosclerotic thrombotic occlusion of the right coronary artery.
Myocarditis and cardiac aneurism.
Mural thrombosis of left ventricle.
Arteriosclerosis.
Chronic adhesive pericarditis.
Chronic passive congestion.
Acute pericarditis.
Hypertrophy and dilatation of heart.
Slight ascites and anasarca.
Chronic pleuritis.

DR. RICHARDSON: This man was rather short, well developed and fairly well nourished. We were not permitted to examine the head. The skin in the region of the base of the neck and the upper part of the chest presented a sallow appearance, with possibly a little underlying yellowish tinge. I notice that color in these old cases of heart disease. I don't know what kind of jaundice it is. It is slight

in amount, and nothing that would be put down in the anatomical diagnosis.

The peritoneal cavity contained a small amount of thin pale clear fluid,—a little ascites. The appendix was negative. The gastro-intestinal tract was out of the picture except that it showed a well marked velvety, juicy mucosa,—passive congestion.

There was a little fluid in the pleural cavities—a few c.c.—and pleural adhesions on each side. The trachea and bronchi showed some reddening of the mucosa and a moderate amount of reddish mucus; otherwise they were negative. The apices of the lungs were frankly negative. There were no areas of consolidation. The tissue generally was spongy and a little leathery, and yielded more or less reddish frothy fluid,—a little congestion, no definite emphysema.

The pericardium contained about fifty c.c. of thin dirty reddish cloudy fluid and a little fibrin, and there was some reddening of the pericardial surfaces,—a frank acute pericarditis. In the region of the anterior half of the left ventricle a band of fibrous adhesions extended between the visceral and parietal pericardium, a broad band of old adhesions tying the two surfaces together pretty strongly, —an old band of chronic adhesive pericarditis besides the acute pericarditis.

The heart weighed 585 grams, considerably enlarged. The myocardium, except in places to be spoken about, was pretty fair, but in these places presented an entirely different picture. The right ventricle in the good places was three mm. and the left ventricle eleven mm., so that where it was more or less normal in appearance it was about as thick as usual. In the region of the band of adhesions on the left ventricle wall, section showed marked fibrosis and thinning of the wall. Above and extending well up the wall of the heart the sections of the heart wall showed fibrosis and in places areas of brownish-yellow more or less necrotic looking tissue,—altogether a marked myocarditis, fibrous, with areas of necrosis, of course suggesting that somewhere some coronary had been blocked to produce the condition. This marked thinning of the ventricle wall in the region of the lower half of the ventricle produced what we call cardiac aneurism, sometimes called aneurismal dilatation. The valves themselves were out of the picture, showing a moderate amount of the usual sclerosis, but otherwise negative. The circumference of the tricuspid was a little increased.

Now for the real condition which underlies all the others. The orifice of the left coronary was free, but a short distance from that

point there was an area of marked fibrosis with marked diminution of the lumen. Then going down from that point along the artery and its branches, there were places here and there where there were small fibrous plaques with diminution of the lumen. But in between, the vessel was in pretty good condition. In the right coronary the orifice was free. But about two cm. from that point there was an area of sclerosis with marked thickening of the wall and diminution of the lumen, and on the intimal surface a small columnar thrombus plugging the artery. The right artery beyond this area was much like the left. We have here again an example of the peculiar localization of sclerosis within a given set of arteries. The condition of the heart from a pathological standpoint furnishes a basis for Dr. Cabot's diagnosis, coronary sclerosis, the background for angina.

The first portion of the aorta was pretty good. There was slight sclerosis in the arch and a small fibrocalcareous area. The other portions showed more or less fibrous sclerosis with some atheroma. The great branches showed a small amount of sclerosis.

The liver showed chronic passive congestion. The gall-bladder, bile ducts, pancreas were negative. The spleen was chunky and thick, the issue elastic; chronic passive congestion. The adrenals were out of the picture.

The kidneys weighed 350 grams and were typical, grossly and microscopically, of passive congestion. They were bluish-brown-red, with a good cortex, the vessels rather engorged. Except for passive congestion they were out of the picture.

The prostate, seminal vesicles and testes were negative.

On the surface of the area of myocarditis in the wall of the left ventricle, up just beneath a papillary muscle there was an area of roughening to which a small frank thrombus was adherent.

An interesting thing from the pathological point of view is the peculiar localization of the sclerosis in the coronary arteries and the great amount of change in the left ventricle wall. Not infrequently we find cases where there is marked sclerosis all along the coronaries with diminution of the lumen, leaving only a slender channel, and with the myocardium showing no definite changes.

DR. CABOT: Was there more change in the right than in the left?

DR. RICHARDSON: There was more change in the wall of the left ventricle, but there was considerable change in the right ventricle wall also. There was much change in the region of the interventricular septum.

DR. CABOT: I suppose that long two-hour pain he had was when that thrombus formed. The acute pericarditis we missed as we usually do. We recognise one out of five cases here.

DR. E. L. YOUNG: I think it is interesting, the number of cases we see here with urinary symptoms and the prostate put down as enlarged, which show nothing post-mortem, as in this case.

### V. RUPTURE OF THE HEART

In one case the cardiac infarction resulting from a much narrowed left *descendens* led to a *rupture of the heart*. "The probe passed easily through the center of an opening four mm. in length with irregular blood-stained margins. This opening is the center of a dark red boggy area in which some branches of the much narrowed left coronary are lost. Cross section of the heart wall in the region of the perforation shows a slender ragged channel which extends through into the left ventricle. The tissues about the channel are blackish red and slightly soft; it is margined by a small area of grayish firm fibrous-like tissue."

Despite all this, the clinical diagnosis is "Acute Bronchitis, Psychoneurosis, Weak Heart." She was not thought to be much ill and was on the danger list for only a few minutes before her death, when she was noticed to be breathing slowly and soon after to be unconscious and pulseless, with great pallor. Until that time the heart and pulse had shown nothing abnormal, but on account of three months' complaint of prickling sensations in the finger-tips, abdomen, legs and back Dr. James J. Putnam had considered her psychoneurotic.

With this may be compared a case of "*traumatic rupture of the heart*" (No. 2249) occurring in a young man of twenty-seven who one hour before he entered the hospital had fallen backward with a heavy iron bar on top of him, sustaining a compound fracture of the left leg with wounds on the scalp and right hand but *no injury to the chest*. The heart was normal in size and valves but "over the left ventricle, midway between the auriculoventricular junction and the apex was an ecchymosis in the middle of which was a linear tear 2.5 cm. long with closely approximate edges running diagonally across the ventricle. The rent entered the interior of the heart to the left of and behind the anterior papillary muscle. Below the opening the heart wall showed hemorrhagic infiltration and at the extreme apex there was some laceration and disorganization over the septum beneath ecchymoses. There was also hemorrhagic infiltra-

tion of the wall of the right auricle with a ragged laceration 2.2 cm. by 1 cm.

There was hemopericardium.

During the hour of his life at the hospital the heart sounds were very irregular and rather faint, with periods of complete standstill lasting several seconds. The radial pulse could be obtained only occasionally and was very faint and irregular. The patient was in muttering delirium and did not answer questions.

Necropsy 1897 was a similar case. A man of twenty-three was struck by a falling telegraph pole. His chest was crushed and his lung ruptured. Death followed swiftly. At necropsy beside the lesions mentioned there were pneumopericardium and rupture of the epicardium.

## CHAPTER VI

## ANGINA PECTORIS

It is not to be expected that anatomical studies will ever throw much light on the clinical manifestations which we call angina pectoris. At the outset it seems improbable that so elusive and transient a phenomenon, varying as it does with some of the most evanescent of vital changes—such as exertion, emotion, and the effects of the nitrites—could be explained by a permanent anatomic lesion. Anything so closely connected as angina is with temporary physiological phases of the body's activities cannot so far as I can see be adequately explained by anatomical lesions which must be present before and after the attack, i.e. in the absence as in the presence of angina itself. Hence hypotheses such as vascular spasm or cardiac fatigue naturally suggest themselves. The difficulty with such theories is that it is hard to see how they can be either proved or disproved.

I am not disappointed therefore that the 138 cases studied here throw no new light on the nature or the origin of angina pectoris. The controversies regarding the relationship of this disease (or symptom) to occlusion of the coronary arteries seems to me to depend, in part, on the similarity of the symptoms following *cardiac infarction* —the "infarction syndrome"—with those of true angina pectoris.

The long and especially the fatal attacks of heart pain unrelated to exertion, unrelieved by rest or nitrites, we certainly must associate with coronary block and its effects. We can hardly suppose any unusual state of things in the aorta as causal here.

But before or after these terrible seizures there are often milder and properly anginoid attacks, typical in the location of pain, in its relation to exertion, emotion and rest and in the relief by nitrite medication. It is certainly tempting to think that some similar mechanism, some cause for insufficient blood flow through the coronaries is at work also in these cases. But the inference is not necessary. There may be some anginoid attacks due to coronary disease and others of a different origin. On the whole I incline to this opinion.

There can be no question, I take it, that the pain of *cardiac infarction* is due not to any changes in the aorta but to the infarct

itself and its results. It is true that *a similar syndrome may occur without infarct* as in the following case (No. 3527): An Irishman of seventy-four while being treated in the ward for dermatitis exfoliativa associated with aortic regurgitation and without any evidence (post-mortem) of arteriosclerosis or of syphilitic aortitis, complained one evening of slight dyspnea and abdominal distress. At one a.m. he had an attack of sharp pain in the precordia. In half an hour the pain had become an intense agony. (Pulse 140, respirations 50.) Morphia $\frac{1}{6}$ gr. and nitroglycerin $\frac{1}{100}$ gr. had no effect and the pain continued until 5:40 a.m. when $\frac{1}{6}$ gr. of morphia was again given and the pain ceased.

On being questioned he now said that he had similar but milder attacks for three or four weeks. He vomited frequently in the final twenty-four hours of his life and after this attack became irrational, restless, dyspneic, with Cheyne-Stokes breathing leading to death. Necropsy showed no cause for the pain.

In spite of occasional cases like this it remains true that there is a group of clinical facts by which we can predict the finding of a cardiac infarct post-mortem with a percentage of diagnostic success equal to that now attainable in most other diseases.

But *from the existence of angina* in the sense of a precordial pain, preceded or followed usually by pain in the left arm (or both arms), always produced in the early stages of its development by emotion or exertion and ceasing after a few minutes' rest, often linked with a sense of constriction and an anticipation of immediate death,*—from this *what can we predict as to the post-mortem?*

In our series if we had predicted that *either* coronary sclerosis or syphilitic aortitis would be found post-mortem, we should have been right in this prediction 32 times and wrong 11 times, apparently a respectable showing of 75% success.

But if we analyze this success it crumbles. Most of the patients here studied were at an age when arteriosclerosis (including some coronary sclerosis) is common and sometimes extreme, *without angina*. Among our 72 *cases of coronary sclerosis without angina* there were four in which both the main coronaries were nearly occluded by arteriosclerosis. I have looked over the necropsies and clinical records of all cases which contained in their anatomical diagnosis a note of marked sclerotic changes in the coronaries, 127 cases in all. To these I have added 11 cases suffering from anginoid

* I make no attempt to enumerate here *all* the phenomena or varieties of angina.

pain without coronary disease of any considerable degree, making 138 cases in all. The whole group can be subdivided into:

| | |
|---|---|
| (1) Coronary narrowing without angina.... | 94 cases |
| (2) Coronary narrowing with angina..... | 33 cases |
| (3) Angina without coronary narrowing.... | 11 cases |
| | 138 |

Of the first group—narrowed coronaries without evidence of angina in life—there is little to say save that the number of cases, 94, is far beneath the true number.* Among the 94 cases which I have come across, there were *eleven* in which the arteriosclerotic process was so extreme that the lumen of one or more arteries was nearly or quite obliterated (see below).

As in all the cases studied in this section there was an enormous preponderance of elderly males,—ten males to every female. The average age was 54. The heart was usually enlarged but in two-thirds of the cases was quite competent, the patients dying of pneumonia, prostatic disease, apoplexy, cancer, etc. Less than one-half showed some fibrous myocarditis but no acute infarctions occurred in this group.

The absence of more damage to the myocardium may be explained by supposing that the collateral circulation, which varies in different hearts, was in these cases sufficient to nourish the heart wall despite the narrowing of certain vessels.

In two of these cases there was syphilitic aortitis, one with gumma of the heart wall, yet no pain. In another, one coronary was occluded at its mouth, yet there was no pain and death was not sudden. In three there was arteriosclerotic occlusion of the left coronary; in three others a clot or a vegetation had practically if not absolutely closed the left coronary.

When the pain is not relieved by rest but lasts on for hours and is associated with pulse failure and fainting, we can much more confidently assert that an organic obstruction of the coronary by syphilitic aortitis, arteriosclerosis or thrombosis is present and is the main cause of the symptoms. But when the pain is brief and not associated

* Had I read over all the 4000 necropsies looking for the descriptions of coronary arteries I should have found many more cases of coronary sclerosis. The 94 cases which I found represent merely those with coronary changes marked enough to be mentioned in the anatomical diagnosis or summary of the findings. Since the accumulation of more cases would merely reiterate a fact already established by many observers (that coronary sclerosis without angina is common), I have not thought it worth while to pile up more of this cumulative evidence.

with evidence of heart failure we cannot say, so far as our material goes, that the coronary arteries are any more diseased than in many elderly individuals who are free from angina. Coronary arteriosclerosis may be present yet insignificant.

Again coronary disease may be wholly absent in patients with definite angina, as is shown in eleven cases of this series. *In 6 of these the whole aorta, carefully examined macroscopically, was normal,** and I see no reason to believe that the microscope would have revealed anything of importance.

These eleven cases *without* coronary disease were in other respects very much like the cases of this series *with* coronary disease. There were ten men to one woman, three over seventy years and three under forty (30, 32, 39). The hearts averaged 598 grams in weight. In one the aorta showed syphilitic aortitis without any narrowing of the coronary orifices; four others showed an arteriosclerotic aortic arch (without coronary disease), and *six showed no changes whatever in the aorta*. Chronic passive congestion was present in ten of the eleven post-mortem. Blood pressures, in one case high, in three cases were normal, in the others not measured.

Some of the phrases used to describe the pain are as follows:

1. For six months sharp piercing pains about the heart.
2. Occasional sharp piercing pains about the heart.
3. For two years sharp sudden pains about the heart on exertion.
4. Burning pains in left chest and down the left arm and wrist on exertion, came one to five times a day and lasted from five minutes to two hours.
5. Severe paroxysmal precordial pains up to and on the day of death despite chronic passive congestion.
6. Precordial pain and pressure for two or three months perhaps fifty times in a night, ceasing with onset of edema.
7. "Typical attacks of angina pectoris" in the ward relieved by nitroglycerin. Chronic passive congestion ante and post-mortem.
8. Intense agony in the precordia lasting five hours despite morphia gr. $\frac{1}{6}$, finally ceasing with another $\frac{1}{6}$. Similar but milder attacks for three or four weeks. (Necropsy: chronic passive congestion, hypertrophy and dilatation of the heart. Aorta smooth, coronaries free. No history of syphilis. Chronic perisplenitis and perihepatitis.)
9. For five months attacks of sharp precordial pain lasting a few minutes. (Blood pressure 160/130.)

* One showed a single smooth yellowish patch.

10. Two years ago attacks of nocturnal precordial pain for three months. (Chronic passive congestion ante and post-mortem.)

Syphilitic aortitis with narrowing of the coronary orifices in the aorta certainly seems to have a closer relation to angina than arteriosclerosis does. It was present in twelve cases of this series, ten of which suffered from angina. In our 92 cases of syphilitic aortitis there were fifteen with angina and several others with epigastric pain, possibly of the anginoid type. This frequency is apparently about the same as in coronary sclerosis, which out of 127 *known* cases is associated with angina in 33. But the *actual* number of cases of coronary sclerosis in this series is presumably far more than 127. For out of 1051 cases recorded as arteriosclerotic in the necropsy records, we can scarcely believe that the coronaries were spared in 970.

But granting that syphilitic aortitis and angina are not infrequently associated, the question still remains, what is the relation between the two? The partisans of the coronary theory of angina can point to a good many cases of angina in which post-mortem the mouth of one or more coronaries was found narrowed or occluded by the syphilitic process. But still more common is the coincidence of syphilitic aortitis and angina when the disease has *not* blocked the coronaries at all, and when these vessels are free, smooth, and dilatable throughout.

In our series, among 15 cases of syphilitic aortitis with angina there are four showing narrowed or occluded coronary openings *and six in which these openings are free.* My chief impression is this: *Syphilis has something to do with angina pectoris, more to do with it than arteriosclerosis has. But the syphilitic disease certainly does not act in all (or in most) cases by any demonstrable effect on the coronary arteries.* It may exert its effect in the way described by Sir T. Clifford Allbutt or in some other way, but certainly not in all cases by way of the coronaries.

Of the 33 cases of angina pectoris with coronary changes, 23 were above the fifty-first year, 21 were men and 12 women. The pain disappeared with the advent of chronic passive congestion in three, *and did not disappear in twelve;* as to the other cases the record is not clear.

Syphilitic aortitis was present in nine of the 33, fibrous myocarditis in fourteen, cardiac hypertrophy in most. The blood pressure showed no rule; it was high, low, or normal.

Among the phrases used to describe the pain fourteen could be classified as typical of angina: Other *non-typical* phrases are recorded as follows:

1. "In bad weather sharp pain shooting from chest down left arm."

2. "Frequent attacks of precordial pain for two months."

3. "Much pain around the heart July 3" (during hospital observation one week before death).

4. "Constant pain in right breast, worse with exertion. Sometimes shoots down left arm."

5. In ward "attacks of great weakness and precordial distress relieved by amyl nitrite or morphia; two to four attacks in twenty-four hours, especially at night."

6. Severe epigastric pain with smothering.

7. Sense of painful constriction in the chest. Sudden death. Arteriosclerotic occlusion left *descendens* with myocarditis.

8. Shooting pain in right arm with slight tightness in chest.

9. "Dyspnea and constriction in the chest at night." (Pernicious anemia.)

10. "Sharp pain in chest radiating to left nipple."

11. "Substernal pain." Arteriosclerosis of coronaries with fibrous myocarditis and intracardiac clot in corresponding region.

12. Dull precordial pain with exertion, radiating down left arm.

13. Severe stabbing pain at cardiac apex following attacks of paroxysmal dypsnea. Sudden death. Acute endocarditis with coronary thrombosis.

14. For three years attacks of sharp non-radiating momentary precordial pain on exertion.

15. Dull throbbing precordial pain radiating to chest and shoulders. Right coronary closed by syphilitic aortitis.

16. Male, sixty-seven years. Eight years epigastric pain in half-hour attacks both with and without exertion, radiating to both arms. Marked arteriosclerosis of the aorta and coronaries.

The following cases are of interest.

*No.* 3424. Housemaid of sixty. For five years she has suffered from attacks of severe precordial pain and intense dyspnea brought on by exertion, worry, or anger, two or three times a year. Pain usually starts in precordia and is as though she were being crushed. Radiates to left shoulder and hand. Lasts about an hour. Followed by intense dyspnea and cough, which last for about a week after the attack.

Last May she had an exceedingly sharp cutting pain starting in the region of the left biceps, radiating across the back to the shoulder and arm and finally to the precordia. Since then (it is now December) she has had no more pain but progressively increasing dyspnea, orthopnea and cough.

Physical examination showed aortic stenosis and regurgitation, hypertrophy and dilatation, chronic passive congestion. Necropsy confirmed these. The aorta and coronaries showed marked arteriosclerosis.

*No.* 3259. Carpenter of fifty-five with diabetes and abscess of right lung. October 5th: "On this date a carbuncle of the back appeared and he began to feel an ache down his left forearm when he walked. A year later he made many visits to the Out-Patient Department on account of precordial pain which began nine minths after the first hospital visit. He describes it as "a dead heavy feeling over the heart, coming on with exertion and lasting ten to fifteen minutes." In attacks he has also headache, vertigo and dizziness and feels as if he was going to die. The pain radiates down both arms, especially the left. The attacks now come on with less exertion and last longer, sometimes an hour.

Sudden death in the hospital. Arteriosclerotic occlusion of the coronary arteries with fibrous myocarditis, chronic pneumonitis and abscess of the lung.

*No.* 3607. A colored fireman of thirty-four complains that for eight years he has noticed a sickening fainting feeling under the sternum with nausea on exertion. Three years ago he began to have constant pain of varying severity in both forearms. This left the right arm after a month but has continued in the left. For the past year it has radiated up the arm and across the back. Mercury gives relief.

Necropsy: syphilitic aortitis. Coronaries not obstructed.

### ANGINA AND MYOCARDITIS

Beside the two familiar but still mysterious associates of angina, aortitis and coronary narrowing, it is linked up with fibrous myocarditis which was present in fifteen of the forty-four cases of angina. The most obvious explanation that suggests itself is that coronary disease, which is certainly responsible for some cases of fibrous myocarditis and myomalacia, may be also the cause of angina and so link the two sets of facts together. But as both angina and myocarditis are noto-

riously diseases of elderly people, their association with sclerotic coronaries proves nothing as to the cause of angina.

*Aortic stenosis* without mitral disease also appears rather frequently as an associate of angina. The latter was present eight times among 28 cases of aortic stenosis. But here as with myocardial fibrosis, we must take account of the factor of age. Aortic stenosis (uncomplicated) is a disease of elderly people, 22 of 28 cases occurring in persons past their fortieth year, and 15 of 28 after the fiftieth year. It may be that influences dominant in elderly people act to favor both the slow emergence of the symptoms of aortic stenosis and the appearance of angina pectoris. There need be no more direct connection between the pain and the valvular lesion.

I have seen three cases of intense and typical angina associated with *pernicious anemia* and without coronary change. One showed moderate sclerosis of the aorta, the others none. All were strictly dependent on exertion and relieved by rest. Such cases, like those seen in convalescence from pneumonia and from other infections, certainly make us skeptical of any etiology based wholly on organic and permanent changes either in the coronaries or in the aorta.

## BLOOD PRESSURE AND ANGINA

Although the recorded data furnished as to blood pressure are scanty in this series of patients with angina pectoris, they are sufficient to support the usually accepted idea that angina has no definite relation either to hypertension or to hypotension. High, low, and normal pressures are found and the high pressures are no more common than in any set of people past middle life and free from angina.

*Cardiac enlargement* is present in a considerable majority of the cases, but like hypertension, myocarditis and aortic stenosis, bears no discernible causal relation to the angina, which can and does exist in persons with hearts of normal size.

## THE CESSATION OF ANGINA WITH THE ADVENT OF GENERAL PASSIVE CONGESTION

It has been noticed by many that angina pectoris often ceases when cardiac compensation fails. Our experience presents three striking instances in confirmation of this. But out of fifteen cases in which this point was especially studied there were twelve in which the angina *persisted*, despite all the evidences of chronic congestive

heart failure. Evidently it is only certain types or phases of congestive failure which give relief to angina.

*Death from angina* without congestive failure is I believe a decided rarity, if we exclude the cases of cardiac infarction with blocked coronary. There were but *thirteen cases* in this series of 44 in which no congestive failure was found post-mortem. This represents all the known deaths from angina in a series of 1906 individuals dying with cardiovascular lesions.

### CORONARY THROMBOSIS AND ACUTE BLOCKING OF THE CORONARY

*I. Cases without Pain.*

1. In *No.* 2393, a boy of twenty suffering with acute endocarditis; the orifices of the coronaries were partly occluded by soft vegetations attached to and above the aortic valve. There were no symptoms corresponding to this obstruction in life, the patient dying of chronic passive congestion and sepsis.

2. *No.* 2578 is a similar case of acute endocarditis occurring in a man of forty-six, who died with symptoms of a general septicemia and without any pain. His death was not sudden or in any way remarkable.

3. In *No.* 2727 a woman of forty-three dying of a widely disseminated lymphoma with no symptoms calling attention to the circulatory system (the diagnosis was lethargic encephalitis in life) was found at necropsy to have a thrombus in the descending branch of the left coronary.

*II. Cases with Pain.*

1. In *No.* 3389 a man of fifty-two suffered from typical angina and also from sharp paroxysmal *epigastric* pain with dyspnea. Post-mortem the left *descendens* was not only sclerotic but occupied by an organizing thrombus. Death was not sudden or painful but is to be counted I suppose as, on the whole, of the anginoid type, since there was no chronic passive congestion or other cause for death discovered.

2. Necropsy 3921 was performed on a woman who died at thirty-one of acute endocarditis and chronic passive congestion. She had had many attacks of severe stabbing precordial pain immediately following on spells of dyspnea and weakness. Death was sudden and was associated with intense pallor and a slight general convulsion. The post-mortem examination showed in addition to the acute endocarditis a partial obstruction of the left *descendens* by a clot.

. . .

In the painless cases we may suppose, I take it, that death followed the coronary obstruction too swiftly for the production either of pain or of myocardial infarct.

Outside of the 4000 cases here analyzed I have records of the following cases which seem of sufficient interest to be added here.

1. *No.* 4116. A married Scotch woman of forty-seven was seen in the autumn of 1915 with the history of seven years of attacks of severe epigastric pain with vomiting and headache. She had had seven such in the last five weeks, relieved only by morphia. Exploratory laparotomy showed no disease in the biliary tract, stomach, duodenum, pancreas, kidney or spleen. An appendix thought to be slightly inflamed was removed. The attacks of pain continued. In September 1920 she had sudden severe substernal distress following exertion and lasting twenty minutes. That night she had another attack while lying quiet in bed. This time the pain radiated to the back, to both shoulders and down the left arm, especially to the fourth and fifth fingers. Dyspnea and orthopnea came with the pain which she said lasted twenty-four hours this time, and had recurred several times since. Orthopnea persisted and six days ago the feet became swollen.

There were paroxysms of fibrillation and at times an apex rate of 190 with a large pulse deficit, but no arrhythmia. Electrocardiogram showed only sino-auricular tachycardia with a somewhat inverted T-wave (digitalis?).

She died in diabetic coma.

At necropsy there was very slight general arteriosclerosis; very marked coronary sclerosis with occlusion of the left *descendens*, with a corresponding area of fibrous myocarditis, and a degeneration and perforation of the interventricular septum. There were mural thrombi in the ventricle, slight hypertrophy and dilatation, chronic passive congestion. Streptococcus sepsis with abscess in a bronchial lymph gland. Also hyalin degeneration of the islands of Langerhans and a few minute concretions in the gall-bladder.

The most natural interpretation of these data seems to be that the patient had survived an infarct of the heart in or before 1915; that the infarct became transformed into a fibrous myocarditis, but that a few days before her death in 1920 the interventricular septum became necrosed and perforated owing to the degeneration of a fresher cardiac infarct.

2. A Finnish laborer of thirty-nine had suffered for three years with attacks of precordial pain and dyspnea, accompanied by severe

pain in the left elbow. The attacks lasted about two minutes. Three months before he came under observation he had an attack which he said lasted nine days without intermission or change in intensity. He sat doubled up and could not sleep or eat. The elbow was not swollen.

Later chronic passive congestion appeared and the pain ceased. X-ray suggested a mitral lesion with dilatation. Blood pressure 105/80. Heart enlarged, regular, 80, soft systolic at apex. Wassermann negative. Electrocardiogram showed a broad notched P-wave in Lead II. The R-wave in all leads was low. Sinoauricular tachycardia. Sudden death after nine-days' observation.

*Necropsy.*—Heart 590 grams. Left coronary occluded for 3.5 cm.; this artery led directly to a large area of fibrous myocarditis with great thinning of the wall (5–10 mm.). Mural thrombus over this. Moderate arteriosclerosis of aorta. A branch of the pulmonary artery occluded by arteriosclerosis with resulting infarct. A broad band of old pericardial adhesions at the cardiac apex. No sign of syphilis.

3. *No.* 4170. A Swiss woman of sixty-five, divorced, had had two still-births and three living children. Was seen in decompensation with attacks of precordial pain and "tightness" relieved by nitroglycerin. Blood pressure 160/65, 175/85, 180/100, 100/45, 96/65. Auricular fibrillation. Heart not apparently enlarged. Parkinson's disease. Died of congestive failure.

*Necropsy.*—Heart 440, moderately enlarged with dilatation confined to the auricles; left coronary sclerosed with considerable diminution of its lumen. Right less so. Myocardium no lesions. Arterioslcerosis of aorta with small dissecting aneurism.

4. *No.* 4200. A married woman of forty with a negative past history began to suffer two weeks before we saw her from excruciating knife-like midepigastric pain radiating to the back beneath the right scapula and lasting two days; morphia alone gave any relief. On the second evening she became unconscious. Next morning the right hand and right facial muscles were weak and the speech thick. But after a week in bed she felt and seemed well and was up for two days. Then dyspnea and orthopnea.

When seen there was a loud friction rub synchronous with the heart's action, heard over and on both sides of the sternum. Blood pressure 120/60. Heart irregular, rapid, no mumurs. Moderate cardiac enlargement. Right hand weak. Staphylococcus albus cultivated from the blood. Death in two days.

*Necropsy.*—Subacute pericarditis with moderate hypertrophy and dilatation (430 grams) and mural thrombus in the left ventricle. Circumflex coronary occluded by an embolus and an infarction of the corresponding area of the heart. Also embolism of superior mesenteric and of iliac arteries. A small amount of fibrous sclerosis in the aorta, none elsewhere.

The pain might conceivably be due to pericarditis, but its intensity, sudden onset, its radiation and the coma soon after it, make the coronary embolism a more probable cause.

5. A man aged forty-eight when seen, had had four years previously an attack of sharp precordial pain radiating to the left shoulder and down the left arm. Physical examination was negative and in a short time he seemed quite well and remained so until during an attack of peritonsillar abscess, which did not disable him from work, he was suddenly seized with terrible pain in the abdomen and legs which caused him to fall to the floor. He died a week later with signs of general peritonitis and gangrene of the legs.

*Necropsy* showed old myocardial scars and complete arteriosclerotic closure of a small branch of the left coronary. Intracardiac mural thrombi on the old myocardial scars had led to mesenteric and iliac thromboses.

## SUMMARY AND CONCLUSIONS

1. If we take care to distinguish the group of symptoms and signs known as angina pectoris from the syndrome accompanying cardiac infarct, we must confess that there are many cases of angina without adequate anatomical basis or explanation.

2. Coronary sclerosis and occlusion is certainly a cause of the pain and collapse seen in cardiac infarction, but bears no clear or stable relationship to angina pectoris proper.

3. The same may be said of disease in the aortic arch of which there is no evidence macroscopically in a considerable group of anginoid cases.

4. Extensive coronary occlusion is not infrequent in the hearts of patients who during life have never suffered from angina.

5. Syphilitic aortitis is associated with angina in about 16% of cases. But in only a small minority of these do we find the coronaries obstructed at their mouths. There must be some other connection between syphilis and angina, perhaps that suggested by Sir T. Clifford Allbutt.

6. Fibrous myocarditis and angina are often associated but there is no evidence of a causal connection. The same is true of aortic stenosis and angina.

7. Angina occasionally ceases with the advent of general passive congestion. But in most cases of our series this was not true.

8. When a coronary is suddenly blocked by a clot death may follow at once with or without pain. (See Cardiac Infarction, Chapter V, 2.)

# CHAPTER VII

## ACUTE AND SUBACUTE ENDOCARDITIS

### DEFINITION AND TERMINOLOGY

The 180 cases studied here have in common this fundamental fact:—on some portion of the endocardium, usually the heart valves, there is situated a patch of *soft* inflammatory tissue composed largely of blood platelets, fibrin and bacteria more or less firmly adherent to the endocardium adjacent. These adherent masses are distinguished from the *hard* fibrous or fibro-calcareous scars of chronic endocarditis, which however may be present along with the acute soft vegetations.

The word "acute" refers primarily to the softness of the vegetations on the endocardium, that is, to the early stage of the inflammation. As will be seen below, some of the cases are *literally* acute in the sense of lasting but a short time and leading rapidly to the death of the patient. Other cases strictly deserve the term "subacute" or even "chronic" since they last for months or even for years. But there is no justification for lumping all these cases under the single termination "subacute" as has been done by some writers. The term "subacute bacterial endocarditis," much used at the present time, seems intended to suggest that there are forms of endocarditis not due to bacteria, an assumption which seems insufficiently grounded. I prefer therefore the general term "acute and subacute endocarditis" though admitting that it has to be stretched to cover some cases lasting too long for the strict application of either term.

### TYPES OF ACUTE AND SUBACUTE ENDOCARDITIS

The cases here studied fall into two main groups: (1) those which *apparently start in the heart* and circulation and not in any septic focus outside it; (2) those that *apparently come to the heart*, originating either in a focus of sepsis or in a gravely weakened condition of the whole organism due to cancer, nephritis, diabetes, or some other debilitating disease.

Within the first group we may distinguish further (a) those which manifest the first attack of disease in the heart, and (b) those which

are recurrent or implanted upon a previously diseased endocardium. Linking these two groups together I shall speak of them as "primary" acute endocarditis.* In our 180 cases, 102 belonged to the primary group. Of these, 36 cases represent first attacks and 66 recurrent or implanted disease.

In contrast with these 102 cases there is a group of 78 in which the endocarditis is relatively a secondary or minor feature while the main cause of death is some such disease as general peritonitis or cancer. Within this group one can distinguish two sub-groups, (1) pyemic and (2) terminal. The *pyemic* group, comprising 44 cases, is made up of those in which it seems clear, both from the post-mortem examination and from the clinical history, that the disease has originated and done its most serious damage *outside* the heart. 25 of the 44 cases belonging within this group showed only soft vegetations, while in 19 these soft vegetations were implanted upon or associated with a fibrous or fibrocalcareous base representing, I take it, a previous attack of endocarditis.

Finally there is the *terminal* group of cases in which the endocarditis plays no part recognizable during life and is often so minute as to be easily overlooked by the post-mortem examination. 34 cases belong to this group.

TABLE 120

| | | |
|---|---|---|
| 1. (*a*) | PRIMARY *acute* endocarditis | 36 cases |
| (*b*) | PRIMARY *acute* endocarditis, recurrent | 66 cases |
| 2. (*a*) | SECONDARY *acute* endocarditis, pyemic | 44 cases (19 recurrent) |
| (*b*) | SECONDARY *acute* endocarditis, terminal | 34 cases (17 recurrent) |
| | | 180 cases |
| | Total recurrent cases (66 + 19 + 17) = | 102 |
| | Total non-recurrent cases = | 78 |
| | | 180 |

**Characteristics of the Main Subdivisions.**—The distinguishing marks of the *primary group of cases*, whether initial or recurrent, may be set down here in the way of an introductory summary of the pages to follow. The characteristic features of this type are as follows:

* I realize fully the limitations of the word "primary" and the obvious fact that endocarditis must in all cases be brought to the heart from outside it and not originate there. The word primary is here used to mean that the bulk and most serious portion of the lesions are situated in the heart itself rather than outside it.

(a) The *absence of any focus of infection* or other important lethal cause *outside the heart.*

(b) The presence post-mortem (in seven-eighths of the cases), of relatively *large shaggy masses or ulcerations* on the heart valves.

(c) *Embolism* present in 60%.

(d) *Nephritis* present in one-third of the cases.

(e) *Cardiac enlargement* ante and post-mortem, associated in most cases with dyspnea and often with edema.

(f) *Constitutional evidences of sepsis*, especially chills and stupor, without any evidence of a septic focus outside the heart.

(g) The presence of *murmurs* other than the insignificant systolic murmur.

(h) The presence in most prolonged cases of an increasing secondary *anemia*.

In contrast with these characteristics, the cases belonging in the second group show:

(a) The presence of an important cause of death outside the heart.

(b) Small lesions on the heart valves (in five-sixths of the cases).

(c) Embolism almost never recognized before death and only in one-third of the cases after death.

(d) Complicating nephritis relatively rare.

(e) Little or no recognizable enlargement of the heart.

(f) Murmurs, systolic or absent.

## AGE AND SEX*

Uniting all groups we find 93 males and 87 females in this series. The sex incidence is essentially the same in all the different subgroups above described. In the primary cases there were 57 males to 45 females, in the secondary group 56 men to 42 women. The predominance of males is in accordance with previous observations on this subject, though it is not as extreme as is suggested by Riesman.†

* In the whole 4000 necropsies among which these 180 cases occurred the males are nearly twice as numerous as the females.

† Journal of the American Medical Association, May 21, 1921.

TABLE 121.—AGE AND SEX IN ACUTE AND SUBACUTE ENDOCARDITIS

| Age | Male | Female | Total |
|---|---|---|---|
| 0– 9 | 3 | 2 | 5 |
| 10–19 | 5 | 11 | 16 |
| 20–29 | 18 | 19 | 37 |
| 30–39 | 25 | 28 | 53 |
| 40–49 | 19 | 9 | 28 |
| 50–59 | 13 | 13 | 26 |
| 60–69 | 8 | 4 | 12 |
| 70+ | 2 | 1 | 3 |
| | 93 | 87 | 180 |

In Table 121 the distribution of the ages is shown in detail. Summing it up we may say that in the primary group the disease affects especially young adults between the twentieth and fortieth year. Fifty-six of the 102 cases fell within these limits. In the secondary group the age and sex are those characteristic of the underlying disease. *In any form the disease is rare in the first decade and not common before the twentieth or after the sixtieth year.*

When we come to note the relation of age to sex we find that among the patients who died before the fortieth year females predominate in the relation of 60 to 51 males, while among those dying after the fortieth year there are 42 males to 27 females. One may conjecture that this difference is due to the fact that in the first group there fall a good many cases in which the disease is implanted upon a previously existing chronic endocarditis of the mitral valve, a site of infection commoner in women than in men. On the other hand, cases falling in the second group have relatively few of the recurrent type which is so prone to attack women.

72 of the 102 primary cases occurred before the fortieth year, and only 30 in persons more than forty years of age.

In the whole 180 cases there were but 32 with a previous history either of rheumatic fever or of chorea. Most of these fell into the recurrent section of the primary group.

### VALVE AFFECTED

The disease was confined to the mitral valve in 77 cases, to the aortic valve in 36 cases, while both valves together were affected in 50. The further details as to the parts affected are shown in Table 122.

TABLE 122

| Valve affected | Primary | | Secondary | | Total |
|---|---|---|---|---|---|
| | I Initial | II Recurrent | III Pyemic | IV Terminal | |
| Mitral | 11 | 17 | 29 | 20 | 77 |
| Aortic | 9 | 11 | 10 | 6 | 36 |
| Mitral and Aortic | 9 | 30 | 5 | 6 | 50 |
| M + A + T | 3 | 3 | 0 | 1 | 7 |
| Tricuspid | 2 | 0 | 0 | 1 | 3 |
| Aortic and Tricuspid | 1 | 1 | 0 | 0 | 2 |
| Pulmonary | 1 | 0 | 0 | 0 | 1 |
| Mitral and Tricuspid | 0 | 3 | 0 | 0 | 3 |
| 4 valves | 0 | 1 | 0 | 0 | 1 |
| | 36 | 66 | 44 | 34 | 180 |

It is of interest here to notice the resemblance between the cases of this series, the chronic non-deforming lesions and the commonest of the chronic deforming value lesions described in Chapter II. The similarities, as will be seen in Table 123 are quite striking.

TABLE 123

| Valve affected | Acute and sub-acute endocarditis | Chr. deform. endocarditis | Chr. non-deforming endocarditis |
|---|---|---|---|
| Mitral | 77 | 107 | 68 |
| Aortic | 36 | 28 | 45 |
| M + A | 50 | 40 | 104 |
| M + A + T | 17 | 33 | 22* |
| Total | 180 | 208 | 239 |

* All combinations other than the above.

I do not wish to press this analogy too far but certainly it contains at least a suggestion that the organism or organisms of acute endocarditis are if not identical with at least closely similar to those which produce chronic valvular heart disease. On the other hand the dissimilarities of incidence obvious in chronic non-deforming endocarditis argue a different etiology.

Another point of interest about these findings is the *absence of any notable tendency to favor the tricuspid valve.* It has often been said that acute endocarditis—the so-called malignant or ulcerative forms—is especially prone to attack the tricuspid valve. But this series shows that in only sixteen cases out of 180 was the tricuspid valve attacked at all, and in only three was it attacked exclusively.*

A curious and interesting point is the relation of sex to the site of the disease. Of 77 cases in which the mitral valve alone was the seat of the acute endocarditis, 49 or nearly two-thirds were women. On the other hand, in the 36 cases involving the aortic valve alone, 23 or ⅔ were men. I have no idea how these figures are to be explained.

One may also note that in the recurrent cases (grouped in the second, third and fourth columns of Table 122)† the preponderance of cases confined to the mitral valve no longer exists. In the group of recurrent cases the disease was confined to the mitral valve in only 38 or 38% and affected other valves in 64. The non-recurrent cases show that the mitral valve was attacked exclusively in 39 or 50% and the other valves also in 39. The difference in the two groups is considerable.

In 117 of the 180 or 64% the disease was confined to a single valve.

### NATURE OF THE INFLAMMATORY PROCESS (TABLE 124)

There appear to be two main morphological types in the pathological anatomy of acute endocarditis: (1) those in which the disease is composed exclusively of soft thrombotic masses or vegetations, and (2) those in which ulceration and destruction of the valve is associated also with these vegetations. The latter or "ulcerative" type is much the less common, occurring in only 34 of 180 cases. But this predominance of the vegetative or polypous type of endocarditis is less marked in the primary group of cases, where we find 29 ulcera-

* Gonorrheal endocarditis though carefully searched for was not found in the 4000 necropsies of this series. The proneness of this organism to attack the tricuspid and pulmonic valves has been noticed by Thayer and others.

† In 19 cases within group III (Table 122) the sepsis was implanted upon an old endocarditis so that these 19 cases are recurrent as well as pyemic.

tive cases out of 102, as compared with five ulcerative cases out of 78 in the pyemic and terminal varieties.

TABLE 124.—NATURE OF ENDOCARDITIS

| | I "Primary" non-recurrent | II Primary recurrent | II + III Septic + recurrent | III Septic | IV Terminal |
|---|---|---|---|---|---|
| Large polyps | 20 | 24 | 4 | 2 | 2 |
| Ulcers and polyps | 14 | 15 | 1 | 4 | 0 |
| "Small" polyps | 1 | 14 | 6 | 6 | 12 |
| Pinhead sized polyps | 0 | 10 | 2 | 1 | 13 |
| Minute polyps | 1 | 3 | 6 | 12 | 7 |
| | 36 | 66 | 19 | 25 | 34 |

Table 124 also shows that the lesions are much larger as a rule in the primary than in the secondary cases. Nevertheless it is notable that even in the latent terminal cases two showed large polypoid masses.

**Embolism.**—86 out of 180 cases showed at necropsy evidences of embolism (135 embolic lesions in all). The distribution of these embolic lesions appears in Table 125.

TABLE 125.—DISTRIBUTION OF EMBOLIC LESIONS

| | I "Primary" non-recurrent 33 in 36 | II Primary recurrent 76 in 66 | III Septic + recurrent 21 in 44 | IV Terminal 5 in 34 | Total 135 in 180 |
|---|---|---|---|---|---|
| Spleen | 10 | 29 | 7 | 2 | 48 |
| Kidneys | 10 | 26 | 7 | 2 | 45 |
| Lungs | 4 | 12 | 3 | 0 | 19 |
| Extremities | 3 | 0 | 1 | 0 | 4 |
| Brain | 2 | 2 | 0 | 0 | 4 |
| Skin | 0 | 4 | 0 | 0 | 4 |
| Coronary arteries | 0 | 2 | 0 | 0 | 2 |
| Parotid gland | 1 | 0 | 0 | 1 | 2 |
| Mesenteric vessels | 1 | 0 | 0 | 0 | 1 |
| Retina | 0 | 0 | 1 | 0 | 1 |
| Paravertebral tissues | 1 | 0 | 0 | 0 | 1 |
| Liver | 0 | 1 | 0 | 0 | 1 |
| Abdominal veins | 0 | 0 | 1 | 0 | 1 |
| Ovary | 0 | 0 | 1 | 0 | 1 |
| Renal artery | 1 | 0 | 0 | 0 | 1 |

Embolic lesions are much commoner in the primary types of acute endocarditis than in the secondary varieties. Thus 66% of the primary cases (Columns I and II above) showed some evidence of embolism after death, while only 24 out of 78 or 32% of the secondary group showed such evidence. Emboli were commonest of all in the recurrent-primary cases, occurring there in 43 out of 66 cases, or 65%; in the non-recurrent primary group, embolic lesions were found in 19 out of 36 or 52%. In the other groups the percentage is progressively less, being only 26 in the terminal cases. Only a minority of these embolic phenomena were recognized during life, 33 as against 86 post-mortem (see Tables 125 and 126). All but two of these 33 cases fall within the primary group. Aside from purpura which some might hesitate to include as an embolic phenomenon, splenic embolism was recognized most often, seven times in all. Renal embolism comes next with six diagnoses, then cerebral embolism with

TABLE 126.—EMBOLISM RECOGNIZED IN LIFE

| | Primary | | Secondary | | Total |
|---|---|---|---|---|---|
| | Initial I | Recurrent II | Pyemic III | Terminal IV | |
| Lungs and spleen | .. | 1 | .. | .. | 1 |
| Brachial | 1 | .. | .. | .. | 1 |
| Radial | 1 | .. | .. | .. | 1 |
| Parotitis | 2 | .. | .. | 1 | 3 |
| Spleen | 1 | 2 | .. | .. | 3 |
| Brain | 1 | 1 | .. | .. | 2 |
| Leg, skin | 1 | .. | .. | .. | 1 |
| Kidney | 1 | 2 | .. | .. | 3 |
| Brain and spleen | 1 | .. | .. | .. | 1 |
| Kidney and spleen | 1 | 1 | .. | .. | 2 |
| Superior mesenteric | 1 | .. | .. | .. | 1 |
| Lungs | .. | 3 | .. | .. | 3 |
| Purpura | .. | 5 | .. | .. | 5 |
| Splenic, renal, brain, subcutaneous peripheral veins | 1 | .. | .. | .. | 1 |
| Kidneys, spleen, brain, lung | 1 | .. | .. | .. | 1 |
| Brain and kidney | 1 | .. | .. | .. | 1 |
| Brain, spleen and kidney | 1 | .. | .. | .. | 1 |
| Brain and skin | 1 | .. | .. | .. | 1 |
| Retina and skin | .. | .. | 1 | .. | 1 |
| | 16 | 15 | 1 | 1 | 33 cases in 172 |

five, and pulmonary embolism with four. There were three cases of acute parotitis, very possibly metastatic or embolic in origin, though the direct entry through the mouth is also possible.

The case of superior mesenteric embolism presented in life only the evidences of acute intestinal obstruction with peritonitis. No endocarditis was suspected, and the case was treated as a surgical emergency.

The central artery of the retina was recognized as plugged in only one case, although from the observations of others I should suppose that some cases must have been overlooked in the present series.

The practical importance of emboli in the diagnosis of the disease will be referred to later. But it is obvious that if we are to make the diagnosis at all we must do so without recognizing emboli in the great majority of cases. For though doubtless many were overlooked in the present series, the fact that we were aware of their presence in only 33 cases out of 180 in the whole series, and in only 31 cases out of 102 in the primary group, leads me to believe that even with the best of clinical study we cannot as a rule recognize any emboli in life.

**Skin Hemorrhages.**—Hemorrhages are recorded during life in only 17 cases, ten of these belonging to the primary group and seven consisting merely of purpuric spots. A generalized hemorrhagic eruption such as is seen in other infectious diseases is seldom recorded in our cases of acute endocarditis.

**Tenderness of the Fingers and Toes.**—Only in three cases of our series was this noted, but I strongly suspect that with better observation we should have recognized this symptom in a larger number of cases. I am convinced however that it is not common.

**Nephritis.**—Some type of nephritis occurred in 45 cases out of 180. This is excluding suppurative infections of the kidney. Table 127 shows that nephritis was much commoner in the primary group of cases than in the others. 33 out of 102, or practically one-third of the cases in the first group showed some nephritis, while only 12 out of 78 cases in the second group showed it. Among these cases of nephritis 24 were of the acute glomerular variety, 10 were classified as subacute glomerulonephritis, and the remaining as chronic nephritis. In the primary group acute nephritis occurred 19 times, subacute 7 times, chronic 7 times. In the secondary group acute nephritis 5, subacute 3, chronic 4. It would appear therefore that there is no single type of nephritis regularly associated with acute endocarditis, as some writers have supposed.

TABLE 127.—NEPHRITIS IN ACUTE OR SUBACUTE ENDOCARDITIS

| | | | | |
|---|---|---|---|---|
| Primary | Group I. Initial | 10 of 36 | Acute | 6 |
| | | | Subacute | 1 |
| | | | Chronic | 3 |
| | Group II. Recurrent | 23 of 66 | Acute | 13 |
| | | | Subacute | 6 |
| | | | Chronic | 4 |
| Secondary | Group III. Pyemic | 5 of 44 | Acute | 2 |
| | | | Subacute | 3 |
| | Group IV. Terminal | 7 of 34 | Acute | 3 |
| | | | Chronic | 4 |

**Meningitis.**—In 11 cases of the first group, three of the recurrent and eight of the non-recurrent types of endocarditis, acute leptomeningitis occurred as a complication. In the secondary type of endocarditis none of these complications occurred.

**Foci of Sepsis.**—In the 78 cases of endocarditis believed to be secondary to disease outside the heart, *general peritonitis* was most often the source of infection and was believed to be the cause in 10 cases. Pneumonia comes next with seven cases, puerperal sepsis in six cases, a septic extremity, arm or leg, in five cases, sepsis in the urinary tract in five cases, empyema in three, lung abscess in two, dermatitis in two, tonsillar sepsis, bed sore, appendicitis, osteomyelitis, post-operative sepsis, once each.

Puerperal sepsis has long been recognized as prone to attack the heart, but I do not think that general peritonitis has received sufficient attention in this connection. The same is true of urinary sepsis. On the other hand, I am surprised to find that osteomyelitis and bed-sores have played so small a part.

By the classification adopted in this chapter no case has been included in the primary group unless we were convinced, the pathologist and I, that the sepsis manifesting itself as acute endocarditis had originated within the circulatory system. Nevertheless there are included within the 102 cases of this group five in which necropsy showed suppurative lesions outside the heart, in our opinion secondary to the endocarditis or independent but not its cause. These lesions were, in one case empyema, in another interlobar empyema with tuberculosis, in a third lupus, in a fourth mastoiditis and deep phlegmon of the neck, in a fifth meningitis, duodenal ulcer

and pneumonia. We recognize that in any of these cases an interpretation of the endocarditis as secondary is possible, and we mention them here to make clear the consequent percentage of possible error in our classification.

**Underlying Disease in Terminal Cases of Endocarditis.**—Neoplasms come first with ten cases, nephritis next with seven, the rest of the causes, as shown in Table 128, are pretty well scattered.

TABLE 128.—ACTUAL SOURCES OF SEPSIS

| | III | IV |
|---|---|---|
| General peritonitis | 10 | |
| Pneumonia | 5 | 2 |
| Puerperal sepsis | 6 | |
| Urinary sepsis | 5 | |
| Septic leg (arm) | 4 | 1 |
| Empyema | 3 | |
| Lung abscess | 2 | |
| Dermatitis | 2 | |
| Osteomyelitis | 1 | |
| Post-operative sepsis | 1 | 1? |
| Appendix | 1 | |
| Bedsore | 1 | |
| Sepsis with fracture | 1 | |
| Pyemia | 1 | |
| Tonsillar | 1 | |

TABLE 129.—UNDERLYING DISEASE

| | |
|---|---|
| Neoplasm | 10 |
| Nephritis | 7 |
| Diabetes | 3 |
| Apoplexy | 2 |
| Pernicious anemia | 2 |
| Splenic anemia | 1 |
| Erysipelas | 1 |
| Leukemia | 1 |
| Pericarditis | 1 |
| Phthisis | 1 |
| Tabes dorsalis | 1 |
| Tuberculous peritonitis | 1 |

## CLINICAL MANIFESTATIONS

**1. Constitutional Sepsis.**—Evidences of the influences of a general infection upon the body (i.e. fever, leucocytosis, anorexia, loss of weight), were present in only 79 of the 102 "primary" cases and in 40 of the 78 "secondary" cases. But these were of value in calling attention to the possibility of endocarditis only in the 102 cases of the first group. In the others they would naturally be attributed to some of the more obvious foci of sepsis present in the body. The type of fever was continued in 17 cases and of the intermittent or picket-fence type in 13 of 36 cases in Group I. *Chills* were noted in only 27 cases out of 172, 23 in the primary group and four in the others. They were especially common in the *non-recurrent primary cases*, making up 17 out of the total of 36 in this subdivision. It is only in this type of case that chills are of any help in diagnosis. In a general way we may say that when chills occur without any recognized cause such as malaria or a known septic focus, there are three places to look for their origin: (1) the biliary tract and gall-bladder, (2) the genito-urinary tract, and (3) the heart. In our experience, however, chills even when present as a symptom of acute endocarditis are rarely of diagnositic value as compared with more localizing and definite evidence such as embolic phenomena or diastolic murmurs.

**2. Mental State.**—In the classical descriptions of acute endocarditis it has repeatedly been noticed that patients with this disease do not feel sick, complain of nothing, want to get up and go about their usual occupations, and sometimes actually do so. This state of euphoria was distinctly noticeable in four of the 102 cases of primary endocarditis here studied, and very possibly would have been noted in others had it been more carefully looked for. In most of the cases however it was certainly not present. One of the cases of striking euphoria in this series occurred in a hospital orderly who up to one week from the time of his death was actively at work carrying patients and attending to the ordinary duties of his position. Ordinarily however either an overwhelming constitutional sepsis or the discomfort attendant upon local foci of sepsis or sufferings from an uncompensated chronic valve lesion prevented possibility from an euphoria while the patients were under observation.

A "*typhoidal state*" was noted in two cases of this group, and a stuporous or comatose condition in eleven. One patient was notably nervous and troubled with insomnia.

**3. Anemia.**—In Table 130 are shown the blood counts of seven patients in the non-recurrent subdivision of the primary group.

TABLE 130.—ANEMIA IN GROUP I

| Case | Necropsy | Age and Sex | Red Cells | Hemoglobin |
|---|---|---|---|---|
| 1 | 915 | 34. M | 2,248,000 | 48% |
| 2 | 745 | 8. M | 2,880,000 | 45% |
| 3 | 2949 | 23. M | 2,800,000 | |
| 4 | 135 | 40. F | 3,100,000 | 41% |
| 5 | 1341 | 22. F | 3,840,000 | 70% |
| 6 | 125 | 41. F | ..... | 55% |
| 7 | 2541 | 24. F | ..... | 60% |

TABLE 131.—LEUCOCYTOSIS

| Leucocytes | I* | II | III | II and III | IV | Total |
|---|---|---|---|---|---|---|
| Under 5000 | 1 | 2 | 2 | 0 | 0 | 5 |
| 5,000–10,000 | 4 | 1 | 1 | 3 | 1 | 10 |
| 10,000–15,000 | 4 | 2 | 2 | 1 | 1 | 10 |
| 15,000–50,000 | 23 | 27 | 9 | 8 | 2 | 69 |
| 50,800–58,000 | 0 | 1 | 2 | 1 | 0 | 4 |
| 78,000 | 0 | 0 | 0 | 1 | 0 | 1 |
| "Leucocytosis" | 0 | 4 | 0 | 0 | 0 | 4 |
| No record | 4 | 29 | 9 | 5 | 30 | 77 |
| | 36 | 66 | 25 | 19 | 34 | 180 |

The anemia here illustrated is of course of the secondary type and is considerable though not extreme in degree. When it occurs, however, it is of very great diagnostic value, supposing that hemorrhage and other obvious causes are absent; for chronic valve lesions and the fevers most likely to be confused with that of acute endocarditis rarely produce anemia. So that I think one may say that if a case appears to be one of ordinary valvular heart disease but is accompanied by fever and especially by anemia not obviously accounted for otherwise, acute or subacute endocarditis is a very probable diagnosis.

*These numbers (I, II, etc.) refer to the sub-groups of acute and subacute endocarditis explained above. "II + III" means a dominant septic focus outside the heart, but also at necropsy an old chronic endocarditis underlying the fresh acute vegetations.

**4. Leucocytosis.**—In Table 131 are shown the leucocyte counts in 103 cases of the present series. 71 of these fall into the class of well-marked leucocytoses, most of them above 25,000 and under 50,000. In five cases the count ranged between 50,800 and 78,000. There is, however, a small but important group of 14 cases with leucocytes in normal numbers. In the absence of other causes for leucocytosis, especially in the group here called primary, the presence of a real leucocytosis is of considerable value, though it must be remembered that intracardiac clots and peripheral infarcts are also capable of producing fever and leucocytosis in the absence of any septic foci inside or outside the heart.

*Circulating Phagocytes.*—In one of the cases of this group the progressive anemia and leucocytosis were associated with an extraordinary number of circulating phagocytes which could be seen actively ingesting both red and white corpuscles. This case has been recorded in detail by Dr. Mary W. Rowley.* A similar case studied by her also occurred in connection with a chronic endocarditis very possibly overlaid by acute endocarditis but not proved to be such, as there was no post-mortem examination. Another case similar to these has been reported by Van Nuys.†

**5. Arrhythmia.**—Unfortunately our records upon this point are worth very little. They suffice merely to show that in a majority of these cases arrhythmia is either absent or so slight that it was not noticed at all during the life of the individual. In 32 cases, 23 of them falling within the primary group, some type of arrhythmia was noticed. In a good many of the other cases the absence of any arrhythmia was definitely stated. In others there is no record.

### SYMPTOMS ON THE PART OF THE CIRCULATION

(a) In Table 132 it appears that in the primary group of cases cardiac enlargement was usually recognized. This is especially true of the recurrent subdivision. In the cases of secondary and terminal endocarditis, on the other hand, cardiac enlargement was recognized only 16 times among 78 cases, as contrasted with 67 times in 102 cases of the primary group.

(b) Very similar figures are seen as regards dyspnea in Table 133, and as regards edema in Table 134.

* Occurrence of Atypical Phagocytic Cells in the Circulating Blood, New York Medical Journal, 1907. Vol. 85, p. 674.

† F. Van Nuys: An Extraordinary Blood. Presence of Atypical Phagocytic Cells. Boston Medical and Surgical Journal, 1907. Vol. 156, p. 390.

TABLE 132.—CARDIAC ENLARGEMENT IN LIFE

| I | II | III | IV |
|---|---|---|---|
| 16 of 36 | 51 of 66 | 10 of 44 | 6 of 34 |

TABLE 133.—DYSPNEA

| I | II | III | IV |
|---|---|---|---|
| 15 of 36 | 52 of 66 | 7 of 44 | 7 of 34 |

TABLE 134.—EDEMA IN LIFE

| I | II | III | IV |
|---|---|---|---|
| 8 of 36 | 47 of 66 | 5 of 19 | 6 of 34 |

From a diagnostic point of view however it is obvious that cardiac enlargement, dyspnea, and edema do not help us to recognize acute endocarditis, but point rather towards a valvular or hypertensive type of chronic heart trouble. Still when these manifestations of impaired circulation are associated with those of constitutional sepsis referred to above, we certainly have a basis for suspecting acute endocarditis, a suspicion which cannot approach definite knowledge unless we have bacterial findings from the blood stream or evidence of embolism. This group presents itself ordinarily under the clinical picture of chronic valve disease and is so diagnosed.

(c) *Cardiac Murmurs.*—Murmurs of some sort were recorded in 118 out of 180 cases. In the cases of primary endocarditis they were recorded in 79 out of 102. The definite absence of murmurs is recorded in only 10 cases of this group; in the remaining 13 there is no record. In the cases of secondary endocarditis, on the other hand, murmurs were recorded in only 35 out of 78 and are definitely recorded as absent in 34. Systolic murmurs alone—which of course have no diagnostic significance—make up the whole record in 55 cases.

Turning to the more *significant murmurs* we find systolic and diastolic murmurs together in 40 cases, systolic, diastolic, and presystolic murmurs combined in 14 cases, systolic and presystolic murmurs in eleven. Obviously none of these murmurs or combinations of murmurs has any peculiar diagnostic significance in relation to *acute* endocarditis, though I believe it is entirely possible that an acute endocarditis, quite unassociated with chronic deformity in

the valve, can, through the presence of perforating ulcerations and blocking polypous masses, produce either a stenosis or a regurgitation. This, however, in my opinion, is rarely the case, the significant murmurs being due in the great majority of cases to chronic underlying lesions. The nature of the evidence on which this belief is based is hard to present. With the heart in one's hand, looking at the vegetations and ulcerations and estimating their size, one arrives at an impression that they are or are not capable of producing stenosis or incompetency of the valve. When the vegetations are so minute that it needs a hand lens to make sure of their presence, it would be generally agreed, I take it, that they could not of themselves produce a leak or a blocking of the valve. On the other hand, when the vegetations are as big as one's thumb it is almost impossible to avoid the conclusion that they have interfered seriously in the function of the valve to which they are attached. Between these extremes there are of course doubtful cases where the presonal equation of the observer must enter in.

I do not believe that the quality or the exact location of murmurs helps much if at all in interpreting them as evidence for or against acute endocarditis. Much more important is a change in the position or quality of the murmur within a few hours or days under the observation of one person. Among the 36 cases of primary non-recurrent acute endocarditis such a change occurred in seven cases out of ten in which there is a definite record on the subject. But in the majority of the cases both in this group and in the others, we have no sufficient study or record upon this point.

(d) *Thrills.*—Largely from the study of cases not included in this group I have come to believe that *a thrill is the most important of all the features in the local examination of the heart as evidence of acute endocarditis.* Realizing of course that a thrill may be produced by a chronic stenosing lesion at any of the valves as well as by congenital defects and by aneurism, I believe nevertheless that the presence of a thrill and especially the appearance or modification of such a thrill, in a febrile case, is of considerable practical value in the diagnosis of an acute lesion. In the present series, however, such thrills were recorded in only 29 out of 180 cases, and 22 of these were in cases associated with a chronic deforming lesion and may well have been due to that rather than to acute endocarditis. My own belief, therefore, as to the significance of a thrill in this disease is not substantiated in the figures here presented. I urge it nevertheless upon the attention of others.

(e) *Chronic passive congestion* was found at necropsy in 86 out of 180 cases, 72 of these belonging to the primary group and 14 to the secondary. It is notable that evidence of general stasis was present in almost every one of the recurrent cases, in the first group, 59 out of 66. This is only what one would expect from the association in this group of a chronic valvular lesion. But it is notable that even in the absence of any such chronic lesion, 13 *out of* 36 *non-recurrent cases in the primary group showed chronic passive congestion after death.* On the other hand, not a single case in the group of pure sepsis with secondary involvement of the heart showed any passive congestion after death, a fact which seems to suggest that sepsis *alone* cannot weaken the heart in such a way as to produce *chronic* passive congestion. About a quarter of the cases with sepsis and secondary involvement of the heart valves implanted upon a previous chronic endocarditis, showed passive congestion after death. But where there was no chronic process on the valve, secondary sepsis alone was never in this series associated with chronic passive congestion.

TABLE 135.—HEART WEIGHTS AT NECROPSY

| | Primary acute | Primary acute recurrent | Pyemic | Pyemic; chronic endo. as well as acute | Terminal (IV) | Total |
|---|---|---|---|---|---|---|
| Normal..... | 13 | 6 | 2 | 0 | 3 | 18 |
| 128–200 | 1 | 2 | 1 | 0 | 0 | 4 |
| 200–249 | 1 | 0 | 5 | 3 | 4 | 13 |
| 250–299 | 1 | 3 | 7 | 5 | 5 | 21 |
| 300–349 | 1 | 6 | 2 | 1 | 3 | 13 |
| 350–399 | 1 | 11 | 3 | 3 | 5 | 23 |
| 400–449 | 2 | 8 | 1 | 2 | 6 | 19 |
| 450–499 | 1 | 10 | 3 | 1 | 2 | 17 |
| 500–549 | 0 | 8 | 0 | 1 | 1 | 10 |
| 550–599 | 2 | 6 | 0 | 1 | 2 | 11 |
| 600–700 | 2 | 4 | 0 | 1 | 1 | 8 |
| 700–800 | 0 | 3 | 0 | 1 | 1 | 5 |
| "Enlarged" | 11 | 2 | 1 | 0 | 1 | 15 |
| 850 | 0 | 1 | 0 | 0 | 0 | 1 |
| 1150 | 0 | 1 | 0 | 0 | 0 | 1 |
| 1273 | 0 | 1 | 0 | 0 | 0 | 1 |
| | 36 | 66 | 25 | 19 | 34 | 180 |

## WEIGHT OF HEART AT NECROPSY

In comment on the facts shown in Table 135, one may say that in 111 out of 180 cases hypertrophy is slight or absent; i.e. in 111 cases the heart weighed 450 grams or less. In the 78 cases of secondary acute endocarditis 38 or approximately one-half showed no hypertrophy whatever. On the other hand, in the 102 cases of the first group there are many very large hearts and only 27 under 400 grams. The largest hearts of this series were associated with chronic pericarditis as well as with acute endocarditis and valve lesions, so that we have no reason to believe that the endocarditis played any considerable part in their size. On the other hand in the non-recurrent cases of the primary group there are eleven cases of hypertrophied hearts the size of which is not explained by any chronic lesion or by any hypertension evident during the period of observation in life, so that it seems possible that acute endocarditis is of itself capable of producing cardiac hypertrophy in some cases. Four cases of this group occurred in persons less than thirty years of age and not the subject of any nephritis or arteriosclerosis. Three of these four were women, one fourteen years of age, one twenty-one, and one twenty-nine. It is hardly probable that hypertension should have produced the cardiac hypertrophy in these cases, and there was nothing whatever in the heart, pericardium or kidneys to account for any such enlargement as was found. Under these conditions we may reasonably believe that there has been no hypertension present to explain the cardiac hypertrophy, and may therefore suppose that the *ulcerations and polypoid masses themselves produce enough disturbance of cardiac function to account for the hypertrophy.*

## BACTERIOLOGY

So little perseverance was devoted to catching bacteria in the circulation during the life of the cases here studied that I shall here record only the results of post-mortem cultures. In 80 out of 180 cases (see Table 136) an organism very probably connected with the endocarditis was cultivated from the blood at necropsy. 54 of these positive cultures were in the first or primary group of cases, and only 26 in the secondary cases. 19 out of 36 of the primary non-recurrent cases of acute endocarditis showed positive cultures. 35 out of 66 of the recurrent cases, 19 out of 44 of the septic cases with secondary endocarditis, gave positive cultures, while seven of the 34 terminal cases were also positive.

TABLE 136.—ORGANISM POST-MORTEM

| | I | II | III | IV | Total |
|---|---|---|---|---|---|
| Streptococcus | 15 | 19 | 16 | 6 | 55 |
| Staphylococcus aureus | 1 | 8 | 0 | 0 | 9 |
| Atypical streptococcus | 1 | 1 | 0 | 0 | 2 |
| Pneumococcus | 2 | 4 | 2 | 1 | 9 |
| Staphylococcus albus | 0 | 1 | 0 | 0 | 1 |
| Streptococcus and staphylococcus | 0 | 1 | 1 | 0 | 2 |
| Streptococcus and pneumococcus | 0 | 1 | 0 | 0 | 1 |
| | 19 | 35 | 19 | 7 | 80 |

The streptococcus was much the commonest organism, being present either alone or in association with other organisms in 75%. No careful study of the type of streptococcus was made in any considerable number of these cases, so that I shall attempt no comparison with the results of others upon this very important point. The staphylococcus aureus was found in nine cases all falling within the sub-group of primary acute endocarditis, the pneumococcus in nine cases, six of which fell in the first group and three in the second, Staphylococcus albus not the result of skin contamination occurred in one case. In two cases there was the association of streptococcus and staphylococcus and in one case of streptococcus and pneumococcus. *No gonococci* or *"influenza bacilli" were found.*

## ASSOCIATED LESIONS

118 of these cases were associated with hypertrophy and dilatation of the heart, and 62 were without any such association. Aside from the question of cardiac enlargement, 54 cases showed no cardiac lesion except the acute endocarditis. 69 were associated with valve lesions and of these 69, 20 had also further pathology in the circulatory system. 20 were associated with chronic non-deforming endocarditis, seven of these being further complicated. There were 9 cases of acute pericarditis and 9 of chronic pericarditis with the acute valvular lesion. 13 of these pericardial cases were still further complicated by other circulatory lesions. 17 cases were associated with arteriosclerosis and 17 with chronic nephritis, nine of these 34 cases having some further cardiac pathology as well. One case is associated with goitre and one with myocarditis and other lesions. The overlapping and duplication of these is obvious.

If one looks for the *gravest* associated lesion in each case the results are as in Table 137.

TABLE 137.—CHIEF* ASSOCIATED LESIONS IN 180 CASES OF ACUTE ENDOCARDITIS

| Acute and subacute endocarditis | Without enlargement | With enlargement |
|---|---|---|
| Alone | 35 | 19 |
| With valve lesions | 7 | 43 |
| With nephritis | 0 | 17 |
| With goiter | 1 | 0 |
| With chronic pericarditis | 1 | 8 |
| With leukemia | 0 | 1 |
| With pernicious anemia | 0 | 1 |
| With acute pericarditis | 4 | 5 |
| With chronic non-deforming endocarditis | 11 | 9 |
| With arteriosclerotic degeneration of the kidney | 2 | 2 |
| With arteriosclerosis | 1 | 12 |
| With myocarditis | 0 | 1 |
| | 62 | 118 |

* To avoid duplications the most important lesion associated with the endocarditis has in each case been counted as *the* associated lesion.

TABLE 138.—PRIMARY ACUTE AND SUBACUTE ENDOCARDITIS

| Duration | Nonrecurrent | Recurrent |
|---|---|---|
| | I | II |
| Less than 2 weeks | 2 | 3 |
| 2– 3 weeks | 6 | 4 |
| 4– 7 weeks | 15 | 5 |
| 2– 5 months | 2 | 21 |
| 6–10 months | 5 | 8 |
| 10–14 months | 2 | 2 |
| 15–24 months | 1 | 0 |
| 24 months | 1 | 3 |
| 2– 3 years | 2 | 2 |
| 4 years | .. | 2 |
| Questionable | .. | 16 |
| | 36 | 66 |

## DURATION

We have no way of calculating the duration of cases in the group of pyemic or terminal endocarditis since there the underlying illness, septic, neoplastic or whatever, may always have produced the earliest symptoms. In the group designated as primary we obtained the figures shown in Table 138, from which I shall draw the following conclusions.

The recurrent cases of primary endocarditis last usually for months. 38 out of 50 with a known duration lasted two months or more. On the other hand, the non-recurrent primary cases lasted as a rule weeks rather than months, 23 out of 36 knowing nothing of any illness until within two months from the day of death. Four to seven weeks is the duration in nearly half of these cases, while two to five months is the commonest duration in the recurrent cases, 19 of which fell within these limits. The majority of the notably long cases, lasting ten months or more, are in the group of recurrent cases, seven falling in this group to six in the non-recurrent group.

Among those in which the duration is stated as two or three years (or four years as in one case), it is to be supposed that as in other chronic infections, for example tuberculosis, there is a waxing and waning of immunity against the disease, periods of latency or arrest in the process followed by the periods of activity. That this was the case in at least two of our cases was shown by the occurrence post-mortem of clean-cut, smooth-edged holes in valves, easily distinguishable from congenital defects or fenestrations, and clearly the result of a previously ulcerative process which had healed.

In one case the patient definitely stated that he had been through exactly the same experience two years previously, had recovered after some months of illness, and then had remained perfectly well until a few weeks before the time when we saw him.

Especially in children and in adolescents I think there is reason to believe that this alternation of periods of activity with periods of quiescence in the inflammation upon the heart valves is not at all uncommon. Many of the cases called "chronic" and listed along with deforming valvular lesions had at certain periods leucocytosis and fever unexplained by any known lesions outside the heart. Some of these cases may well have suffered from a brief and relatively mild attack of acute endocarditis, overcome, like the milder tuberculous infections, without ever subjecting the system to any observable strain or producing any but the mildest evidences of a constitutional reaction.

In the primary non-recurrent group of cases the average *stay in the hospital* up to the time of death was 17 days. Leaving out of account four relatively long cases which were in the hospital between 37 and 90 days each, we have for the remaining cases an average of ten days from the time when they were sick enough to be brought to a hospital until the moment of death.

**Diagnosis.**—31 cases out of 102 of the primary group were diagnosed in life. None of the secondary group was recognized during life.

## SUMMARY

1. Acute and subacute endocarditis can be divided into the "primary" cases which appear to be confined to the circulatory system, and the "secondary" group which result from a septic focus outside the heart (e.g. general peritonitis or a septic uterus) or from a chronic debilitating disease (neoplasm, nephritis, etc.). In these secondary cases the endocarditis is probably a terminal event.

2. In 56% of cases, acute or subacute endocarditis is a recurrent process implanted upon an old area of healed inflammation. This percentage rises to sixty-four if we consider only the "primary" cases.

3. It occurs especially between the twentieth and the fortieth year and affects the two sexes about equally.

4. The mitral valve is attacked in 76% of cases, and is the only valve attacked in 43%. In general the frequency of occurrence of the disease on the different valves is about the same as in chronic deforming endocarditis. There is no special tendency to attack the tricuspid or pulmonary valves.

5. *Embolism* is shown post-mortem in nearly two-thirds of the "primary" cases, but is recognized during life in only one-half of these, or one-third of all "primary" cases. The skin, spleen, kidneys, brain, and lungs are most often affected. In the "secondary" cases emboli are but one-half as common as in the "primary."

6. *Nephritis* occurs in 40% of all cases and in 33% of the "primary" group.

7. *Diagnosis* is often, perhaps usually, impossible. Fever and chills in a patient with a diastolic murmur and a palpable thrill should always make us suspicious. If these are associated with an unexplained secondary anemia and especially with evidences of embolism (purpura, hematuria, enlarged spleen) we may expect that assiduous search will demonstrate organisms (usually streptococci) by culture in the blood.

In many cases the diagnosis is masked by the more striking evidences of chronic valvular disease and general passive congestion.

8. Decided *cardiac enlargement* occurs in 37%. Most of these cases can be accounted for by an associated valvular disease or by hypertension. But in a few cases it seems probable that the acute vegetations themselves disturb cardiac function enough to produce hypertrophy.

9. The *duration* is usually weeks or months, occasionally years.

10. *Recovery certainly occurs* and in children is probably not uncommon.

### *Points of Especial Interest to the Writer*

1. The frequent occurrence of a palpable thrill.
2. The occasional occurrence of cardiac hypertrophy unexplained.
3. The absence of any evidence that the right side of the heart suffers more often than in chronic valvular disease.
4. The proof of healed ulcerative lesions.
5. The absence of the gonococcus and the influenza bacillus.
6. The rarity of an association with acute pericarditis (only 9 cases in 180).
7. The absence of the sternal tenderness mentioned by Libman.

### ILLUSTRATIVE CASES

#### Necropsy 2700

A housewife of twenty-nine entered August 20 with a diagnosis of Banti's disease made by an intelligent internist. She had always considered herself healthy. She had had no rheumatic fever, pleurisy, or malaria. She had had two miscarriages and no subsequent pregnancies. Her catamenia were usually profuse, regular and painless; ceased the May before admission. During the present illness she had urinated once or twice at night. Her bowels were always costive. Her best weight was 99½ pounds.

Eight months before admission she was curetted on account of a miscarriage. Some time after this she began gradually to lose strength, color and weight. In May she was in bed a week with nausea, fever and pain in the right side (abdomen?). Two weeks later she noticed peculiar numbness in the right arm; no paralysis; but at this time she had difficulty in enunciation and next day complete aphasia which lasted forty-eight hours but left her quickly.

Two months before admission she had severe pain in the left upper quadrant for four weeks. This kept her awake at night, was not associated with any lump, and gradually passed away. Her bowels were increasingly constipated, though there was no pain or change in the stools so far as she knew.

The chief complaint was of increasing pallor and weakness, so that for the two months before admission she kept to her bed. There was no jaundice or bleeding from the gums. For the past two months she had noticed crops of red spots on the legs.

Examination showed a fairly well developed, poorly nourished woman with obvious loss of flesh. The skin was muddy, but not really jaundiced. There were purpuric spots over both legs below the knees. The apex impulse of the heart was in the fourth space, 7 cm. from the midsternal line. There was no right-sided enlargement. A blowing systolic murmur was heard at the apex transmitted to the axilla and back, also heard at the base. The sounds were of good quality. The pulmonic second sound was not accentuated. The lungs showed dullness, diminished voice and breath sounds at the left base extending up about halfway to the angle of the scapula and continuing around the chest to the front. There was a pleural rub at the right base behind on deep inspiration. Over an area extending from the umbilicus to the pubes and corresponding roughly with the area which would be produced by a greatly distended bladder there was distension, tympany, visible peristalsis and a succussion sound. In the region of the spleen there was a hard mass with a definitely nodular edge moving freely with respiration, on pressure not tender, well felt from the flank. There was slight dullness in both flanks, not shifting with change of position. There was some diastasis of the recti. Vaginal examination showed nothing of importance. The rectal examination was negative. The extremities and reflexes were normal.

During more than seven weeks of observation in the hospital the patient had waves of fever lasting a week or ten days each, rarely reaching more than 103°. There were only three or four days in this whole period when she was free from fever. As a rule the daily swing was about 2°, but at times it was 4° or more. The pulse was continuously elevated, usually between 100 and 120. The respirations were 20 to 25 during most of the course. The output of urine was 40 to 70 ounces, the specific gravity 1.002 to 1.005 at five of seven examinations, 1.016 at the other two. Traces of albumin were always present, sugar absent. The sediment usually

showed a few hyalin and granular casts; nothing else of importance. At entrance the red cells were 2,500,000, the leucocytes 6,000, the hemoglobin 38% (Sahli), the polynuclears 83%. The stained smear showed moderate achromia, slight variation in size and shape, no blasts. The blood plates counted by Dr. J. H. Pratt were 141,000. August 29 the blood showed very little change. September 9 the red cells were 2,280,000, September 13 2,056,000, September 17 2,000,000, September 24 1,900,000. There were no important changes in the look of the blood smear except that the achromia grew more and more marked and polychromatophilia appeared. Blood cultures August 22, 24, 25 and September 5 showed always a profuse growth of atypical pneumococci. A urine culture August 25 and September 8 showed the same organism. September 19 a blood culture showed a somewhat less definite organism, the surface growth showing diplo-



FIG. 104.—Rash following medication.

cocci while the water of condensation showed chains of streptococci. A Wassermann August 24 was positive. August 26 20 minims of a catheter specimen of urine were injected into a guinea-pig. Necropsy on this animal October 3 was negative.

Further study of the mass in the left hypochondrium seemed to show that it was a tender spleen. At entrance the patient was given ten grains of potassium iodid three times a day. Forty-eight hours later a very marked rash developed on the face and arms. (See Figs. 104 and 105.) August 26 this rash was pustular, but cultures taken from the pustules showed no growth. The mass in the region of the bladder at first suggested the retention of urine. The patient was catheterized with removal of thirty-six ounces of urine, but the tumor still persisted. Apparently it was due to protrusion of intes-

tines through the rectal diastasis. About August 25 she had several sharp attacks of pain in the spleen with increase of its tenderness. On the 26th a loud harsh systolic murmur was heard at the apex of the heart apparently different from that heard at entrance. Dr. E. Lawrence Oliver pronounced the cutaneous eruption to be due to KI taken.

September 7 there was a slight colitis, which cleared up in a few days. About this time the patient began to have constant severe pain in the region of the spleen, and its lower edge was exquisitely tender. At midnight on the eighth she suddenly developed deep noisy breathing and when seen by the nurse was in a convulsion. Next morning the reflexes of the right arm were increased and there was right ankle clonus. The patient was drowsy and stupid all day, the white count still, however, remaining low. At this time the

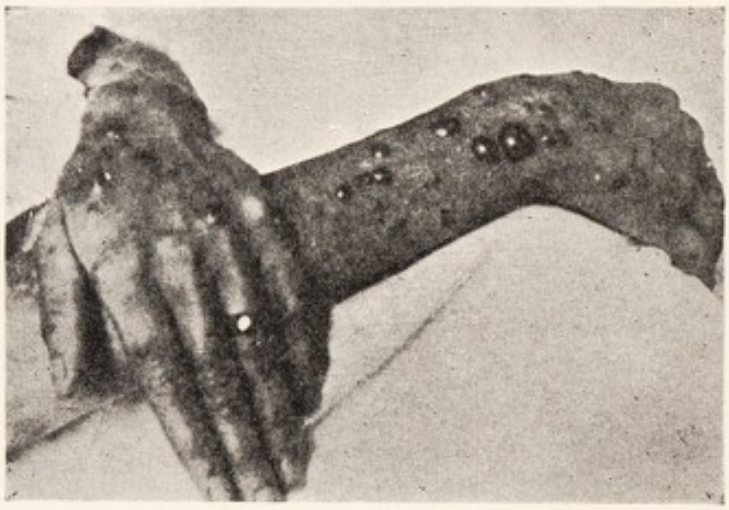


FIG. 105.—Rash following medication.

cardiac murmur replaced the first sound altogether. September 11 the left chest showed dullness to flatness from the angle of the scapula to the base, with moist râles and absent breath sounds over this area. The left clavicle was slightly tender. By September 19 the splenic tenderness had disappeared and the signs in the left chest were gone. Her appetite was good, her spirits much improved, yet the patient was steadily growing weaker. There was marked ankle clonus, especially on the left. September 27 the liver was quite tender. Toward the end of life she had to be kept under morphia and became incontinent of urine and feces, so that a bedsore developed over the sacrum. October 9 she died.

*Clinical Diagnosis (from Hospital Record).*—Subacute infectious endocarditis.

Multiple infarcts.

Septicemia.

Chronic passive congestion.

*Dr. Richard C. Cabot's Diagnosis.*—Septicemia, streptococcus-pneumococcus.

Acute endocarditis of the mitral valve.

Hypertrophy and dilatation of the heart.

Chronic passive congestion of the lungs.

Infarcts of spleen, liver, lungs.

Perisplenitis.

Decubitus.

*Anatomical Diagnosis.*—Septicemia, atypical pneumococcus.

Acute and slight chronic endocarditis of the mitral valve.

Hypertrophy and dilatation of the heart.

Infarcts of the spleen and kidneys.

Slight chronic passive congestion, general.

Hemorrhages of the liver.

Focal fibroid myocarditis.

Slight chronic pleuritis, right.

Decubitus.

DR. RICHARDSON: The gastrointestinal tract showed nothing at all except some reddening of the mucosa, the initial stages of passive congestion. The abdominal wall was very thin, the muscles pale, and while there was no distinct separation, yet the thinness of the wall would easily permit the bulging forward of the intestines that Dr. Cabot mentioned.

DR. CABOT: Do you think that just part of the general emaciation?

DR. RICHARDSON: Yes.

DR. CABOT: We said: "Why was there diastasis of the recti?" The answer is, there was not any. The mystery is wiped out. The patient was very thin everywhere, so thin that the intestines came forward.

DR. RICHARDSON: The cultures showed the organism already mentioned, an organism belonging to the streptococcus-pneumococcus group and that particular one known as the *viridans*. That was proved true in life, proved true in the heart blood and from the spleen, establishing the streptococcus septicemia.

The heart showed only very slight hypertrophy and dilatation. The right wall of the heart was 4 mm. thick, slightly thickened, the left 10 mm., not thickened at all. The cavities were only slightly enlarged. That is important, because there was only a slight amount

of chronic passive congestion and not much dilatation. The aortic, tricuspid and pulmonary valves were negative. On the mitral valve in a few scattered places there were small polypoid vegetations, taken together a very small mass. So that within the heart there was no definite cause for hypertrophy and dilatation, and there was not much. The aorta and its branches were negative. So that although there was a polypoid mass of vegetations on the mitral valve, they were so small that they produced but little decrease in the circumference. She was a very small woman, and the valve would not ordinarily measure more than eight cm.; there was very little change. That makes very little deformity of that valve. That is a point Dr. Cabot has always insisted on. Where there is real stenosis of a valve there is with it deformity or such change as can be called deformity. There was nothing in the cavities, and no ball thrombus.

The spleen was very large as has been described. It weighed 465 grams; that is considerably enlarged. Scattered through it were infarcts the outer surfaces of which showed acute splenitis resting over these areas of infarction. The spleen tissue itself was a little soft, as is apt to be the case in infections, although not always so.

The pleural cavities showed no fluid. The lungs showed a slight amount of chronic passive congestion, and there was a slight amount of chronic passive congestion in the liver, which is of course in harmony with the character of the heart. The liver weighed 1660 grams—a little large—and the interesting thing was that scattered through it were some small areas of hemorrhage. Those were in the nature of purpura, and probably are to be associated with the infection present and also with the anemia.

Dr. Cabot: Why do we have purpura in infections?

Dr. Richardson: I don't know. As the cases come here we find it associated with infections and anemia. Those are the two common things, and then again we find it associated with nothing that we can put our finger on. We do not know whether it is change in the quality of the blood or in the character of the vessel walls which produces purpura.

A Physician: How much purpura hemorrhagica do you see?

Dr. Cabot: It is a very rare disease. We often go a full year in this hospital without seeing any.

Dr. Richardson: We get cases here of unexplained purpura, something akin to hemorrhagic diathesis. One of the most marked cases I have seen was in connection with tuberculosis. That of course was infection.

The microscopic examination confirmed the gross picture, showing the vegetations on the heart valve laden with bacteria. Another interesting point is that of the focal myocarditis of the left ventricle. There was a small area of softening in the wall, and the vessel leading to that area was occluded; it was definitely an area of infarction, a focal myocarditis.

A PHYSICIAN: Is this case infection from the heart vegetation itself?

DR. CABOT: I do not think anybody can answer the question whether it starts in the blood and goes to the heart or starts in the heart and goes to the blood. My own guess is that it starts in the blood.

**Necropsy 2910**

A colored laborer of twenty-eight entered August 19. His past history was negative except that he was kicked in the leg by a horse a month and a half ago. For the past three weeks he had been drinking two bottles of beer and one of gin daily. He formerly drank much whiskey and beer. He smoked from two to six cigars a day. He had "chancre" three weeks before admission.

A few days after the chancre appeared he began to have "rheumatism" in his ankles, calves, and left wrist, so painful that he had difficulty in walking. For three weeks his appetite had been poor, especially in the morning. August 12 examination in the Orthopedic Room of the Out-Patient Department showed slight swelling of the left wrist and right ankle. He was given aspirin. August 15 he is reported that the pain was relieved, but that he now had pain in the left lower quadrant. The leucocyte count was 16,000. August 19 the abdominal pain was still present. Physical examination showed no tenderness. The constant dull ache in the left lower quadrant had persisted. He had vomited a little white watery material during the past two days, without relation to food. He thought he had had slight edema of the legs. He urinated once or twice nearly every night.

Examination showed a well-nourished negro. The right epitrochlear glands were the size of a pea; the left were not palpable. The heart showed no enlargement to percussion. The sounds, action, pulses and artery walls were normal. There was a soft blowing systolic murmur at the apex, transmitted to the axilla. The systolic blood pressure was 115. The lungs were clear. The spleen seemed somewhat enlarged to percussion. The edge was not

felt. There was a small indurated area on the corona of the penis with a moist surface and slight discharge. No urethral discharge was discovered. There was slight tenderness under the outer malleolus of the left ankle. The left wrist was slightly painful on extreme flexion or extension. The pupils were slightly irregular. The right reacted sluggishly to strong light; the left reacted well. The reflexes were normal.

The temperature was 101° to 105°, the pulse 84 to 160, the respiration 28 to 80. The amount of urine was 25 to 81 ounces, the specific gravity 1.012 to 1.020. There was the slightest possible trace to a trace of albumin at all of five examinations, a few red blood corpuscles at all, rare hyalin and granular casts at two, a large number of granular casts, a few hyalin, and many casts with red and white blood corpuscles attached at the last two. The hemoglobin was 100%. There were 25,000 to 52,000 leucocytes, 91% polynuclears. A Wassermann was negative. Blood cultures August 19 and 23 showed *streptococcus pyogenes*. The stools were dark red, showed many red blood corpuscles, and gave a positive guaiac. A skin consultant reported, "I think that the lesion of the penis is probably a primary lesion. I do not believe his temperature is due to syphilis, and should think his present acute medical condition of more importance than his syphilis."

August 25 the heart murmur had a muscial quality which disappeared over night. Many fine crackles were heard at the lower right axilla, and August 26 many crackles at the left base behind. August 28 he had a chill. Although he gradually lost ground he felt comfortable and ate his meals with evident relish. August 29 there were many fine moist crackles at the right base in the axilla and in front. September 4 the pulse and respirations rose. Autogenous vaccines were given. The systolic murmur continued loud and harsh, occasionally with a musical tone. Râles were heard at both bases. September 7 he died.

*Clinical Diagnosis (from Hospital Record).*—Streptococcus septicemia.

Syphilis.

*Dr. William H. Smith's Diagnosis.*—Streptococcus septicemia.

Ulcerative endocarditis of the mitral valve.

Possibly multiple foci of bronchopneumonia.

Passive congestion?

Infarction of the lungs? of the spleen? of the left kidney?

*Anatomical Diagnosis.*—Septicemia, streptococcus.

Acute endocarditis of the mitral valve with occlusion of the valve orifice.

Extensive infarcts of the spleen and kidney with purulent softening of a large splenic infarct.

Thrombosis of the right common iliac artery, the splenic artery, the branch of the left renal artery.

Hypertrophy and dilatation of the heart.

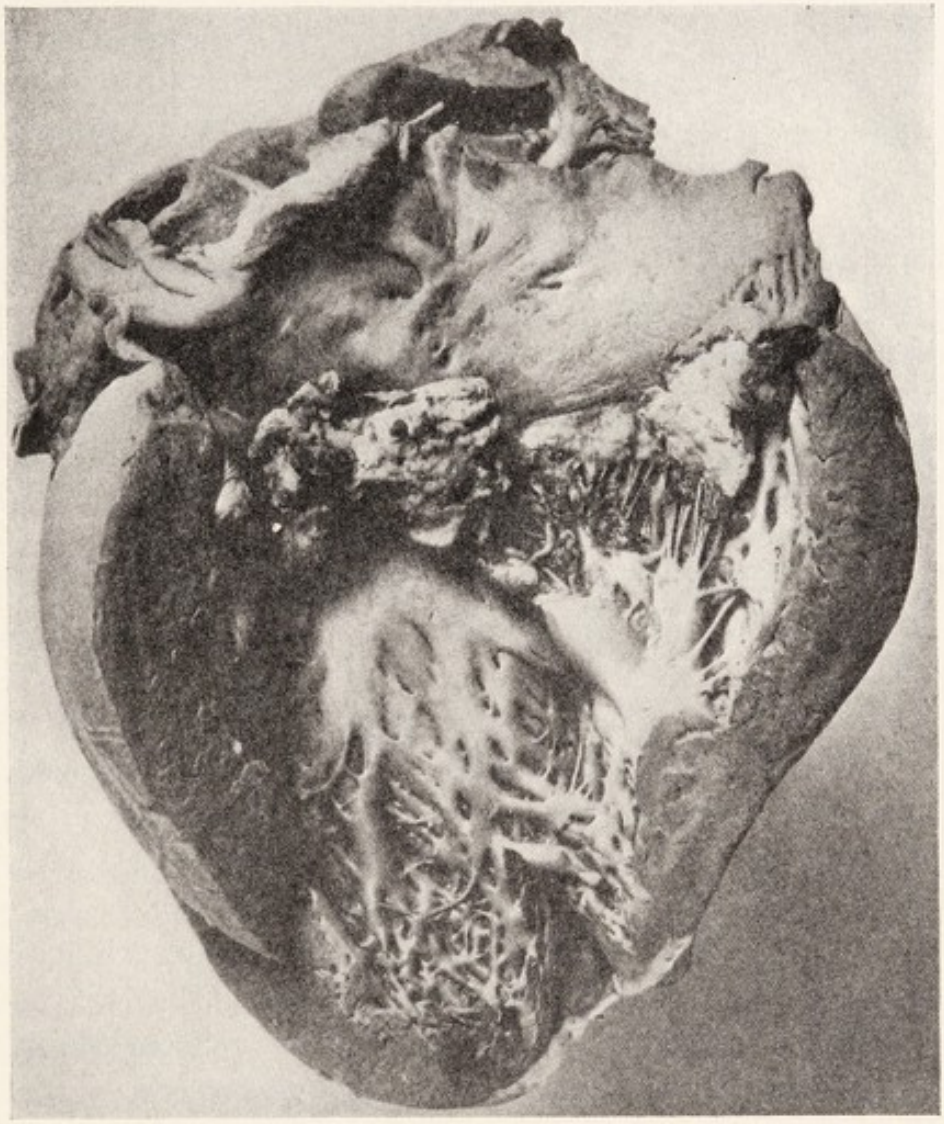


FIG. 106.—Acute polypous endocarditis of the mitral valve with a huge mass occluding the mitral orifice. (Photograph by Lewis S. Brown. Dr. Oscar Richardson.)

Chronic passive congestion, general.

Acute glomerulo-nephritis.

Slight chronic pleuritis, left.

DR. RICHARDSON: The illustration (Fig. 106) shows the mitral valve with the huge mass on it. When all its edges are brought together there is little space for the circulating fluid to go through.

It was gradually closing down. This was a case of streptococcus septicemia with which was associated the large polypoid mass of vegetation on the mitral valve and the acute glomerulo-nephritis. By the latter I mean a condition in which there is proliferation of the endothelial cells of the capillaries of the glomeruli with the occlusion of the glomeruli. At this time we associate the condition with organisms of the streptococcus-pneumococcus group. From the picture of the mitral valve it is plain to see how small particles could be washed off and carried into the various vessels. We do not always find the plugs in the vessels as we did in this case, where we found them in three places. That picture is rather uncommon. The question arises in these cases of infection with this group of organisms as to whether it may not be an arteritis of the vessels and the thrombi erected at that point. We had a case the other day which supports this hypothesis well. In this particular place we do have a definite source for the thrombi, that is, the thrombi are embolic. The emboli come down, get plugged there, and become adherent to the wall. In the other case the valves were perfectly smooth and free, and there was marked infarction of the kidneys and the thrombus in the renal artery.

The heart weighed 363 grams and was moderately dilated. With the exception of the mass on the mitral valve it showed nothing remarkable.

We made a careful examination for gonococci and spirochetes, not only by the cover glass method but also by staining sections according to Levaditi's method. There was no evidence of syphilis or gonorrhea.

The lungs showed chronic passive congestion.

The liver and the gastro-intestinal and the genito-urinary tracts were negative with the exception of the kidneys. They weighed 404 grams, rather large. (Normally 200–400.) They showed the infarcts mentioned in the anatomical diagnosis and a rather wide cortex, 6–7 mm. The glomeruli were visible at points.

Although this man was a negro, in whose race the prevalence of tuberculosis is well-known, there is no tuberculosis recorded; even the lymphatic glands were negative.

### Necropsy 3030

An Irish laborer of thirty entered January 8. His past history was negative except for "fever" for a few days as a child. He drank six glasses of whiskey and eight of beer a day, sometimes more.

For a year he had had ulcers on his legs. Three months before admission they practically healed, but soon broke down and since this had not healed. He had never noticed any varicosities, and denied all symptoms of syphilis.

For six weeks he had had a troublesome cough with a moderate amount of sputum. He had been increasingly dyspneic, especially at night. His feet and ankles had been swollen at times. He urinated three or four times at night. He slept poorly, especially after drinking heavily. He had anorexia and was constipated.

Examination showed a well-nourished man tossing restlessly about, with occasional unproductive cough. The mucous membranes were slightly dusky. The apex impulse of the heart was in the fifth space. The borders of dullness were 14.5 cm. to the left, 4 cm. to the right, the substernal dullness 6 cm. The action was rapid and slightly irregular. The sounds were of fair quality, heard with difficulty on account of noises in the bronchi and lungs. There was a loud blowing systolic murmur at the apex transmitted to the axilla, a to-and-fro murmur at the second right rib transmitted to the neck. The aortic second sound was accentuated. The pulses were irregular. The blood pressure was 150/80 to 120/70; the systolic later fell to 100. The lungs showed poor expansion. Expiration was somewhat prolonged, especially on the right. There were many coarse crackles and groans. There was moderate dullness throughout the right side of the abdomen, not shifting. The liver dullness extended from the fifth space to 3 cm. below the costal margin. The edge was not felt. The genitals were normal. There was considerable pitting edema of the lower legs and ankles. On the lower legs were two ulcers, discrete, with well defined edges and granulating centers, and a small healed scar surrounded by an area of scaly brownish skin. The pupils and reflexes were normal.

The temperature was 95.7° to 101.8°, the pulse 65 to 116. The respirations were 17 to 36, after February 1 not remarkable. The amount of urine was 17 to 75 ounces, the specific gravity 1.030 to 1.012. There was albumin in large amounts in twenty-four of twenty-eight examinations, a slight trace at the other four. Hyalin and granular casts were seen at all examinations, in large numbers at the first three, red blood corpuscles at all, leucocytes at nine. A culture from the urine showed a few bacilli. The hemoglobin was 80 to 60%. There were 7,200 to 12,600 leucocytes, 75% polynuclears. The smear at entrance was not remarkable; March 18 the reds were

2,648,000. Four Wassermanns were negative. The fundi at admission and February 13 were normal.

The patient was very uncomfortable at entrance. January 13 he was resting better at night, the edema was clearing, and the heart borders coming in. He continued to improve slowly. January 21 the left border of dullness was 16 cm., the substernal dullness 8 cm., the apex impulse diffuse in the 5th and 6th spaces. There was a short rough musical systolic over the left precordia, a short rough systolic of different quality in the aortic area, a soft diastolic

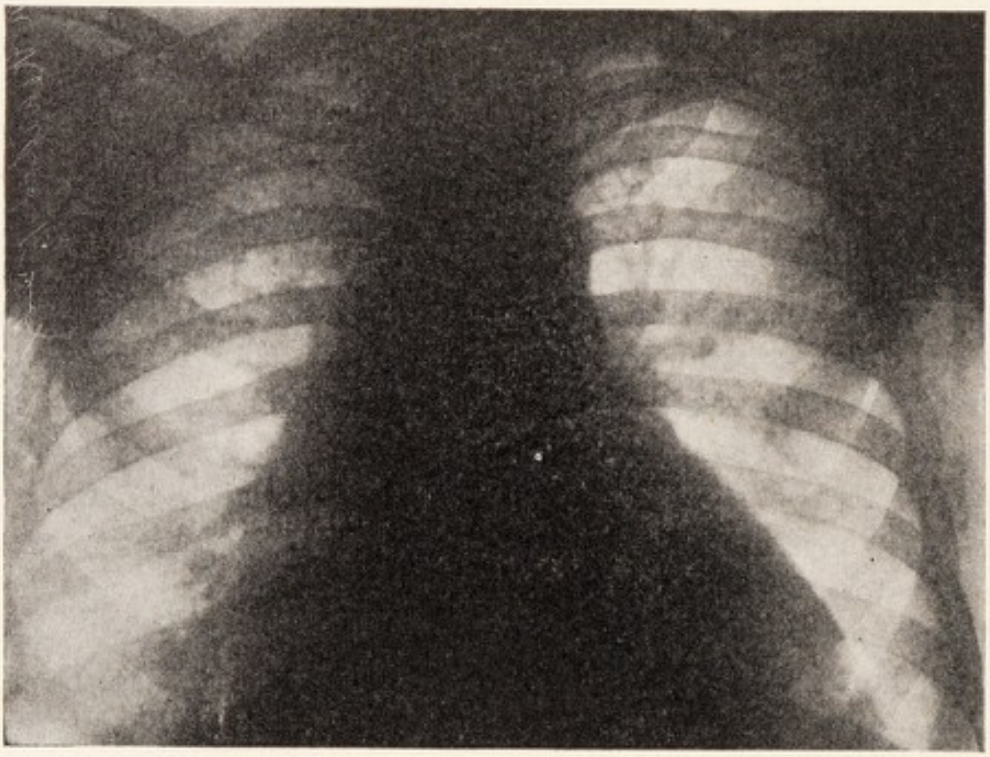


FIG. 107.—The heart is greatly enlarged to the left. There is a shadow at the right of the sternum probably due to peribronchial thickening. The aortic arch is not enlarged. General peribronchial thickening and glandular enlargement. By fluroscope no abnormal pulsation was made out.

in the aortic area and along the right sternal margin. There were a few râles at the bases of the lungs. The ulcers of the legs were improving rapidly with corrosive dressings. X-ray January 24 showed the chest as in Fig. 107. He was put upon mercury salicylate and potassium iodid. Because of marked coryza and lachrimation the potassium iodid was omitted next day, and because of salivation the mercury was omitted January 31. February 4 the systolic in the aortic area was barely audible. He was running a slight irregular temperature without obvious cause. Blood cultures were negative February 4 and 21 and March 2. March 5 the temperature was no longer elevated. He was allowed to sit on the porch. He was given

two carbon dioxid baths and felt better after them. There was a slight amount of fluid in the abdomen.

March 14 the right ear and mastoid region were painful, the mastoid tender on pressure. This drum was incised by an ear consultant, with a free discharge of pus, which continued. March 21 there was a butterfly patch of dull red over the nose, slightly tender and indurated. The patient had a slight chill. Two days later the patch had disappeared, but the mastoid tenderness was exquisite. The patient was dull, often irrational, complaining of no pain. The aural consultant advised postponing operation on account of the general condition. There was considerable edema of the legs, later of the hands, and marked edema and tenderness down under the right jaw. March 30 the eyes were turned to the right; there was slight nystagmus. The knee-jerks were feeble. The patient was noisily delirious, and died that day.

*Clinical Diagnosis (from Hospital Record).*—Chronic glomerulo-nephritis.

Chronic endocarditis.

Aortitis?

Chronic middle ear.

Chronic mastoid.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the aortic and mitral valves, with stenosis of each.

Hypertrophy and dilatation of the heart.

Subacute glomerulo-nephritis.

Secondary anemia.

Terminal streptococcus infection.

Chronic passive congestion.

*Anatomical Diagnosis.*—Acute vegetative endocarditis of the aortic valve.

Chronic ulcerative endocarditis of the aortic valve.

Subacute glomerulo-nephritis.

Septicemia, pneumococcus streptococcus.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Hydropericardium.

Hydrothorax.

Ascites.

Anasarca.

Scar in the region of the right mastoid with sinus formation.

Phlegmon of the deep tissues of the right side of the neck.

The heart weighed 584 grams (normally 200–300). The cavities were considerably enlarged. The mitral valve was 12 cm. in circumference (normally 10 cm.). The valve was not otherwise remarkable. The circumference of the aortic valve laid open was 8 cm.; but there was a large fibrocalcareous mass 3 cm. × 2 cm. which fused two of the cusps together and pushed up into the orifice of the valve, thereby considerably decreasing its actual circumference. The circumference of the tricuspid valve was 14 cm. (normally 12–13). Except for enlargement the valve was not remarkable. The pulmonic valve measured 9.75 cm. (normally 8–9 cm.), not remarkable.

*Note by Dr. G. W. Holmes.*—In reviewing the X-ray plates in the light of the outcome it may be noted that the general shape of the heart, roughly triangular, suggests fluid in the pericardium.

### Necropsy 4577

An Italian clerk of twenty-three entered August 4, 1923, for relief of general weakness, malaise, dyspnea, slight cough, palpitation, and slight aching in the joints. His general health had always been good. He had the usual diseases of childhood and possibly scarlet fever. He had occasional head colds and slight headaches. Since tonsillectomy at seven he had had occasional sore throats. Several years before admission his nose was injured.

Four and a half years before admission, after a mild attack a week and a half in duration of influenza and tonsillitis associated with general malaise, slight fever and some chilliness, he had his first attack of painful and swollen joints. He was confined to bed for nine weeks. On returning to work he developed sore throat, soon followed by mild general constitutional symptoms. One morning on waking he had considerable swelling of all the joints, and pain which increased in severity until it was almost impossible to bear the weight of the bed clothes. There was local heat but no redness. He felt slightly feverish and at times chilly, but had no actual chill. At the initial attack all the joints became involved simultaneously. Active or passive motion aggravated the pain considerably. In addition to it there was swelling of the precordia. The acute condition did not last very long. Most of the nine weeks of his illness was a gradual recovery. In addition he spent a few months in recuperation. From that time he was free from symptoms until two months before admission, when he had a second attack of painful and swollen joints. A month later cardiac signs were discovered. During the past two weeks cardiac symptoms had developed. He

attributed the present attack of debility to worry about his mother's condition. A short time before the joint symptoms developed he had slight malaise and weakness. Then followed pain and stiffness in the left calf muscle, and soon afterwards pain and swelling in the right ankle, with rapid involvement of the left ankle. He gave up work. A few days after the ankles had been involved, when the inflammation was subsiding, the knee-joints became involved, and soon after this the ankle-joints again, with the left shoulder joint. Until a few weeks ago he was up and about the house. For three weeks his joints had given him no trouble, but he had developed at first a "full feeling" in the epigastrium, with gradual development of slight dyspnea, which in the past week had increased so that he slept in a more or less semi-recumbent position. His sleep was still considerably disturbed, especially in the past few days. The "fullness in the stomach" had increased until it had several times caused him to vomit. In the past few days he had had palpitation and slight precordial pain. Recently he had had slight unproductive cough and had urinated once or twice at night.




FIG. 108.—Measurements by percussion.

Examination showed a poorly nourished, rather pale man. The lungs were clear. The apex impulse of the heart was felt in the fifth space, diffuse. The heart was enlarged to the right and left. The percussion measurements were as shown in Fig. 108. There was forceful bounding pulsation, best seen and felt in the fifth space one cm. outside the nipple line, faintly felt in the sixth space. The whole precordia lifted with the apex. There was an apical thrill. A systolic murmur replaced the first sound at the apex and the base. A short diastolic was heard at the aortic area and the apex. The blood pressure was 130/50–0. The abdomen was dull, perhaps because of feces. The liver and spleen were palpable. The liver edge was felt. The fingers were some what clubbed. The pupils and reflexes were normal.

The temperature was 98° to 103.1°, the pulse 91 to 129, the respiration 18 to 36. The output of urine was 42 to 83 ounces, the specific gravity 1.010 to 1.018. There was a very slight trace to a trace of albumin at all of five examinations. Red and white blood corpuscles were present at all but one, twice in large numbers, granular casts

at three. The renal function was 50%. The hemoglobin was 65%. There were 13,600 to 34,500 leucocytes, 3,580,000 to 4,160,000 to 3,100,000 reds, moderate achromia, only slight variation in shape, with an occasional tendency toward tailed forms. One blood culture was negative, one showed streptococcus. One Wassermann was

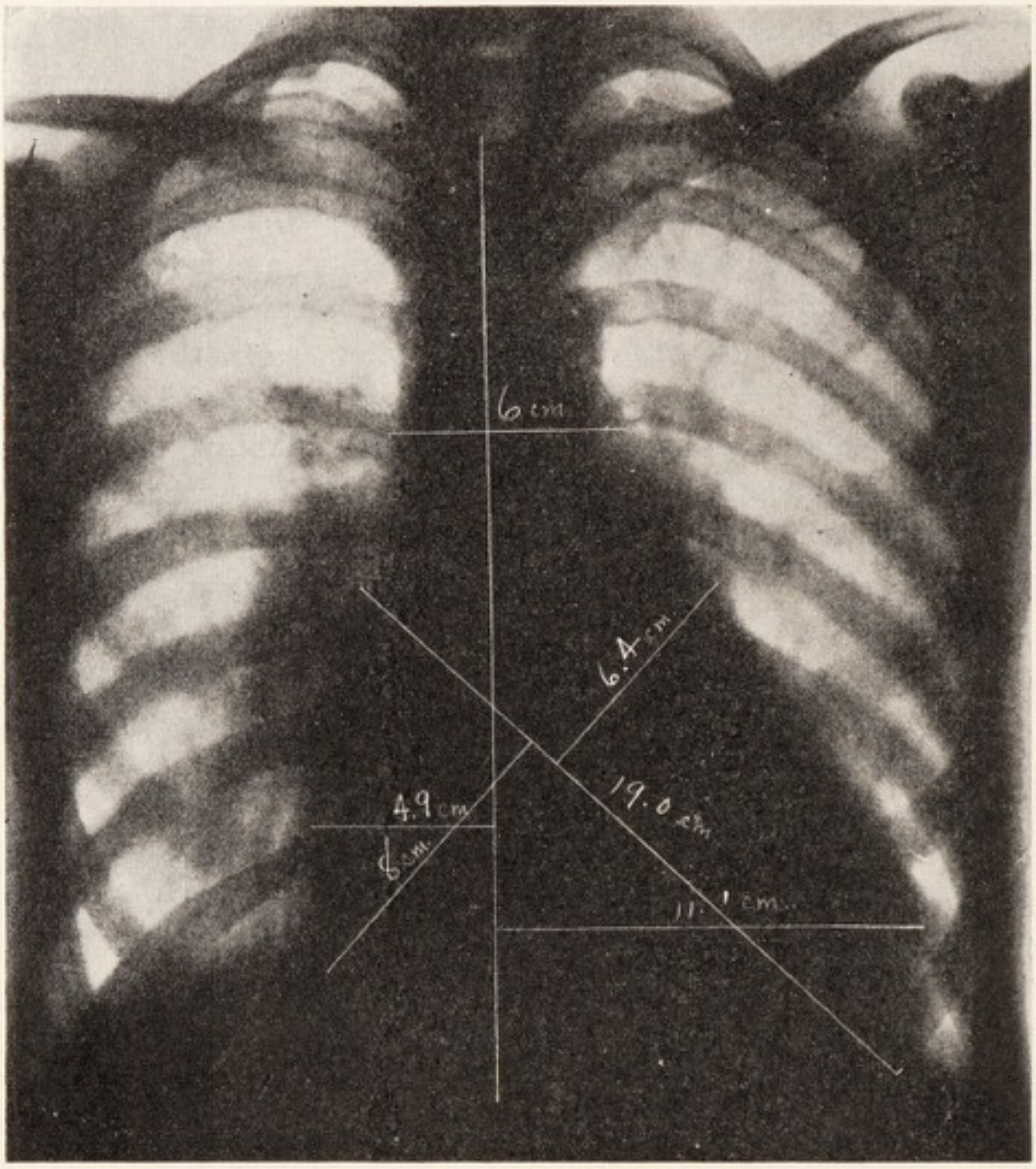



FIG. 109.—Chest 25.5 cm. Marked enlargement of the heart shadow. Increase apparently marked across the base in the region of the auricles. No change in the supracardiac dullness. Marked increase in the hilus shadows on both sides, with thickened prominent lung markings extending well out toward the periphery of the chest, and a large amount of mottling between the larger lung markings. These changes apparently do not reach the periphery or apices, and may be cardiac in origin.

weakly positive, one negative. The X-ray findings August 17 are shown in Fig. 109.

August 16 transfusion of 510 c.c. was followed by a chill for a short time. The red count was not markedly changed. August 21 the patient was discharged with instructions for rest and hygiene.

September 18, 1923, he reentered. During the greater part of the interval since his discharge he had been in bed, in the afternoon walking about for a short time. His condition had remained in general about the same, perhaps growing somewhat worse, definitely so for the past few days. After he had been home a short time he began having bloating and a full feeling in the epigastrium, so that it embarrassed his breathing a good deal toward the evening and usually after eating. With the bloating he usually had cough with watery mucoid sputum, and frequently vomited with relief. After the vomiting he frequently had palpitation and precordial pain which at times radiated to the right side. A few days before reentry he noticed for the first time toward evening pitting edema of the ankles. He was dyspneic when at rest, especially when bloated. He had had

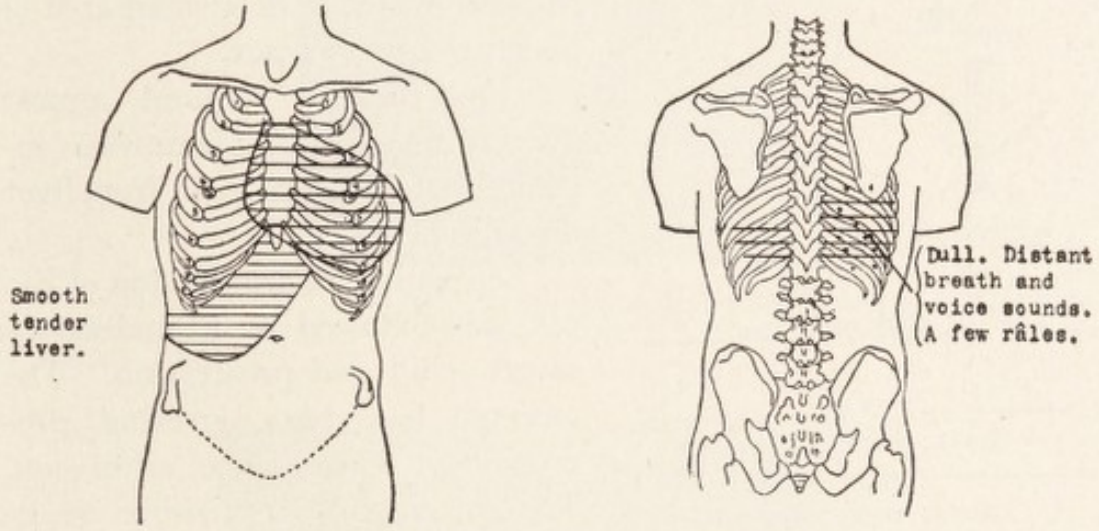



FIG. 110.—Chest sign in Case 4577.

no joint symptoms except some pain and stiffness in the right calf a few weeks ago. He thought he had some evening rise of temperature.

Upon examination he was very emaciated, with slight mucoid cough. The lung signs were as shown in Fig. 110. The heart was very large, with diffuse heaving precordial impulse and thrill. There was a systolic blowing murmur over the precordia, best heard at the pulmonic area, and a crisp short diastolic. The heart was heard throughout the chest. The blood pressure was 105/50. The spleen was not felt because of abdominal distension. The liver was as shown in the diagram. There was great edema of the feet and ankles. Over the shins were red petechiae.

The temperature was 99° steadily falling to 94°, the pulse 130 falling to 86, the respiration 34 to 16. The output of urine was 20

to 33 ounces, the specific gravity 1.018. There were occasional leucocytes, no albumin. The hemoglobin was 50 to 55%. There were 32,800 to 33,200 leucocytes, 2,980,000 reds, 89% polynuclears. The mononuclears showed large vacuoles in cytoplasm. The red blood corpuscles showed considerable variation in size, typical macrocytes, a few microcytes. Most of the fields showed red cells well filled with hemoglobin; others showed slight achromia. There were a few tailed forms and oblong cells. The platelets were strikingly decreased and small. A Wassermann was moderately positive. The non-protein nitrogen was 95 mgm. X-ray showed the general appearance of the heart practically the same as at the previous note, possibly slightly more enlarged. The right antrum was dull. The right superior bicuspid tooth supporting a bridge showed evidence of a small area of absorption at its apex.

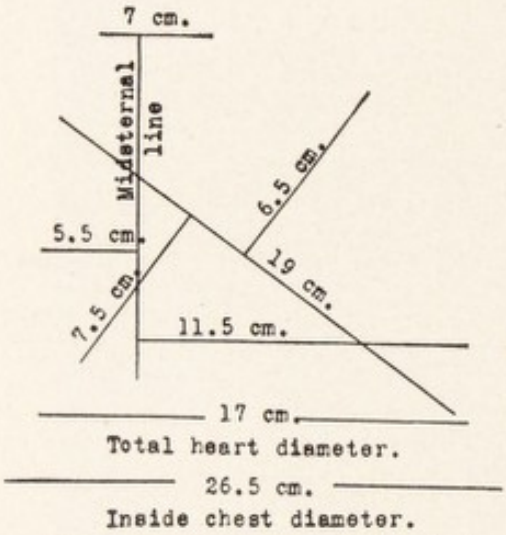



FIG. 111.—Measurements by X-ray in September.

The patient vomited almost everything taken. There was considerable abdominal pain from liver distension.

September 18 transfusion of 500 c.c. was followed by a moderately severe chill and prostration. The patient had been growing progressively worse since admission. The evening of September 18 he became weaker, andthe pulse failed decidedly, though it improved with caffein. Early the next morning he quietly died.

*Clinical Diagnosis (from Hospital Record).*—Bacterial endocarditis.

Streptococcus infection of blood.

*Dr. Richard C. Cabot's Diagnosis.*—Acute and chronic endocarditis of the aortic and mitral valves.

Hypertrophy and dilatation of the heart.

Chronic passive congestion.

Acute nephritis (?).

Chronic pericarditis (?).

*Anatomical Diagnosis.*—Vegetative endocarditis of the mitral and aortic valves.

Vegetative endocarditis of the endocardium of the right auricle.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Infarcts of lungs, spleen and kidneys.
Chronic glomerulo-nephritis, capsular.
Ascites.
Edema of the ankles.

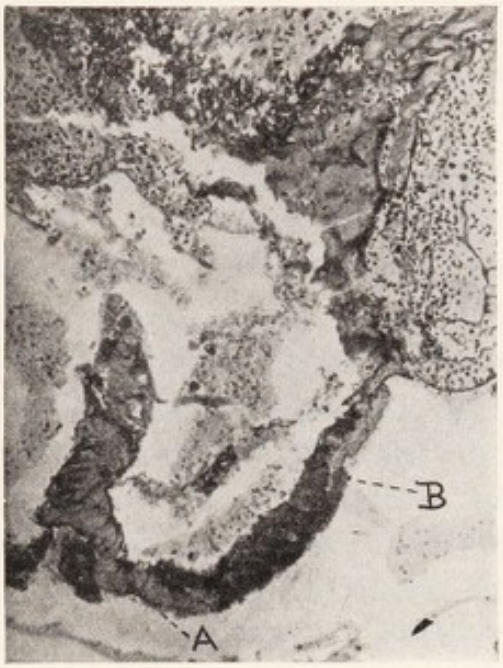



FIG. 112.—Case 4577. *Streptococcus viridans* endocarditis. Section of valve A, B, bacterial fringe. (× 1000). (Photomicrograph by Dr. Albert E. Steele. Dr. William H. Smith.)

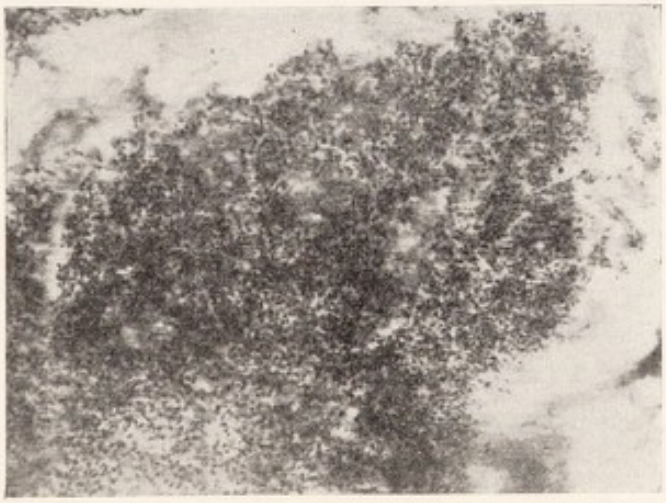


FIG. 113.—Case 4577. *Streptococcus viridans* endocarditis. High magnification of the area at A in Fig. 112. (× 1500.) (Photomicrograph by Dr. Albert E. Steele. Dr. William H. Smith.)

DR. RICHARDSON: We were not permitted to examine the head. There was edema of the legs. There were red spots scattered over the feet and the anterior aspect of the right arm.

There was considerable thin clear fluid in the peritoneal cavity,—ascites. The liver was seven cm. below the costal border. The dia-

phragm was at the fifth rib on the right, at the fifth interspace on the left.

In the pleural cavities there were fifty c.c. of fluid on the right and forty c.c. on the left, with a few adhesions on each side. The lungs were rather voluminous, but showed nothing for note except for a few small dark red areas scattered in the lung tissue.

There was fifty c.c. of clear fluid in the pericardium, the beginnings of hydropericardium. The heart weighed 670 grams,—a very large heart for the lesions mentioned. The myocardium was three to four mm. on the right, eight to nine on the left. The cavities showed some dilatation and were filled with blood clot. The valve measurements were as follows: mitral 12 cm., aortic 7.5 cm., tricuspid 14 cm., pulmonic 9.5 cm. That is an increase in the circumference of the mitral, with the others about as usual, except that the tricuspid is a little large. The tricuspid and pulmonary valves showed no lesions. But on the mitral, extending over practically the whole length of the free margin of the curtain, there were numerous smaller and larger frank vegetations. These vegetations extended up high on the wall of the auricle. The aortic cusps showed a number of vegetations, not so large though as those on the mitral.

DR. CABOT: Was there anything chronic on those valves?

DR. RICHARDSON: I did not see any; and a mitral valve of twelve cm. of course indicates that the amount of chronicity, if there was any, was very slight. The extension of the vegetative patches upon the auricle is rather characteristic in these *viridans* infections.

The spleen weighed 395 grams and was moderately enlarged. We must remember that this man was an Italian. There were a few infarcts in it.

The kidneys weighed 582 grams. This means they were enlarged. The microscopical examination showed capsular glomerulonephritis and infarcts.

The culture from the heart blood showed a streptococcus of the *viridans* type.

DR. CABOT: There was no pericarditis?

DR. RICHARDSON: None.

DR. YOUNG: Would you get as much nitrogen retention as this in the terminal hours of any other condition than one causing passive congestion? This was done the day of death.

DR. CABOT: I remember 125 in a case of typhoid fever; we scratched our heads over that a lot. Of course there is passive congestion. So that I should not be able to say I had seen it in cases

without passive congestion. But I do think it is important not to make a diagnosis of nephritis on a high blood retention unless we have other evidence.

A PHYSICIAN: May I ask Dr. Richardson if the lungs showed any changes?

DR. RICHARDSON: Nothing but chronic passive congestion.

DR. CABOT: The X-ray did not lead us to expect anything more than passive congestion.

A PHYSICIAN: I have been interested in the changes in the lungs in passive congestion. I wondered whether that mottling was wholly due to passive congestion or whether there was a true fibroid condition there.

DR. CABOT: But our general impression is that we can get just as much mottling from passive congestion alone, that we must not conclude that we have an organic condition in the lungs if we have something else like passive congestion to account for the mottling.

A PHYSICIAN: How do you explain the weakly positive Wassermanns?

DR. CABOT: I do not explain them. We have a great many here, although the test is done as carefully as possible. Our rule in all departments of this hospital, including the syphilis department, is never to make a diagnosis of syphilis on a weakly or a strongly positive Wassermann without any other evidence of syphilis.

A PHYSICIAN: What is the antigen used?

DR. CABOT: I don't know what antigen we are using now. But I think it is a generally accepted observation that one should never make a diagnosis of syphilis on a Wassermann reaction no matter who made it or what kind of antigen was used, unless there is other evidence in the patient's history or body to support it. I think a great deal of harm is done by concluding from a weakly or moderately or strongly positive Wassermann that a patient has syphilis.

### Necropsy 4081

An English housewife of thirty-two entered May 3. From the age of twelve she had attacks of dull frontal headache lasting a few hours, usually relieved by vomiting, at first very frequent and severe, later occurring once in three or four weeks. She had had none for ten years. She had always been near-sighted, and for twenty years had worn glasses. At fifteen she was in bed ten weeks with chorea, and ever since that time had had palpitation, slight

dyspnea and rapid pulse after exertion. At twenty-seven she was ill two weeks with mumps. She had had four or five attacks of severe bleeding from both nostrils lasting two to six hours, relieved by packing. The last attack was four years ago. She had one or two "colds" each winter. At twenty-two she weighed 135 pounds, her best weight. A year before admission she weighed her usual weight, 128 pounds.

Five months ago she gave birth to her first child. She was in labor for more than ten hours and finally was delivered with forceps under ether. She stayed in bed for two weeks and felt well. She had slight bloody discharge for eight or nine days. She was said to have a normal puerperium. The baby was breast fed for four weeks, then put upon artificial feeding because it was not getting enough. Meanwhile the mother felt sick and tired easily. There was a gradual onset of slight irritating cough with or without being preceded by a tickling sensation of the throat. The cough was unproductive and was more frequent towards the evening then the rest of the day. She felt alternate cold and warm sensations over her body. There was insidious onset of night sweating. She had a feverish sensation, usually worse in the afternoon, and at times her temperature rose to 101° or more. She had taken many kinds of tablets and syrup for the cough without any relief. Her weakness and cough became more marked and she had been compelled to stay in bed for the past three and a half months. For three or four weeks the temperature rose as high as 104° in the evening on several occasions. The night sweats became much worse. Her appetite became very poor and her sleep much disturbed. Her bowels were moved once in one or two days by salts or enema.

Examination showed a poorly developed and nourished woman with some mental confusion simulating partial aphasia. Over the chest and abdomen were numerous scattered pin-point purpuric spots. The scalp was yellow, scaly, the hair thin. The mucosae were slightly pale. There was slight pyorrhea. The tongue was protruded in midline with coarse tremor. The chest expansion was slightly greater on the right than on the left. The lungs showed slight dullness over the left top in front and harsh breath sounds throughout, with high-pitched inspiration. The heart sounds were transmitted throughout both backs. There were a few scattered medium moist râles at both bases posteriorly. The apex impulse of the heart was seen and felt in the fifth space 10 cm. to the left. There was a systolic thrill. The action was very rapid (140). The pulmonic

second sound was markedly accentuated. At the apex was a short presystolic roll. A snapping first sound and a loud blowing systolic murmur were heard over the precordia. The pulses were of small volume and tension. The blood pressure was 150/70 to 90/50. The liver dullness extended from the fifth rib to two centimeters below the costal margin. The edge was not felt. There was a firm smooth mass in the left upper quadrant extending 6 cm. below the costal margin to the midline, probably spleen. The rectal examination showed nothing of importance. There was slight cystocele and rectocele. Pelvic examination showed a lacerated perineum. The anterior lip of the cervix was cystic. There was slight edema of the feet, more on the right. The fingers showed slight tremor. The right pupil was greater than the left. Both were slightly irregular, with poor reactions to light and distance. The reflexes were normal except for poor position sense in the toes on the left.

The temperature was 98° to 103.2°, the pulse 108 to 147, the respiration 20 to 38. The output of urine was 11 to 42 ounces, the specific gravity 1.014 to 1.030. The urine was cloudy at all of five examinations and showed a very slight trace to a trace of albumin at all, leucocytes at all, red cells at three, granular casts at two. The hemoglobin was Tallqvist 70%, Sahli 72%. There were 4800 to 8600 leucocytes, 78% to 90% polynuclears, 4,012,000 to 3,136,000 reds, showing slight achromia. The platelets were slightly increased at one examination, the reds and platelets normal at another. No endothelial leucocytes were seen in sixteen smears. A Wassermann was negative. May 3 a blood culture showed streptococcus and Gram-positive bacilli in one flask, staphylococcus and Gram-positive bacilli in another. May 7 there was streptococcus viridans in both flasks. May 11 streptococcus in both and staphylococcus albus in one.

The patient lost ground and was very confused at times. The pulse rate was 130. New purpuric lesions appeared on the neck and feet. The presystolic murmur was variable. May 11 there was marked hallucinosis and her fingers were very cyanotic. May 13 she was failing rapidly. The pulse rate varied from 80 to 135 from minute to minute, and was very poor in quality. There was slight edema over the upper lumbar region. There were frequent new crops of ecchymoses, or rather purpuric spots, on the chest, abdomen, back and mostly on the neck, occasionally on the finger tips and toes. There was no change in the heart sounds except occasional extrasystoles. May 20 for a few minutes she had twitching of the right

hand and leg. May 22 she was more or less stuporous, with a fixed stare. Cyanosis of the hands was marked at times. The anemia was progressive. The temperature was normal, but the condition was poor. Early in the morning of the 23rd she suddenly cried out, and at once went into a series of violent convulsive movements of the whole body, slightly more marked on the left side. Her eyes were turned to the left and her legs drawn up. The respirations became weaker, and she died.

*Clinical Diagnosis (from Hospital Record).*—Malignant endocarditis.

Mitral stenosis.

Secondary anemia.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic and acute endocarditis of the mitral valve.

Mital stenosis.

Septicemia, streptococcus.

Infarcts of the spleen.

Cerebral embolus.

Secondary anemia.

*Anatomical Diagnosis.*—Septicemia, streptococcus viridans.

Malignant endocarditis, mitral valve.

Small vegetation on one of the cusps, aortic valve.

Hypertrophy and dilatation of the heart.

Slight hydropericardium.

Hydrothorax and ascites; slight anasarca.

Slight chronic passive congestion, general.

Infarcts of the spleen and kidneys.

Cholelithiasis.

Obsolete tuberculosis of a bronchial gland.

DR. RICHARDSON: This case showed a streptococcus viridans septicemia, established during life and from the heart blood at necropsy.

The heart weighed 350 grams; considerable enlargement and moderate dilatation. Except for a small vegetation on one of the aortic cusps the aortic valve was negative and the tricuspid and pulmonary negative. The mitral valve showed a very extensive area of vegetations forming a thick mat extending along the free margin and from the posterior cusp extending up along the auricular wall. This mass taken all together when the valve was closed produced considerable obstruction to the valve. But so far as any extensive chronic process goes, that was not found.

The kidneys were negative for glomerulo-nephritis, but showed extensive infarcts. The spleen was markedly enlarged and showed numerous infarcts. The circulatory apparatus elsewhere was negative and the uterus and adnexa negative. The gall-bladder was negative save for about eight stones of pretty good size.

(Specimens of heart and spleen.) The spleen weighed 850 grams. We can plainly see the infarcts showing here and there. Some pieces were cut from the valve, but the marginal process is evident. If we supply the gaps with material like that extending all along the free margin of the valve and note the process on the auricular wall and where it extends down on the chordea tendineae, we have a typical picture of malignant endocarditis. The appendices in this heart were free. Small masses broke off from what we see here and passing into the vessels plugged them up, with the production of infarcts.

**Necropsy 3945**

A divorced Irish-American woman of twenty-nine, unoccupied, entered September 21 for relief of weakness. She had always been "nervous." She had measles, pertussis, chicken-pox and scarlet fever before she was seven. At eight and at twelve she had chorea, and about the same time two attacks of inflammatory rheumatism. *Since childhood she had sweat so heavily that her clothes were very wet most of the time.* She had hysterical attacks about once a month, at the time of catamenia. At twenty-one she was anemic and did not menstruate for two months. At twenty-seven she was divorced from her husband, a drunkard and drug addict of sixty-one. She had one miscarriage. She had dysmenorrhea, and for a year had had leucorrhea. Once or twice a month she had cramps in the legs, often awakening her from sleep. Her best weight was 130 pounds, her present weight 116.

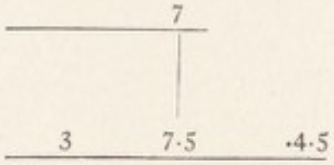



FIG. 114.—Heart measurements by percussion.

For thirteen years she had had palpitation. For five years she had had "gas pains" and epigastric distress once or twice a week either shortly after meals or when she was nervous, relieved by belching, by induced vomiting, and by indigestion pills. She was afraid to eat because of gas pains. For two years she had had chills, especially in damp weather, one to three in a fortnight, lasting two

minutes, relieved by hot drinks. For two months she had been growing more nervous and had had increasing weakness, especially in the legs. She had a horror of being alone, and when left so had palpitation. Her hysterical attacks caused more weakness than formerly. For four to five weeks she had had swaying sensations in the head brought on usually by worry or excitement. The day before admission for the first time she had sharp gnawing pains in the abdomen for five minutes.

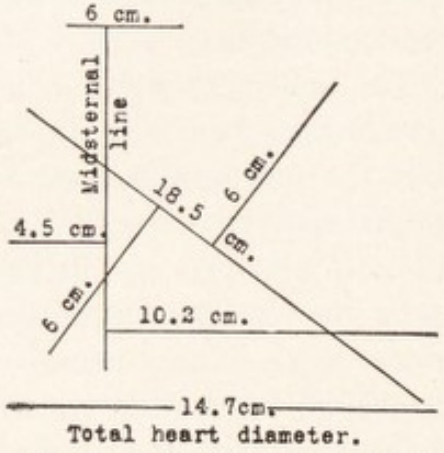



FIG. 115.—Heart measurement by X-ray.

Examination showed a well-nourished woman with marked pulsation of the vessels of the neck. The throat was reddened. The apex impulse of the heart was in the fifth space, 12 cm. to the left of the midsternum. The measurements by percussion and X-ray are shown in Figs. 114 and 115. Short high-pitched systolic

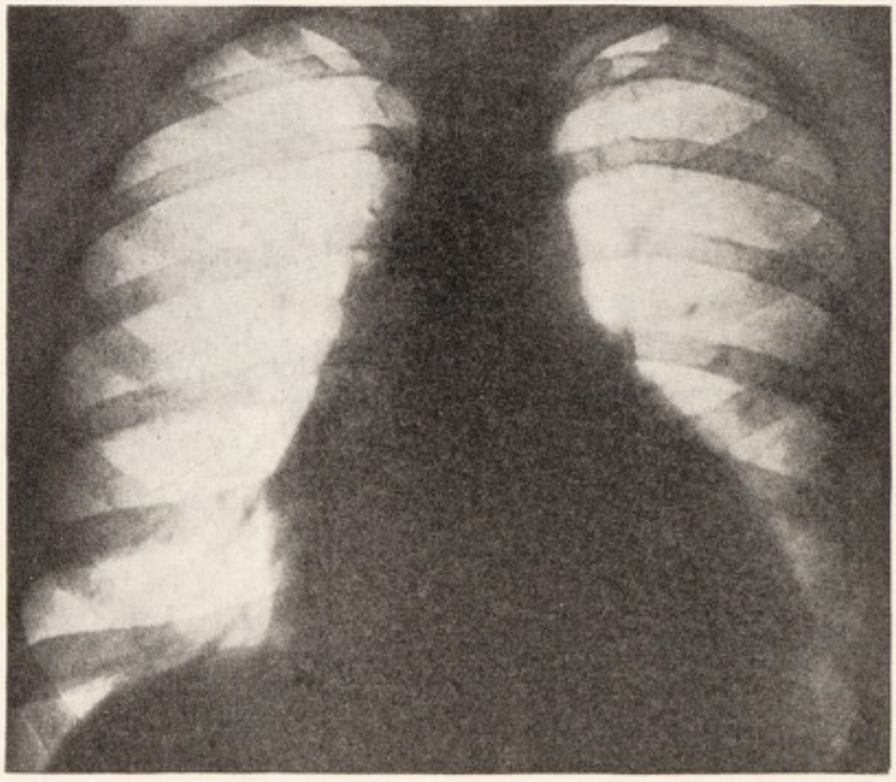


FIG. 116.—September 29. Enlarged and calcified glands at the lung roots. Increased density of the lung markings, noticeable in the ascending trunks. The signs are more marked on the right. The heart shadow is symmetrically enlarged.

and diastolic murmurs were heard at the apex and in the left axilla, a rough loud systolic at the base transmitted upward, and a long

rough blowing diastolic transmitted downward along the left sternal border. The pulses were synchronous, of good volume, high tension, Corrigan in type. Pistol shot was well heard over all the large vessels. The fingers showed capillary pulse. The systolic blood pressure was 160 to 170, the diastolic 50. The artery walls were palpable. The abdomen was normal except for a dynamic aorta. The extremities, pupils and reflexes were normal.

The temperature was 96.7° to 99°, the pulse 71 to 128, the respirations normal except for one rise to thirty-one the day before discharge. The output of urine was 17 to 63 ounces, the specific gravity 1.010 to 1.020. The urine was cloudy at three of four examinations. There was no albumin. The sediment was full of pus at the first examination and showed many to occasional leucocytes at two others. Two more specimens, taken by catheter, showed very rare leucocytes. A urine culture showed a slight growth of staphylococci. The renal function was 30% in the first hour; no urine the second hour. The hemoglobin was 80%. There were 6200 to 22,000 leucocytes, 61% to 74% polynuclears. A Wassermann was negative. The blood nitrogen was 42 mgm. per 100 c.c. of blood. A stool was negative to guaiac. A vaginal smear showed no gonococci. The fundi were normal. X-ray September 22 showed evidence of a small amount of fluid at the left base. September 29 (see illustration) there were enlarged and calcified glands at the lung roots, increased density of the lung markings, noticeable in the ascending trunks. The signs were the most marked on the right. The heart shadow was symmetrically enlarged. The kidney outlines were obscured by gas. There was no evidence of stone.

The condition changed very little. She complained of pains in the right lower quadrant. After September 29 the leucocyte count ranged from 16,200 to 22,000. There was a considerable neurotic element in the case. The patient had many pains and complaints for which no cause could be found. October 2 the digitalis was omitted, and October 5 KI gr. x t.i.d. was ordered. October 9 she was discharged relieved.

March 21, *two years and a half later*, she reentered. She had worked steadily as cashier or as maid in a club. All the summer before readmission she had felt tired. In January she began to have dyspnea, then edema of the legs and some cough. Four weeks before readmission she gave up work because of edema, increasing dyspnea and some orthopnea. For three weeks her abdomen had been swelling. For two weeks she had been in bed.

Examination was as before except for the points noted. She was fairly well nourished, very dyspneic, with jumping carotids—the chest shook with every heart beat. The mucosae were cyanotic. There were moist râles in both lungs behind below the angles of the scapulae. The apex impulse of the heart was seen and felt all over the chest and in the seventh space in midaxilla, with systolic retraction at the apex. The left border of dullness was in the midaxillary line. The other borders were as before. The action was regular and rapid (124), the sounds of good quality, the pulmonic second sound accentuated. Murmurs replaced all the heart sounds. At the apex the first sound was sharp and replaced (*sic*) by a loud blowing systolic transmitted to the axilla and back; also a loud blowing diastolic. At the base both a systolic and a diastolic were heard. The aortic second sound was gone. All the murmurs were heard over the back. There was pistol shot in the groin. A presystolic thrill was felt at the apex. The pulses were of poor tension, the artery walls not palpable. The systolic blood pressure was 180 to 145, the diastolic 50 to 60. The liver edge was 6 cm. below the costal margin, tender and pulsating. The extremities showed marked tremor. There was edema of the legs and lower back. The shins were smooth. The knee-jerks and ankle-jerks were sluggish.

The temperature was 95° to 99.2°, the pulse 82 to 142, the respiration 21 to 48. The output of urine was 9 to 32 ounces, the specific gravity 1.018 to 1.032. The urine was muddy or cloudy at four of five examinations and showed a very large to slight traces of albumin at all, diacetic acid at two. The sediment was red at one examination and showed red blood corpuscles at three, leucocytes at four, a few hyalin casts at two. The hemoglobin was 95%. There were 11,600 to 24,000 leucocytes, 71% polynuclears. A Wassermann was negative. The non-protein nitrogen was 19 mgm. per 100 gm. of blood.

The day after admission the chief of service found a suggestion of presystolic thrill at the apex and of systolic thrill at the base. March 23 the heart started fibrillating. There was marked orthopnea. March 29 there was some distension, relieved by enema. March 30 the patient was fairly comfortable, slept most of the time, and had the slowest pulse since admission, 84, with no pulse deficit; still absolutely irregular. April 2 she became very dyspneic and cyanotic and complained of a sense of great oppression in the chest. The heart was rapid, weak, and absolutely irregular. Caffein gr.

iii s.c. gave relief. There was a return of distension. The patient became very orthopneic, and complained of pain in the right scapular region. There was pulse deficit 5–10. When asleep she had Cheyne-Stokes breathing. She grew steadily worse. The edema increased. April 15 there was ptosis, external strabismus and dilatation of the pupil of the left eye, and some difficulty in talking. Soon after this she was comatose for a while, with rapid contraction and dilatation of the pupils, and was afterwards unable to articulate clearly. April 17 she died.

*Clinical Diagnosis (from Hospital Record).*—Cardiac decompensation.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of aortic and mitral valves with stenosis and regurgitation of each.

Hypertrophy and dilatation of the heart.

Cardiac thrombosis or acute endocarditis.

Chronic passive congestion, general.

Infarcts of spleen and kidneys.

*Anatomical Diagnosis.*—Chronic endocarditis of the mitral, aortic and tricuspid valves; stenosis.

Acute endocarditis of the mitral valve.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Infarcts of the right lung.

Hydrothorax, double.

Hydropericardium.

Slight ascites.

Anasarca.

Slight arteriosclerosis.

DR. RICHARDSON: The heart in this case weighed 772 grams (normally 200–300). It was greatly hypertrophied. It was dilated markedly on the left, slightly on the right. The mitral circumference was reduced to 7 cm. (normally 10), the tricuspid to 5.5 (normally 12–13). The aortic was 7 cm. (normal), but the cusps were greatly shortened by the fibrosis.

DR. CABOT: Were there no infarcts?

DR. RICHARDSON: None except one in the lung.

DR. CABOT: And no thrombi in the heart?

DR. RICHARDSON: There was an acute endocarditis on the mitral which was a thrombus mass on the valve. This of course could be a source for emboli.

The kidneys were negative.

### Necropsy 3542

An Irish-American painter of forty-five entered October 11, 1915, for relief of edema and dyspnea. He had "typhoid-pneumonia" at ten years. For eighteen years he had been a carriage and automobile painter, and until 1910 used much lead paint. In 1900 he had lead poisoning. Since that time he had been careful about washing, etc. For five years he had used no lead. For ten years he had had a sharp stabbing pain in the left axilla, worse when he had a cold or cough. Until 1910 he used to have slight attacks of dizziness. From 1910 until six weeks before admission he had daily dull frontal headaches coming at any hour, sometimes at night. For over a year he had had dull epigastric pain half an hour after eating, associated with an "awful feeling like a bunch in the epigastrium" with nausea and vomiting. Vomiting, and nothing else, gave relief. There was some pyrosis. For a year he had had one loose movement a day and had urinated six or seven times by day, five or six at night. His best and usual weight was 142 pounds, his weight a year before admission and at present 118.

In 1911 he had a pain over his heart on exertion travelling to the axilla and back, sharp for twenty-four hours, then growing dull. With it was dyspnea. Every two or three nights he had attacks of nocturnal dyspnea. He had occasional edema of the legs and ankles in the evening. His appetite began to be poor. For two years he had had a real chill every morning and afternoon, followed by fever. For six months he had had swelling of the abdomen. He stopped work because it was slack and in order to rest. His dyspnea, edema and precordial pain grew worse, so that two months ago he could hardly walk upstairs. He slept on three pillows. He now had constant "dragging" precordial pain radiating to the back and the epigastrium. Six weeks before admission his knees, ankles, and great toe were red, hot, swollen, stiff, and so painful he could not stand up, but remained in bed. For five days he had had a short, dry cough with very little sputum.

Examination showed a fairly well developed and nourished man, slightly dyspneic. The skin had a slightly yellowish pallor. The mucous membranes were pale. There was much pyorrhea; no lead line. The chest expansion was greater on the right than on the left. The apex of the heart was in the fifth space, 12 cm. to the left of midsternum, ½ cm. outside the nipple line. The left border of percussion dullness was 13.5 cm. outside the nipple line, the right border 3 cm. to the right of midsternum, the substernal dullness 7 cm.

The sounds were of fair quality. There was an occasional dropped beat and extrasystole with compensatory pause. The aortic second sound was accentuated. At the base the sounds were faint and there was a faint systolic murmur. At the apex there was a rough systolic murmur loudest just inside the nipple line, following a rough first sound. The second sound was reduplicated. The rhythm and sounds changed from minute to minute. The pulses were of fair volume and low tension, with an occasional dropped beat. The artery walls were felt. The lung signs are shown in Fig. 117. The abdomen bulged, and showed tenderness under the left costal margin. The liver dullness extended from the sixth rib to two centimeters above the costal margin. The genitals were negative. The

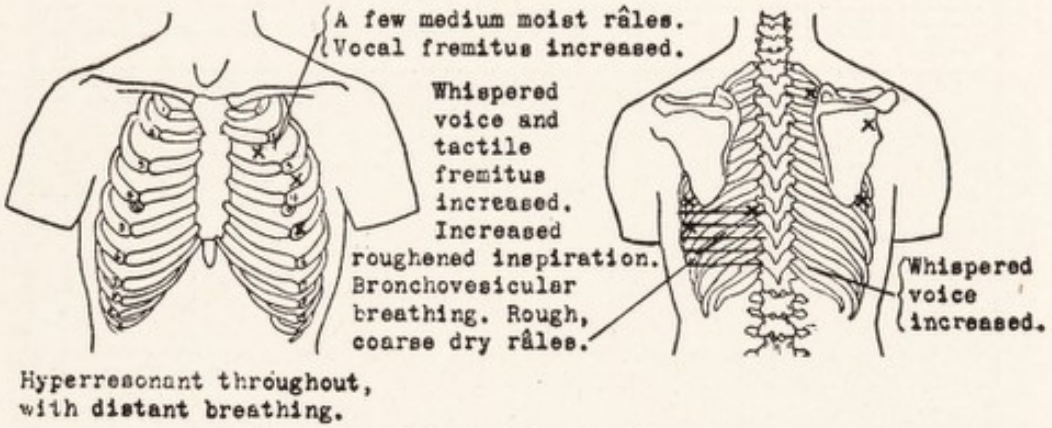



FIG. 117.—Physical signs in Case 3542.

extremities showed no edema. The pupils were slightly irregular, reacted to light and distance through a small arc; the right was greater than the left. The reflexes were slightly exaggerated. The fundi were normal except for arteriosclerosis.

Until November 1 the temperature was usually 98° to 100°; the later chart is shown (Fig. 118). The pulse was 68 to 140, the respiration 19 to 37. The blood pressure was 200/100 to 105/70. The output of urine was 10 to 72 ounces, usually 15 to 40 ounces, the specific gravity 1.006 to 1.012 at seventeen tests. There were small amounts of albumin at all, granular casts at 12, hyalin at 9, fatty at 3, cellular at one, leucocytes at 3. The renal function at three tests was 10%, at one test 15%. The hemoglobin was 65 to 45%. The leucocytes were 10,100 to 24,600, the polynuclears 82 to 66 to 87 %, the reds 4,600,000 to 1,840,000, with slight achromia and variation in size, very slight poikilocytosis at two of six examinations, slight polychromatophilia. The non-protein nitrogen was 66 mgm. per 100 gm. of blood. A Wassermann was negative. The stools

gave a negative guaiac at three tests. A Mallein skin test was negative. In an X-ray of the kidneys the outline was fairly well seen. There was no evidence of stones. Plates of the teeth showed multiple tooth roots and pus pockets. The sinuses were negative.

October 16 the knees and great toe joints were stiff and painful, the left knee hot, tender, and slightly fluctuant. Next day there was a loud roughened systolic at the apex, faintly heard in the axilla and at the base, and the aortic second sound was more accentuated. The left knee was more painful October 22. The heart action was regular, the second sound accentuated, the murmur heard in the neck.

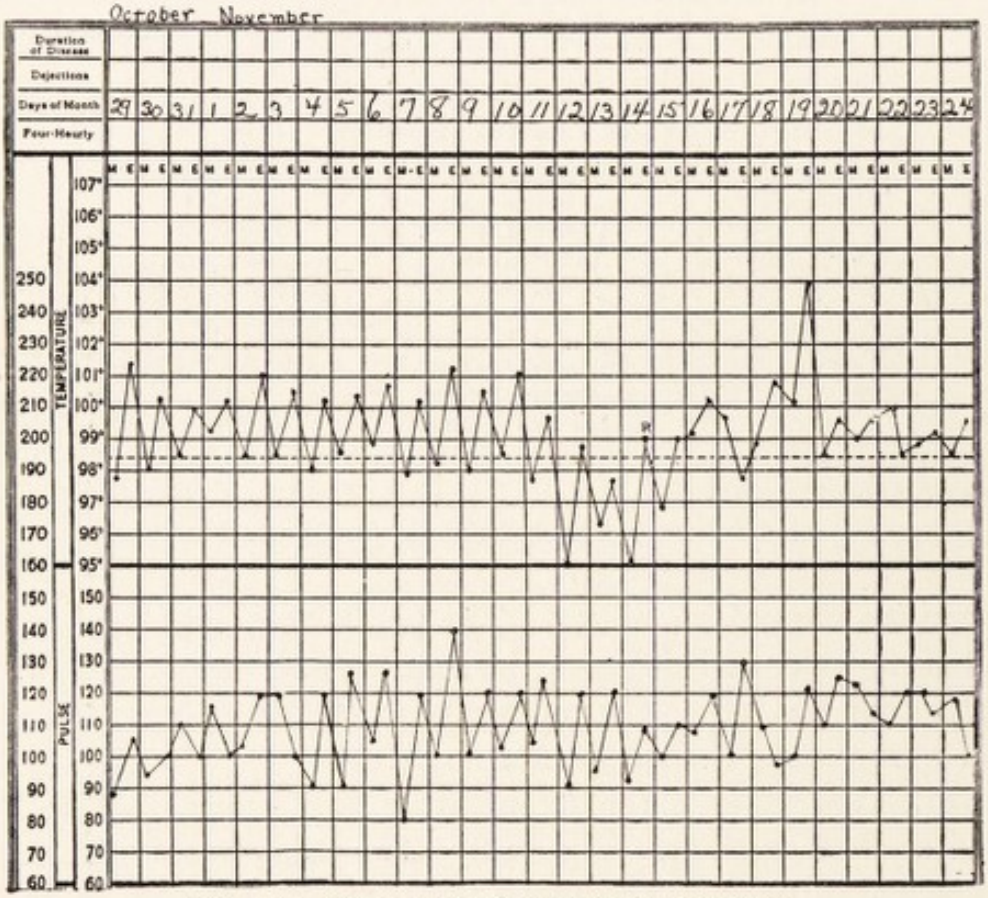



FIG. 118.—Temperature and pulse in Case 3542.

The apex impulse was 9.5 cm. from midsternum, 1.5 cm. inside the nipple line, the right border 4.5 cm. to the right, the supracardiac dullness 6 cm. October 25 the knees were better but the ankles were stiff and painful. A throat consultant found no evidence of a focus of infection in the tonsils or sinuses but was in doubt about the teeth. October 27 several teeth were extracted for drainage of pus pockets. Two days later the joint condition had subsided and the right cardiac border had come in to 3.5.

November 2 the temperature began to run from 100° to 103.4° and the pulse from 80 to 125 with no discoverable cause. The joints were not troubling. The murmurs and the aortic second sound were as at entrance. The urinary output was up and down. By December 2 the temperature was down, the appetite better. Decem-

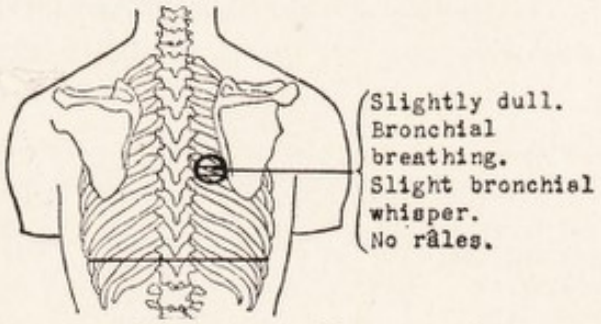



FIG. 119.—Physical signs Dec. 15 in Case 3542.

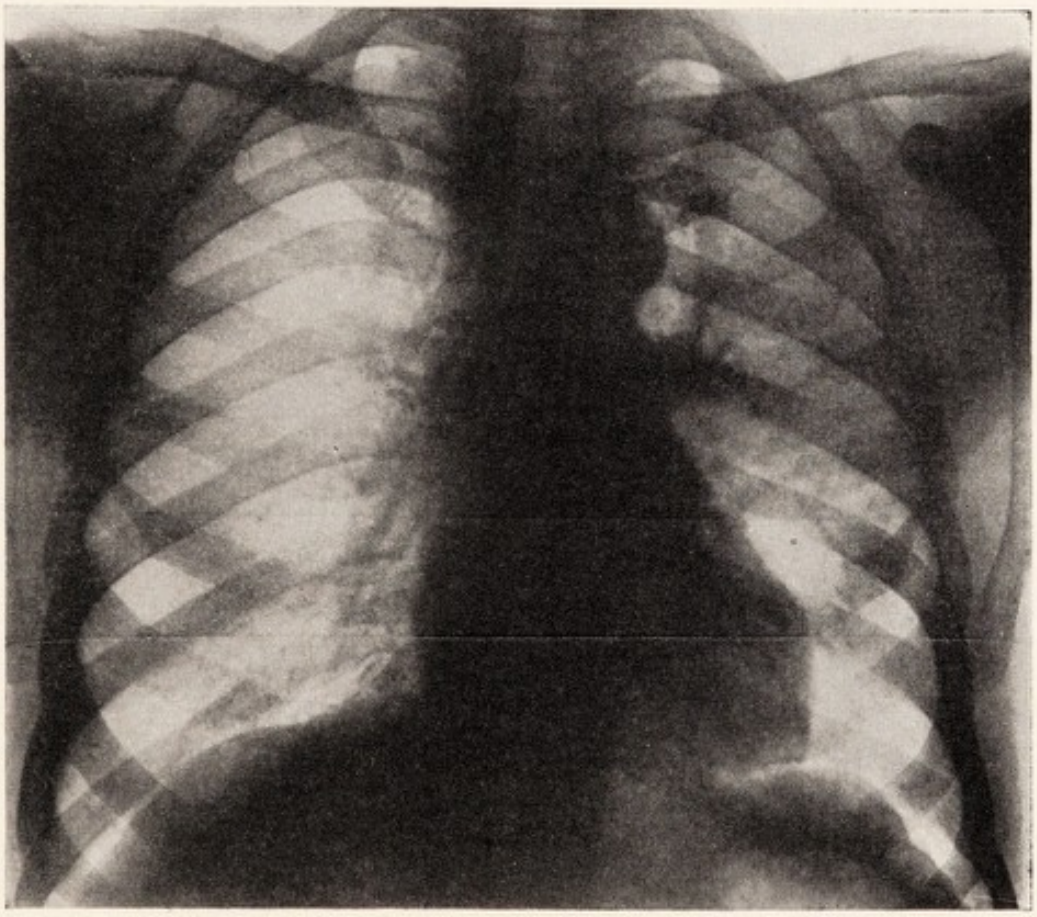


FIG. 120.—Heart shadow normal. Prominent aortic knob. Mottled opacity, left lung, from the apex of the fourth rib.

ber 13 he was coughing less. December 15 the lung signs were as in Fig. 119. The breath was very foul, especially at night, and the patient was spitting up about an ounce of mucopurulent material daily. X-ray of the left chest (see Fig. 120) showed a rather mottled

opacity extending from the apex to the fourth rib in front. The heart shadow was not displaced. The left chest expanded less than the right. The right lung appeared normal. Electrocardiogram showed normal rhythm.

December 24 the left upper back was dull, with increased breathing and whisper. December 28 the lung signs were as shown in Fig. 121. January 3 he was coughing more, with still increasing amounts

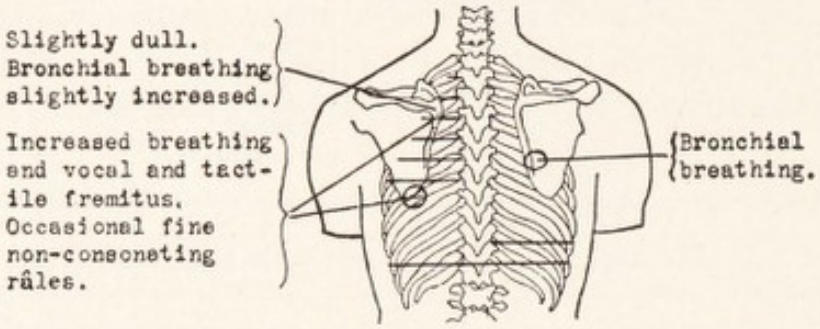



FIG. 121.—Physical signs Dec. 28.

of sputum. January 6 the lung signs were as in Fig. 122. He grew weaker. The signs became most marked in the left upper back. January 24 he died.

*Interpretation of X-ray, December* 16.—Pneumonic process in the left upper lobe.

Abscess.

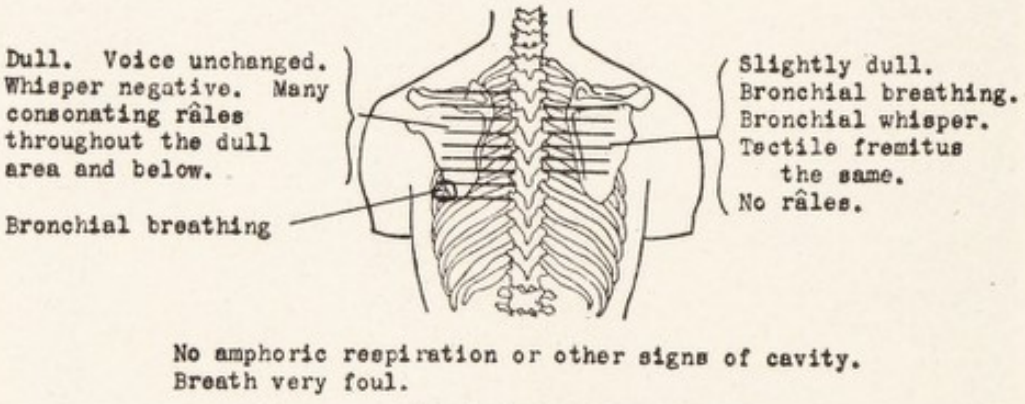



FIG. 122.—Physical signs Jan. 6.

*Clinical Diagnosis (from Hospital Record).*—Chronic nephritis.
Lung abscess.
Arteriosclerosis.
Secondary anemia.
Bronchopneumonia.
Extraction of teeth.

*Dr. Richard C. Cabot's Diagnosis.*—Arteriosclerosis.
Arteriosclerotic kidney.

Hypertrophy and dilatation of the heart.
Abscesses of the left lung.
Terminal pneumonia of the right lung?
Secondary anemia.
Acute endocarditis?
*Anatomical Diagnosis.*—Arteriosclerosis.
Arteriosclerotic nephritis.
Hypernephroma of the kidney.
Acute endocarditis of the aortic valve.
Chronic endocarditis of the aortic valve.
Hypertrophy and dilatation of the heart.
Abscess of the left lung.
Infarct of the spleen.
Anemia.
Chronic pleuritis.

The heart weighed 282 gm. The organ was slightly enlarged. The right ventricle wall measured 3 mm., the left ventricle wall 12 mm. The cavities on the right were moderately increased, on the left slightly increased. The mitral valve measured 10.5 cm., the aortic in the region of the aortic ring 6.5 cm., with 4.5 cm. for the circumference of the cusp margins. The tricuspid measured 13 cm., the pulmonary 7 cm. The mitral and tricuspid valves showed a slight amount of fibrosis. The pulmonary valve was negative. In the region of the junction of the anterior and the posterior cusps of the aortic valve there was a small irregular fibrocalcareous mass of material about 1 cm. in greatest dimension. Adherent and erected upon this was a cauliflower-like mass 1.5 by 1 cm. by 6 mm. consisting of pale grayish granular rather friable material mottled with spots and flecks of adhering blood-like material. The mass rested upon the ventricular aspect of the cusp. On the right posterior cusp, ventricular aspect, and in the region of the corpus Aurantii there was a group of small, rather flat vegetations, otherwise similar to the vegetations already described. The cusps elsewhere showed a slight amount of fibrosis. In the endocardium beneath the cusps there were a few small fibrous areas.

**Necropsy 4522**

An American student of twenty-one came to the Emergency Ward May 3, 1923. At twelve he had rheumatic fever. Since that time he had been active except that he had not taken part in strenuous games.

April 21 he came down with influenza. After three days in bed he returned to school. April 28 he was obliged to go to bed again, and had remained there, with a fluctuating temperature up to 102°. He was feeling better until four o'clock the day of admission, when he was seized with a sudden left-sided hemiplegia with spasticity. At admission he was unable to talk.

Examination showed a well nourished, semistuporous boy with left-sided paralysis, the left arm and the fingers of the left hand rigidly flexed and the left leg fixed in one position. He restlessly moved his right arm and leg. He was incontinent of urine. He made a feeble attempt to put out his tongue and open his mouth; it was not possible to determine whether there was paralysis here or not. The cranial nerves were not tested. There was slight tremor of the tongue on extension. The neck was possibly slightly stiff. There was no neck sign. The heart was slightly enlarged to the left. The percussion measurements were not recorded. The point of maximum intensity of the beat was outside the nipple line. There were thrill and systolic and diastolic murmurs at the base. The blood pressure was 130/68. The lungs, abdomen, genitals and pupils were normal. The reflexes were increased on both sides, more on the left. There was clonus of the left ankle. Babinski and Kernig were positive. The knee-jerks were active, especially the left.

The temperature was 100°, the pulse 91 to 108, the respiration 20 to 36. The amount and specific gravity of the urine were not recorded. There was the very slightest possible trace of albumin at the single examination, many hyalin and cellular casts, thirty to forty red blood corpuscles and eight to ten leucocytes per high power field. The hemoglobin was 80%, the leucocytes 17,000, the polynuclears 83%, the reds and platelets normal. The Wassermann is not recorded. A pharyngeal culture showed pneumococci and streptococci. Lumbar puncture gave 10 c.c. of slightly cloudy pearly whitish fluid. The specific gravity is not recorded. The initial pressure was 170, the pressure after withdrawal of 5 c.c. 140, after withdrawal of 5 c.c. more 120. The pulse, respiration and jugular compression were normal. There were 860 cells, 70% polynuclears, 30% lymphocytes; no organisms. A Wassermann was negative, alcohol a trace above normal, goldsol 0011100000, total protein 68, sugar .714%. Neurological examination showed bilateral spasticity, more on the right, a very spastic flexed left arm, some spasticity of the right, with some voluntary motion. The knee-jerks were very greatly increased, the right more than the left. There was

bilateral Oppenheim and Gordon. The fundi showed clear discs, the margins normal, the vessels full; not so well seen on the left. An eye consultant reported early choked discs, both eyes, the left more than the right.

The morning of May 4 the patient was more stuporous, the breathing was Cheyne-Stokes in type, and the reflexes were more active than the night before and slightly more active on the right side than at the previous examination. He was not able to respond at all to questions, and made no attempt to comply with commands to put out his tongue, etc. The cardiac condition, temperature and blood pressure remained unchanged. It was hard to be certain whether Kernig was present or whether the limitation of motion was due to spasm. Both sides reacted in about the same way, but the left Kernig seemed slightly more pronounced than the right.

In the afternoon he was still more stuporous and there were many loose moist coarse tracheal râles. He made no attempt to move. The neck was still stiff. The pupils reacted sluggishly, and there were slight slow lateral movements. That evening he quietly ceased breathing. The heart continued to beat for five minutes after the respirations stopped. At first the beat was regular, then coupled, then in triads.

*Clinical Diagnosis (from Hospital Record).*—Rheumatic heart disease (mitral stenosis).

Left hemiplegia (embolus).

Meningitis.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis, aortic (and mitral?); aortic stenosis and regurgitation.

Acute endocarditis, aortic.

Hypertrophy and dilatation of the heart.

Cerebral embolism and abscess.

Acute meningitis.

*Anatomical Diagnosis.*—Chronic and acute endocarditis of the mitral valve.

Embolic occlusion of middle cerebral arteries with large areas of infarction of the brain.

Hypertrophy and dilatation of heart.

Slight chronic passive congestion.

Infarcts of spleen.

Soft hyperplastic spleen.

DR. RICHARDSON: There was much pale froth running from the nostrils. Opening the skull we found considerable edema of the pia

but no definite meningitis. The left cerebral artery was solidly plugged, and the anterior half of the brain, especially on the right side, showed marked softening and disintegration. The sinuses and middle ears were negative, the pituitary and pineal glands negative. In other words there was an embolus plugging the middle cerebral artery, with softening of the brain. It is fair to say that the specimen was kept intact because it is not so very usual to find such a typical picture of embolus plugging the middle cerebral. It is possible that the original embolus was infected, coming from the vegetations on the valve. But there was no definite meningitis, and if there was any infection present it was probably in the disintegrated tissue.

There was nothing in the peritoneal cavity. An appendix was recorded only two cm. in length. That is very small, but otherwise there was nothing the matter with it.

The gastro-intestinal tract showed streaks and areas of reddening of the mucosa,—beginning chronic passive congestion, not very extensive. The diaphragm on the right was at the fourth rib, on the left at the fifth rib.

The pleural cavities showed no fluid and no adhesions. The trachea and bronchi contained much reddish frothy fluid. The lung tissue was leathery, brownish-red, yielding considerable frothy fluid,—beginning chronic passive congestion, not very marked as yet except for considerable edema. There was no hydrothorax, no hydropericardium, but the fluid was beginning to infiltrate into the lungs.

The heart weighed 329 grams, not much enlarged. The myocardium was good, the right ventricular wall four mm., a little thick, the left twelve mm., about what it should be. On the left the cavities showed slight dilatation. That goes very well with the picture we have already mentioned. The mitral valve was seven cm. in circumference,—a little decrease. In places it showed fibrous thickening, and for a length of about three cm. there was a thick fibrous base on which was erected a mass of thrombotic material, a little firm at the base, but as we came to the surface soft spongy acute endocarditis. From that of course the bit was washed off that went up into the brain.

In the spleen, which was definitely enlarged at one pole, a definite infarct. The kidneys were free from infarcts.

The aortic valve was six cm. in circumference, the tricuspid twelve, the pulmonary seven, and those valves were frankly negative.

The intima of the aorta and great branches was smooth, showed no evidence of sclerosis. The pulmonary artery, veins, and vena cava were negative.

The liver showed beginning chronic passive congestion. The gall-bladder and bile-ducts were negative. The kidneys weighed 245 grams, and macroscopically and microscopically showed nothing except a little bit of congestion. The pelves, ureters, bladder, prostate, seminal vesicles, and testes were negative.

Culture from the blood at the time of necropsy gave no growth. But in these cases of infection with an organism of the *viridans* group, as this one is, it is not infrequent to get negative cultures.

DR. CABOT: Is there any way in which that soft area of the brain could have communicated with the spinal canal so that the lumbar puncture findings might be explained?

DR. RICHARDSON: It was pretty well disintegrated. The whole artery was shut off.

DR. CABOT: We want to account, if we can, for this very queer lumbar fluid.

DR. RICHARDSON: That is the only way,—i.e., by the softening. But there was no definite abscess so far as we dissected, and it would be just the same as sometimes happens in an infarct of the spleen, where I have in some cases recovered the organism from the infarct and not from the blood. That of course immediately points the finger at me. It would have been a good idea if we had taken a culture from the infarcted area of the brain. It is a possibility.

DR. CABOT: It seems possible for us if we want to be obstinate to say that this was an abscess of the brain which communicated with the spinal cord and so gave us this fluid as we supposed. We have no culture. I do not feel at all sure that it is so. But in the absence of culture it is very hard to say that it is not. Unless we say something of that kind it is very hard to account for the spinal fluid.

DR. RICHARDSON: Did they describe the macroscopic appearance of that fluid?

DR. CABOT: It was "pearly white,"—that is, not clear.

DR. RICHARDSON: I should not expect to find many leucocytes in a pearly fluid.

DR. FOSTER: A very interesting thing about this case is that the only history of any possible heart incapacity was at school. The doctor there had told him he should not take part in any strenuous athletics. At the beginning of his illness a local doctor examined his

heart and said there were no murmurs. He was here at the necropsy and was about ready to give up medicine. I was wondering whether or not this murmur could have developed as rapidly as that.

DR. CABOT: Of course the murmurs of mitral stenosis in a well-compensated case are just the kind that most physicians of this country do not hear. A presystolic roll with very little else most people miss. But if there was as little as you got it might depend perfectly well on the acute endocarditis.

A PHYSICIAN: In a leucocytosis of the blood ordinarily you would not get such a spinal fluid?

DR. CABOT: Nothing like that at all.

### Necropsy 2439

An Irish stone cutter of fifty-eight entered September 2 for relief of difficulty in urination of two or three years' duration. He had always had the best of health though he had lost considerable weight, he did not know how much. He denied venereal disease. Five days before admission he had complete retention. A physician catheterized him and left the catheter in for two or three days. Since the morning before admission he had passed some urine with difficulty. He voided last four hours before admission. Since the onset of the acute retention he had had continued pain in the bladder.

Examination showed a man looking sick and old and showing evidence of the loss of considerable weight. His color was fair, his mucous membranes rather cyanotic. The location of the apex impulse of the heart is not recorded. There was no enlargement to percussion. The radials were slightly hard. There was moderate lateral excursion of the brachials. The action was regular, the sounds faint but of fair quality. There were no murmurs. The prostate was slightly nodular but not enlarged or tender. The examination was otherwise negative. The temperature and pulse are shown in Fig. 123. The respiration was normal until September 7, afterwards 24 to 30 with a rise to 40 on the last two days. The urine was normal in amount, cloudy, alkaline at one of two examinations, the specific gravity 1.006–1.018, a slight trace of albumin at the second examination. The blood is not recorded.

The patient was put immediately upon constant drainage. There was a residual of four ounces of slightly turbid urine. He complained of considerable pain in the perineum. The catheter was removed and the perineum found to be fluctuant and tender about the bladder. Pressure expressed pus from the meatus. At operation September

5 a sound met obstruction in the deep urethra. Incision was made in the perineum with the evacuation of two ounces of thick cloudy urine. The urethral bulb was surrounded by slough. A rubber catheter was passed through the perineum to the bladder. The patient did not gain as he should have done after the operation. The urine was kept up by rectal salt solution. September 13 he was worse and markedly jaundiced. There was bile in the urine and in the stools. The amount of urine was greatly diminished.

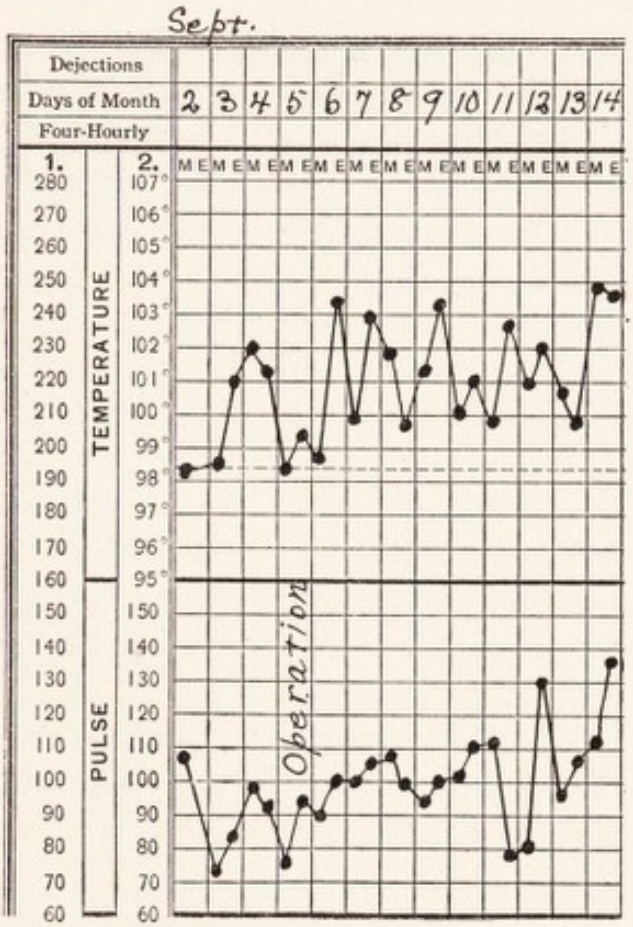



FIG. 123.—Chart in Case 2439.

During the last few days of his life a tremendous systolic blow was heard principally over the apex beat. September 14 he died.

*Clinical Diagnosis.*—Perineal abscess.

Malignant endocarditis.

*Dr. Edward L. Young's Diagnosis.*—Obstructing prostate.

Periurethral abscess.

Septicemia.

Acute endocarditis.

*Anatomical Diagnosis.*—Malignant endocarditis of the mitral valve.

Septicemia, streptococcus.

Infarcts of the spleen and kidneys.

Icterus.

Operation wound.

DR. RICHARDSON: The incision in this case was a short one in the anterior abdominal wall. We could not remove any of the organs. Icterus was well marked. There was no evidence of peritonitis, no stones in the gall-bladder. The hepatic, cystic and common bile-ducts were free, the mucosa negative, the bile flowed freely. The pancreas was not remarkable, the ducts of Wirsung free. There was no evidence of new-growth in any of the organs seen, no ulcers in the lower end of the ileum. There was a soft, hyperplastic spleen with infarcts. The kidneys were of normal contour and size and showed infarcts. The heart was somewhat enlarged and the mitral valve presented a frank mass of vegetations, three by two cm. The lungs were negative as nearly as could be made out.

The growth was streptococcus from the spleen. The vegetations on the mitral valve and endocardium showed the usual hyalin material, blood plates and masses of micrococci.

DR. YOUNG: Did you open the bladder?

DR. RICHARDSON: No.

DR. CABOT: As you hear the history, Dr. Richardson, would your guess be that this endocarditis originated in the conditions of the bladder and the instrumentation? He had had no heart symptoms, had he?

DR. YOUNG: There were none according to the record. They report the location of the apex impulse not recorded, no enlargement to percussion, the action regular, the sounds faint but of fair quality, no murmurs till near death.

DR. RICHARDSON: When was the first rise in temperature?

DR. YOUNG: He came in with enough sepsis so that he had a temperature the day after he came in. The first rise was right after the operation. I believe he came in with a septicemia.

DR. RICHARDSON: But it shoots up on the day after operation. I think that is fair enough, but there is also the other side, that he might have had the septic condition mentioned and that following the operation it extended and set up the endocarditis.

### Necropsy 3336

A man of thirty-eight entered the hospital March 30. His past history was negative except for recurring attacks of left lower quadrant pain with alternating diarrhea and constipation. For a

year he had been subject to great mental and physical strain. He used alcohol and tobacco in moderation.

A week before admission he had an attack of tonsillitis. On the fourth day a peritonsillar abscess was opened with marked relief. The next day his throat was much better, but he was chilly and feverish.

Examination showed a well nourished man, bright, cheerful, free from pain, but a little delirious at night. The throat showed

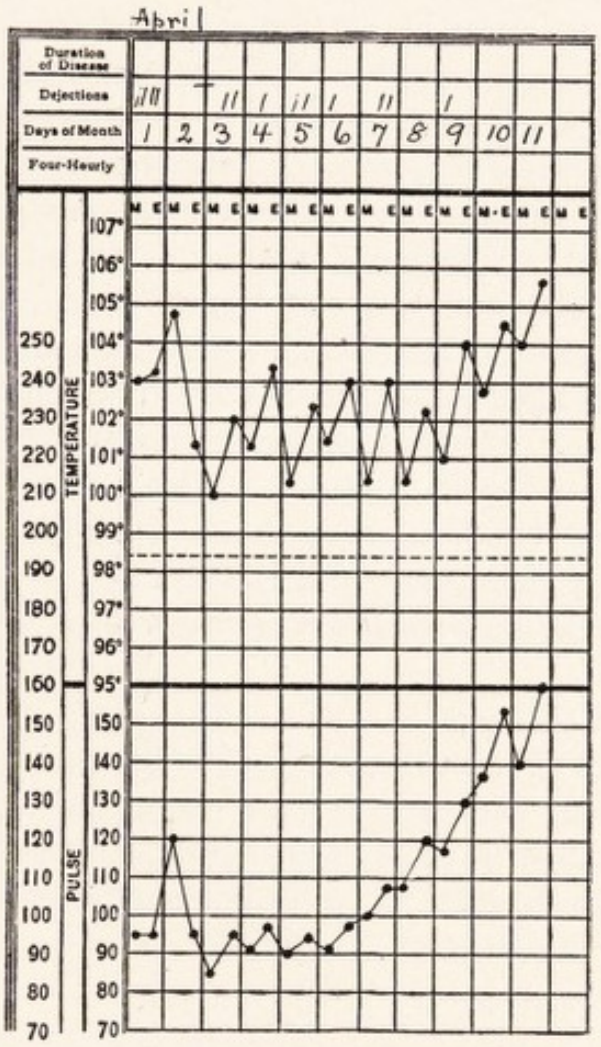



FIG. 124.—Temperature and pulse in Case 3336.

marked injection and swelling, especially on the right, where there was a small whitish scar (incision for peritonsillar abscess). There were several submucous hemorrhages in this area. Examination was otherwise negative.

The temperature and pulse are shown in Fig. 124. The respiration was 25 to 45. The blood pressure is not recorded. The output of urine on the day of entrance was 165 ounces, after that 50 to 85

ounces. The specific gravity was 1.010 to 1.017. There was the slightest possible trace to a very slight trace of albumin, an occasional red cell and a rare granular cast. There were 14,200 leucocytes, 90% polynuclears. The reds were normal. *A blood culture showed very large numbers of streptococci.*

April 2 the patient was still delirious, with a temperature of 104°. A painless red spot the size of a half-dollar appeared on the right wrist. The next day this was painful. April 5 the patient seemed better, slept well with opiates, and was delirious only occasionally. The right wrist also was better, but a similar spot appeared on the left calf.

April 6 both these spots were better, but the left wrist and eye were now red and painful, and the patient more uncomfortable and delirious. Next day the eye was worse, and was pronounced by an eye consultant to be a case of embolism of the retinal artery. The throat, the wrists, and the calf were better. Nevertheless the patient seemed to be losing ground. A systolic murmur at the apex was now noted as having grown more marked since being first observed three days before. The pulse was going up, though of good quality. Petechiae appeared over the back. With no new symptoms, but constantly rising temperature, pulse and respiration, the patient grew increasingly toxic, and April 12 died.

*Clinical Diagnosis (from Hospital Record).*—Pyemia.

Endocarditis.

Peritonsillar abscess.

*Dr. Richard C. Cabot's Diagnosis.*—Streptococcus septicemia.

Probably acute endocarditis, (left side of the heart).

Probably emboli in the kidney, perhaps in the spleen and liver, possibly in the lungs, retina and subcutaneous tissues.

Probably acute glomerulo-nephritis.

*Anatomical Diagnosis.*—1. Chemical or physical origin of fatal illness.

Septicemia, streptococcus.

| | |
|---|---|
| | Acute vegetative endocarditis of the mitral valve. |
| | Hypertrophy and dilatation of the heart. (Weight 473 grams.) |
| | Infarcts of the spleen and kidneys. |
| 2. Secondary or terminal lesions. | Fibrinous pleuritis, double. |
| | Soft spleen. |
| | Central degeneration and foci of necrosis of liver. |
| | Suppurative nephritis. |
| | Purpura. |
| | Focal hemorrhage in wall of small intestine |

The kidneys weighed 512 grams. Wide cortex. There were many purplish spots, and in the right kidney three yellow infarcts; both these lesions embolic.

The cardiac hypertrophy is not explained. The patient was athletic and took much exercise; but most athletes have no cardiac hypertrophy.

## CHAPTER VIII

## CHRONIC NON-DEFORMING VALVULAR SCLEROSIS OR ENDOCARDITIS

After excluding cases characterized by soft or acute vegetations on the valves, there remains a group of cases usually labelled by the pathologist as "chronic endocarditis" on the basis of fibrous thickening of the valves, to which may be added stiffening and fibrocalcareous changes. From the clinical point of view the important thing about these lesions is to *distinguish those which deform the valves and so interfere with their function, from those which do not.* This we have attempted to do by the careful study of the records in all cases to which our memory of the post-mortem examination did not extend. After excluding the cases with definitely deformed valves believed to cause stenosis or regurgitation, there are left in our 4000 necropsies 237 cases in which the valves, despite some endocarditis, remain flexible and apparently quite capable of opening and shutting in the normal way. There is apparently little if any interference with their function. These cases of non-deforming sclerosis or fibrosis of the valves we have further subdivided on the basis of the pathologist's judgment, into those which represent (a) "the usual sclerosis of age," and (b) those in which the changes seem greater in degree or different in kind from anything that can be accounted for in this way.

In our series Group a represents the condition of the valves in almost every elderly patient, quite independent of the cause of his death, and recognizable only by careful comparison with the heart valves of younger persons. Of these we have made no special enumeration or further study, believing that there is no considerable chance of their being of any clinical importance. After setting these on one side there remain as already said 237 cases which are not to be included under Group a but yet which do not deform the valves.

Further study of these 237 cases brings out the following facts as to

## ETIOLOGY

There appear to be two causal factors. One of these factors acts especially on younger people, up to the thirtieth year, and acts on both sexes alike. In this group there were twenty-five males and seventeen females. But as this is almost exactly the relation (66 to 34) of male and female in the total group of 4000 necropsies in which these fall, we believe that the figures do not represent any excess of males. In this group there is very little cardiac enlargement or arteriosclerosis, very little chronic passive congestion, and fewer murmurs than in the other group.

The other factor is one which acts especially on persons over the thirtieth year and which acts twice as often in males as in females. There is certainly reason to suspect that this factor is arteriosclerosis. At any rate it can be called an *old man's factor*. This latter factor (arteriosclerosis?) usually *affects both the mitral and the aortic valves*. It was present in 73 males to 31 females, and especially in the decades between the sixtieth and eightieth years.

On the other hand the cases in which the mitral valve alone is affected are about equally males (36) and females (31), figures which when properly weighted in relation to the distribution of males and females in our necropsy statistics, as given above, seem to show that females are somewhat more affected by this predominantly "mitral factor," which we may surmise to be rheumatism, as we know that the infection associated with this syndrome has a particular tendency for affecting the mitral valve alone and the female sex predominantly.

TABLE 139.—AGE, SEX AND VALVE INCIDENCE IN NON-DEFORMING VALVE THICKENINGS

| | Pure mitral | | Mitralandaortic | | Pure aortic | | Other valves | | Total |
|---|---|---|---|---|---|---|---|---|---|
| | Male | Female | Male | Female | Male | Female | Male | Female | |
| Under 10 yrs. | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 3 |
| 10–19 yrs. | 3 | 2 | 1 | 0 | 1 | 0 | 0 | 0 | 7 |
| 20–29 yrs. | 3 | 5 | 3 | 1 | 0 | 1 | 0 | 0 | 13 |
| 30–39 yrs. | 7 | 4 | 4 | 2 | 1 | 0 | 2 | 0 | 20 |
| 40–49 yrs. | 8 | 11 | 6 | 3 | 6 | 2 | 2 | 1 | 39 |
| 50–59 yrs. | 8 | 5 | 11 | 10 | 14 | 3 | 1 | 1 | 53 |
| 60–69 yrs. | 3 | 2 | 27 | 8 | 6 | 4 | 5 | 2 | 57 |
| 70–79 yrs. | 3 | 1 | 18 | 3 | 4 | 1 | 4 | 0 | 34 |
| 80–80 yrs. | 0 | 0 | 3 | 3 | 1 | 0 | 4 | 0 | 11 |
| | 36 | 31 | 73 | 31 | 33 | 11 | 18 | 4 | 237 |
| Total | 67 | | 104 | | 44 | | 22 | | |

There remains a third group affecting the aortic valve alone. In this there are thirty-three males, eleven females, and only 11 out of the forty-four cases were under the fiftieth year, 8 of these 11 being males and three females. This is presumably an arteriosclerotic group of cases.

The cases with tricuspid involvement were eighteen of them in males, four in females. These last groups are apparently like the second group in respect to age and sex distribution. In other words, when this disease gets beyond the mitral valve it is a lesion of elderly males, i.e., arteriosclerotic (?).

My chief interest in the study of these cases has been not only their possible etiology, just dealt with, but has concerned itself with the following questions:

(1) Do these lesions cause functional impairment of the heart as shown in chronic passive congestion *post-mortem?*

(2) Do these lesions produce cardiac murmurs?

(3) Are they associated with cardiac hypertrophy?

(4) Do they lead to cardiac symptoms and complaints recognized by the patient himself (dyspnea, palpitation, pain, etc.)?

1. Pursuing the first inquiry whether these non-deforming valvular lesions weaken the heart's action so as to produce passive congestion, we find that at necropsy there was present chronic passive congestion in:

15 out of 44 cases affecting the aortic valve alone.
13 out of 67 cases affecting the mitral valve alone.
33 out of 104 cases affecting the mitral and aortic valves.
6 out of 22 cases affecting the mitral aortic and tricuspid valves.

Total, 67 out of 237 or 28%.

What can we conclude from these facts? Since only 28% showed any passive congestion, we cannot say that these lesions usually cause passive congestion, for a "cause" must act in more than 28% of the cases in which it is present. But now analyzing further these sixty-seven cases in which chronic passive congestion was present, we find the following factors, other than the valve lesions, to account for it: (See table 140.)

Since most of these complicating lesions are in themselves well recognized causes for chronic passive congestion without the concurrence of any valve lesions, I conclude that there is no reason to believe that these non-deforming valvular lesions so interfere with the heart's action as to produce chronic passive congestion.

TABLE 140.—ASSOCIATED LESIONS IN THE 67 CASES OF NON-DEFORMING VALVE SCLEROSIS, WITH STASIS AT NECROPSY

| | |
|---|---|
| Arteriosclerosis | 29 cases |
| Chronic nephritis | 11 cases |
| Chronic pericarditis | 8 cases |
| Fibrous myocarditis | 6 cases |
| Syphilitic aortitis | 4 cases |
| Acute endocarditis | 3 cases |
| Acute pericarditis | 7 cases |
| Acute nephritis | 4 cases |
| Hypertrophy and dilatation without "cause" | 6 cases |
| Pernicious anemia | 1 case |
| Suppurative myocarditis | 1 case |
| | 80 cases |
| I.e., multiple factors in 13 cases | 13 |
| | 67 |

2. *Did These Lesions Produce Cardiac Murmurs?*—Part of the answer to this question can be given at once in statistical form. Only 79 out of 237 cases, or 33%, produced any murmurs that were recognized in life. These murmurs were systolic in 78 cases and diastolic in 19; that is to say, all but one of the diastolic murmurs accompanied systolic murmurs. One or both of these murmurs were heard in:

| | |
|---|---|
| 21 out of | 67 mitral cases. |
| 18 out of | 44 aortic cases. |
| 31 out of | 104 mitral and aortic cases. |
| 9 out of | 22 mitral, aortic and tricuspid cases. |
| 79 | 237 |

Obviously then, as with the question of chronic passive congestion in its relation to these lesions, so with the murmurs, we may say that a lesion associated with murmurs in only 33% shows but a feeble correlation and can hardly be said to be a cause of importance.

But now if we study further the 79 cases associated with murmurs we find present the following conditions, all of them by themselves often associated with murmurs and ordinarily believed to have a causal relation to them. (See table 141.)

This table shows that in the twenty-one "pure mitral" cases with murmurs there was no arteriosclerosis at all in the vessels post-mortem. How about the "pure mitral" cases *without* murmurs? Six of the forty-seven cases without murmurs had arteriosclerosis.

TABLE 141.—VALVES AFFECTED

| | Mitral and aortic | Mitral | Aortic | Mitral, aortic and tricuspid |
|---|---|---|---|---|
| Arteriosclerosis with hypertrophy and dilatation | 24 | 0 | 11 | 7 |
| Chronic nephritis | 6 | 1 | 4 | 0 |
| Hypertrophy and dilatation without known cause | 6 | 5 | 3 | 2 |
| Chronic pericarditis | 3 | 4 | 1 | 0 |
| Acute pericarditis | 2 | 1 | 0 | 1 |
| Myocarditis | 1 | 1 | 3 | 2 |
| Aneurism | 1 | 0 | 3 | 0 |
| Acute nephritis | 0 | 2 | 1 | 0 |
| Unknown or doubtful | 0 | 7 | 2 | 0 |
| Acute endocarditis | 0 | 0 | 1 | 0 |
| Pernicious anemia | 0 | 0 | 0 | 2 |

In other words, arteriosclerosis was a very small factor in these "pure mitral" cases. (The six cases just referred to occurred at the following ages: 78, 60, 56, 55, 54, 31.) This tends to show (what is usually recognized) that arteriosclerosis has very little to do with the lesions of the mitral valve.

Combining by causes the figures of the last table we see that arteriosclerosis with enlarged heart was present in forty-two of the cases with murmurs. Enlarged heart without arteriosclerosis, nephritis or any other known "cause" was the only tangible lesion in sixteen cases. Chronic nephritis (with enlarged heart), chronic adhesive pericarditis, fibrous myocarditis, are also common lesions in the cases showing murmurs. There were but nine cases out of 79 in which no gross lesion of any kind could be found to account for these murmurs, not even cardiac enlargement, which the following table shows to be an outstanding fact in the whole series except in the pure mitral cases which evidently form a group by themselves.

3. *Is Cardiac Hypertrophy Associated with Cases of Non-deforming Endocarditis?* (See table 143.)

Obviously these figures need not mean that the often-associated hypertrophy was *due* to the slight thickening of the valves. For first of all the hypertrophy was present in but two-thirds of the cases (160 out of 237) and in the pure mitral cases only in 28 of 67, or two-fifths. Moreover there were present in the 160 hypertrophied cases the following traditional "causes" for such a condition.

TABLE 142.—ASSOCIATED LESIONS IN 160 CASES OF NON-DEFORMING ENDOCARDITIS WITH CARDIAC HYPERTROPHY

| | |
|---|---|
| Arteriosclerosis | 72 cases |
| Chronic nephritis | 24 cases |
| Chronic pericarditis | 11 cases |
| Fibrous myocarditis | 9 cases |
| Acute pericarditis | 10 cases |
| Syphilitic aortitis | 7 cases |
| Acute nephritis | 2 cases |
| | 135 |
| Counted more than once | 15 |
| | 120 |
| No cause | 40 |
| | 160 |

No "cause" for hypertrophy (unless the non-deforming endocarditis is so considered) was found in forty of the 160 hypertrophied cases. If mere correlation is to be considered "causal," these forty cases could be held to indicate that non-deforming endocarditis leads once in four times to cardiac hypertrophy, though not to chronic passive congestion and not to murmurs. But there is no need of any

TABLE 143.—CARDIAC HYPERTROPHY AND DILATATION IN NON-DEFORMING VALVULAR SCLEROSIS

| Mitral and aortic | Mitral | Aortic | Mitral, aortic and tricuspid | Total |
|---|---|---|---|---|
| 81 of 104 | 28 of 67 | 33 of 44 | 18 of 22 | 160 of 237 |

such conclusion. For it is well recognized that anatomical "causes" for cardiac hypertrophy are not always to be found; 154 of 1209 in our series were without any recognized "cause." Many cases of such enlargement exist not only without any of the "causes" listed in the last table but without any valve lesion, deforming or non-deforming. Hence it seems to me probable that the correlation of hypertrophy and non-deforming valve lesions in these forty cases is a coincidence. They may have been cases of hypertension without known cause.

4. *Does Non-deforming Endocarditis Produce in Life "Cardiac Complaints" (Dyspnea, Palpitation, Edema, Etc.)?*—In only fifty-one of our 237 cases, or 21% were any such cardiac complaints recorded:

TABLE 144

| VALVE AFFECTED | SYMPTOMS IN | |
|---|---|---|
| Pure mitral | 8 cases out of | 67 or 1/9− |
| Pure aortic | 10 cases out of | 44 or 1/4+ |
| Mitral and aortic | 25 cases out of | 104 or 1/4− |
| Mitral, aortic and tricuspid | 8 cases out of | 22 or 1/3+ |
| | 51 | 237 |

In these fifty-one cases other well recognized causes for cardiac complaints were found (arteriosclerosis with hypertrophied and dilated heart 17 cases, chronic nephritis 10 cases, hypertrophied and dilated heart without known cause 6 cases, fibrous myocarditis 5 cases, chronic pericarditis 4 cases, acute pericarditis 4 cases, aneurism 2 cases, stomach cancer with anemia 1 case, pernicious anemia 1 case, acute endocarditis 1 case).

In the mitral group in which other factors such as arteriosclerosis, nephritis, myocarditis etc. are at the minimum, in which therefore we most often find the non-deforming endocarditis *standing out as the only lesion in the heart*, we find fewer cardiac complaints, fewer murmurs, less chronic passive congestion and less cardiac hypertrophy than in the other groups. These four evidences of disturbed heart function fall off rapidly as we get nearer to isolating the non-deforming endocarditis from confusing accompaniments.

## CONCLUSIONS

1. It seems to me from these considerations that chronic non-deforming endocarditis (or sclerosis) of the heart valves:

(a) Does not cause cardiac complaints (or symptoms).

(b) Does not cause chronic passive congestion.

(c) Does not cause cardiac murmurs.

(d) Does not cause cardiac hypertrophy.

And so it may be said to have no clinical significance.

2. Pathologically it is a result, I believe, either of a slight attack of rheumatic endocarditis (especially when it occurs in young people on the mitral alone) or (more often) of the general causes of bodily wear and tear which are associated with advancing age, arteriosclerosis and the male sex. In the latter group of cases it usually affects two or more valves. In both groups it is functionally and practically insignificant.

## CHAPTER IX

## ACUTE PERICARDITIS

**1. Age and Sex.**—In 186 cases of acute pericarditis the outstanding and altogether mysterious fact is the very decided excess of males. More than three-fourths of all (141 out of 186) cases were in that sex. This is all the more surprising because we have been in the habit of thinking of acute pericarditis as frequently, at least, rheumatic in origin, and it is certainly true that women are more prone to

TABLE 145.—AGE IN ACUTE PERICARDITIS

| | |
|---|---|
| 0– 9 | 14 |
| 10–19 | 14 |
| 20–29 | 34 |
| 30–39 | 29 |
| 40–49 | 31 |
| 50–59 | 28 |
| 60–69 | 27 |
| 70–79 | 6 |
| 80–89 | 2 |
| Unknown | 1 |
| Total | 186 |

TABLE 146.—SEX IN ACUTE PERICARDITIS

| | |
|---|---|
| Males | 141 |
| Females | 45 |
| | 186 |

rheumatic infection than men. But so far as our statistics go, the fact seems to be that *rheumatism in the ordinary sense*—that is to say, a generalized infection prone to attack the joints as well as the heart—*is only a minor factor in the production of pericarditis*. If we put on one side the cases in which the pericarditis can be regarded

as a terminal phenomenon and not as the main illness from which the patient suffered, then it will doubtless appear that *in the remaining group of cases* rheumatism is an important factor. That this group is small appears at once probable from the fact that we have a clear rheumatic history in only twenty-four cases out of 186, not much more than 12%.

That pericarditis is primarily a terminal affair is further suggested by the *ages* of the patients in our series: only 62 of 186, or 33%, were under the thirtieth year at the time of death.

**2. Associated Lesions.**—One of the facts which has surprised me most in the study of this series of cases is that there are *so few examples of acute endocarditis occurring in connection with the pericarditis.* We find only sixteen undoubted cases in this series, or 8%. It used to be taught that in acute pericarditis, underlying valvular murmurs are often covered up by the noise of the pericardial friction, and that we must always be on the watch for this association of acute endocarditis and pericarditis. This series seems to prove that this is not the case. There is, on the other hand, a rather small proportion of *chronic* endocarditis associated with acute pericarditis, which serves chiefly to confirm our estimate of the amount of rheumatism in this group of cases. In the history, rheumatism appeared clearly twenty-four times, a figure remarkably close to the number of cases of chronic endocarditis as found *post-mortem*,—twenty-eight cases in 186.

**3. Fever and Leucocytosis.**—Another surprise in the study of these cases has been the absence of fever in 45, or 26% of the 172 cases in which a definite record was made. Many of these patients were the subjects of wasting, debilitating diseases often associated with subnormal temperature. The *terminal* nature of acute pericarditis is further emphasized by this group of facts.

The leucocyte curve corresponds very well with the temperature curve; that is, we have leucocytosis in 115 out of 156 cases in which a definite record is made. The absence of leucocytosis in 41, or 26% may be reasonably held to indicate (like the 26% of afebrile cases) that the patient does not sufficiently react against the invading infection.

**4. Diagnosis.**—In my previous studies of pericarditis I have repeatedly called attention to the fact that our diagnostic resources are very scanty. The reason for this is obvious if we remember that the diagnosis of acute pericarditis rests in the majority of cases on nothing but the presence of an audible or palpable friction rub. When the diagnosis of any disease rests upon but a single sign

or symptom, it is obviously insecure, and particularly so when, as is shown in this series, that sign is very frequently absent in proved cases of the disease. Only in 40, or 21% of 186 cases was any friction heard. In many of these cases—though I cannot say exactly how many—the pericarditis had been suspected owing to the presence of an accompanying pneumonia or endocarditis. Therefore it was searched for and listened for again and again, as in several of these cases I can personally recall.

Only in ten cases was there evidence of pericardial fluid that could be regarded as approximately distinctive. Seven of these cases are included with the forty already mentioned as showing friction. If therefore, we add together the cases in which pericarditis was suggested either by the presence of friction or by signs suggesting pericardial effusion, or by both sets of data, we get a total of forty-three, the figure representing our percentage of diagnostic success, 23%.

*Precordial pain* was even less reliable as a clue to the existence of pericarditis in this series. In only twenty-six cases or 14% was it definitely complained of.

Of particular interest to me is the list of underlying causes of death in this series of cases, as shown on page 642. First of all comes the group of blood infections, septicemia or pyemia, whether associated with a known focus of infection or not. Within this last group comes the particularly interesting type of pericarditis occurring in children with staphylococcus sepsis,—seven cases. All of these had abscesses of the myocardium, so that the pericarditis may reasonably be assumed to have spread through the myocardium.

In addition to these there is a considerable group of cases due to the streptococcus, in which the pericardium is inflamed along with other portions of the body; or, in other words, in which the pericarditis complicates an empyema, an acute endocarditis, a general peritonitis, an abscess of the lung. *After septicemia three diseases stand out in the etiology: pneumonia, chronic nephritis, and cancer.*

*Chronic passive congestion* was found in sixty-seven necropsies out of 186, or 35%. In twenty-four of these it was accounted for by chronic nephritis; in seven by hypertension with arteriosclerosis, in eight by valvular disease, in four by chronic pericarditis, a total of forty-three out of sixty-seven.

**Septic Heart Failure and Dropsy (Chronic Passive Congestion).** Beside these mechanical reasons for general stasis (applicable to about two-thirds of our cases) there may be considered a septic-

infectious group of causes which are not well recognized as causes of poor heart function and so of dropsy. Even lesions like tonsillitis or dental sepsis which seem small, local, and not septicemic, are generally and I think correctly believed to weaken the heart's action. Even after a "cold" we are sometimes surprisingly feeble and dyspneic. But it is, I suppose, the more overwhelming and virulent infections that might produce the most serious effect on the heart,—such infections as show themselves simultaneously in several places. In our sixty-seven cases of acute pericarditis associated post-mortem with evidence of chronic passive congestion, there were sixteen which showed in life only the following septic lesions as possible causes for the circulatory failure:

TABLE 147.—SEPTIC LESIONS ASSOCIATED WITH STASIS AT NECROPSY IN ACUTE PERICARDITIS

| | |
|---|---|
| Acute pericarditis *only* | 4 |
| Pneumonia and empyema | 4 |
| Acute endocarditis | 3 |
| Acute empyema | 2 |
| Acute polyarthritis | 1 |
| Acute pleuritis and peritonitis | 1 |
| Acute chorea and phthisis | 1 |
| | 16 |

**Acute or Chronic Passive Congestion?**—In sixteen of the remaining cases the pathologists recorded after death a *chronic* passive congestion, although in life the entire illness (so far as the patient or his friends knew) had lasted but a short time, e.g. two weeks (No. 921, pneumonia and acute endocarditis), one week (No. 1801, acute pericarditis the only cause of death), three weeks (Nos. 3053, 3242, acute polyarthritis and pericarditis). In the majority of these sixteen cases, however, a chronic lesion such as chronic nephritis, mitral stenosis, mediastinitis, was present before the acute symptoms showed themselves, so that the pathologist's findings are in all probability correct. But in a few acute septic cases like those just quoted, one wonders whether the pathologist was correct in recording chronic rather than acute passive congestion. Can this distinction be made in all cases from *post-mortem* evidence alone?

**Pericardial Effusion.**—Out of 186 cases twenty-seven showed 100 c.c. or more of exudate at necropsy. The fluid was measured with the following results:

TABLE 148.—AMOUNT OF FLUID IN PERICARDITIS WITH EFFUSION

| | | |
|---|---|---|
| 100 c.c. | in | 7 cases |
| 150 c.c. | in | 5 cases |
| 200 c.c. | in | 4 cases |
| 250 c.c. | in | 1 case |
| 200–300 | in | 2 cases |
| 300 c.c. | in | 4 cases |
| 300–400 | in | 1 case |
| 400 c.c. | in | 1 case |
| 500 c.c. | in | 1 case |
| 700 c.c. | in | 1 case |
| | | 27 |

In addition to these, nineteen showed a "considerable," fourteen a "moderate," and two a "large" amount of fluid. In four cases the sac is described as "distended" or "full." One contained "an excess," and one "a quantity" of fluid. Together these make up sixty-eight out of 186 cases in which we suppose that an effusion possibly capable of producing physical signs was present. In the remaining 118 cases (or over three-fifths) the fluid is recorded as "a small amount," a "slight excess." a "few c.c.," or was absent and the familiar shaggy or "bread-and-butter" exudate was described.

Of these sixty-eight cases 7 or 10% were recognized in life. The terms used at necropsy to describe these cases are:

"Distended"
"Markedly distended"
"500 c.c."
"Considerable amount"
"300 c.c."
"250 c.c."
"300 c.c."

In two other cases there was no excess of fluid in the pericardium at the time of necropsy, but this does not prove the prior diagnosis of effusion to be wrong.

The evidence used for diagnosis was in all cases a wide percussion area in the third or fourth interspaces to the right and left of the sternum, aided in one case by an X-ray picture.

*Paradoxical pulse* is mentioned in three of the nine cases. Its absence is once noted. Muffled or distant heart sounds are recorded in three other cases (also in one more with a diagnosis only of "dry pericarditis"). In one all heart sounds were replaced by murmurs.

In some of the cases with the largest effusions (e.g. 700 c.c.) only friction was noted in life.

*Weakness or muffling of heart sounds* was noticed in nine of the unrecognized pericardial effusions. Indeed in one of these the sounds are recorded as "inaudible." But the presence of pneumonia or other causes for poor heart action no doubt prevented these facts from suggesting pericarditis.

**Bacteriology.**—I have already alluded to the well-known fact that acute pericarditis is not usually or primarily a local lesion, like a gum-boil or an inflamed hemorrhoid, but rather the manifestation of a general septicemia. This is shown by its frequent association with pneumonia, peritonitis, acute pleuritis, and arthritis. It is further evident from the frequency of positive blood cultures *post-mortem*.

113 out of 186 cases showed positive cultures, the details being as follows:

TABLE 149.—ORGANISMS FOUND IN BLOOD AT NECROPSY IN PERICARDIAL INFLAMMATION

| | | |
|---|---|---|
| Streptococcus | 39 | |
| Pneumococcus | 33 | |
| Staphylococcus aureus | 14 | |
| Staphylococcus albus | 3 | |
| Staphylococcus unspecified | 4 | |
| Questionable | 9 | |
| B. coli | 3 | |
| Pseudo-Pneumococcus and colon | 1 | |
| Streptococcus and colon | 1 | |
| Streptococcus and pneumococcus | 1 | |
| Steptococcus and staphylococcus | 2 | |
| Streptococcus and mucosus | 2 | |
| B. mucosus capsulatus | 1 | 113 |
| Culture negative | | 73 |
| | | 186 |

The staphylococci were oftenest found in the cases of juvenile osteomyelitis with myocardial abscess, the pneumococci in the pneumonic cases, and the B. coli in those associated with ulcerative colitis.

**Murmurs in Acute Pericarditis.**—In seventy of these cases, murmurs, believed to be endocardial, not pericardial, were recorded. In sixteen cases the cause of these murmurs can naturally be put down as the valvular disease which necropsy showed was present in addition to the pericarditis. But there remain fifty-four in which this explanation will not hold. Of this fifty-four, thirty-nine showed only systolic murmurs.

TABLE 150.—DIASTOLIC OR PRESYSTOLIC MURMUR IN ACUTE PERICARDITIS

| No. | Age and sex | Murmurs | Heart weight | Chronic passive congestion | Remarks |
|---|---|---|---|---|---|
| 233 | 35 M | Presystolic at apex. | 465 | + | Chronic nephritis accounts for dyspnea (1 yr.), cardiac hypertrophy with congestion. Mitral 8.5 cm. but not deformed. |
| 529 | 64 M | Presystolic. | 520 | + | General arteriosclerosis. Mitral 10 cm. Aortic 8. "Atheromatous endocarditis and dilatation of aortic valve." |
| 1038 | 53 M | Diastolic at base. | H. & D. | 0 | Pernicious anemia. |
| 1668 | 50 M | Diastolic near left sternal edge. | 610 | 0 | Chronic nephritis. General arteriosclerosis. Mitral 10.5, aortic 7.4. |
| 3206 | 61 M | Diastolic, left sternal edge. | 696 | + | Chronic nephritis. |
| 3693 | 18 M | Diastolic along left sternal edge. | 566 | + | Acute glomerulonephritis. Chronic non-deforming endocarditis of aortic mitral and tricuspid valves. |
| 3760 | 50 M | Faint diastolic at apex and base. | 485 | + | Chronic nephritis. |
| 3357 | 68 M | Soft diastolic at apex (transient). | 370 H. & D. | 0 | General arteriosclerosis. Fibrous myocarditis. |
| 2590 | 75 M | Diastolic at the base. (2d right and 2d left interspaces). | 570 | + | General arteriosclerosis. Mitral 11.2, aortic 8.3, tricuspid 14, pulmonic 9. |

15 *remain in which diastolic or presystolic murmurs were heard in cases without deforming valve lesions post-mortem.* In six of these fifteen there were acute vegetations (921, 1814, 2800, 3242, 3561, 3797) which may have played some part in producing the murmurs, though in four of these the acute vegetations were so small that it is hard to imagine how they could produce a presystolic or diastolic murmur. (They are described as (1) "Pin-point sized," (2) "Match-end sized," (3) "Flat ulcers 5 × 8 mm. across," (4) "Minute or small masses.")

In at least 9 cases, however, there is nothing whatever in the endocardium to explain these murmurs. In Table 148 the facts are presented in detail.

These nine cases include four of chronic nephritis and three of general arteriosclerosis, lesions very commonly associated with an

hypertrophied and finally insufficient heart. In such cases presystolic murmurs at the apex (as in No. 233) have often been described. Diastolic murmurs might well be produced in the same way, though they have not been so often recorded so far as I know.

In Necropsy 1038 (pernicious anemia) the murmur may perhaps be considered "functional." I have elsewhere described such a murmur in association especially with the anemia of chronic nephritis.*

In Necropsy No. 3693 a boy of eighteen previously well is taken with "grippe in the joints" five weeks before entering the hospital and, after this infection, has dyspnea on exertion but no acute respira-

TABLE 151.—MAIN CAUSE OF DEATH IN ACUTE PERICARDITIS

| | |
|---|---|
| General septicemia* | 50 |
| Pneumonia and pneumococcus sepsis | 37 |
| Chronic nephritis | 32 |
| Neoplasms (and 1 case of leukemia) | 18 |
| Valvular heart disease | 7 |
| Arteriosclerosis | 3 |
| Aneurism | 3 |
| Syphilitic aortitis | 2 |
| Trauma | 5 |
| Meningitis | 3 |
| Pernicious anemia | 2 |
| Hepatic cirrhosis | 2 |
| Cardiac infarction | 3 |
| Acute and subacute nephritis | 2 |
| Appendicitis | 3 |
| Status lymphaticus | 1 |
| Strangulated hernia | 1 |
| Actinomycosis | 1 |
| Pericarditis alone | 9 |
| ? | 2 |
| | 186 |

* I class here cases in which several serous sacs or other cavities were involved, i.e., peritoneum, pleura, joints, endocardium, with or without positive blood cultures.

tory distress until two days before his death five weeks later. His entire illness thus covered ten weeks only. Examination showed pericardial friction and fluid (300 c.c. post-mortem); also a presystolic thrill and roll at the apex and a diastolic blowing murmur along the left sternal border. No murmur in the second right interspace.

* Richard C. Cabot, M. D. and Edwin A. Locke, M. D. of Boston: On the Occurrence of Diastolic Murmurs without Lesions of the Aortic or Pulmonary Valves (Johns Hopkins Hospital Bulletin, May, 1903. Vol. XIV, No. 146).

Pulmonic second accentuated. After five weeks of febrile illness with a leucocytosis of 12,700 to 3400 and a "picket-fence" temperature he died.

Necropsy showed in addition to the acute pericarditis a hypertrophied and dilated heart (566 grams), chronic passive congestion, and ascites. There was a chronic *non-deforming* endocarditis of the aortic, mitral and tricuspid valves. (Mitral 8.5 cm., aortic 6.5 cm., tricuspid 12. cm., pulmonary 7.5 cm.)

No. 3797 presents a good example of the simulation of valvular disease by this "marplot" pericarditis. In this case there was no pericardial friction and no evidence of fluid in the sac though both were searched for. There was a systolic murmur at the apex replacing the first sound and widely transmitted. A diastolic was heard in the second and third left interspaces near the sternum. One of our best diagnosticians signed the record as "Acute endocarditis, mitral stenosis and regurgitation, aortic regurgitation. Adherent pericardium (?)."

*Post-mortem* there was acute endocarditis of the aortic and mitral valves, chronic and acute pericarditis, but no deformity of the valves.

**Cardiac Hypertrophy in Acute Pericarditis.**—In 117 out of 186 cases in this series cardiac hypertrophy is recorded after death. In thirty-four of these the hypertrophy is associated with chronic nephritis and in three with arteriosclerosis. Nine were associated with valve lesions, two with chronic pericarditis in combination with the acute process, and two with pernicious anemia. This accounts for 50 out of 117 cases, including all the largest hearts.

In the remaining 67 cases there are only nine hearts over 520 grams. The two largest weighed 664 grams (male aet. 67) and 602 (male aet. 50). Nineteen other hearts weighed between 400 and 483 grams. Thirty-one weighed under 400 grams. Eight are recorded merely as "large" or "rather large." For the most part therefore it is only a moderate or a rather slight hypertrophy that we must try to explain in these cases.

Twenty-two of these patients died of pneumonia, the pericarditis being presumably associated with this. Twenty-eight died of some form of septicemia associated with acute peritonitis, arthritis, ulcerative endocarditis, septic knee, urinary sepsis, etc. In five cases the chief cause of death seemed to be a neoplasm, in three cases infarction of the heart, in two cases trauma, in one syphilitic aortitis, in one cirrhosis of the liver.

In only five was the infection manifested in the pericardium alone.

Twenty-eight of the 67 cases of cardiac hypertrophy without any traditional "cause" were thus associated with nothing more definite than a wide-spread sepsis, and twenty-two with pneumonia, and remain totally unexplained unless we can conceive that an acute infection is itself a cause of cardiac hypertrophy, or unless we assume an earlier permanent hypertension.

I do not see how the sepsis in the pericardium or elsewhere can produce hypertrophy of the heart. My best guess therefore would be that these 28 patients (like many others of the 67 above mentioned) had previously suffered from a "primary" hypertension, had thus acquired cardiac hypertrophy, and had then caught a sepsis and died. The necropsy then shows evidence of two stories: (a) a long one in which "essential" hypertension built up some degree of cardiac enlargement, and (b) a short one of sepsis and death with acute pericarditis. These two stories need have no important connection with each other. Neither need we assume any connection between the pericarditis and the enlarged heart. (This subject is also discussed under acute endocarditis.) The details in nine of these cases are shown in Table 152.

TABLE 152.—CARDIAC HYPERTROPHY WITH ACUTE PERICARDITIS AND WITHOUT OTHER SUPPOSED "CAUSES" FOR CARDIAC HYPERTROPHY

| Age and sex | Necropsy No. | Heart weight | Duration of fatal illness | Chronic passive congest | Remarks |
|---|---|---|---|---|---|
| 19 M | 3930 | 483 | 1 week | + | Syphilitic aortitis with no involvement of the aortic ring. |
| 18 M | 3693 | 566 | 2 weeks | + | |
| 50 M | 2955 | 603 | 2–3 weeks | + | Traumatic death. Chronic interstitial orchitis. Angina pectoris. Coronaries free. |
| 48 M | 2675 | 372 | ..... | o | |
| 42 M | 39 | 440 | 2 weeks | o | |
| 20 M | 2290 | 389 | 11 days | o | |
| 40 M | 1711 | 429 | 10 days | o | |
| 38 M | 1231 | 480 | 10 days | o | |
| 20 M | 89 | 483 | 1 week | + | |

**Plastic Pericarditis.**—Out of 110 cases of this type, eighteen were correctly diagnosed in life. In nine more our diagnosis was of pericarditis without fluid though at necropsy a considerable effusion was found. A friction rub and nothing else was recognized in life.

**Diagnosis of Pericarditis in General.**—In all we recognized the presence of some sort of pericarditis in forty-three cases or 23%. In thirty-three of these the evidence was friction alone, in three cases fluid, in seven cases both. The reason for our 77% of failures is in part the terminal nature of the process and its frequent occurrence in surgical cases where attention was otherwise engaged. But this is not the whole, or I believe the main reason. Even when we expected pericarditis and sought for it again and again, we often failed to find it, though it was there at necropsy, and not always a recent affair either. I think we must conclude that in some, perhaps in most, cases, there are no physical signs, no friction rub or any evidences of fluid.

From my own and others' clinical blunders I know it to be a fact *also* that we often hear a friction rub and diagnose pericarditis when the necropsy, shortly following, proves that pericarditis has never occurred (See p. 684). Some of these frictions may be due to pathological dryness of the tissues (uremia or pyloric obstruction).

## SUMMARY

1. Acute pericarditis is three times as common in men as it is in women. Why this should be so it is hard altogether to explain. In part it is perhaps accounted for by the fact that chronic nephritis and arteriosclerosis—both commoner in the male sex—often lead to a terminal pericarditis.

2. Other cardiovascular lesions (except hypertension and its results) are not often associated with acute pericarditis. The disease occurs either alone or with chronic nephritis or arteriosclerosis. Acute endocarditis is a complication in only 8% of cases.

3. A quarter of the cases are afebrile, presumably because of low bodily resistance in terminal states.

4. Pericarditis itself was the main cause of death in only 9 cases out of 186. In most instances pericarditis is only one localization of a generalized septicemia (often with pneumonia), or else represents a terminal infection in chronic diseases such as cancer, or nephritis.

5. Organisms of the streptococcus-pneumococcus group can generally be cultivated post-mortem from the exudate or from the blood. The staphylococcus—especially in children with myocardial

abscess and general septicemia—is not an uncommon invader (twenty cases in 186).

6. 117 cases of enlarged heart (hypertrophy and dilatation), with chronic passive congestion in sixty-six cases, were present in our 186 cases of acute pericarditis. 50 or about ½ of these cases are easily explainable by the other diseases present (chronic nephritis, etc.). But there remains a majority not so easily explained. It seems improbable that acute pericarditis is in and of itself a cause for cardiac hypertrophy and congestive heart failure. An earlier unrecorded hypertension is more probably the explanation for these hypertrophies.

7. Pericardial exudates of considerable size were present in 36% of cases.

8. Cardiac lesions are often wrongly diagnosed in acute pericarditis on account of murmurs believed to be of endocardial origin. Fourteen cases were of this sort. In these diastolic or presystolic murmurs were heard or supposedly heard, though they can hardly be accounted for by anything found in the endocardium. *When there is evidence or suspicion of pericarditis no diagnostic conclusions should be based on murmurs no matter how clearly they seem to be of endocardial origin.*

9. As I have previously proved in a smaller series of necropsies* we failed to recognize acute pericarditis during life in nearly four-fifths of the cases in which it was present post-mortem. It is also a fact, though not proved here, that a diagnosis of acute pericarditis is often made in life when none is present at necropsy. Errors of commission as well as of omission are frequent in the diagnosis of this disease.

10. Pericardial effusion was recognized during life in nine cases out of the sixty-five proved at necropsy. With more frequent use of radioscopy this record can be greatly improved. An extraordinarily wide area of transverse percussion dullness is our best diagnostic sign when X-ray evidence cannot be had. The character of the pulse and of the heart sounds gives but little aid.

### POINTS OF SPECIAL INTEREST TO THE WRITER

1. It seems at least possible that in a few cases of this series hypertrophy and dilatation and chronic passive congestion were due to the acute pericarditis itself, though it is always possible to class

* Diagnostic Pitfalls Identified during a Study of 3000 Autopsies: Journal of the American Medical Association, December 28, 1912, Vol. LIX, 2295–2298.

these cases in the group of hypertensive cardiovascular disease with complicating or terminal pericarditis.

2. *Pericarditis (acute or chronic) is a diagnostic marplot* when we are searching for valvular heart disease.

3. When there is no considerable effusion the diagnosis of acute pericarditis is so often missed or falsely asserted that its correct diagnosis may be said to be in many cases impossible. When effusion is present in considerable amount it should usually be recognized by the X-ray but not often otherwise.

## ILLUSTRATIVE CASES

### Necropsy 3418

An American painter of forty-four entered November 24. The complaints were swelling of the legs and heart trouble. His father died at fifty-six of heart trouble and dropsy, his mother at sixty-five after two years of "pleurisy." A sister died of tuberculosis. The patient was living at home when the mother died.

At thirty-two he had a small growth in the rectum the nature of which he did not know. There had been no recurrence. The same year he had an attack of lead colic characterized by sharp pain in the "pit of the stomach;" no paralysis or muscle weakness. He had "malaria" and rheumatic fever nine years ago, sore throat every winter, an ulcerated tooth a year ago. He had had a slight hacking cough all his life. His stools were black. He had never seen blood in them. He chewed one plug of tobacco every other day. He slept poorly. He did both inside and outside painting. He was not careful about washing his hands. His best weight was 210 pounds twenty-three years ago, his usual weight (one year ago) 170, his present weight 179.

For nine years he had noticed that when he stood up all day his ankles were slightly swollen at night. Six years ago he noticed dyspnea on exertion. As the dyspnea progressed he became conscious of his heart's beating. For the past two years he had vomited after breakfast about once a week. The vomiting was preceded by a dull pain which it relieved. The vomitus contained no blood or food of the previous day. Sometimes during these attacks his eyesight was blurred. For the past two years he had nycturia, with marked urgency. One year ago he noticed that he stumbled in the dark, his eyes were more frequently blurred, he had edema below

the eyes, and severe frontal and occipital headaches on waking, at times lasting several days and accompanied by nausea. His feet felt heavy. Six months ago the headaches obliged him to give up work. Since that time he had been at home, quiet, with no relief of symptoms. For six weeks his legs and abdomen had been swollen, with tenderness in the right upper quadrant. He now urinated eight times a day, five times at night. For the past two weeks he had had slight epistaxis in the morning. The dyspnea was now very marked.

Examination showed a pale, slightly cyanotic man. Pyorrhea. Mucosae pale. The apex impulse of the heart was felt in the fifth space. The percussion measurements are shown in Fig. 125. The

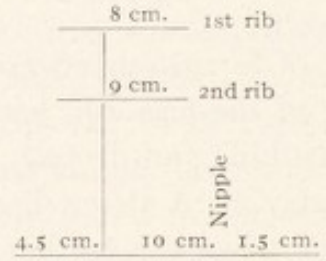



FIG. 125.—Cardiac percussion records in Case 3418.

aortic second sound was markedly accentuated. There was a slight musical systolic murmur at the apex, a low, faint systolic at base.

The blood pressure was 200/140–180/120. The artery walls were palpable. The lungs were negative except for coarse sonorous râles at both bases behind. The abdomen was held tensely. There was dullness in the flanks, shifting slightly. The liver dullness extended from the fifth rib to 3 cm. below the costal margin; the edge was just felt. There was edema and tenderness of both ankles. The knee-jerks were normal. The right pupil was greater than the left; both were irregular, reacted to light and distance.

The temperature was normal except for an occasional rise to 100° in the later days of life. The pulse was 60–102. The respirations were normal. The output of urine was 40–90 ounces until December 26, when it fell from fifteen ounces to almost nothing. The specific gravity was 1.010–1.012. There was a slight trace of albumin at all of five examinations, rare leucocytes at four; one finely granular cast at one; a few hyalin casts at one. The renal function was 6%. The hemoglobin was 55–80%. The leucocytes were 6000–12,300, the reds 3,408,000–4,736,000, with marked variation in size, some in shape; no stippled cells or polychromatophilia. The polynuclears

were 71%. A blood culture was negative. The blood showed on non-coagulable nitrogen determination 176 mgm. in 100 grams of blood. A Wassermann was negative, a gonococcus fixation test deeply positive. November 30 the fundi showed narrowing of the retinal arteries, occasional hemorrhage; diagnosis, retinal arteriosclerosis. December 23 there was retinitis of both eyes. The right eye was normal. The left eye showed a few small hemorrhages seen just above the optic nerve. A radial tracing showed no irregularity or alternation. There was a tendency to plateau pulse. X-ray November 28 showed cardiac enlargement and dilated arch.

The patient felt very well in bed. December 4 and 5 he was allowed to sit up. December 6 he felt weak, and was returned to bed. December 12 his left thumb was swollen and tender. An X-ray of the hand was negative except for thickening of the soft tissues. December 16 his ankles were beginning to become infected. December 21 he was very uncomfortable, requiring an occasional dose of morphia. The throat and dental departments were consulted to see if a possible focus could be found. The throat examiner reported, "No probable source of infection." X-ray of the teeth showed definite thickening of the left antrum, and small pockets about a number of teeth.

By December 25 the patient still required morphia and was incontinent of urine and feces. The joints were swollen, tender and hot. December 27 a friction rub was heard all over the precordia. He was having anginoid attacks, relieved for an hour or two by nitroglycerin gr. $\frac{1}{100}$. The friction rub persisted. The joints grew somewhat less painful. There was great diminution in the urinary output. He gradually became more delirious, and December 31 died.

*Clinical Diagnosis (from Hospital Record).*—Uremia.
Arteriosclerosis.
Acute dry pericarditis.
Chronic nephritis, arteriosclerotic.
Enlarged aortic arch.

*Dr. Richad C. Cabot's Diagnosis.*—Arteriosclerosis.
Coronary sclerosis.
Arteriosclerotic nephritis.
Hypertrophy and dilatation of the heart.
Acute pericarditis.
Terminal septicemia, streptococcus.

*Anatomical Diagnosis.*

| | |
|---|---|
| 1. Primary fatal lesions. | Arteriosclerotic nephritis.<br>Slight arteriosclerosis. |
| 2. Secondary or terminal lesions. | Hypertrophy and dilatation of the heart.<br>Edema of the lungs.<br>Fibrinous pericarditis.<br>Septicemia, streptococcus. |
| 3. Historical landmarks. | Slight chronic pleuritis, right. |

A cathether specimen of urine taken after death showed no phthalein output.

*Note by Dr. Cabot.*—I see no reason to modify the original opinion that lead poisoning was at the basis of this man's trouble.

### Case CXXXIX

A boy of twelve was seen May 1, 1920.

He had had an attack of measles of ordinary severity from which

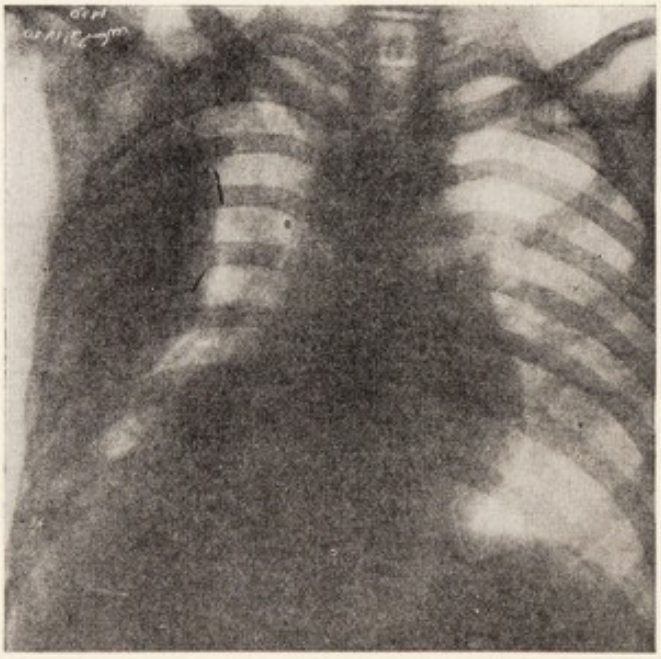


FIG. 126.—Empyema, left, and pneumonia, right. (Roentgenological Department, Massachusetts General Hospital.)

he was apparently convalescent April 26 and 27, with normal temperature. April 28 he had a sudden chill with high temperature. The temperature had remained about 104°. The white count was 11,200 on the 29th, 27,200 on the 30th. The pulse and temperature had been constantly elevated and there had been considerable dry cough.

Examination of the lungs showed respiration extremely limited. Breathing shallow. Movement more restricted on the left. In the left back from midscapula to base marked dullness, increased whisper and tactile fremitus, loud bronchial breathing, crepitant râles. The left axilla showed slight hyperresonance with occasional fine râles.

May 3 there was severe pleuritic pain on the left. The temperature was still about 104°. Examination showed flatness in lower back and axilla suggesting the possibility of fluid. May 8 there was flatness in the left axilla and back with faint distant breath sounds and diminished tactile fremitus. A needle was inserted just below the angle of the left scapula and 4 c.c. of straw-colored turbid serum

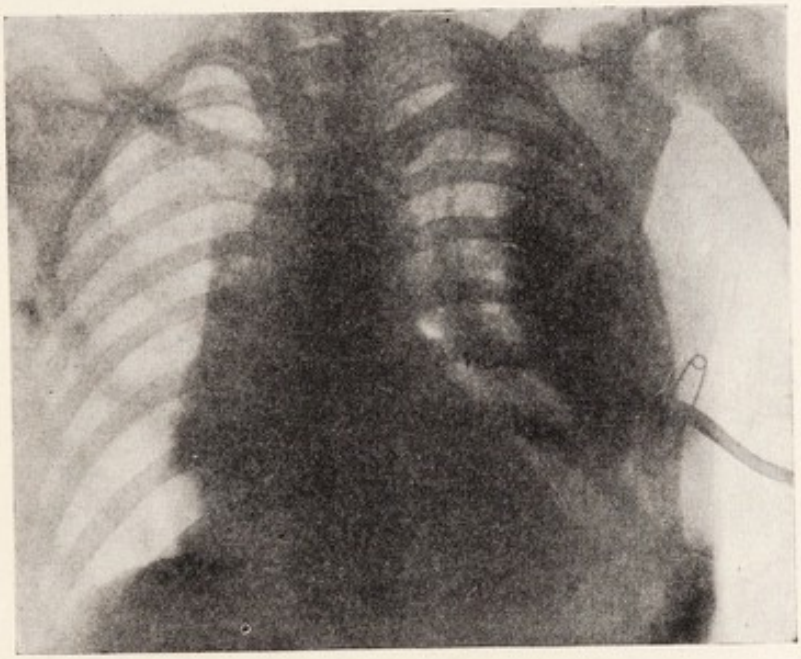


FIG. 127.—Shows tip of catheter draining left pleural cavity. (Note the axillary position of the exudate.) Dr. Wyman Whittemore. (Roentgenological Department, Massachusetts General Hospital.)

removed. The sediment showed an excess of leucocytes and many bacteria, mostly in pairs, with occasional short chains of three or four cocci. A few of the bacteria appeared to have pointed ends and to be morphologically like pneumococci, but on the whole more suggestive of streptococci.

May 9 the patient was moved to a hospital. X-ray (Fig. 126) showed empyema on the left with pneumonia on the right. Operation was done. Forty-eight hours later the left ear was discharging pus. The right ear was opened by an ear specialist.

May 15 X-ray (see Fig. 128) showed the heart shadow enormously increased and the empyema cavity empty. Aspiration of the pericardium in fifth left space just outside the nipple line and inside the

border of dullness gave thin pus showing under the microscope many leucocytes and a rare organism resembling the organism from the empyema fluid. This organism proved later to be pneumococcus type I.

The pericardium was drained. 1200 c.c. of pus was removed during the first twenty-four hours following operation. Fig. 129 was taken about a week later. The amount of pus gradually diminished until at the end of about four weeks there was less than 2 c.c. obtained by suction in twenty-four hours. Then drainage was

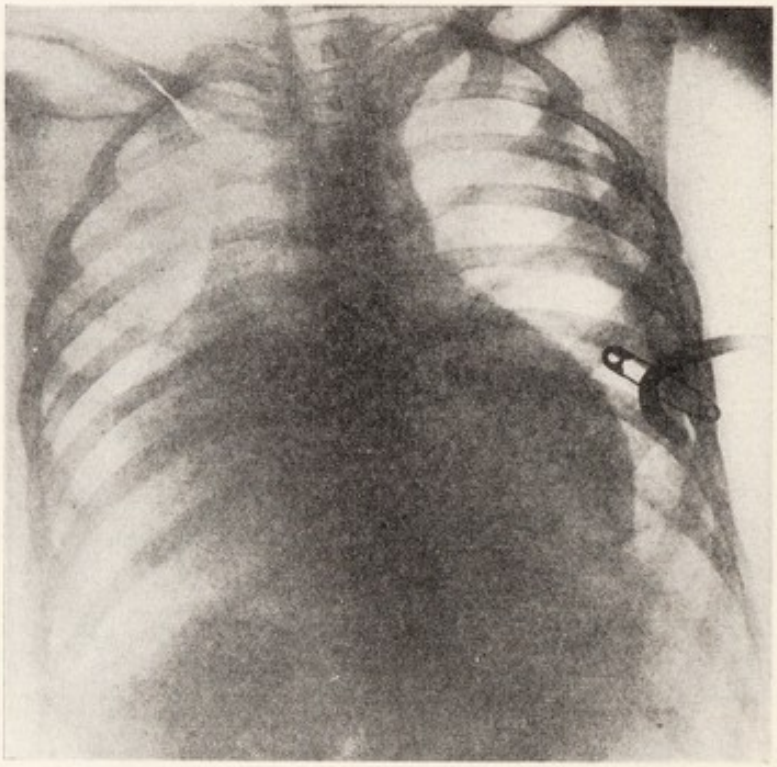


FIG. 128.—Acute pericarditis. Catheter still in left pleural cavity. Empyema practically gone. Dr. Wyman Whittemore. (Roentgenological Department, Massachusetts General Hospital.)

removed. Twenty-four hours later the drain from the pleural cavity was removed. Smear taken from both cavities showed less than one pneumococcus in five fields and a rare staphylococcus. Both sinuses promptly healed up, and at the end of eight weeks the patient was out walking around the hospital with a temperature no higher than 99°.

The skin over both mastoids was found to be edematous at this time. Both mastoids were immediately operated on under ether. The patient made an uneventful convalescence, and left the hospital at the end of three weeks.

*Dr. Wyman Whittemore's Pre-operative Diagnosis.*—Double pneumonia.

Empyema, left, encapsulated.

*First Operation.*—Local anesthesia. Left pleural cavity drained by a closed suction technique.

*Dr. Whittemore's Pre-operative Diagnosis.*—Acute pericarditis.

Mastoiditis, double.

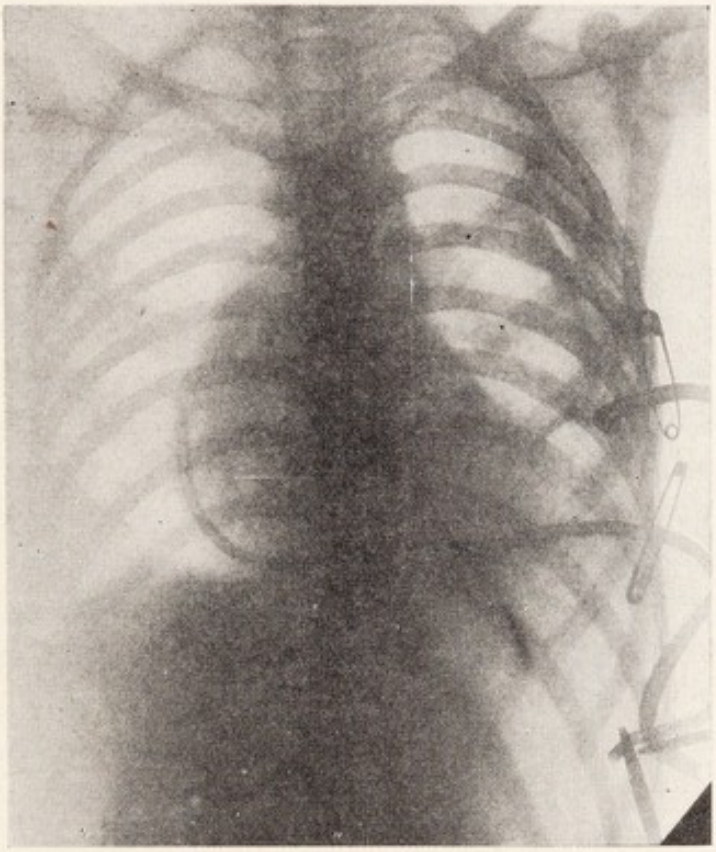


FIG. 129.—Catheter coming from left, going under base of heart, and draining posterior sulcus of pericardium. Dr. Wyman Whittemore. (Roentgenological Department, Massachusetts General Hospital.)

*Second Operation.*—Local anesthesia. Pericardium drained by a closed suction technique.

*Diagnosis.*—(Measles.)

Pneumonia, double.

Empyema, left, encapsulated.

Mastoiditis, double.

Acute pericarditis.

Operations: drainage of empyema; drainage of pericarditis; curettage of mastoids.

### Necropsy 4945

An American student of twenty-two entered August 19, 1922. He had measles and whooping cough before he was a year old. Since that time he had always been healthy and athletic. He was earning his way through college as a musician, playing an hour and a half every noon and four nights a week until one o'clock, afterwards studying often until three. His best weight was 156 pounds, a year before admission, his usual weight 150 to 155 during the summer, about 140 in winter. In the summer of 1922 it had remained at about 140.

Six months before admission he noticed a hard swelling on his left forearm. It gave him very little discomfort except that it sometimes interfered with rapid finger work. During the next two months it gradually increased to its present size. One spot was slightly tender to touch. Three months before admission he developed a dry cough which had persisted, with a little white sputum occasionally in the morning, and dyspnea, worse when he lay down. A month before admission for four days he had sharp cutting pain in the right lower back only on deep inspiration or motion of the trunk, and absent when he lay on his left side. A week before admission he had gradual onset of severe dull aching pain in the right thigh, becoming so severe that he required several doses of morphia in two days. After it subsided a slight pain remained on any motion which put strain on the quadriceps group. He went to a hospital, where 50 c.c. of bloody cloudy fluid was withdrawn from the left chest.

Examination showed a poorly nourished man with slightly cyanotic lips and an acneiform eruption over the face and upper torso. The posterior pharynx was reddened. The tonsils were enlarged, inflamed and cryptic. The left chest bulged slightly. The lung signs, abdomen, and apex impulse of the heart were as indicated in Fig. 130. The heart action was rapid. The sounds were of poor quality. The aortic second sound was greater than the pulmonic second. The right pulse was greater than the left. The blood pressure was 100/75 to 115/90. A firm fusiform tender non-inflammatory swelling apparently involved the left ulna, starting with the subcutaneous tissues. The skin could be rolled over the mass. The tendons and muscles had normal function. The arm at the tumor area was about one and a half times the diameter of the right arm. Another tumor mass appeared from palpation to lie over the right femur, more diffuse than the one in the arm, and tender. It could be rolled over the femur. The pupils and reflexes were normal.

The temperature was 96.3° to 104°, with periods of elevation August 21 to 22, August 26 to September 7 and September 19 to 23; after September 24 there was no elevation. The pulse was 80 to 170; the greatest rise was August 27 to 30. The respirations were 17 to 55. The amount of urine was normal, the specific gravity 1.015 to 1.028. The urine was cloudy at three of five examinations, alkaline at two, the slightest possible trace to a large trace of albumin at three, leucocytes at three, red blood corpuscles at one. The hemoglobin was 80%. There were 13,000 to 23,200 to 9200 leucocytes, with 55 to 87% polynuclears, and 6% eosinophils at entrance. August 28 one examiner recorded, "Mononuclears atypical. Many granules of neutrophilic type. Nucleus slightly large and cell very regular in outline." Another recorded, "The atypical cells are somewhat larger than a polynuclear, with a large nucleus filling most of the cell, indented but not bean shaped. The cytoplasm contains many neutrophilic granules." The reds were normal August 19 and

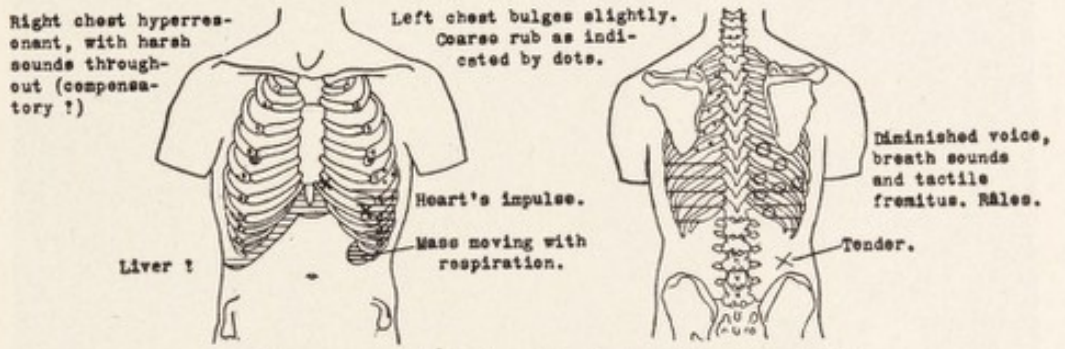



FIG. 130.—Chest signs in Case 4945, Aug., 1922.

28; September 21 there was slight achromia and an occasional polychromatophilic cell; September 27 slight achromia, anisocytosis. A Wassermann was negative. The non-protein nitrogen was 30.3 mgm., creatinin 1.68 mgm. The sputum showed no blood or tubercle bacilli; there were streptococci, occasional staphylococci, Gram-positive and Gram-negative bacilli, and a small amount of pus. August 20 a chest tap in the eighth interspace over a very dull area near the posterior axillary line gave no fluid. Solid material was encountered, grating against the needle and causing pain. Another tap in the eighth interspace in the posterior axillary line gave 10 c.c. of bloody fluid which clotted rapidly. There were 912,000 red blood corpuscles, 2100 leucocytes, 5% vacuolated mononuclears (pleural? tumor?), 55% polynuclears, 38% lymphocytes, 2% large mononuclears. A culture and a smear were negative. A blood culture showed staphylococcus albus. X-rays August 21 are shown in

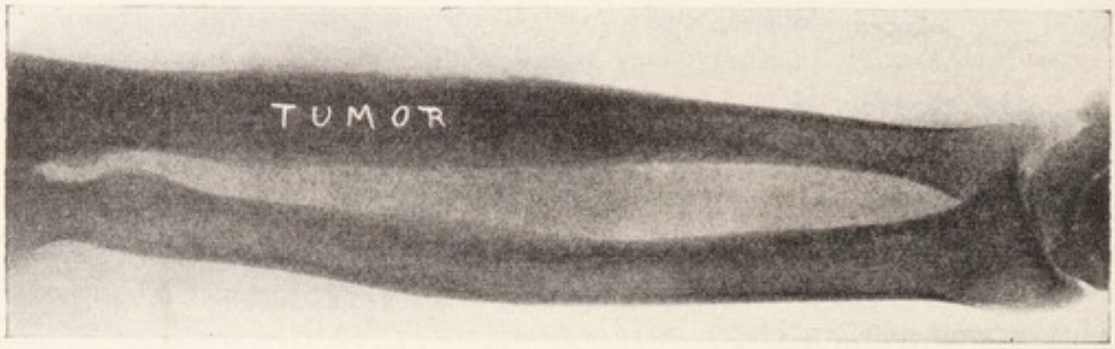



FIG. 131.—Forearm August 21, first admission, before treatment. Shows fusiform thickening of the middle third of the ulna, apparently due to proliferative changes in the region of the periosteum. Small but distinct ray formations on the outer aspect of the ulna. Soft tissues in region of involved area considerably thickened. No definite tumor outline visible. Bones show no evidence of atrophy or destruction.

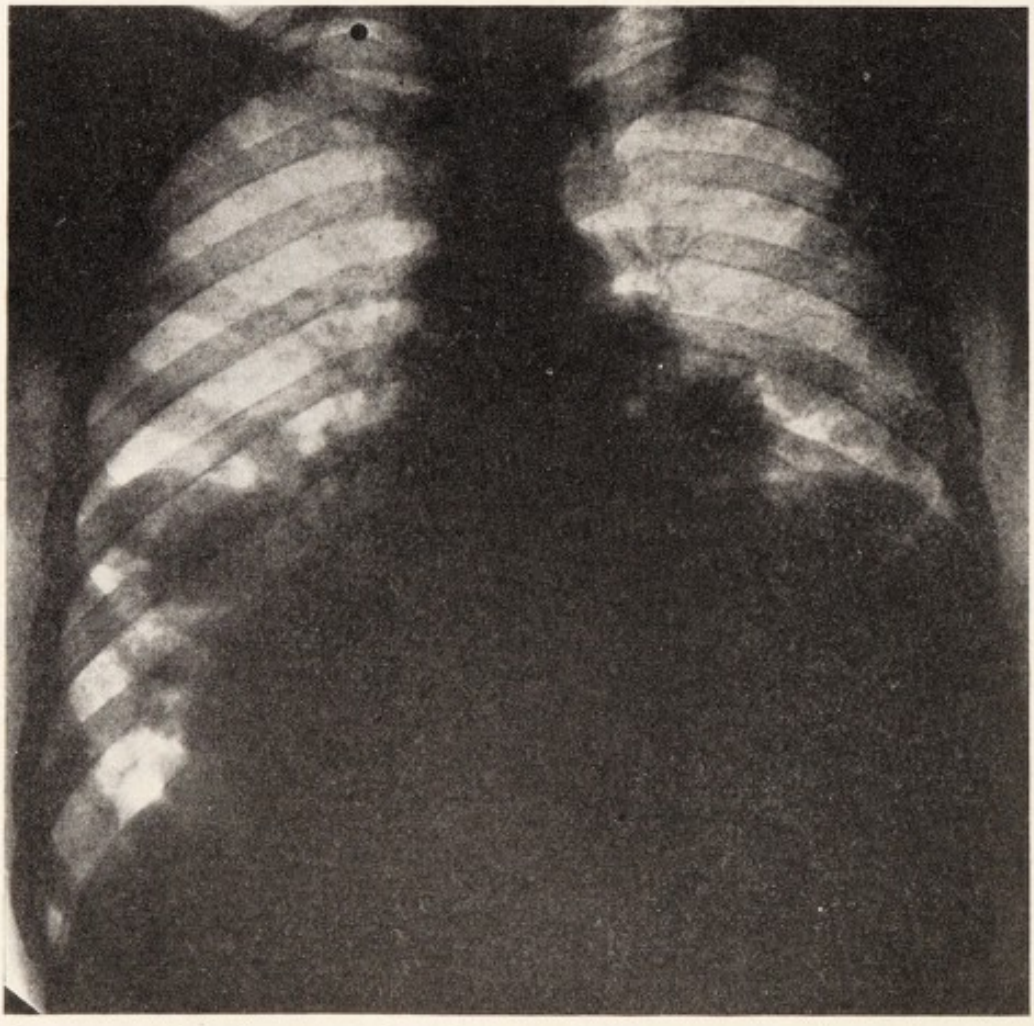


FIG. 132.—Chest August 21, first admission, before treatment. Large dense sharply defined shadows obscuring outline of heart and angle between it and diaphragm on both sides. Similar small shadows well out in periphery of lung. No evidence of cavity formation. Apices clear.

Figs. 131 and 132. A plate of the femur showed a line suggesting separation of the periosteum from the bone over the inner aspect of the middle third. No soft tissue changes were visible. August 28 a plate of the chest was difficult to interpret because of motion. Apparently there was little increase in the size of the dull areas in the chest. The outline of the diaphragm was still visible. September 5 and 7 there was no definite change in the appearance of the chest or the forearms. The vital capacity September 11 was 3060 c.c.; September 13 at a single reading only 1110. The forced respiration caused such violent coughing that a second determination was not attempted.

August 27 the patient was given neavy radiation. This was followed by severe reaction and the maximum rise in temperature, pulse and respiration. He required two grains of morphia in less than twenty-four hours. For two weeks he had dyspnea, marked cyanosis, and paroxysms of cough, usually unproductive.

September 12 a biopsy showed an atypical specimen on which the pathologists disagreed.

September 23 another X-ray treatment was given with no bad results. He had had four by September 29, and showed remarkable improvement. His nights were better than they had been at any time. October 3 the lungs showed about the same dullness to percussion as before, but striking absence of the showers of râles previously heard. X-ray showed some lessening of the areas of involvement. October 6 he was discharged with advice for further X-ray treatment.

After leaving the hospital he went for a month to the country, where his weight increased from 125 pounds to 143. November 24 his vital capacity was reported as 3200 c.c. On his return to the city he developed a "pleurisy" with severe pain in the left posterior chest, constant, but worse on deep inspiration or movements of the body. This persisted except for occasional remissions following X-ray treatments. From June 20 to 27 it became more severe and was associated with pain deep in the right thigh and knee joint, becoming so severe that he was given morphia. June 26 this pain made him cry out. June 27, 1923, he reentered the hospital.

Upon examination he was poorly nourished, with flushed face, warm and moist skin, and cyanotic lips. There was brown pigmentation over the chest, very marked on the left. The bone beneath the scar on the left forearm was roughened. There were pea-to-bean-sized firm non-tender cervical lymph nodes. The left lower

chest bulged posteriorly. The left chest moved very slightly. The right was almost normal. The lung signs were as shown in Fig. 133. The apex impulse of the heart was in the fourth space 7 cm. to the left, not forceful. The midclavicle was 8 cm., the right border

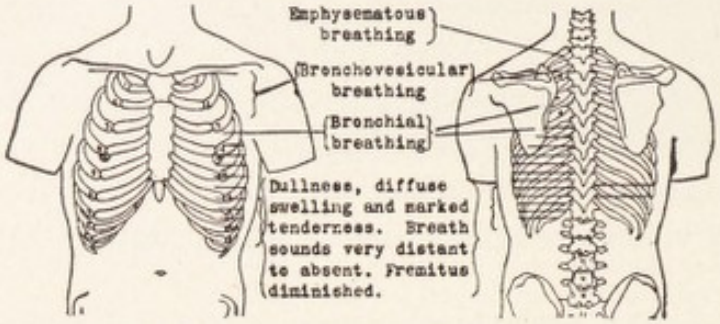



FIG. 133.—Chest signs in Case 4945, June, 1923.

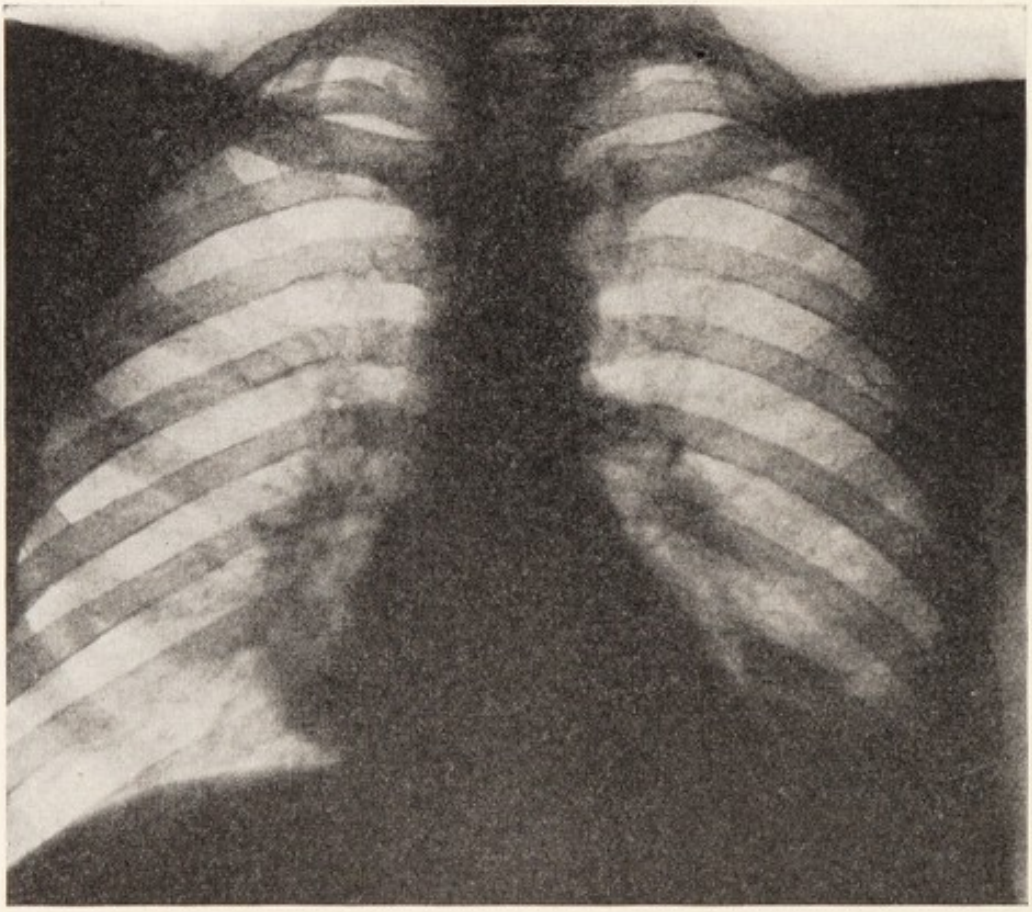


FIG. 134.—Chest in July, eleven months after first admission, after X-ray treatment. Looks practically clear except for an area of somewhat increased density at the right base close to the heart border. Diaphragm on the left apparently high. Left costovertebral angle hazy.

of dullness 5 cm. The pulse was fair, the rate 110. The blood pressure was 116/66. The abdomen was very tense. The spleen was enlarged to percussion, the liver not enlarged.

The temperature was 97.3° to 103.5°; there was no elevation July 4 to 6 or 12 to 17. The pulse was 90 to 140, the respiration 16 to 30. The output of urine was ℥ 31 to 91, the specific gravity 1.010 to 1.024. The urine was alkaline at two of three examinations and showed the slightest possible trace of albumin at one, occasional leucocytes at one. The hemoglobin was 70 to 80%. The leucocytes were 12,200 to 8800, the polynuclears 80 to 91%, the reds normal except for slight anisocytosis at the first examination. The X-ray is shown in Fig. 134.

A surgical consultant found no evidence of perinephric abscess. The patient improved before as well as after his first X-ray treatment. July 17 he was discharged, to return for X-ray treatment.

July 23, two days after his third X-ray treatment, he suddenly developed "shingles" involving the left side of the chest wall from spine to midline over a breadth of four inches. This lasted three

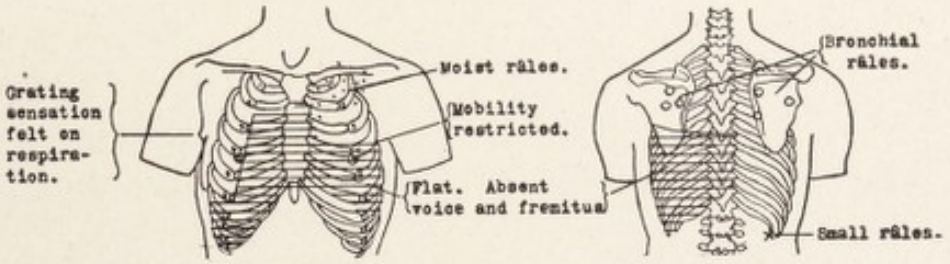



FIG. 135.—Physical signs in Case 4945, Oct., 1923.

weeks, and continuous pain and tenderness or hyperesthesia two or three weeks longer. September 15 he went to the country for three weeks, and felt well and active until three days before leaving. Then he developed dyspnea and a feeling of hardness in the left lower chest. He went home and rested. The dyspnea disappeared, but returned October 19. During the next few days he had severe pain in the right upper chest and shoulder on deep inspiration. His weight had remained the same; his appetite had been steadily fair. The left forearm swelled at times for no apparent reason, becoming swollen and firm with edema which in about a week subsided. Twice since his discharge he had very distressing dull aching pain in the outer thigh along the sciatic nerve (?) subsiding in a week or two. He had been told that the prognosis was hopeless. When the pains became too bad he took a pill with marked relief.

Examination at his third admission, October 26, 1923, showed him emaciated, sweating profusely, lying on his left side in much respiratory distress, with occasional attacks of cough with little sputum. The skin over the left chest and abdomen was tense, shiny,

and scaly. The left chest moved little with respiration. Two substernal glands were felt, also glands in the left axilla and both groins. The lung signs were as shown in Fig. 135. The heart was apparently pushed to the right. The left border was not determined. The right border was 7 cm. to the right of mid-sternum. The sounds were heard only to the right of the sternum. There was a to-and-fro rough murmur sounding like a pericardial rub. The belly-wall showed almost board-like rigidity in the upper quadrants and considerable in the lower quadrants. It was impossible to palpate through it. The superficial veins were distended. There was no tenderness. In the middle of the left forearm was an oval non-tender bony mass with a smooth surface, merging with the bone. The right thigh was slightly tender along the course of the sciatic nerve.

The temperature was 99° to 96.3°, the pulse 118 to 140, the respiration 34 to 20. The amount of urine was normal. The urine

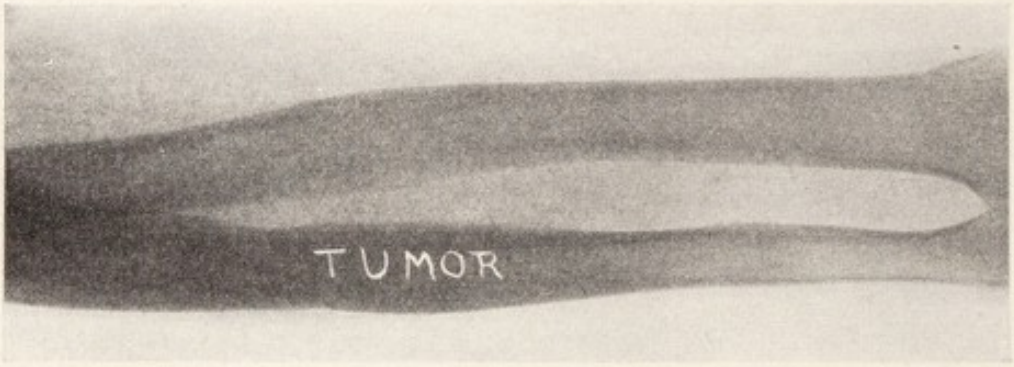



FIG. 136.—Forearm October 5, three weeks before third admission, after X-ray treatment.

was cloudy, the specific gravity 1.034. There was a very slight trace of albumin. The hemoglobin was 65%. There was 16,200 leucocytes, 85% polynuclears, 4,160,000 reds, slight achromia. The chest was tapped October 26 in the eighth interspace at the angle of the scapula and in the posterior axillary line. After an hour of manipulation of needle and position with strong suction, 30 c.c. of thick slightly viscous bloody fluid which clotted almost immediately was obtained. It showed 80% large mononuclears, 10% small fibroblastic (?) cell types, 5% polynuclears, 5% fragmented cells, many red blood cells, four mitotic figures, 36,000 leucocytes. The fluid clotted too quickly for a count of the red blood cells, but it must have been very high—2,000,000–3,000,000. October 28 a tap in the eighth left interspace just below the scapula gave 2 c.c. of thick bloody fluid which clotted quickly. On manipulating the trocar it seemed as though there were adhesions along the chest wall.

The patient was propped up in bed and kept comfortable with morphia and caffein. October 28 he died.

*Pre-operative Diagnosis* (*Sept.* 12, 1922).—Tumor of ulna.

*Operation.*—Local novocain. Incision made over middle of ulna. Bone partly solid, partly moth eaten, surrounded by thickened periosteum which did not appear characteristic of sarcoma, but suggested syphilis or chronic osteomyelitis. Specimen of periosteum and bone removed for examination. Wound closed.

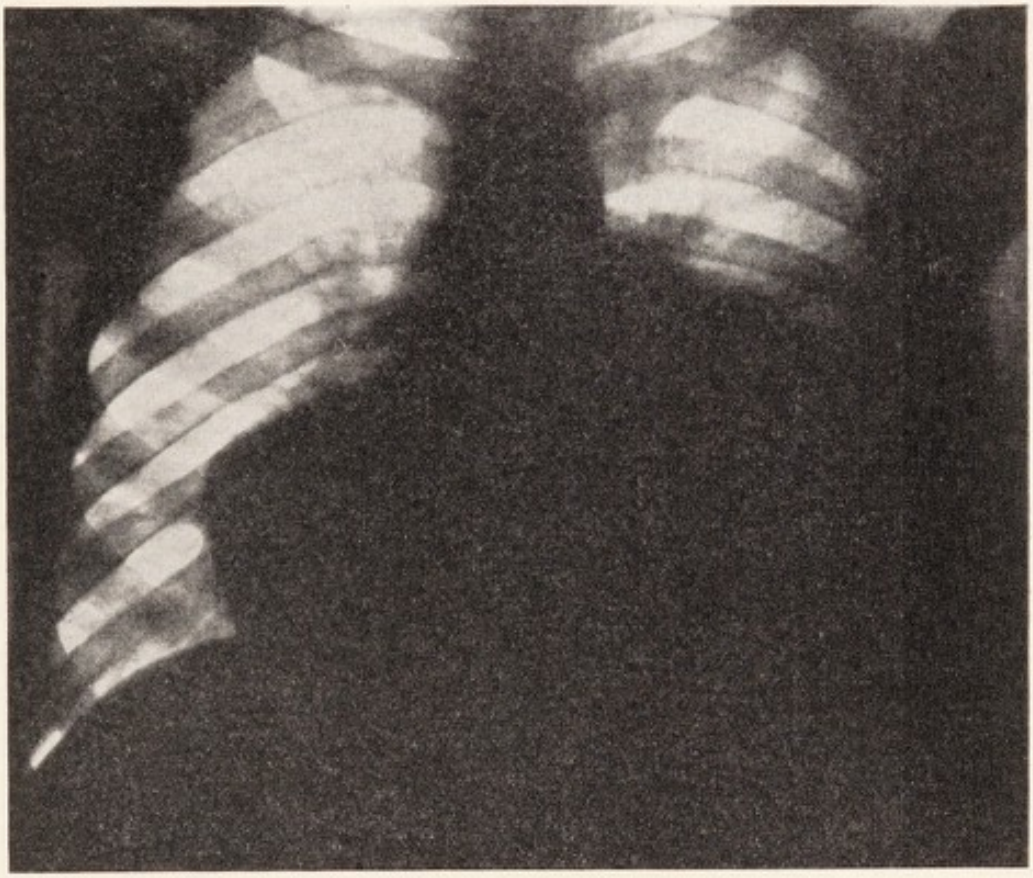


FIG. 137.—Chest October 5, three weeks before third admission, showing recurrence and displaced heart.

*Pathological Report.*—Microscopic examination of small fragments of bone showing clusters of atypical cells suggesting osteoblasts, with fibrils and areas of osteoid tissue.

Osteogenic sarcoma.

H. F. HARTWELL.

*Bacteriological Report.*—Culture from bone negative.

*Clinical Diagnosis* (*from Hospital Record*).—Osteosarcoma of left ulna with metastases to left lung and pleura.

*Dr. Richard C. Cabot's Diagnosis.*—Osteosarcoma of the ulna with metastases in the chest.

*Anatomical Diagnosis.*—(Osteogenic sarcoma of left ulna.)

Metastatic osteogenic sarcoma of lungs, bronchial lymph glands and paravetebral and retroperitoneal tissues.

Acute pericarditis.

DR. RICHARDSON: There was a large bulging area on the left chest just above the costal border, and the cutaneous vessels of the anterior wall of the thorax, the shoulders and along the sides of the abdomen were prominent. There was no particular deformity of the left forearm at the time of necropsy, and we were not allowed to go any further with the examination. The organs had to be replaced in the body.

There was a little thin pale fluid in the peritoneal cavity. The mass had pushed down the stomach, spleen and intestines so that the lower border of the stomach was six cm. below the umbilicus, the small intestines practically in the pelvic cavity, and the transverse colon skirting along the top of the bladder. Below the diaphragm, except that this mass pushed down in the retroperitoneal tissues, there was nothing except that on the right side of the pelvic cavity there was a mass of new growth tissue similar to that in the lung. It was hemispherical, rather discrete, and measured $7 \times 3\frac{1}{2}$ cm. The diaphragm on the right was at the fifth interspace, on the left at the eighth rib. A mass of new growth tissue in the region of the left lung pushed the diaphragm down to within a few cm. of the crest of the ilium; down along the retroperitoneal tissues. It pushed the spleen forward so that its long axis was parallel to the median line, and its lower pole only eight cm. above the crest of the ilium.

There was no fluid in the pleural cavities, and no adhesions on the right; the left was bound down to the tumor in that region. The bronchial glands were slightly enlarged and some of them showed new growth tissue. In places in the new growth tissue there were peculiar rust-colored areas of necrosis. The right lung was voluminous and showed metastases here and there, some of them of pretty good size, but nothing in comparison with the other side. On the left side the ovoid mass of new growth tissue measured $31 \times 24 \times 24$ cm. In some places the pleura was thickened and retracted, with possibly some streaks and areas of fibrosis lying along the tumor tissue. Scattered through the large mass of new growth tissue were in places large rust-colored areas of necrosis.

The pericardium showed *acute pericarditis*. The heart weighed 275 grams. The left border was in line with the left border of the sternum, the right at a line perpendicular to the junction of the middle and outer thirds of the right clavicle. The valves and cavities were negative, the aorta and branches negative except that the ovoid mass flattened the ascending thoracic portion of the aorta markedly.

The abdominal organs require no comment. The bones of the body, as far as dissection permitted, were negative.

Microscopical examination of the kidney, liver, spleen, etc., was negative.

DR. CABOT: Have you any doubt that this man's life was prolonged by radiation?

DR. HOLMES: No, I have no doubt of it. Without radiation he would have died sooner; but what is more important, he was able to carry on very comfortably until a very short time before death.

DR. CABOT: It was a prolongation not merely of existence but of actual work. What seem to be the prospects for improving therapy in cases of this kind? We have had one or two cases where you had extraordinary temporary results in banishing from the chest great masses of tumor, but with recurrence. Do you look for gain? Do you think we are likely by the methods we now control to be able to hold this lesion off longer?

DR. HOLMES: There is not a great deal of evidence that points towards cure, but I think that we are going to be able to make it easier for the patient while undergoing treatment; that there will be less Roentgen sickness. As for the actual prolongation of life, it does not look as though we were going to be able to accomplish a great deal once widespread metastasis has taken place. There are some interesting data in the recently published accounts of German clinics. In the clinics where the radiation treatment is controlled by the surgeon they are using radiation for all cases of malignant bone tumor rather than surgery. I think there is some question whether cutting into a tumor or even amputation of the primary tumor does not shorten the life of the patient. It is better to treat the primary growth with radiation rather than to remove it. It is possible that work along that line may enable us to carry these cases longer and more comfortably.

DR. MERRILL: Dr. Holmes told us that when that appearance was seen in the lung the question arose whether it was recurrence or fibrosis. Knowing as we do in this case that it was not fibrosis, but

recurrence, in another similar case, were we not deterred, what would be the prospect of giving him another lease of life?

DR. HOLMES: I think it would be pretty good.

DR. MERRILL: And even presuming that it was a condition of fibrosis and there still remained a question of recurrence, what would be the great harm in producing a condition of fibrosis? A man can exist with a large amount of fibroid change in his lung. Suppose we treated him, what harm would it do?

DR. HOLMES: I do not know that it would do any. I think we were unduly scared. But there has been a good deal of experimental work recently that has given us a rather bad scare. Some of the patients develop, for example, a Roentgen cachexia which goes on to death. So that we feel rather shaky about going ahead. I think perhaps if we had it all to do over again we should recognize we made a definite mistake in not doing it. Of course the mass that developed in the abdomen would probably have gone on and we should not have been able to control it. Other cases similar to this have developed metastases in the brain which we could not control. The lymphoma cases, which are much more susceptible to X-ray, we can control in the chest, but they die of metastases in the brain. So that I think with metastasis we are going to lose the patient anyway.

### Necropsy 4269

An American boy baby of four months entered October 11, 1921, for relief of constipation and vomiting. His parents had lost one child of pulmonary tuberculosis a year previously. The mother had had one miscarriage. The child was normally delivered at eight months, weighing less than five pounds. He was put at once upon a formula. He occasionally regurgitated, and then began to have loose green stools. At seventeen days old he was taken to the Floating Hospital, where he remained for three weeks. On the boat he caught "impetigo." At discharge he was still vomiting a little. His weight had increased from four and a half pounds at admission to five and three quarters. In a few days he began to vomit more and then to have eight or nine loose green slimy stools a day. By direction of a dispensary clinic he was put upon a different formula and was well for three days, then began to vomit again with great force, and one night had convulsions lasting five minutes in which the face and mouth twitched more than the rest of the body. The temperature was 102°. He entered a hospital, from which he was discharged September 30 against advice.

Upon his discharge from the hospital his bowels were constipated and his buttocks very raw. His mother started giving him an ounce or prune juice at night. Since that time he had been having two or three firm yellow stools a day until the day of admission when they were looser. October 10 he vomited with great force five minutes after each feeding and apparenly lost the entire feeding, which was sour and contained curds.

Examination showed a poorly developed and nourished baby with excoriations over the lower abdomen, buttocks and inner sides of the thighs. The mucosae were pale. There were white patches in the mouth. There was umbilical hernia. There was well formed rosary and slight enlargement of the epiphyses.

The urine was normal, the hemoglobin 50%. The leucocytes 9000, the polynuclears 32%, the reds 3,232,000, six nucleated cells, moderate variation in size and shape. A Pirquet was negative on two occasions. A Wassermann was negative.

On admission the child was put upon whole milk 15, water 15, ℥ 5 every four hours. The next day two tablespoonfuls of malt soup was added. He vomited once on each of the first two days, but not at all after that, and after the first day the green watery stools became soft solid. On the 18th the malt soup was increased to three tablespoonfuls. On the 20th he had nasopharyngitis, with a temperature of 102.6°. The right ear was opened on the 22nd. He vomited twice on each of the two days before discharge without apparent cause other than the otitis media. The stools, however, were normal. At admission the weight was 6 pounds 9 ounces, on discharge October 24 7 pounds 11 ounces.

After the child's discharge the mother was unable to get the malt soup, so she gave him whole milk 32, water 32, cane sugar 1 tablespoonful; ℥ 8 every three hours. The baby took all of it, did not vomit, had normal stools, and was well until November 20.

That day he began to have snuffles, with a nasal discharge. He was very fretful and coughed. His temperature was about 103° the whole week. November 24 and 25 he was worse. His bowels were constipated. The night of November 24 he was given a dram of castor oil. He cried much of the time. He took the bottle well until the morning of admission, November 26.

Examination showed a well developed and fairly well nourished, very pale baby, not appearing very sick. The anterior fontanel was 3 × 4 cm., level. The head veins were large. There was slight craniotabes. The sclerae and mucosae were very pale. The ears,

nose and mouth were negative. The lungs showed diminished resonance over the entire right back, more marked at the base, with bronchial breathing, increased voice sounds and fine crackling râles. The apex impulse of the heart was not seen or felt. The left border was 4½ cm., the right border not made out. There was sinus arrhythmia. The epiphyses were enlarged. There was a suggestion of clubbed fingers. The pupils and reflexes were normal. There was no Kernig or neck sign.

The temperature and pulse were as shown in Fig. 138. The respirations were 84 to 46. The urine is not recorded. The hemoglobin was 60%. There was 13,000 leucocytes, 66% polynuclears, 3,680,000 reds, slight achromia. The platelets were normal. A Pirquet was negative.

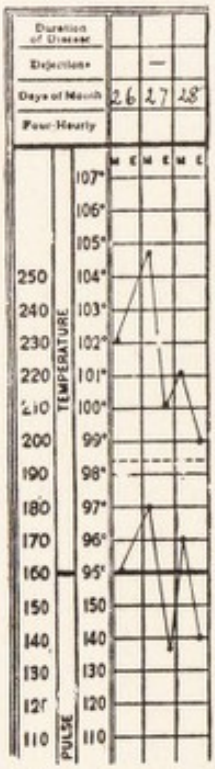



FIG. 138.—Temperature and pulse in Case 4269.

The day after admission the temperature was 104.6° and the child seemed dried out. He did not eat well for the first time. The fontanel was depressed. After 300 c.c. of normal saline was given intraperitoneally he seemed much better. The morning of November 28 he seemed in good condition. The temperature was 100.2°, the fontanel level, the lower lobe showed fewer râles. He did not however take his feedings well. At midday it was noticed that he was spitting up a little milk with foam and had a peculiar ash-gray look and rapid and labored breathing. His whole body was stiff, and the arms and hands twitched. The fontanel had become tense and the child fretted constantly. Lumbar puncture gave fluid under increased pressure, 2370 cells, 88% polynuclears; globulin positive; Fehling's not reduced. Gram-negative intra- and extracellular diplococci were found. 15 c.c. of antimeningococcus serum was given intraspinously and 30 c.c. intravenously. The child stopped crying after it and seemed more comfortable. He would not take the bottle, so was tube-fed. At midnight he began to fret again and breathed rapidly and with difficulty. The fontanel was again becoming tense. Another lumbar puncture gave 20 c.c. of fluid more cloudy than the first. 15 c.c. of serum was given intraspinously. Two minutes after the needle was withdrawn he began to have respiratory difficulty and was definitely sicker. The needle was inserted again in order to withdraw some fluid with the idea that the serum was causing increased pressure.

There did not seem, however, to be increased pressure, and the condition did not improve. Artificial respiration was given, but the child died after a few breaths.

*Clinical Diagnosis (from Hospital Record).*—Lobar pneumonia.

Cerebrospinal meningitis.

*Anatomical Diagnosis.*—Fibrinopurulent pleuritis (influenza bacillus and pneumococcus).

Fibinopurulent pericarditis (influenza bacillus and pneumococcus).

Acute leptomeningitis (influenza bacillus).

DR. RICHARDSON: A well developed and poorly nourished infant. In the region of the anterior fontanel there was evidence of the puncture mentioned. The vessels of Willis, the sinuses, the middle ears, and the mastoids were negative. Was there a history of middle ear?

DR. TALBOT: Yes.

DR. RICHARDSON: Apparently he had got over that at this time. The pia in the region of the convexities and scattered along the base was coated with a thin layer of pale yellowish exudate, and there was a small amount of thin cloudy fluid at the base, but there seemed to be no excess of fluid in the ventricles. The brain weighed 550 grams, and on section presented no lesions. Anatomically of course it was a leptomeningitis, extending down presumably into the region of the cord. In the anterior abdominal wall in the left lower quadrant there were two brown puncture-like spots.

DR. TALBOT: Normal saline had been introduced into the peritoneum.

DR. RICHARDSON: On the back were the puncture spots mentioned. The subcutaneous fat was small in amount, the muscles were pale, the peritoneal cavity, appendix and gastro-intestinal tract negative. The mesenteric and retroperitoneal glands were negative.

The pleural cavities: on the right side there was a small amount of yellowish purulent fluid and much membranous yellowish fibrinous exudate. This was plastered over the visceral and parietal pleura; on the left side there was nothing. Other than for the sticking of the lung on the right by the fibrinous exudate there were no adhesions.

The thymus gland was present, rather small, negative. The bronchial glands were negative.

The lung tissue generally was pinkish, spongy, yielding considerable pale pinkish frothy fluid; negative.

The pericardium contained much yellowish purulent fluid, and the two layers were thickly coated with membranous, opaque yellow-

ish fibrinopurulent material. The circulatory apparatus generally was negative. There was nothing in the liver, gall-bladder, pancreas, spleen, adrenals or kidneys.

DR. CABOT: Those intracellular diplococci—what were they?

DR. RICHARDSON: They said they were not stained by Gram, and they were probably influenza bacilli.

DR. TALBOT: They must have been pretty small.

DR. RICHARDSON: They were. Clinically which do you think came first, the infection in the chest or in the meninges?

DR. TALBOT: I think the clinical description there was of a lobar pneumonia first, and although the description is not very detailed one might infer that it had cleared up and we had a pleuritis either with it or secondary to it, and that the meningitis came last.

DR. RICHARDSON: When you tapped him the first time you got the pus—it was pus the first time?

DR. TALBOT: Yes.

DR. RICHARDSON: Anatomically here we have no evidence to indicate which was first.

DR. CABOT: You cannot swear that he had not had pneumonia and got over it?

DR. RICHARDSON: He probably did not. The lungs did not look like that. The condition found was infection of the pleura and pericardium.

DR. TALBOT: How much fluid was there in the chest?

DR. RICHARDSON: A small amount. Generally speaking the character of the exudate in the pericardium and pleura resembled more that of the pneumococcus. The exudate over the meninges was different. It did resemble more, I think, the epidemic form of meningitis than it did the pneumococcus. The condition there was not so marked. There was not so much membranous fibrinous exudate as on the pleura and the pericardium. I have been trying to think if we have had a discussion before on an influenza meningitis.

DR. CABOT: I think not.

DR. TALBOT: Of course we could not expect the antimeningococcus serum to do any good to influenza.

MISS PAINTER: Why did the baby seem to be better after the first injection?

DR. TALBOT: I suppose the lumbar puncture relieved the pressure.

**Necropsy 4174**

An Italian-American schoolgirl of ten entered March 2, 1921. She did not remember any illnesses or any attacks similar to the present one. The morning before admission she had a stomach ache, which rapidly became worse and caused her to vomit. As the pain increased it localized more or less in the lower quadrants and more particularly on the left side. She continued to vomit during the day. The day of admission the pain was intense and the vomiting continued. Her bowels had moved on both days.

Examination showed a well nourished girl with reddened and injected tonsils and slight cervical adenopathy. The lungs were clear and resonant. The heart was normal except for rapid action. There was exquisite tenderness in both quadrants and over the pubes, more marked on the left side. There was definite spasm of the muscle in the lower quadrants, while the abdomen above was soft. No organs or masses were palpable. The rectal examination was negative except for slight tenderness on movement of the uterus. The extremities, pupils, and reflexes were normal.

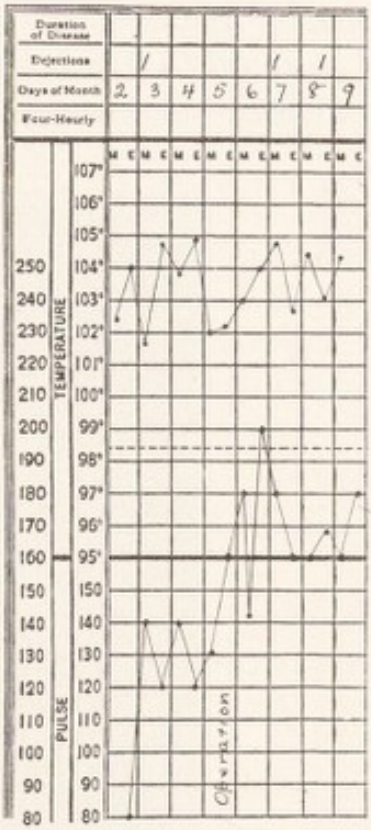



FIG. 139.—Temperature and pulse in Case 4174.

The temperature and pulse were as shown in Fig. 139. The respirations were normal until the evening of March 3, then 28–50. The amount and the specific gravity of the urine are not recorded. There was a trace to a large trace of albumin at both of two examinations, diacetic acid at the first. The hemoglobin is not recorded. There were 40,000 to 48,000 to 38,000 leucocytes, 89% polynuclears.

The evening of March 3 the patient vomited and was delirious. A medical consultant thought the case might mean a crisis of pneumonia, but was not typical. March 4 the patient looked worse. A medical consultant found no focus of pneumonia. He thought there was a little suggestion of dullness at the left base. Another consultant found some suppression of breath sounds over the right midlobe and inferior lobe, but said the pulmonary signs did not explain the temperature, etc. Fluoroscopic examination showed the lung fields

clear. The right diaphragm did not move so freely as the left, especially in the inner half. The costophrenic angles were clear. The plate showed in addition a band of slightly increased mottled density in the right chest in the region of the base of the upper lobe. The child's body was cold. She was not entirely oriented. There were three pulse beats to a respiration. There was no sweating. That night she was delirious and restless. A subpectoral was given. March 5 the abdomen was stiff and rigid and tender throughout.

Late that evening operation was done.

*Clinical Diagnosis (from Hospital Record).*—Idiopathic peritonitis.

Ether. Incision and drainage of peritonitis.

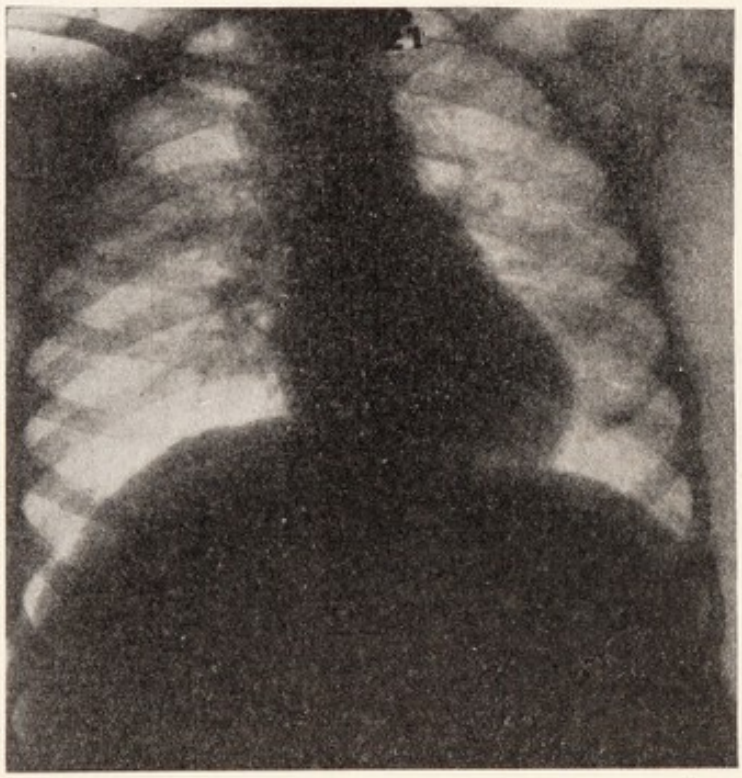


FIG. 140.—Costophrenic angles clear. A band of slightly increased mottled density in the right chest in the region of the upper lobe.

Next day two subpectorals were given, and March 7 two more. A culture from secretions from the nose was negative. March 8 there was rapid breathing, the nostrils moving with each respiration. The general condition was growing worse. There was considerable drainage. She retained a little rectal glucose. A sore was found on the left buttock, and two blue streaks in the same region. There was high color in her cheeks. March 9 she was distinctly worse. She roused and cried occasionally. In the middle of the morning she suddenly became cyanotic and had labored breathing. Next day she died.

*Dr. Hugh Cabot's Diagnosis.*—General septicemia with widespread generalized peritonitis.

Possibly pleuritis,

Possibly pneumonia,

Possibly pericarditis.

*Anatomical Diagnosis.*—General fibrinopurulent peritonitis.

Empyema, double.

Acute pericarditis.

Operation wound.

Slightly defective closure of the foreamen ovale.

*Note by Dr. Richardson.*—Culture from the peritoneal pus showed streptococcus.

It is not uncommon in cases of general peritonitis to find a culture from the blood stream showing no growth. In this case a few diplococci were recovered from the heart blood culture,—beginning septicemia. It is also not uncommon in cases of general fibrinopurulent peritonitis to find fibrinopurulent pleuritis. This infection of course comes either through the diaphragm or by the blood stream. At times it is difficult to say which is the avenue. In this case there are no definite lesions of the lungs,—only the empyema associated with the peritonitis.

*Further Discussion by Dr. Cabot.*—I am interested that Dr. Richardson finds a streptococcus, as this is the organism which is much more likely than any other to cause this picture.

## CHAPTER X

## CHRONIC PERICARDITIS

Starting from the *post-mortem* facts as revealed in the 112 examinations of this series, we find that chronic pericarditis shows itself, in the first place, as the so-called "milk spot" or patch of superficial fibrous thickening of the pericardium to which we have no reason to pay any attention. When adhesions exist between the two layers of pericardium, they may consist (a) of *slight* cases in which the few loose bands of adhesions presumably do not interfere with the work of the heart and are essentially historical landmarks, witnessing the presence of some past infection. Next (b) we have the *extensive cases* in which the whole pericardium is adherent to the surface of the heart so that no pericardial cavity any longer exists. Here there is at least the possibility of interference with the work of the heart, though, as will be seen presently, it does not appear that such interference can always be demonstrated. Lastly, (c) we have the group of *cases with mediastinitis* in which, in addition to obliteration of the pericardial sac, we have adhesions between the external surface of the pericardium and the mediastinal tissues,—pleura, sternum, diaphragm. As a source of interference with cardiac function, this group seems to be the most important.

In the first of these groups, wherein the pericardial cavity is not altogether obliterated, we may find acute pericarditis associated with the chronic process, just as the two are linked together in the endocardium. Acute pericarditis with the chronic process was present in 5 of 112 cases. Indeed it seems quite possible that we may have a series of attacks or recrudescences of pericarditis, as we certainly do of endocarditis, the membranes becoming with each attack more extensively vascularized and therefore more subject to the invasion of bacteria.

On the basis of these three types of pathological anatomy, we should expect to find more than one clinical type or picture of the disease, and as we run over the clinical histories corresponding with these necropsies, this is to some extent the case. Clinically there are three groups, I, Vestigial, II, Primary rheumatic, III, Secondary rheumatic.

## CHRONIC VESTIGIAL PERICARDITIS

In the largest group (see Table 154) the typical case is that of an elderly man who has never had rheumatism, who complains of no symptoms which can be in any way referred to the circulatory system, but dies of cancer, of Addison's disease, of urinary sepsis, pulmonary tuberculosis, or some other disease outside the heart. In the patients of this group no one suspects or ought to suspect pericarditis during life, and no one would look especially for it post-mortem. Only as a part of routine thoroughness in necropsy work it is discovered at all. In the 64 cases of this group brought together in Table 154, only 12 had any rheumatic history; more than $\frac{4}{5}$ were of the male sex; the average age at death was 53, the average weight of the heart only 430 grams, most of the cases showing little or no hypertrophy. In view of all these facts one would expect that in this group of cases the pericardial adhesions found at necropsy would be few and far between, in other words that we are dealing in this group with some of the results of relatively mild attacks of the disease. But in fact the number of cases with extensive, practically obliterating pericardial adhesions slightly outnumber those of the mild and incipient type. There are 36 of the obliterating type to 28 of the partial. Mediastinal adhesions, however, were present in only 8 out of 64 of this series and this is the essential point.

I have already pointed out that in this group we have had little or no evidence of cardiac disturbance during life. Only 20 of the 64 cases, or $\frac{1}{3}$, showed any dyspnea or other cardiac symptoms during life. 6 of these patients had chronic nephritis, so that their dyspnea might easily be accounted for, either as a result of the cardiac hypertrophy and dilatation which is linked with that disease, or as a uremic phenomenon. 2 others had pneumonia to explain dyspnea and 6 others were explainable without regard to the pericarditis. Only 16 of the cases showed evidences of chronic passive congestion, i.e. of poor heart function, post-mortem, while 48 showed none. 12 of these 16 had other lesions *post-mortem* which would account for the passive congestion without any relation to the pericardial adhesions. The other 4 cases, however, are not as easily explained and deserve some further description. One of these was a man of fifty-three whose heart at necropsy weighed 628 grams, although the pericardial adhesions were but slight and there was no nephritis or endocarditis to explain this hypertrophy. His history *stated that he had had orthopnea ever since he was sixteen years old*, an almost incredible assertion. He had marked evidences of stasis both before and after

death. I suppose that he really *suffered* from the hypertensive type of heart disease though he happened to *show* only the chronic pericarditis post-mortem.

Another patient, strangely enough, had in her history the same curious statement about *prolonged dyspnea*, which in this case was said to have existed since the age of six. The patient, a woman of twenty-two, died of cancer of the stomach. The heart in this patient weighed 308 grams and was considered by the pathologist to be but slightly hypertrophied. The pericardial adhesions were extensive but there was no mediastinitis, no history of rheumatism, no valvular or other lesion to account for dyspnea, and no evidence of passive congestion at necropsy. The blood pressure was 110/50.

Of 2 remaining cases one was a post-operative death, the other a crush.

Leaving out of account these 4 cases we may say that this group is *vestigial* because chronic pericarditis is, in these instances, of no clinical importance, a mere vestige of some early and apparently harmless infection. It is merely an item conscientiously recorded in the complete *post-mortem* findings.

### PRIMARY RHEUMATIC GROUP

Sharply contrasted with this group we find a smaller series of twenty-three cases, in almost all of which the pericardial disease constituted an important obstacle to the normal function of the heart. In 17 of these the pericarditis was clearly the main cause of death by passive congestion. 3 had no passive congestion, 2 died mainly of valvular disease, and 1 of pneumonia.

*The end of life came relatively early in this type of the disease, the average age at the time of death being twenty-eight years, contrasting with the average age, fifty-three, in the vestigial group.* There were 15 men to eight women. 18 out of twenty-three of these cases had a well-documented history of rheumatic fever, sometimes of many attacks.

While in the "vestigial group" described in preceding paragraphs, the adhesions were extensive in a little more than half the cases, they were extensive in 19 out of twenty-three of the group now under consideration. On the whole, then, we may say that these "*primary rheumatic*" *cases occur in young people and are characterized by extensive pericardial adhesions.* Mediastinitis was present in 11 out of twenty-three.

That the pericarditis interfered with the work of the heart is strongly suggested by the cardiac weights. In the primary rheumatic group the average weight was 655 grams. (Contrast this with the average weight in the vestigial group, 430 grams.) As will be seen in Table 155, this group contains some of the largest hearts seen in the whole series of our 4000 *post-mortem* examinations since 1896. In one case the cardiac weight, 1328 grams, was greater than in any necropsy of the whole 4000, and in three others, it was also extraordinary; 1205, 1150 and 1158 grams. There was not a single case in the series in which the absence of hypertrophy was demonstrable. Moreover, 19 of the 23 cases showed no cause for cardiac hypertrophy *except* the pericardial adhesions. In the remaining four cases there was subacute nephritis in two, general arteriosclerosis in one, and in the remaining case stenosis of the mitral, aortic and tricuspid valves.

## SECONDARY RHEUMATIC GROUP

Besides these two fairly well delimited types, our series contains an additional group of 25 cases characterized by the fact that other "rheumatic" heart lesions probably played the chief part in bringing about death, while the pericarditis was of secondary importance. This group affected relatively young individuals, the average age of death being thirty-two years. There were 13 males and 12 females in the group, suggesting, for reasons above alluded to, a slight excess of females. The heart weights averaged 508 grams. The adhesions were extensive in 12 while in 13 cases they were slight. Mediastinitis was present in only 8 cases. The accompanying cardiac infections are seen in the last column of Table 156.

## AGE AND SEX

Looking at the whole group of 112 cases we find that there are 32 females to 80 males, so that—as in acute pericarditis—the males make up 72% or nearly three-fourths of the whole series. The ages and the sex are shown in the following table.

. . .

TABLE 153.—AGE AND SEX IN CHRONIC PERICARDITIS

| Age | Males | Females |
|---|---|---|
| 0–10 | 0 | 4 |
| 11–20 | 7 | 3 |
| 21–30 | 8 | 5 |
| 31–40 | 16 | 7 |
| 41–50 | 15 | 5 |
| 51–60 | 19 | 4 |
| 61–70 | 8 | 1 |
| 71–80 | 7 | 1 |
| 81–90 | .. | 1 |
| ? | .. | 1 |
| ..... | 80 | 32 |

Obviously the disease affects women earlier than men. 42% of the males are over 50, while only 22% of the females exceed that age (provided the ages are correctly recorded). At the other end of the age scale, where there is perhaps less temptation to lie about it, we have 37+% of the women under the 31st year, while only 18+% of the men are of this age.

## DIAGNOSIS

It remains to inquire whether there are any signs or symptoms on the basis of which one may make the diagnosis of chronic pericarditis during life. In answer to this question, the outstanding fact is that in very few of these 112 cases was the diagnosis even suspected before death, and in only six was it actually made (Nos. 2297, 3009, 3290, 3345, 3520, 3648). In three cases acute pericarditis was the clinical diagnosis (1063, 3242, 3496) and in two cases this diagnosis was correct, though insufficient. Among the 46 cases in which *some* cardiovascular lesion was considered in the diagnosis, chronic valvular disease was most often thought to be present. This was the diagnosis in 22 cases. In 13 of these the mitral valve was

accused (mitral stenosis 7, mitral regurgitation 3, both 3) and seven of these were right so far, since there *was* mitral stenosis as well as chronic pericarditis at necropsy. Myocarditis was the only diagnosis in 5 cases, all of them wrong. Acute and chronic endocarditis was predicted in 4 cases, all correct diagnoses as far as they went. Of acute endocarditis alone there were six diagnoses, all correct though partial. Arteriosclerosis was all we recognized in 2 cases and aneurism (correctly) in one.

Thus we may say that in 66 or nearly ⅔ of the cases we failed to consider any cardiovascular lesion, that in the 46 cases remaining we got part of the diagnosis—the accompanying acute endocarditis, acute pericarditis or valvular disease—in 25. Systolic retraction of the interspaces near the heart or between the ribs of the left back is a sign of little or no value in the diagnosis of pericardial adhesions. It has been amply demonstrated by Tallant* and others that systolic retraction of interspaces is a common phenomenon in patients who show after death no evidence of pericarditis. Whenever a markedly hypertrophied heart is acting strongly within a relatively thin and elastic chest wall, the interspaces will fall in with each systole simply as a result of negative pressure. On the other hand we have searched in a number of these cases for systolic retraction of interspaces because we had some more or less distinct suspicion that adhesive pericarditis was present. But we usually failed to find it even when such adhesions were present after death.

Radiologists tell us that pericardial adhesions can be recognized by the X-ray, in case they are associated with chronic mediastinitis and especially with diaphragmatic adhesions. In such cases we are told that the respiratory movements of the diaphragm and the contractions of the heart can be seen by the fluoroscope to be limited. All this may well be the case, but I have yet to see an instance in which a diagnosis of chronic pericarditis has been suggested by a radiologist and then verified *post-mortem*.

Aside from these two signs, the falling in of interspaces and the limitation of cardiac or respiratory movement as seen by the fluoroscope, I know of no sign that even purports to give us any direct evidence of chronic pericarditis. But some *suspicion* of its presence may be aroused when, in a relatively young patient with a rheumatic history and evidences of cardiac hypertrophy and poor cardiac function, we are unable to account for them by any valvular

* Dr. Alice Tallant: Some observations on the occurrence of Broadbent's sign, Boston Medical and Surgical Journal, 1904, Vol. 151, p. 457.

TABLE 154.—GROUP I. CHRONIC VESTIGIAL ("LANDMARK") PERICARDITIS

| Number | Age | Sex | Rheumatism | Extensive or slight | Mediastinitis | Heart weight | Chronic passive congestion | | Remarks |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Before death | After death | |
| 156 | 40 | M | o | Slight | o | 427 | o | o | Pneumonia. |
| 425 | .. | F | + | Slight | o | 365 | + | + | Rh. 3 years ago. Goitre. |
| 439 | 48 | M | o | Extens. | o | 300 | o | o | |
| 448 | 38 | M | o | Slight | o | H & D | o | o | |
| 449 | 38 | F | o | Extens. | o | 165 | + | o | Addison's disease. General peritonitis. |
| 534 | 55 | M | o | Slight | o | 469 | + | o | Chronic nephritis. |
| 546 | 44 | M | o | Slight | o | 314 | o | o | |
| 563 | 49 | M | o | Very sl. | o | 261 | o | o | General peritonitis. |
| 568 | 32 | F | + | Subac. | o | H & D | o | + | Post-operative death. |
| 631 | 72 | M | + | Slight | o | 370 | o | + | Chronic nephritis. Bronchiectasis. |
| 739 | 79 | M | o | Slight | o | 680 | o | o | Obstruction. Cancer. |
| 796 | 59 | M | o | Slight | o | Sl. hypertr. | o | o | |
| 823 | 45 | F | o | Extens. | o | 308 | o | o | |
| 858 | 56 | M | o | Extens. | o | 254 | o | o | |
| 938 | 50 | F | o | Extens. | o | 245 | o | o | |
| 1158 | 54 | M | o | Slight | o | 313 | o | o | |
| 1228 | 54 | M | o | Extens. | o | 362 | o | o | |
| 1234 | 47 | F | o | Extens. | o | 210 | o | o | |
| 1279 | 27 | M | o | Extens. | o | 490 | + | o | Amyloid nephritis. |
| 1306 | 43 | M | o | Extens. | + | 609 | o | o | Chronic nephritis. |
| 1317 | 58 | M | o | Extens. | o | 827 | + | + | Arteriosclerosis. |
| 1386 | 57 | M | o | Slight | o | 393 | o | o | Duodenal ulcer. Chronic nephritis. |
| 1475 | 60 | M | o | Extens. | o | 388 | o | o | |
| 1483 | 73 | F | o | Extens. | + | 510 | + | + | |
| 1620 | 28 | M | o | Extens. | o | 250 | o | o | |
| 1652 | 51 | M | o | Slight | o | 325 | o | o | |
| 1673 | 53 | M | o | Slight | o | 628 | + | + | Heart weight not explained. "Orthopnea since 16." |
| 1700 | 69 | M | o | Extens. | o | 625 | o | o | Weight not explained. |
| 1875 | 85 | F | + | Slight | o | 650 | + | o | Cancer of sigmoid. |
| 1922 | 67 | M | o | Extens. | o | 342 | o | o | |
| 1935 | 60 | M | o | Slight | o | 398 | o | o | |
| 2198 | 60 | M | o | Slight | o | 562 | o | o | Subac.glomerulo-nephritis. |
| 2314 | 36 | M | o | Slight | o | 297 | o | o | |

TABLE 154.—GROUP I. CHRONIC VESTIGIAL ("LANDMARK")

PERICARDITIS.—(*Continued*)

| Number | Age | Sex | Rheumatism | Extensive or slight | Mediastinitis | Heart weight | Chronic passive congestion | | Remarks |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Before death | After death | |
| 2319 | 72 | M | o | Extens. | o | 560 | o | o | Coronaries occluded. Arteriosclerosis. |
| 2344 | 39 | M | o | Slight | o | 347 | o | o | |
| 2402 | 69 | M | o | Slight | o | 380 | + | + | Traumatic death. |
| 2504 | 56 | M | o | Slight | o | 400 | o | o | Calcified peric. Pn. |
| 2521 | 70 | M | o | Extens. | o | 430 | + | + | Pneumonia. |
| 2553 | 50 | M | o | Extens. | o | ... | o | o | |
| 2657 | 34 | M | o | Slight | o | 245 | o | o | |
| 2660 | 26 | F | o | Slight | o | 284 | o | o | |
| 2737 | 40 | M | o | Extens. | o | 305 | o | o | |
| 2786 | 53 | M | + | Extens. | o | 279 | o | o | |
| 2797 | 45 | M | + | Slight | o | 820 | + | + | Syphilitic aortitis. |
| 2849 | 75 | M | + | Extens. | o | 597 | o | + | Chronic nephritis. |
| 2891 | 63 | M | + | Extens. | + | 663 | + | + | Arteriosclerosis. |
| 2948 | 44 | F | o | Slight | o | 588 | + | + | Chronic nephritis. |
| 3208 | 37 | M | + | Slight | .. | 705 | + | o | Apoplexy. |
| 3026 | 53 | M | + | Slight | o | 739 | + | + | Subac. glomerulo-nephritis |
| 3113 | 50 | M | + | Extens. | o | 498 | o | o | Arteriosclerosis. |
| 3323 | 65 | M | o | Extens. | + | 435 | o | o | |
| 3520 | 40 | M | o | Extens. | + | 425 | + | + | Pneumonia. Sudden death. |
| 3597 | 51 | M | o | Extens. | o | 401 | o | o | |
| 3630 | 71 | M | o | Slight | o | 335 | o | o | Pneumonia. Phthisis. |
| 3692 | 48 | M | o | Slight | .. | 410 | o | o | Streptococcus sepsis. |
| 3714 | 22 | F | o | Extens. | o | 308 | + | o | Dyspnea since 6. Died of gastric cancer. |
| 3733 | 58 | M | o | Slight | o | 303 | o | o | |
| 3759 | 47 | M | + | Slight | o | 580 | + | + | Rheumatism 25 years ago. Gangrene of leg. Phthisis. |
| 3763 | 57 | M | .. | Slight | o | 380 | o | o | Cancer. Operation. Sepsis. |
| 3843 | 68 | M | o | Slight | Sl. + | 600 | + | .. | Aneurism. Cardiac Infarct. |
| 3728 | 80 | M | o | Slight | + | 501 | o | o | Streptococcus sepsis. |
| 3738 | 47 | M | o | Extens. | + | 356 | + | + | Streptococcus sepsis. Acute nephritis. |
| 3927 | 79 | M | .. | Slight | o | 405 | o | o | Prostate operation. |
| 3941 | 58 | M | o | Extens. | o | 265 | o | o | |

TABLE 155.—GROUP II. PRIMARY RHEUMATIC PERICARDIAL ADHESIONS

| No. | Age | Sex | Rheumatism | Extensive | Mediastinitis | Heart weight | Chronic passive congestion | | Remarks |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Ante Mortem | Post Mortem | |
| 266 | 31 | M | + | + | + | 1328 | 0 | 0 | No evidence of stasis ante or post mortem. |
| 281 | 26 | F | + | + | 0 | Hypertrophy and dilatation | + | + | Stenosis, mitral, aortic and tricuspid. |
| 282 | 13 | M | 0 | + | 0 | 625 | + | + | |
| 603 | 15 | M | + | + | + | Hypertrophy and dilatation | + | + | |
| ?662 | 33 | M | + | + | 0 | 403 | 0 | 0 | |
| 755 | 26 | M | 0 | + | 0 | Hypertrophy and dilatation | 0 | + | Acute endocarditis. Subacute nephritis. |
| 893 | 11 | F | + | + | 0 | Hypertrophy and dilatation | + | + | Rheumatism 4 years ago and again six months ago. |
| 1063 | 11 | M | + | + | + | 799 | + | + | |
| 1415 | 55 | F | + | Slight | 0 | 480 | + | + | |
| ?1535 | 31 | F | + | + | 0 | 337 | + | 0 | |
| 1907 | 28 | F | + | + | 0 | 377 | 0 | 0 | Subacute glomerular nephritis |
| 1975 | 52 | M | + | + | 0 | 1150 | + | + | Acute endocarditis. |
| 1980 | 11 | M | + | + | + | 380 | + | + | |
| 2640 | 9 | F | + | + | 0 | Hypertrophy and dilatation | + | + | Acute endocarditis and pericarditis. |
| 3340 | 56 | F | + | Slight | + | 435 | + | + | General arteriosclerosis. |
| 3345 | 19 | M | + | + | + | 1158 | + | + | Diagnosis made in life. |
| 3496 | 49 | M | 0 | + | + | 500 | + | + | |
| 3520 | 40 | M | 0 | + | + | 425 | + | + | |
| 3797 | 18 | M | 4 x | Slight | − | 510 | + | + | Acute rheumatism at end. Acute endocarditis at end. |
| 2952 | 32 | M | + | Slight | − | 600 | + | + | Mycotic aneurism. Acute nephritis. |
| 3009 | 23 | F | 0 | Extensive | + | 448 | + | + | Pick's disease. |
| 3290 | 31 | M | + | Extensive | + | 1205 | + | + | Diagnosis made in life. |
| 3648 | 33 | M | + | Extensive | + | 600 | + | + | Acute endocarditis. |

TABLE 156.—GROUP III. SECONDARY CHRONIC RHEUMATIC PERICARDITIS

| Number | Age | Sex | Rheumatism | Adhesions extensive? slight? | Mediastinitis | Heart weight | Chronic passive congestion | | Valve lesions, etc. |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Ante mortem | Post mortem | |
| 1775 | 13 | F | + | Slight | 0 | 420 | 0 | 0 | Mitral stenosis. Acute pericarditis. |
| 2276 | 32 | M | + | Ext. | + | 560 | + | + | Mitral stenosis. |
| 2366 | 15 | F | + | Slight | 0 | 386 | + | + | Acute endocarditis, aortic and mitral. |
| 2550 | 30 | M | + | Ext. | 0 | 1273 | + | + | Subacute nephritis. Chronic and acute endocarditis. |
| 3736 | 33 | M | + | Ext. | 0 | 315 Slight hypertrophy and dilatation | 0 | + | Ulcerative endocarditis. |
| 3483 | 28 | M | + | Sl. | 0 | 680 | | | Mitral stenosis. Acute pericarditis (700 c.c. in sac). |
| 2802 | 46 | F | + | Ext. | + | 510 | + | + | Mitral stenosis. |
| 2803 | 65 | F | 0 | Sl. | 0 | 386 | + | + | Mitral stenosis. |
| 2635 | 43 | M | + | Sl. | + | 486 | + | + | Mitral stenosis. Acute endocarditis. |
| 1995 | 39. | M | + | Sl. | 0 | 528 | + | + | Mitral stenosis. |
| 421 | 53 | F | 0 | Sl. | 0 | 539 | + | + | Mitral stenosis. Acute endocarditis. |
| 1550 | 49 | M | + | Ext. | 0 | 532 | 0 | 0 | Mitral stenosis. |
| 3535 | 28 | M | 0 | Ext. | + | 532 | 0 | + | Mitral, aortic and tricuspid. Stenosis. |
| 3596 | 34 | F | + | Sl. | 0 | 466 | + | + | Mitral, aortic and tricuspid. Stenosis. |
| 3827 | 48 | M | + | Ext. | 0 | 560 | | | Mitral and aortic stenosis. |
| 999 | 42 | M | + | Ext. | + | 733 | + | + | Mitral and aortic stenosis. |
| 785 | 37 | F | + | Sl. | 0 | 508 | + | + | Mitral and aortic stenosis. |
| 2213 | 35 | F | + | Ext. | + | 415 | + | + | Mitral, aortic, tricuspid and pulmonary stenosis. |
| 2297 | 37 | F | + | Ext. | ? | ? | + | + | Mitral stenosis. |
| 3476 | 20 mos. | F | 0 | Extensive | + | 80 Hypertrophy and dilatation | 0 | + | Empyema, brain abscess. Subacute glomerulo-nephritis. |
| 2437 | 14 | M | + | Slight | 0 | 360 | + | + | Chronic and ulcerative endocarditis (aortic). |
| 2949 | 23 | M | + | Slight | − | 567 | + | + | Acute endocarditis. |
| 2965 | 25 | M | + | Slight | 0 | 558 | + | + | Mitral and aortic stenosis. |
| 3242 | 4 | F | + | Very slight | − | 170 | + | + | Acute endocarditis. |
| 1133 | 51 | F | + | Ext. | + | 640 | + | + | 300 c.c. in pericardium. |

lesion, by chronic nephritis, congenital heart defect,—in short by anything else except by supposing that the patient's rheumatism has obliterated the pericardium. In a few cases, by this concatenation of facts, suspicions may certainly be aroused and those suspicions are strengthened when the patient shows an unexplained, predominating, and recurrent ascites which, as has been pointed out by Pick and others, is occasionally associated with chronic pericarditis when the process works through the diaphragm to produce a capsular form of cirrhosis,—the so-called "Pick's syndrome." This was recognized in one case of the present series and should have been recognized in another which I published a good many years ago.*

There was very little pain in most of these cases, and when it was present there was no good reason to associate it with the pericarditis.

Cardiac murmurs were present in 64 out of 112 cases. 27 of these could be accounted for by endocardial lesions. Not infrequently, however, one sees post-mortem cases in which a well-marked diastolic or presystolic murmur has led to the diagnosis of valvular heart disease in life, yet in which *post-mortem* no valve lesion is found.

### DIAGNOSTIC MARPLOTS

Leaving out the 27 cases in which systolic murmurs alone were audible, we have left 37 cases in which one could hear a presystolic or a diastolic murmur or both. At necropsy the endocardium was normal in 5 of these. These presystolic murmurs were best heard at the cardiac apex and the diastolics along the left sternal edge.

These 5 cases constitute the deceptive group, the *diagnostic marplots* of the series. There were 4 males, 1 female. Ages 18, 28, 37, 38, 58.

There were diastolic murmurs in 4 of the 5 cases, presystolic alone in 1. The heart weights include the largest in our series (1328 grams) and average 749 grams. The adhesions were extensive in 3 out of 5. Mediastinitis was also present in one.

To increase the diagnostic difficulty of these cases a palpable thrill accompanied the murmur in 2 cases. Acute pericarditis was also present in 2. Three of these cases were included in the group called Primary Rheumatic (above).

* Obliterative Pericarditis a Cause of Hepatic Enlargement and Ascites, Boston Medical and Surgical Journal, May 19, 1898.

TABLE 157.—DIASTOLIC AND PRESYSTOLIC MURMURS IN CHRONIC PERICARDITIS

| Number | Necropsy number | Rheumatism | Age and sex | Thrill | Murmurs | Heart weight | Adhesions—slight or extensive | Mediastinitis | Ascites | Acute endocarditis | Acute pericarditis | Chronic passive congestion | Blood pressure |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 266 | + | 31. M | 0 | Systolic and diastolic | 1328 | Extensive | + | 0 | 0 | 0 | 0 | |
| 2 | 448 | 0 | 38. M | 0 | Systolic and diastolic | 427 | Extensive | 0 | 0 | 0 | 0 | 0 | |
| 3 | 1317 | 0 | 58. M | 0 | Systolic and diastolic | 827 | Extensive | 0 | + | 0 | 0 | + | |
| 4 | 1907 | + | 28. F | + | Presystolic at apex | 377 | Slight | 0 | + | 0 | 0 | 0 | |
| 5 | 3208 | + | 37. M | 0 | Systolic and diastolic | 705 | Slight | 0 | 0 | 0 | 0 | 0 | 240/140 |

The cause of these murmurs and thrills is a matter of speculation. We can class them with the "Austin Flint" group first described in connection with aortic regurgitation. But there was no evidence of aortic regurgitation in any of them. We can call them "functional" which merely covers up our ignorance of their cause. To me it seems best to stress especially their association with the hypertrophy and dilatation of the heart chambers, which is one feature of cases of chronic pericarditis.

I believe that in the group of cases first noticed by Flint it was the cardiac enlargement and not the aortic regurgitation which was most clearly related to the murmurs.

Other causes of cardiac hypertrophy, such as chronic nephritis, are also associated, now and then, with apical diastolic or presystolic murmurs.

The only important conclusion seems to be that *in cases of marked cardiac hypertrophy, apical or left parasternal murmurs, whatever their time, sometimes do not mean valve lesions* and should be very cautiously interpreted when occurring without other evidence of heart disease.

*Arrhythmia* was present in 25 cases out of 103 of this series, excluding 11 cases of arrhythmia associated with valve lesions, but was of no special diagnostic significance in relation to the pericarditis.

The pulmonic second sound was accentuated in 47 cases out of 82, the aortic second in 17.

Feebleness of the heart sounds was recorded in 22 cases, but in the great majority there was nothing remarkable about them.

*Friction sounds* were heard in 9 cases but in only one of these was any acute pericarditis found at necropsy. (cf p. 645.)

We have no reason to believe that chronic pericarditis can of itself produce pericardial friction, since this sign is not infrequently recorded in our cases when *post-mortem* examination later shows no pericarditis, acute or chronic, and no overlying pleuritis such as might cause a pleuro-pericardial friction. In some of these cases of unexplained pseudo-pericardial friction, there was an abnormal dryness of the tissues owing to persistent vomiting or other causes. Very possibly the heart within the normal but desiccated pericardium is capable of giving rise to friction sounds.

*Ascites* was present post mortem in 38 cases. In nine of these it was probably the result not so much of the pericardial adhesions as of the accompanying valve lesions. In the remaining 29, the ascites was part of a general dropsy in 17 and in one of the remainder it was presumably due to a pancreatic cancer with metastases.

Only in two cases of this whole series (Nos. 662, 1907) was there present at the time of necropsy any predominating or solitary ascites such as is seen in many cases of hepatic cirrhosis. Pick's syndrome is rare in chronic pericarditis.

The necropsy *cultures* were positive in 39 out of 112 cases, the streptococcus being found in 29, the bacillus coli in 4, the pneumococcus in 4, the bacillus mucosus capsulatus in 1, staphylococcus albus in 1. These cultures have no special connection with the subject under study and are to be regarded probably as evidence of a decrease in the general bodily resistance owing to the patient's exhaustion with the approach of death.

## SUMMARY AND CONCLUSIONS

I. (a) Chronic pericarditis, especially when extensive and accompanied by mediastinitis, may be the essential cause of an enlargement and weakening of the heart which results directly in death. Such cases are usually of known rheumatic origin and recur mainly in youths or young adults.

(b) The pericardial adhesions may be part of cripppling rheumatic infection which attacks also and perhaps with greater damage the heart valves.

(c) More often the pericardial adhesions may be found post-mortem in persons dying of some non-circulatory disease, so that the pericarditis is of little or no importance—a mere historical landmark or vestige of an earlier healed infection.

To these three types of the disease I have given the names:

1. Vestigial.
2. Primary rheumatic.
3. Secondary rheumatic.

The vestigial cases are mostly in elderly people (average age 53) and 4/5 of them were of the male sex with a relatively slight development of the pericardial adhesion and without mediastinitis.

The primary rheumatic type averages 28 years at death. Two-thirds of the patients are of the male sex. In the secondary rheumatic group the average age is 32 and the sexes are about evenly divided. It is in these two groups (which make up together 48 or nearly 1/2 of the 112 cases in this series) that the pericarditis is of importance, sometimes dominating, always contributing to the clinical picture.

II. Only 6 cases in 112 of all three types were recognized in life; the X-ray thus far gives us no help.

III. Diastolic and presystolic murmurs occur in many of the cases and lead to so many erroneous diagnoses that primary rheumatic pericarditis deserves the name of a "*diagnostic marplot.*" Most of these deceptive murmurs occur in patients with very large hearts.

IV. Chronic pericarditis produces in young rheumatic patients the largest hearts known.

V. Only in 2 cases out of 112 was the presence of ascites without general dropsy a notable feature.

## ILLUSTRATIVE CASES

### Necropsy 4502

An American of seventy, formerly a whaler and fisherman, for five years a laborer, entered March 15, 1923. One sister died of tuberculosis. Except for the usual diseases of childhood he could remember no illnesses. At fifteen he had a questionable chancre, rash, sore patches in the throat and pustular scabs on the tibiae. At thirty he had gonorrhea.

For four months he had had dyspnea and weakness accompanying any emotion. Four weeks before admission his lower legs began to itch and he noted an eruption of small papules, and some time after

this of weeping and scaling lesions on the anterior surface of the tibiae. This spread over the calves. Because of itching he pulled off the scabs and some pieces of dead skin. The ulcerations had been in their present condition for three weeks. Since they had ulcerated he had been unable to walk or to take care of himself and had become very filthy. Slight efforts tired him, and the dyspnea and weakness on emotion were much worse. His feet swelled occasionally. His physician reported that a Wassermann by the State Board of Health was positive.

Examination showed a thin man with dry, scaling and very hard skin (fish skin), somewhat pigmented. The lesions on the lower legs involved the superficial subcutaneous tissue. The edges, though ragged, were not undermined, but rather sharply punched out and precipitous. The shallow base was covered by desquamating dead skin, crusts of dried serum and yellow dry doughy material. The ulcerations were confluent and formed a large area extending around the leg from just below the knee to the middle of the tibia. Several narrow areas extended down the back of the leg to the heel. The lesions had a vile odor. The mucous membranes of the pharynx were injected and the tongue was covered with a dry brown coating. All the superficial lymph nodes were palpable. The apex impulse of the heart is not recorded. The measurements by percussion were, supraclavicular dullness 5 cm., right border 5 cm., left border 8 cm.; the midclavicular line was 10 cm. A loud systolic murmur was heard all along the left border and across the sternum radiating to the right axillary line, loudest at the xiphoid and stopping very sharply 5 cm. to the left of the sternum, not heard at the apex. The veins in the neck and arms pulsated markedly at a different rate from that of the brachial artery. The latter and all the visible arteries were markedly tortuous and thickened. The blood pressure was 145/75. The lungs were not abnormal except for occasional crepitant râles at the apices and bases posteriorly. The abdomen was protuberant and very tympanitic. Dullness was found rather high in the flanks. The liver edge could be felt just below the costal margin, smooth, not tender. There was a moderate amount of subcutaneous edema over the back and chest, a slight amount in the legs. The extremities showed slight tremor. The pupils were irregular, reacted to light and distance. The left was smaller than the right. The inferior turbinates (?) obstructed the breathing through the nose somewhat. The knee-jerks were equal. There was questionable Romberg; no clonus, Babinski or Kernig.

The temperature was 96.7° to 100°, with a terminal rise and drop to 104.3°–100.4°. The pulse was 77 to 98, with a terminal rise to 120. The respiration was normal except for a terminal rise to 30. The output of urine was 32 to 30 ounces when recorded, the specific gravity 1.030 to 1.018. There was no albumin. A few leucocytes were seen at one of three examinations. The renal function was 0. The hemoglobin was 85%, the leucocyte count 10,800 to 33,000, with 89% polynuclears. There was moderate anisocytosis. Two Wassermanns were negative.

After three days' use of Dakin's solution the odor from the ulcers had decreased remarkably. The application of the solution caused some pain. The lips appeared slightly cyanotic. Dyspnea was marked. All movements grew increasingly difficult. By March 20 the ulcers were quite clean and granulations were growing up from the surface with great rapidity. The edges were smoothing off and a delicate line of new epithelium was working over the sloping base from the edges.

The next day the patient's jowls were swollen. The potassium iodid was immediately decreased. That day the heart weakness was more evident than ever before. March 22 the patient was failing rapidly. The edema of the face, hands, back and chest was somewhat increased. He was put in semi-Fowler position. The heart sounds were less clear and the action weaker. That afternoon he began to cough up frothy sputum, and moist bubbling râles were heard in the lower chest, especially over the bases laterally. He could not be moved without great discomfort, so the back was not examined. The liver was not tender. There was marked passive congestion of the lungs, but no pneumonic patches were noted. The abdomen was distended with gas. The patient improved a great deal after atropin and the respiratory discomfort subsided somewhat. An enema and stupes also helped.

The night of March 25 the temperature rose suddenly. Nothing was found. The next morning however a rather cyanotic red area was found arising around the left ear and extending on the temple involving the left eyelids, part of the cheek, and all of the ear. The border was sharp and raised. The area was tender. The leucocyte count rose to its maximum. That day the patient died.

*Clinical Diagnosis (from Hospital Record).*—Cardio-renal disease.
Syphilis?
Erysipelas.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic nephritis.

Arteriosclerosis.
Hypertrophy and dilatation of the heart.
Erysipelas (streptococcus septicemia).
Passive congestion of lungs, posssibly of liver.
Pneumonia?
Acute pericarditis?
*Anatomical Diagnosis.*—Arteriosclerosis.
Chronic adhesive pericarditis.
Hypertrophy and dilatation of the heart.
Chronic passive congestion, general.
Serofibrinous pleuritis.
Serofibrinous peritonitis.
Fatty metamorphosis of the liver.
Marked edema of the lungs.
Chronic pleuritis.
Obsolete tuberculosis of a bronchial gland.
Tumor of right epididymis.

DR. RICHARDSON: The legs were bandaged, and I did not remove the bandages. I have no doubt he had erysipelas and the other things on the surface of his skin.

The peritoneal cavity contained 1500 c.c. of thin cloudy fluid and fibrin and some reddish fibrin scattered over the peritoneum. That is a serofibrinous peritonitis, terminal of course, in association with the other expressions of infection.

In the right pleural cavity there was a serofibrinous pleuritis, another expression of the same thing, and a few old adhesions besides. The left cavity was obliterated by old adhesions.

The gastrointestinal tract showed congestion, and we can leave it that way.

One bronchial gland showed obsolete tuberculosis, and there was a mass in one of the testes which was fibrocalcareous. And then the the question arises, is it tuberculosis or is it syphilis. He had tuberculosis somewhere else, and I think possibly that was an old tuberculosis.

I could not make out any pneumonia in the lungs. There was congestion.

The pericardium was obliterated by old adhesions,—chronic adhesive pericarditis, which occurred many years ago.

The heart weighed 660 grams. The valves showed some of the usual sclerosis associated with age, but otherwise were out of the picture. The heart in general was hypertrophied and dilated.

The myocardium generally was rather thick, five mm. on the right, fifteen mm. on the left. Back of that all I found was arteriosclerosis. The sclerosis did not begin in the ascending thoracic, in fact was not observed until we came to the descending thoracic. From there on there was plenty of it. That distribution of course is rather against syphilis.

There were a few bands of adhesion between the liver and the diaphragm, perihepatitis and congestion of the liver, but no definite cirrhosis. The liver was a little enlarged, the vessels engorged.

The kidneys weighed 340 grams,—certainly not those of chronic nephritis. Here the condition seemed to be chronic passive congestion. The vessels were a little prominent. There was a good width of cortex. The kidneys showed nothing that we could put our hands on except as mentioned.

The prostate, seminal vesicles and testes were negative except for the fibrocalcareous mass mentioned. As a matter of fact I did try to decalcify that, and have some sections, but it would bother us a little to say whether the sclerosis present was the end result of tuberculosis or of syphilis. He had one tuberculous lesion, and I do not see why we should not let it go as old tuberculosis. I think the laboratory report of the kidney function must be wrong.

Dr. Cabot: We are accustomed to divide our post-mortem into three general groups, the underlying cause, the terminal infections supplying the factor necessary to turn the scale,—erysipelas, pleuritis—and then the historical landmarks, things mentioned in a conscientious account of everything that was found, having nothing whatever to do with the case, such as this tuberculosis of the lymph glands. In old times pathologists used to try to be entirely dispassionate and not arrange their findings at all,—put them in alphabetical order. It seems to me we are trying to do better in trying to make these reports mean something and supply a basis for our efforts during life.

Serofibrinous peritonitis was present. Nothing is said about tenderness or about spasm, which we so often see as part of an infectious process. We did not suspect in any way adhesive pericarditis, and we shall do just the same thing next time probably. We do not diagnose those things.

The zero reading in the renal function test was probably due in part to failing absorbtion of the dye owing to edema.

Case 4475

An American slaughterhouse foreman of fifty-six entered January 19, 1923, for relief of weakness a year and a half in duration.

His father and one son died of diabetes. An aunt died of tuberculosis. His wife had three or four miscarriages. Several children died in infancy.

Whooping cough was the only disease he remembered in childhood. At fifteen and again at thirty-seven he had inflammatory rheumatism, the first attack lasting two months, the second a whole winter. As long as he could remember he had had attacks of "biliousness," formerly every two weeks, of late years rather infrequently. For two or three days before the attacks his skin and eyes became yellow and his urine very dark. The attacks began with dimness of vision, than severe pain over both eyes. In a few hours he vomited green material and the attack was over. He had a cold sore removed from his lip seven years ago. One gland on the left side of the neck swelled occasionally. His bowels had always been constipated. He sometimes urinated once at night.

He took beer very moderately. Best weight 250 pounds, eight years ago; usual weight 230–240, weight a week ago 205.

Four years before admission there was gradual onset of pain in the lower back, fatigue, and a feeling of fullness and discomfort in the epigastrium. This last was constant and was always made worse by food. With it was marked jaundice. His bowels were constipated. These symptoms lasted four months, during which time he did not work. Finally on account of increasing severity of the symptoms he went to an osteopath. After treatment the pain and jaundice disappeared and he went back to work. The pain had never reappeared. He was then entirely well for a year except that he had a very sore tongue for three or four months. Three years ago following domestic trouble he became very nervous and fretful and lost weight. The nervousness and also the domestic trouble had persisted more or less to the present time. He also began to have a feeling of weakness, bad for a year and a half. Fifteen months ago he began to have pain in midsternum, only after exertion or hard work. With this there was also pain in the left forearm from the elbow to the wrist. He was very weak, did not feel right, and he was told that he looked queer. He felt chilly easily and got out of breath on moderate exertion. After two months he recovered completely. Eight months ago the symptoms returned, and with them jaundice. Again after two months he was well. Very soon after this he had another

breakdown from which he quickly recovered. From six months ago to four months ago he was well and worked every day. Four months ago the present attack began with jaundice, weakness, dyspnea and midsternal pain on exertion, palpitation and pounding in the ears. With this attack his fingers and toes were numb. For the past two weeks his appetite had been very poor and he vomited easily. For ten days he had been coughing and raising thick yellow sputum.

Physical examination showed a well nourished white haired man looking exhausted. Scattered tiny petechiae on the upper chest. Small white round scars on the anterior chest wall. Pigmented scars on the left lower leg and a purplish depressed scar on the right lower leg. Skin and sclerae pale and yellow, the sclerae more yellow at the periphery and slightly injected. One petechia noted under the conjunctival surface of the lower lid. Slight pyorrhea. Tongue margins very smooth. Papillae atrophied on margin. Atrophied areas on under surface and a few petechiae. Moderate discrete enlargement of the cervical and axillary glands. Chest: abnormally wide intercostal angle with depression above a wide upturned xiphoid. Apex impulse of the heart not localized. Sounds of rather poor quality. Soft blowing systolic murmur all over precordia, loudest at apex and base. Blood pressure January 19, 108/35, January 29, 132/60, February 3,90/45, February 16,112/50. Electrocardiogram. Normal rhythm. Rate 90. Diaphasic $T_2$. Abdomen negative except for slight retraction in the epigastrium. Pupils normal. Fundi. Both showed pale, hazily outlined small hemorrhagic areas scattered throughout, some apparently absorbing, others darker and and fresher. Reflexes. Knee-jerks hyperactive, with clonic contractions. Poor position and motor sense in both legs and arms. Slight numbness of toes.

Temperature 96.9°–100.8° except for two periods of elevation, January 19 102°, February 12–18 98.1°–106.5°. Pulse 71–160, rising with the rise of temperature. Respiration 16–29. Urine ℥ 25–80. Sp. gr. 1.015–1.020. Slight trace of albumin at one of two examinations, rare to occasional leucocytes at both. Blood January 19. Hgb. 40%, leucocytes 3200, polynuclears 56%, reds 1,030,000,–1,120,000, marked anisocytosis and poikilocytosis, many large red cells well filled with hemoglobin, considerable variation in staining, many microcytes, platelets very rare, reticulated cells 1.9%. January 23. Hgb. 40%, leucocytes 2200, reds 1,500,000, reticulated cells 1.7%. January 27. Before transfusion hgb. 40%, reds 920,000, reticulated reds 2%. After transfusion hgb. 40%, reds 1,300,000.

February 1. Hgb. 50%, leucocytes 4400, reds 1,320,000, reticulated reds 0.3%. February 7. Hgb. 60%, leucocytes 4100, reds 1,300,000, reticulated cells 2.8%. February 10. Before transfusion hgb. 50%, leucocytes 6400, reds 1,350,000. After transfusion hgb. 60%, reds 1,750,000. February 14. Hgb. 60%, leucocytes 3500, reds 1,900,000, reticulated cells 1.1%. February 17. Leucocytes 2500. Serum dilution 1:60. Clotting time 18–25–24–24–20 minutes. Fragility. Hemolysis began at 0.46 and was complete at 0.26. Throat exudate examination on dark field showed no spirochetes or organisms of Vincent's angina. Vital capacity 4300 c.c. Basal metabolism +21%. Gastric analysis. Fasting contents. 33 c.c. yellow-white mucous material. No free HCl. Total acid 5. Guaiac positive. 3–4 red blood cells and 2–4 leucocytes to a high power

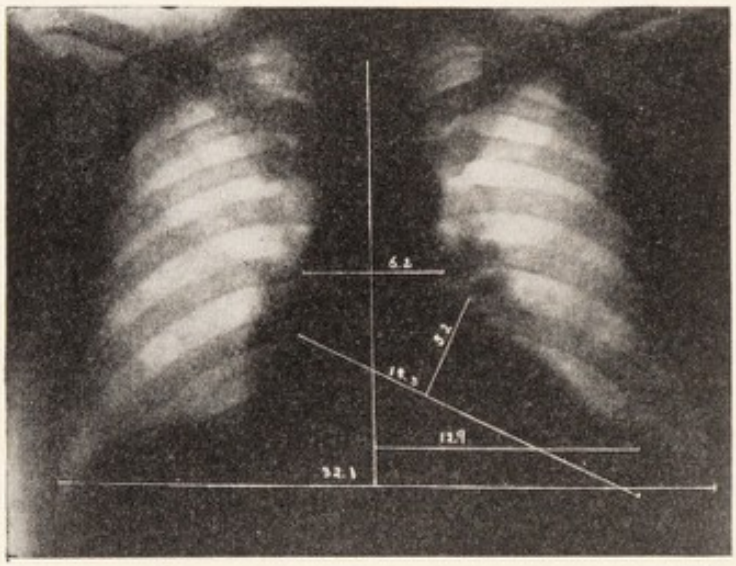


FIG. 141.—Pernicious anemia with hypertrophy and dilatation of the heart. Definite increase in transverse measurement both to right and to left. Cardiac shadow not definitely abnormal otherwise.

field. Test meal. 24 c.c. whitish bread remnants. No free HCl. Total acid 4. Guaiac positive. X-ray. Heart as in illustration. No evidence of pathology in any part of the gastro-intestinal tract. February 17 left frontal sinus distinctly less radiant than the right. Antrum on this side also a little cloudy.

January 27 transfusion of 600 c.c. was done. There was very slight reaction after it. Next day he felt great subjective improvement which continued for several days. February 10 a second transfusion was done. Following it he had fever. February 16 he complained of sore throat and a very sore tongue. On the soft palate

were several small areas of superficial ulceration. The right half of the tongue was markedly swollen, red, and extremely tender. The next day the tongue was more swollen and there were several very tender submaxillary nodes on both sides. During the day marked edema of the neck developed. The saliva showed many streptococci. That night he was much worse. The tongue filled the whole mouth and pharynx, causing obstruction controlled by a tube. Early the next morning, February 18, he died.

*Clinical Diagnosis.*—Pernicious anemia.
Streptococcus septicemia?
Acute glossitis.
Acute cellulitis, mouth and neck.

*Dr. Richard C. Cabot's Diagnosis.*—Pernicious anemia.
Streptococcus septicemia with glossitis.
Hypertrophy and dilatation of the heart.

*Anatomical Diagnosis.*—Pernicious anemia.
Septicemia, streptococcus hemolyticus.
Hyperplasia of bone marrow.
Hematopoiesis of the liver and spleen.
Chronic adhesive pericarditis.
Arteriosclerosis.
Hypertrophy and dilatation of the heart.
Acute glossitis.
Edema of epiglottis.
Hemorrhagic edema of lungs.
Wet brain.
Fatty infiltration of the pancreas.
Myoma of stomach.
Chronic pleuritis, localized, slight.
Obsolete tuberculosis of one bronchial lymph node.
Cholelithiasis.

DR. OSCAR RICHARDSON: I think we can put the anatomical basis under most of the questions that have arisen.

We examined the head in this case. The pia showed a little edema, but the vessels of Willis, sinuses, and middle ears were negative, and the brain tissue outwardly showed no definite lesions..

The spinal cord outwardly showed nothing definite, but as Dr. Cabot said, things can look all right outwardly. In this case however the cord showed no lesions either macroscopically or microscopically. The bone marrow of the femur showed the typical picture of the marrow of pernicious anemia.

The skin showed a pale brownish-yellow sallow color, a peculiar color—something of the pernicious anemia color and a little bit more, but not what we should call a very definite jaundice. In the cubital spaces there were purplish spots,—the transfusion punctures. In a few places there were minute pale purplish spots. Just below the jaw on the right the subcutaneous tissues were slightly swollen. This man had a perfectly definite glossitis and a streptococcus septicemia. The culture from the heart blood showed a typical growth of the streptococcus hemolyticus.

The mucosa of the stomach seemed a little pale; but we have been unable to demonstrate definite lesions in any of the examinations made on stomachs in pernicious anemia.

The intestines and glands were out of the picture. There were a few old pleural adhesions on each side; no fluid in the cavities. The thyroid gland was negative. No thymic tissue was found. The bronchial glands were slightly enlarged, and one on the right showed obsolete tuberculosis. The lungs were edematous, the tissue spongy, saturated with thin red bloody fluid which streamed from the section surfaces,—a frank hemorrhagic edema of the lungs such as is associated usually with a streptococcus infection.

The pericardial cavity was obliterated by dense tough old membranous adhesions,—a perfectly clear cut chronic adhesive pericarditis. The heart weighed 520 grams,—considerably enlarged. (Normally 200–400.) There were three factors here. (1) The chronic adhesive pericarditis. That in and of itself, with no associated chronic mediastinitis or pleuritis, would not be sufficient to produce a heart of 520 grams. (2) There was a little arteriosclerosis. (3) The third factor responsible for the hypertrophy and dilatation of the heart was pernicious anemia.

The arteriosclerosis in this case was interesting. There was a slight amount in the aorta and great branches, but the coronary arteries showed well defined fibrocalcareous sclerosis with much thickening of the walls and some decrease in the lumen. The valves were negative except for increase of the circumferences.

The liver was rather large and pale. The gall-bladder added another interesting fact in this case. It was moderately distended with bile and contained at least 200 small concretions. At the time of necropsy the bile ducts were free and showed no particular dilatation.

The pancreas was of good size and on section was seen to consist of fatty tissue in which were embedded here and there islands of pancreatic tissue,—so-called fatty pancreas or lipomatous pancreas,

we used to think associated with sugar. We have got over that. There was a soft hyperplastic spleen, due in part to the septic process and for the rest to the pernicious anemia.

The kidneys were rather large, weighing 480 grams (normally 200–400), but they were normal. The gastro-intestinal tract was out of the picture.

DR. CABOT: Dr. Holmes, I should like to ask you, with the facts that you have on the fluoroscopic report and the X-ray plate here, do you see any evidence from which you might have suspected adhesive pericarditis?

DR. G. L. HOLMES: In the note there is just one statement; that is the indefinite outline of the heart. The X-ray findings indicative of adhesive pericarditis are: (1) Limited respiratory movements; the left border of the heart as a rule does not move to the same extent that it normally does. That is not indicated on this tracing. Whether they were unable to get any motion or whether they failed to make the record I do not know. (2) The next important finding is the obliteration of the various chambers, so that we are unable to differentiate the auricle from the ventricle. (3) The third is the indistinct pulsation. Apparently they did get some of that.

DR. CABOT: But as to the chambers you did not make any observation. There is no reason why the respiratory mobility of this heart should be diminished? It was not hitched up to the pleura or mediastinum?

DR. RICHARDSON: No. The great pull comes when it is hitched up with mediastinitis and pleuritis.

DR. CABOT: There is no reason why this heart should not go up and down as well as any other way?

DR. HOLMES: I may be wrong about this. I have never had any definite proof. But I believe that as a heart moves up and down it rotates, and any adhesion to the surface of the heart would limit its movement.

DR. RICHARDSON: You say *rotation*. One would think it would lift up.

DR. CABOT: Can you see the rotation in the fluoroscope?

DR. HOLMES: We can see the change in shape which corresponds. So I should think this case would have given some evidence of limitations of respiratory movement.

### Necropsy 3738

A physician of forty-seven entered March 30. One brother had an arrested case of tuberculosis. The patient had had no definite exposure. His general health had been good. For the past twenty-five years he had never been ill.

Since March 3 he had had gradual onset of malaise, backache, and pain in the head, with chilly sensations and irregular temperature, sometimes as high as 102°. He had been up and about most of the time, growing steadily worse. March 21 he went to bed. For the first three days his temperature was usually 101° to 102°, with occasional rises to 104° and frequent sweats. March 24 the leucocytes were 19,000. March 25 a Widal was negative (paratyphoids?). March 26 the leucocyte count was 9,000. A blood culture was negative. He had severe pain in the abdomen, somewhat relieved by a hot water bottle. Half an hour later he had a severe chill, after which the temperature was 103°. After this he had repeated chilly feelings and sweats, with temperature normal in the mornings and ranging up to 103° in the evening.

Examination showed a fairly well developed, emaciated man, unresponsive and apparently very ill. The skin was dry, with a small firm purpuric nodule on the left thigh. The mucous membranes were dry and pale. The lips showed sordes. The sclerae showed questionable slight icterus. The tongue had a furry white coat. The heart showed no enlargement. The action was slow and regular. The sounds were distant. The pulmonic second sound was greater than the aortic second. The first sound at the left border of the sternum was rumbling. There were questionable systolic and presystolic murmurs at the apex and the left border of the sternum. The blood pressure was 95/70 to 78/45. At the apex of the left lung anteriorly was slight dullness with diminished breathing and voice sounds; no râles. The abdomen showed slight general spasm. The right kidney was easily felt, not tender. The genitals, extremities, pupils and reflexes were normal.

The temperature was 97.5° to 106°, the pulse 78 to 162, the respiration 10 to 44. The amount of urine was 10 to 52 ounces. The urine was pink at entrance, cloudy at four of twelve examinations. The specific gravity was 1.009 to 1.026. There was the slightest possible trace of albumin at two examinations, a trace of sugar at one. The sediment showed hyalin casts at two examinations, granular and rare waxy casts at one, an occasional cylindroid with red blood corpuscles attached at one, red blood corpuscles at another. The hemoglobin

was 90%. There were 33,000 to 12,600 to 60,400 leucocytes. A blood culture was negative. March 30 a Widal was positive in 1/10 and 1/50 dilutions. Loss of motility was slow and not complete. Agglutination was complete. Paratyphoids α and β remained free and motile. The patient had had typhoid inoculation six months before admission. The renal function was perhaps 40%; the color was very bad. The blood nitrogen was 30 mgm. per 100 c.c. of blood. A throat consultant found nothing in the nose and throat to account for the symptoms. A dental consultant found no involvement of the right antrum. The lower first molar showed large definite foci for absorption. There were no other findings to account for the temperature. X-ray showed enlarged dense glands at both lung roots, mottled shadows at the right apex, and thickening of the markings running to the apices on both sides. The excursion of the diaphragm was fairly good on both sides. The left side was a little higher than normal. The conclusion was, tuberculosis of the right apex. Pus pockets were found at the roots of two teeth. The right antrum and right frontal sinus were cloudy.

Two days after entrance the temperature was 103° and the pulse 140. A systolic murmur was heard definitely at the apex and the left border of the sternum. April 8 a right lower molar was extracted. A pus sac was found between the roots and a small osteomyelitic area below the roots. It was thought this area might account for the temperature.

April 13 the patient was irrational, then semicomatose. Although he could not be roused he was sensitive to pain, and showed very slight wincing on pressure of the right mastoid tip and more on pressure over the right molar. The left knee-jerk was increased. There was no definite Babinski. There was clonus on the left, not well sustained, and very suggestive Kernig. The neck was stiff. Lumbar puncture gave clear fluid showing 27 red cells, no white cells, a few diplococci of pneumococcus morphology, possibly contamination. Culture from the spinal fluid was negative. After the lumbar puncture the patient showed a somewhat clearer mental condition. A friend now said that before entrance he had complained at times of severe headaches and had said that he felt as he imagined a patient with meningitis would feel.

April 15 he was unconscious (?). Later in the day he recognized his sister. Fifteen c.c. of spinal fluid was removed and 30 c.c. of anti-meningococcic serum returned. Next day 12–15 c.c. was removed and 20 c.c. of serum returned. A slight chill followed the

injection. April 17 18 c.c. of cloudy fluid was removed at a pressure of 110. The fluid showed 500–600 cells, 90%–95% polynuclears. The rest was largely made up of mononuclear cells resembling endothelial phagocytes. No organisms were demonstrable.

April 18 the patient seemed brighter. Right facial paralysis was more marked than at any time previously. The knee-jerks were present and about equal. There was neck sign, marked Kernig, and indefinite Babinski. Next day he was much brighter than at



FIG. 142.—Section of the heart valve in Case 3738, showing the masses of bacteria impinging on the valve. (× 225.) (Photomicrograph by Lewis S. Brown. Dr. William H. Smith.)

any time since the 14th. He said he felt comfortable and had no headache, though he had had some. Dr. Cushing thought the condition due to obscure sepsis rather than brain abscess, and the left facial paralysis a peripheral palsy rather than central. The temperature was subnormal in the morning, the pulse 100. Lumbar puncture gave 10 c.c. of clear fluid at a pressure of 90. Next day the patient had a chill. He voided without catheterization for the first time in days. Lumbar puncture gave 10 c.c. of clear fluid. Ten c.c. of serum was injected. General trembling followed. A systolic bruit, possibly transmitted from the aorta or a transmitted murmur

from the heart, was noted. Between April 15 and 21 the white count ranged from 19,600 to 60,400.

April 21 lumbar puncture gave 8 c.c. of slightly yellow fluid under no increase of pressure, showing 110 cells. April 22 the temperature again reached 102°. The breathing was somewhat Cheyne-Stokes. April 23 the patient was bright and talkative. The temperature was 102°, rising in the evening to 104°. The patient was delirious. April 24 750 c.c. of blood was transfused. Later the patient had a chill, and next day three more. The temperature ranged from 98°–105°. The mental condition was at times cloudy, occasionally clear.

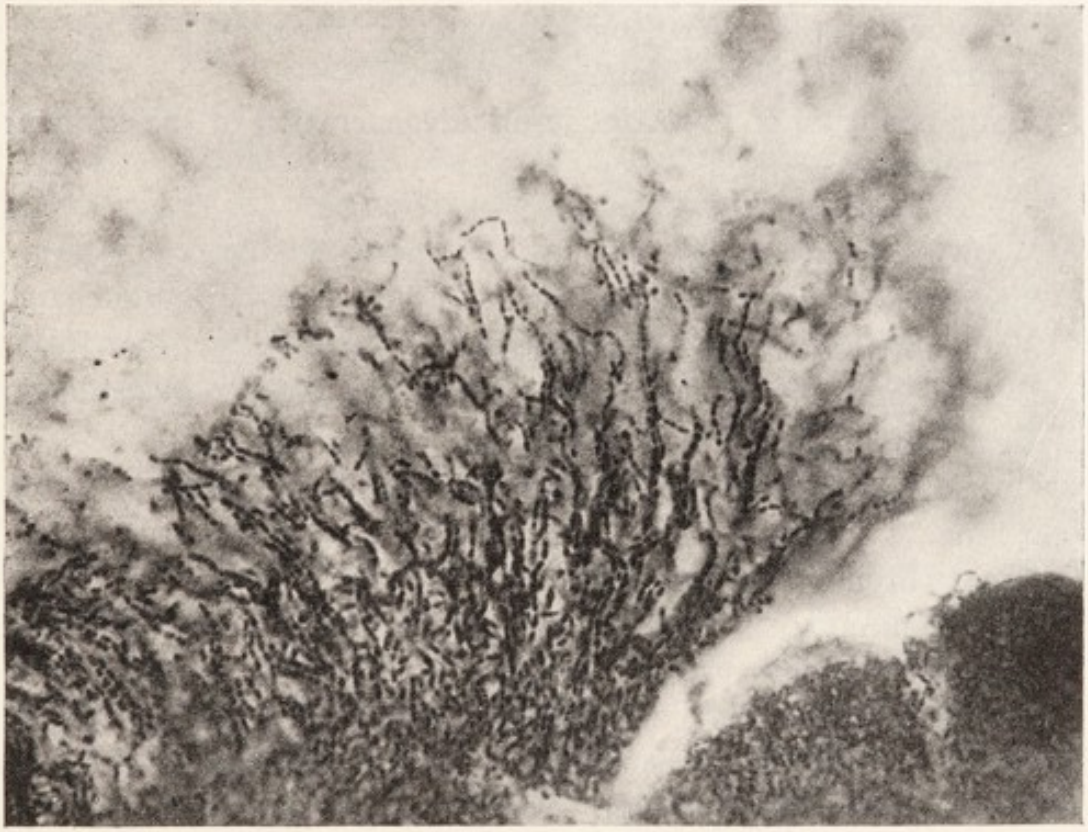


FIG. 143.—Detail of Fig. 142. (× 1000.) Small capsulated streptococcus. (Photomicrograph by Lewis S. Brown. Dr. William H. Smith.)

The urine showed occasional red blood corpuscles, no albumin, sugar, or casts. One blood culture showed staphylococcus albus and questionable spore-bearing bacilli. Another showed no growth. The rises in temperature after the chills were now as high as 106°. May 6 the lungs were full of non-consonating and consonating râles both front and back, with no definite areas of dulness. A loud harsh systolic murmur was heard over the back and in both axillae. The patient grew progressively weaker, continued to have chills, and was a little irrational. Purpuric spots developed at every point touched by the bed clothes. The lungs gradually filled up. May 23 he died.

*Clinical Diagnosis (from Hospital Record).*—Malignant endocarditis.

*Dr. William H. Smith's Diagnosis.*—Septicemia.

Purpura.

Endocarditis of the mitral valve, chronic or acute or both.

Terminal pneumonia or infarction of the lungs.

*Anatomical Diagnosis.*—Chronic adhesive pericarditis.

Chronic interstitial myocarditis with necrosis.

Slight chronic endocarditis of the mitral valve.

Acute endocarditis of the mitral valve.

Hypertrophy and dilatation of the heart.

Streptococcus infection (small capsulated form).

Infarcts of the spleen, kidneys, and brain.

Acute nephritis and arteriosclerotic degeneration of the kidneys.

Purpura.

Chronic passive congestion, general.

Hydrothorax.

Slight ascites.

Slight fatty metamorphosis of the liver.

Edema piae.

Decubitus.

Obsolete tuberculosis of the bronchial glands and the apex of the right lung.

Slight chronic pleuritis, right.

DR. RICHARDSON: This was a case of infection with a minute capsulated streptococcus.

The culture from the blood stream in this case was taken from the inferior vena cava, and was negative. Usually in these cases we get cultures from the blood stream. Once in a while we do not, but may at times get a culture from the spleen. I think the reason is that the organisms get into the blood stream and then disappear from it. The peculiarity about the organism from the anatomical standpoint was its apparent association with septic infarcts produced in various parts of the body.

Polypous endocarditis of the mitral valve means simply that there was a large mass of vegetations there. As a result of this we found infarcts of the spleen, kidneys and brain.

It was a straightforward, definite picture of infection and the production of septic infarcts throughout the body.

The heart did not show much hypertrophy and dilatation; it weighed 356 grams (normally 200–400). The most striking thing

about it was the condition of myocarditis. As far as the coronary arteries could be examined they were negative. The myocarditis was probably of infectious origin. That brings us to the question as to how that was brought about. The vessels probably became plugged with these bacteria, a metastasis of these masses of organisms extending into the vessels, with consequent degeneration and replacement of the muscle tissue by fibrous tissue. In some places the tissue was cut off so sharply that it was necrosed.

The spleen in these cases usually shows the largest infarcts. There was a large infarct in this one. I have seen a very large spleen consist practically of one great infarct.

The microscopical examination of the cardiac valves showed masses of these bacteria, beautifully stained by Dr. Smith. (See Figs. 142 and 143.)

Examination of the myocardium showed the replacement by fibrous tissue of the muscle, and in places necrotic material.

It was a typical picture of the end results of infection by this particular organism, the character being that of the production of septic infarcts, metastasis from some common source, which in this case seemed to have had its main depot on the mitral valve.

### Necropsy 4152

An unmarried American nursery governess of nineteen entered November 5, 1919, for relief of exhaustion. Her general health was not very good. She had had whooping cough, measles and varicella. She had rheumatic fever four times between the ages of eight and twelve, confining her to bed with painful, red, tender, swollen joints. She had had tonsillitis four or five times, confining her to bed from one to three weeks. A hospital reported that her tonsils and adenoids were removed in 1913. "At the time of discharge the rheumatism had cleared up, but the heart condition remained the same."

She had always been subject to nosebleeds, and once a month had severe attacks lasting intermittently for several days. These had been less for the past year. Two years before admission she was ill five weeks with "pleurisy." Every week or two she was ill in bed for a day with frontal headache, with vomiting at intervals all day and swimming of the eyes. These attacks had been less frequent recently. She had been told she was yellow after some of these headaches. She had been at a nerve sanatorium for the summer. Her catamenia were regular every three weeks, lasting five to eight

days with profuse flowing. Previous to six months before admission it sometimes ceased for a week and returned with four or five days of further flowing. She was very weak after periods. There was no family history of excessive bleeding of any sort. So far as she knew she was normal at birth, with no bleeding from the cord.

A year before admission she began to have less strength and to be tired all the time. She did not lose color. She found that her gums bled easily on brushing her teeth. Often she found a blood spot the size of a fifty-cent piece on the pillow in the morning. She seemed to bleed easily. A small cut might bleed half an hour. After her tonsillectomy (see below) she bled for several hours.

Examination showed a well nourished girl. The gums were not spongy. There was no bleeding. The tongue was protruded in the midline with slight tremor. The throat was red. The tonsils were ragged, reddened, with pus pockets. The lungs were clear. The apex impulse of the heart was seen and felt in the fifth space 7.5 cm. to the left. The percussion measurements were 9 cm. to the left of midsternum, 2.5 cm. outside the nipple line, 2.5 cm. to the right, supracardiac dullness 4.5 cm. The pulmonic second sound was slightly accentuated. The pulses and artery walls were normal. The systolic blood pressure was 125, the diastolic 80. The abdomen, genitals, extremities, pupils and reflexes were normal.

The temperature was 98° to 99.4°, the pulse 74 to 93, the respirations normal. The output of urine was 19 to 31 ounces, the specific gravity 1.012 to 1.030. The urine was cloudy at all of three examinations, alkaline at one. The hemoglobin was 70%, the leucocytes 5000 to 8200, the polynuclears 82%, the reds 5,272,000, the smear normal. There was ½% reticulated cells. A Wassermann was negative. X-ray showed the respiratory movements of the diaphragm normal, its outlines regular. The costophrenic angles were clear. The heart shadow was enlarged in the region of the left ventricle. The hilus glands were increased and some apparently were calcified. There was also considerable thickening of the markings running upward and outward on the left side. The changes did not reach the apex or the periphery of the lungs. The appearance was that of a peribronchial process with enlarged bronchial glands.

A culture from the tonsils showed no hemolytic streptococci or diphtheria bacilli. November 10 she was discharged unrelieved.

January 21, 1920, she reentered for examination and tonsillectomy, having had a blood examination by Dr. Bock a month earlier showing 6,696,000 reds, hematocrit reading 33% cells, 77% plasma;

hemoglobin (Palmer) 110%, polynuclears 64%, bleeding time 3 minutes, clotting time 17 minutes.

Examination was as before except as noted. The skin was very dark. The tongue was protruded without tremor. The pulmonic second sound was not accentuated. The apex impulse of the heart was 11 cm. to the left. The percussion measurements were 12.5 cm. to the left of midsternum, 5.5 cm. outside the midclavicular line, 3.5 cm. to the right, supracardiac dullness 6.5 cm. The blood pressure was 130/95. The temperature was 98° to 100°, the pulse 72 to 100, the respirations 16 to 24. The hemoglobin was 80%, the leucocytes 5200 to 15,800, the polynuclears 78%, the bleeding and clotting time as in December.

January 22 tonsillectomy was done under ether. Pathological examination showed no tuberculosis or tumor. January 27 she was discharged relieved to a convalescent home.

July 6, 1920, she returned for her third admission with the following note from a physician: "Since January 27 she has been well except for severe headaches of migraine type for three or four weeks. She has had less asthenia than before tonsillectomy. Her handkerchiefs have however shown purulent and occasional bloody mucus. This attack began July 1 after swimming, with headache, malaise, July 2 some precordial pain, with pulse 130 and fever 102.3°. Ice bag to chest relieved pain. Aspirin and aconite brought down fever. July 3 fever in a.m. 102.5°, reduced in p.m. to 99.8°. Pain severe in morning. At night severe precordial pain required two doses of morphia gr. ¼ by mouth. July 4 fever between 101.5° and 99°, less pain. The ice bag kept on continuously. July 5 fever in the morning, subnormal at noon. Pain—arches of feet complained of July 3, shoulders and knees July 5. Aspirin started, gr. x every two hours, July 4. Mixed vaccine given by a physician July 5."

Examination showed the throat and tonsils slightly injected. The apex impulse of the heart was seen and felt in the fourth space 12 cm. to the left, coinciding with the left border of dullness. The right border was 3 cm. to the right, the supracardiac dullness 6.5 cm. The action was rapid (114). The sounds were of fair quality. The pulmonic second sound was greater than the aortic second, not accentuated. There was a slight systolic murmur at the apex, not transmitted. The pulses and artery walls were normal. The systolic blood pressure was 105 to 115, the diastolic 65 to 80. Both shoulders, particularly the right, showed slight limitation and pain on

motion. The knees and ankles were slightly painful on motion. The examination was otherwise negative.

At entrance the temperature was 101°, the pulse 90, the respirations 28. Afterwards the temperature and pulse were as noted in the history, the respirations 16 to 30. The hemoglobin was 90%, the leucocytes 8400 at entrance, afterwards 5000 to 9000, the reds 3,950,000 to 5,120,000 the polynuclears 70%. A Wassermann was negative. X-ray showed the frontal sinuses rudimentary; no evidence of definite sinus involvement. Four molars were un-

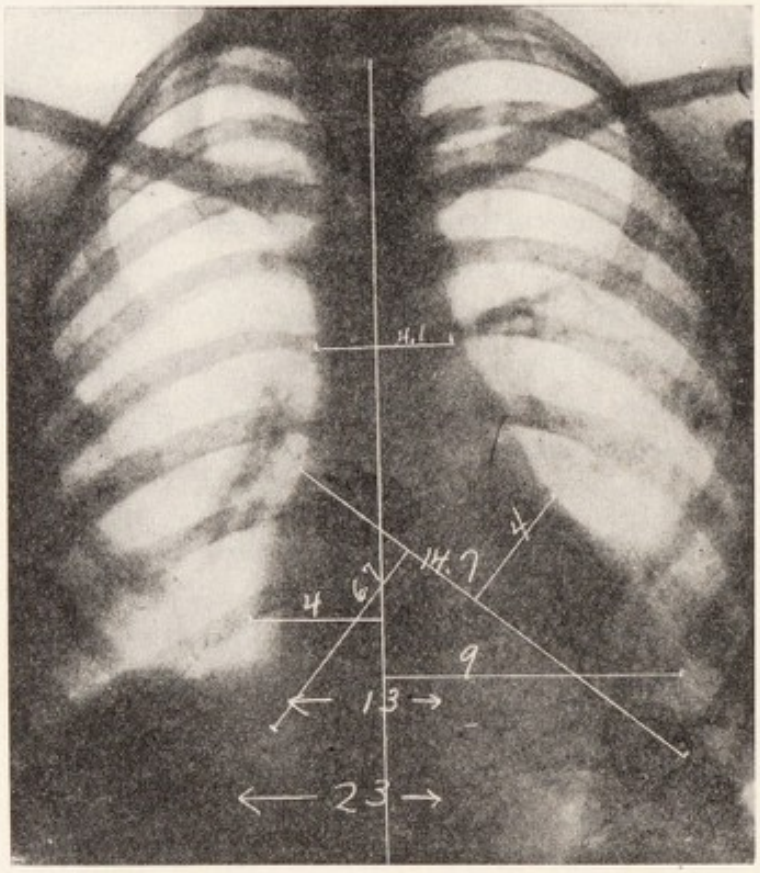



FIG. 144.—Necropsy 4152. Chronic adhesive pericarditis with hypertrophy and dilatation of the heart. Measurements by X-ray at seven feet six months before death. (Roentgenological Department, Massachusetts General Hospital.)

erupted, two rudimentary. A seven foot plate of the heart is shown in Fig. 144. There was moderate general increase in the heart measurements with some prominence in the auricular regions. Fluoroscopic examination showed the lung fields clear, the diaphragm moving freely on both sides; no definite evidence of active tuberculosis. The hilus shadow was considerably increased on both sides. There was evidence of enlargement of the bronchial and peribronchial glands. A throat consultant found nothing in the nose or throat to

suggest focal infection. A nasopharyngeal culture showed organisms like diphtheria bacilli. A blood culture was negative.

The patient was given soft solids. Fluids were forced. She was ordered aspirin gr. xv every two hours until toxic. The temperature was normal by the second day and remained so. July 18 the apex of the heart was 14 cm. from midsternum almost in the anterior axillary line in the fifth space. The action was regular and not rapid. The sounds were of good quality. There was a soft blowing systolic murmur at the apex transmitted to the axilla; also a definite suggestion of a diastolic at the apex in the left lateral prone position. In the recumbent position the left border was not more than 12 cm. from midsternum. The apex shifted normally with change in position. The lungs were negative. July 25 Dr. Edsall advised absolute rest in bed for a considerable period. Slight pain in the right shoulder persisted, and July 26 there was pain and stiffness in the left shoulder and right ankle. July 18 the patient was up for the first time, rather weak. August 8 the temperature had been normal for three weeks. When she began to get up for a few hours a day the temperature rose slightly, between 99° and 100°, the pulse 85 to 95. August 14 the systolic murmur at the apex was rougher than before. No diastolic was heard. The pulmonic second sound was reduplicated. August 18 X-ray of the chest was negative. That day Dr. Paul D. White found the heart practically as at the last examination.

August 27 she developed mumps, which had disappeared by September 5. She began to get up slowly, but developed a slight gastric upset with slight rise in temperature, passing off in a few days. September 24 there was another rise of temperature and pulse and she complained of an intermittent substernal ache lasting from a few minutes to an hour. She was kept in bed again. By the end of October she was able to be up three or four hours without elevation of temperature above 99° or pulse above 90. November 29 she was discharged.

On leaving the hospital she was taken in an automobile and was carried upstairs and put to bed. Next day she dressed, but did not walk about, and lay on a sofa without exerting herself in any way. The following day she had some pain in the joints with precordial pain and fever. The fourth day the pains increased, the temperature was 103°, and she felt very weak. These symptoms persisted.

December 4, 1920, she entered the hospital for the fourth time. Upon examination the apex impulse of the heart was very definitely palpable in the second space 8 cm. to the left of the midsternum in

the third, fourth and fifth spaces in the anterior axillary line. The cardiac dullness was increased. The action was rapid, with the first sound weak, the pulmonic second sound accentuated. A soft systolic murmur was heard over the precordia. There was no definite diastolic. The right pulse was much greater than the left; the latter was barely perceptible. The blood pressure was 100/60. The large joints were tender, especially the ankles. As before there was no swelling, redness or heat. She showed moderate jaundice. The examination was otherwise as before.

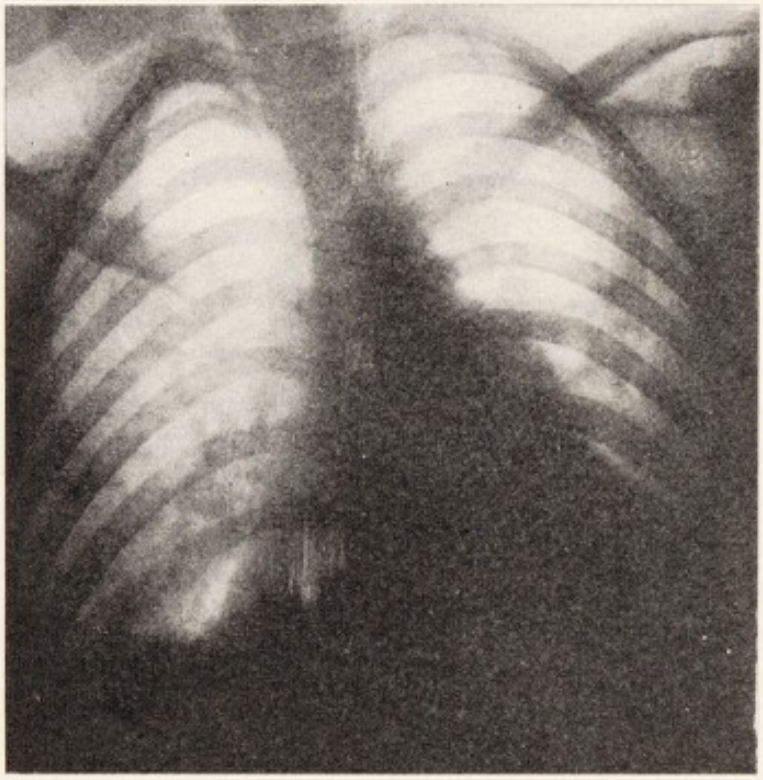


FIG. 145.—Necropsy 4152. The same, one month before death. Heart shadow still roughly triangular and distinctly abnormal. (Roentgenological Department, Massachusetts General Hospital.)

The temperature was 97.4° to 104.1° with daily swings of one to four degrees. The pulse was 100 to 166. The respirations were 25 to 51. The amount of urine was normal when recorded. The urine was cloudy, the specific gravity 1.012 to 1.020. There was the slightest possible trace to a trace of albumin at all of seven examinations, bile December 11. The hemoglobin was 80%, the leucocytes 39,600–11,200–23,300, the polynuclears 75%, the reds and platelets normal. A blood culture showed no hemolytic streptococcus. The X-ray findings were not the same as before. The heart shadow was now roughly triangular and distinctly abnormal. (See Fig. 145.)

December 15 the heart impulse was very forceful, visible in the neck. There were crepitant râles at the bases, more on the left, and signs of fluid at both bases. A left chest tap gave 20 c.c., the first 10 c.c. cloudy, opaque, straw-colored, the remainder apparently almost pure blood. The resulting fluid clotted within two or three minutes; apparently a transudate. Culture was negative. December 18 the chest signs were more marked, and there was a definite diastolic murmur. The respiration was very rapid, and there were râles in the left upper chest in front. A right chest tap gave 480 c.c. of deep orange opaque fluid, specific gravity 1.018, cells not counted; smear, 52% polynuclears, 48% leucocytes, many red blood cells; Esbach 0.7%. December 20 there was less pain and the temperature was lower. There were very few râles. Next day there were many râles throughout both chests and peculiar râles in the left front in the region of the fourth and fifth ribs. Two days later the right base was very dull. The patient was kept under morphia. When not under it she complained of pain in the joints and precordia. The dullness at the right base increased to flatness with diminished signs. December 29 there was a questionable soft diastolic in the pulmonic area. January 9 the patient died.

*Clinical Diagnoses (from Hospital Records).*—First Entry: Secondary anemia.

Second Entry: Chronic tonsillitis.
Metrorrhagia.

Third Entry: Endocarditis, subacute.

Fourth Entry: Acute infectious arthritis.
Acute endocarditis.
Hydrothorax.

*Dr. Ricahrd C. Cabot's Diagnosis.*—Chronic adhesive pericarditis.
Acute endocarditis?
Myocarditis?
Passive congestion of the lungs.
Right hydrothorax.
Chronic streptococcus septicemia.

*Anatomical Diagnosis.*—Chronic adhesive pericarditis.
Hypertrophy and dilatation of the heart.
Chronic passive congestion, general.
Slight ascites.
Slight hydrothorax, right.
Chronic pleuritis.
Slight chronic endocarditis of the mitral valve.

Subacute endocarditis of the aortic valve.

Slightly defective closure of the foramen ovale.

DR. RICHARDSON: We were not permitted to examine the head in this case. The gastro-intestinal tract, other than for some reddening of the mucosa, passive congestion, was negative. There was slight ascites.

In the right pleural cavity there was about 200 c.c. of thin pale fluid, and a slight amount on the left,—a slight hydrothorax on the right. There were scattered fibrous adhesions on each side. The lungs showed chronic passive congestion and nothing else.

The pericardial cavity was obliterated by membranous adhesions, and the two layers were firmly bound together. The heart weighed 650 grams, markedly enlarged. There was considerable dilatation and a slight amount of chronic endocarditis of the mitral valve, and a patch on the aortic valve.

The liver and spleen showed chronic passive congestion, well marked. The spleen weighed 313 grams. There was no definite nephritis.

DR. CABOT: There was no deformity of either of those valves?

DR. RICHARDSON: A fibrous ridge extending along the mitral; but the note specifies that the deformity was slight. The most extensive lesion was of the pericardium.

The culture made at the time of necropsy was negative, but in these cases we may get a negative culture when there may be foci of infection in the body.

DR. MERRILL: It might not be out of place to mention that that fluid was found several months after the last chest examination, which was in August. The fluid found at the time of death was undoubtedly a subsequent development.

DR. CABOT: Yes; the heart was doing fairly well when you examined her; later it gave out.

### Necropsy 4470

An American drug clerk of twenty-nine was brought to the Emergency Ward March 23, 1921. The history was given by his father. The patient had measles and chickenpox in childhood. At twenty he had an operation on his nose. He had rare moderate headache and occasional head colds. Skating and running did not trouble him. His best, usual and present weight was 160 pounds.

He had been doing some extra work and for the past few days had not felt well because of cold in the head. A day or two before

admission after eating pork he felt nauseated. Since that time he had eaten little. He had slight cough. March 22 he came to his father's house very weak and apparently very ill.

Examination showed a fairly well nourished man with dry and cyanotic skin. The mucous membranes were cyanotic, with gray pallor. There was slight pyorrhea. The veins in the neck were dilated. There was dullness to flatness with diminished breathing and many moist bubbling râles at both bases posteriorly. The apex impulse of the heart was diffuse in the fifth space 11 cm. to the left of midsternum, 2 cm. outside the midclavicular line. The right border of dullness was 5.5 cm. to the right, the supracardiac dullness 6.5 cm. The action was irregular, very rapid (168). The sounds were snapping. The pulmonic second sound was accentuated. An explosive systolic murmur was heard. A systolic thrill was felt at the apex. The pulses were normal. The artery walls were palpable. The blood pressure was 150/90 to 125/40 to 120/30. The liver dullness extended from the fourth rib to 5 cm. below the costal margin. The edge was felt. There was edema of the sacrum. The genitals, extremities, pupils and reflexes were normal.

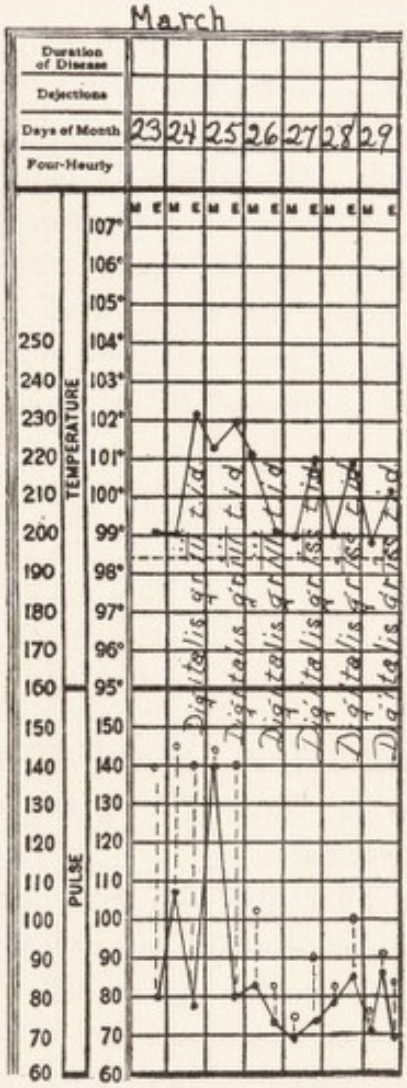



FIG. 146.—Temperature and pulse in Case 4470 (first entry).

The temperature and pulse for the first week were as shown in Fig. 146. Afterwards the pulse was 89 to 42. After April 4 the temperature was not above normal. The respirations were 47 to 15. The output of urine was 22 to 63 ounces, the specific gravity 1.017 to 1.025. The urine was cloudy at three of six examinations, alkaline at three. It showed the slightest possible trace to a trace of albumin at all, was loaded with red blood corpuscles at the first, showed hyalin casts at two and a few large cellular casts at one. The renal function was 35 to 55%. The hemoglobin was 80%, the leucocytes 31,800 to 5400, the polynuclears 86%. A Wassermann was negative. A blood

culture was negative. The non-protein nitrogen was 73.2 March 24, 39.6 April 7. The sputum was slightly bloody, with many disintegrated red cells and occasional staphylococci in tetrads. The stool showed blood and a positive guaiac at one of four examinations.

The patient showed marked orthopnea. Bleeding helped him more than anything else at first. It was found that he had taken digitalis for some time. By March 28 he was comfortable and his color was good. April 5 he had hardly any dyspnea and looked like an entirely different man. Some edema of the ankles was developing which disappeared four days later after bandaging. Edema of the dorsum of the feet persisted, however, while the patient was absolutely at rest in bed. April 13 he was discharged.

After leaving the hospital he lay in bed for over a week, then got up for his meals, then began to walk a little, and improved steadily for three weeks. After that he had to wait on himself more or less, and was worried. He soon found he felt weaker and had increased palpitation and dyspnea. His doctor ordered him

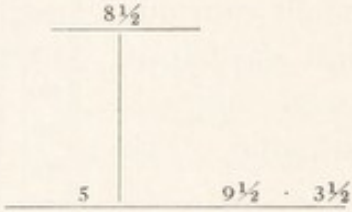



FIG. 147.—Measurements by percussion.

back to bed. May 20 he began to have trouble with his digestion, due he thought to eating a bad orange. May 21 he vomited his breakfast.

That day he returned to the hospital. He was orthopneic, had palpitation very noticeably when he lay on his left side, and felt tired, weak, and slightly nauseated. He urinated once at night.

Examination was as before except for the points noted. There was considerable dyspnea and orthopnea. The skin showed a slight yellowish tint. The mucous membranes were rather pale. There were some carious teeth; no pyorrhea. The lungs showed slight dullness and a few moist râles at both bases posteriorly. The diaphragm excursion was equal. The apex impulse of the heart was diffuse, seen and felt in the fourth, fifth and sixth spaces as far out as the anterior axillary line. Percussion measurements in cm. are shown in the diagram. The action was rapid, absolutely irregular in force and rhythm. The sounds were not distant. The pulmonic second sound was greater than the aortic second but not markedly accen-

tuated. The sounds were difficult to make out because of irregularity, but there seemed to be a definite systolic murmur all over the precordia, loudest at the apex, a sharp first sound, no definite diastolic murmur, pulses equal, synchronous, of varying volume and tension. The artery walls were not palpable. The blood pressure was 130/75–110/60–135/60.

The temperature and pulse were as shown in the chart (Fig. 148) until June 3. Afterwards the temperature was 96.5° to 98.4°, the

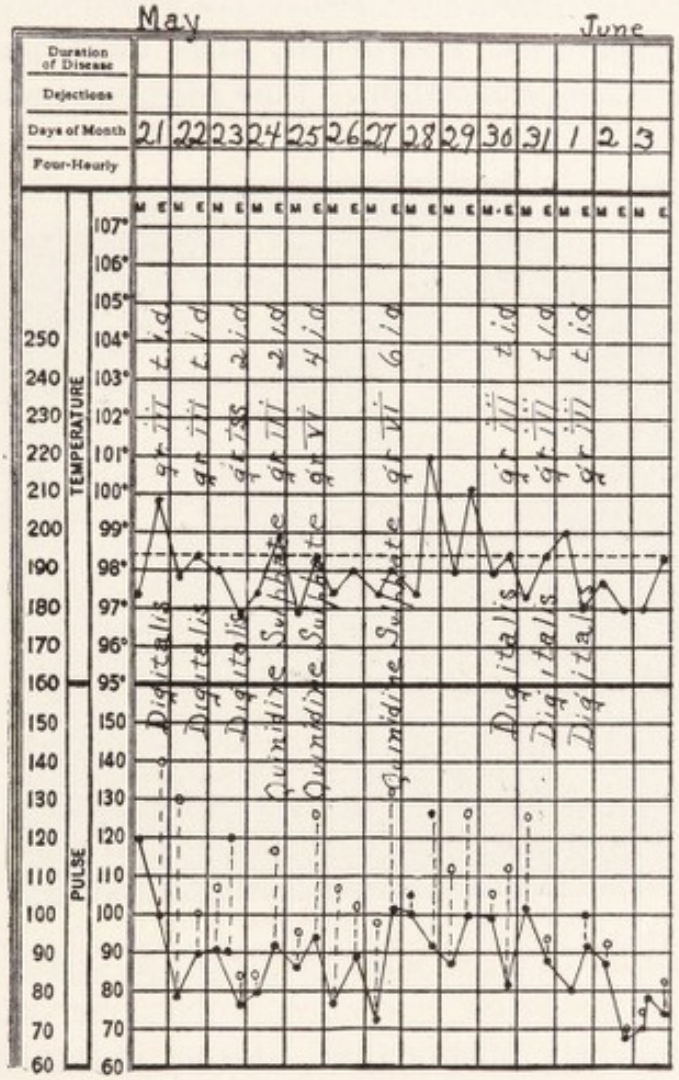



FIG. 148.—Temperature and pulse in Case 4470 (second entry).

pulse 62 to 92 except for one rise June 7. The respirations were not remarkable. The output of urine was 15 to 120 ounces, the specific gravity 1.026 to 1.008. The urine was cloudy or muddy at two of seven examinations, alkaline at two, showed the slightest possible trace to a trace of albumin at all, very rare red blood corpuscles at one, many at another, leucocytes at two, hyalin casts at two, granular at three, cellular at two. The hemoglobin was 80%, the leucocytes

13,200 to 9600, the polynuclears 59%. The non-protein nitrogen was 53.4 mgm. A blood culture was negative. X-ray is shown in Fig. 149.

In the left lateral position there was limited excursion of the left border of dullness and the maximum impulse; probably some fixation, but no retraction at the back. At the apex were a sharp first sound, a presystolic murmur, a high pitched early systolic, a rumbling middiastolic with split second sound, and a systolic thrill.

By May 31 the patient felt well. The heart was still irregular but slower. There was less deficit. The exact part played by quinidine was felt to be uncertain. June 4 the pulse was down to 70.

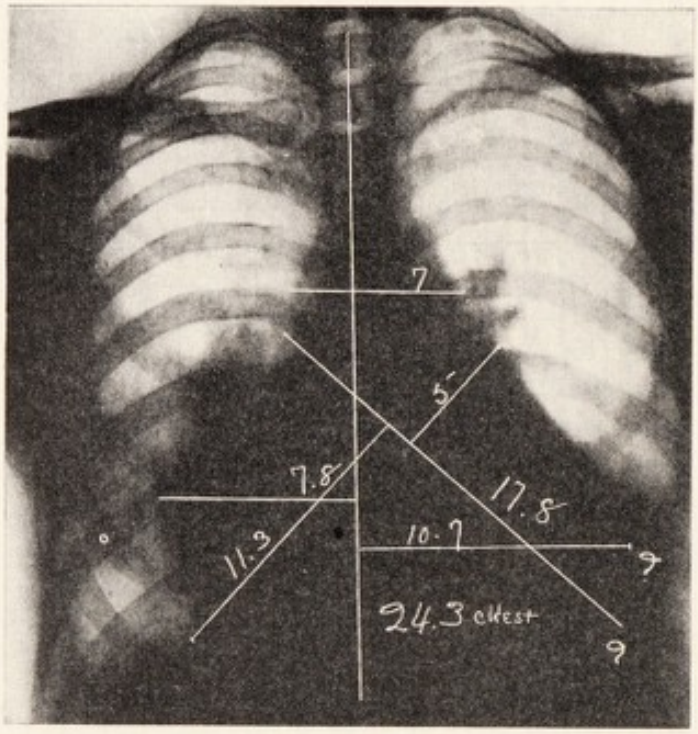



FIG. 149.—Heart shadow is very much enlarged and roughly triangular in shape. The greatest increase is in the region of the right auricle.

There was headache, probably, it was thought, from digitalis, which was discontinued. By June 8 there was no more headache. The pulse was down and steadier and by June 12 there was no deficit, though there was irregularity at times. June 22 he was discharged relieved.

At his third admission, February 23, 1923, the history was unsatisfactory because the patient was in too poor condition to talk. Since his discharge he had been on his feet most of the day, but had not lost a day's work until the onset of this attack. He had been taking pills given him by Dr. Paul White regularly until the attack.

Two months before admission he found that he could not work so hard as formerly. He tired easily and was troubled in climbing stairs. He paid no attention to these symptoms however until the first of February, when he had to go to bed because of a cold in which the predominating symptom was a "tired feeling." The second day of his illness he was found lying with his head thrown over the side of the bed, unable to speak, his right arm and leg paralyzed. Since that time the arm had slowly improved until at admission he could write with some difficulty. He could move his leg and could speak, also with difficulty. Since his "stroke" his memory had been poor, he had used three pillows instead of one, had been very dyspneic, had had cough with about a cupful a day of tenacious sputum, had urinated three times at night, and had felt his heart beat very forcefully at times and at other times apparently stop beating. Since his cold he had had edema of the legs.

Examination showed an extremely weak, well nourished man propped up in bed, slightly cyanotic, dyspneic, coughing frequently,

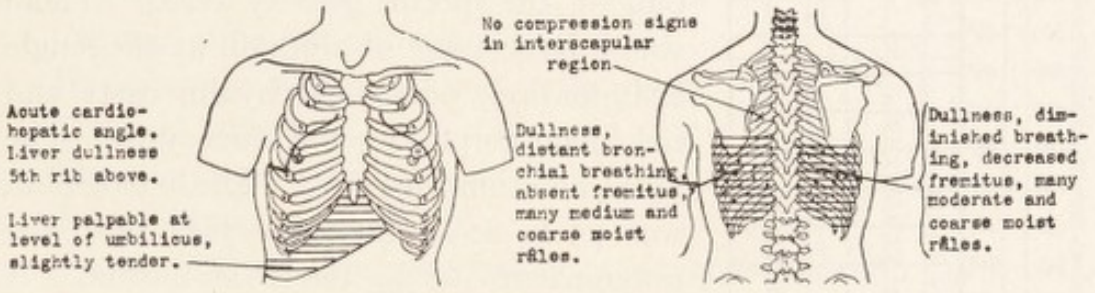



FIG. 150.—Physical signs in Case 4470 (last entry).

raising sputum with great difficulty. He seemed to understand what was said and to be rational, but was unable to express what he wanted to say and had great difficulty in naming objects. The skin was moist and clammy, the face decidedly dusky. The mucous membranes were pale and slightly cyanotic. There were a few scattered glands in the cervical region. The apex impulse of the heart was diffuse and heaving, seen and felt in the third, fourth, fifth and sixth spaces, maximum in the sixth, 14 cm. from midsternum in the anterior axillary line. There was a well marked systolic thrill at the apex. No diastolic was made out. No cardiac pulsation could be made out in the left back. In turning from the right to the left lateral position the apex impulse stayed in the same spot. The first sound at the apex was short and snapping but not very loud. There was a rather loud and reduplicated second sound. The sounds were absolutely irregular in force and rhythm, very rapid (130). A loud

high pitched blowing systolic murmur was heard at the apex following almost immediately after the first sound and filling most of systole, transmitted well out in the axilla. There was a questionable rumbling middiastolic at the apex. The blood pressure was 165/75 to 145/75. The lung signs and the abdomen were as shown in Fig. 150. There was moderate edema of the dorsal surfaces of the feet and slight edema of the ankles. Neurological examination showed in addition to the previous findings normal fundi, questionable slight numbness of the right side of the face, and weak grip on the right. The abdominal reflexes and the jaw-jerk were absent. The right triceps, biceps, radial and wrist reflexes were increased.

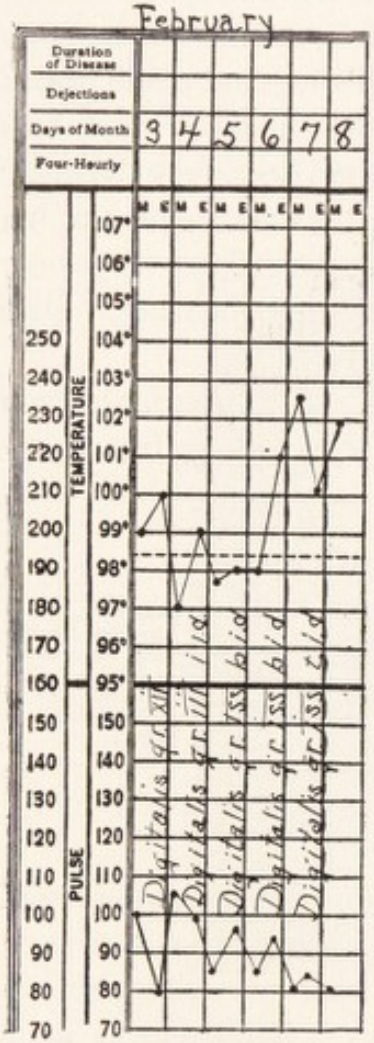



FIG. 151.—Temperature and pulse in Case 4470 (last entry).

The temperature and pulse are as shown in Fig. 151. The respirations were 22 to 40. The output of urine was 40 to 33 ounces, the specific gravity 1.020. There was a large trace of albumin at the single examination, occasional hyalin casts and red blood corpuscles and leucocytes. The renal function was 15%. The hemoglobin was 75 to 80%, the leucocytes 8800, the polynuclears 68%, the reds normal. A Wassermann was negative. The non-protein nitrogen was 59.2 mgm. An electrocardiogram showed auricular fibrillation, ventricular rate 80 to 100, diphasic $T_2$; aberration as before. The sputum was mucopurulent. The presenting organism was a Gram-positive lanceolated diplococcus. There was an occasional streptococcus-like chain of Gram-positive diplococci slightly curved and well encapsulated. A few Gram-negative bacilli, moderately thick, were seen, a few Gram-positive bacilli, long and straight, and a few with a slightly narrowed center, a fair number of diphtheroids, a slight to moderate number of influenza bacilli, extracellular, and one questionable endothelial phagocyte with phagocytized bacteria.

February 5 the heart rate was slower. Two diastolic murmurs were audible, a soft blowing early diastolic along the left border of

the sternum and a low-pitched rumbling middiastolic murmur heard best at the apex. The general condition was much improved.

February 7 the temperature rose to 103° and the patient was much sicker. Throughout the whole right lower back were many moist râles different from the râles on the other side. There were no definite areas of consolidation. There was considerable abdominal distension. The abdomen remained distended in spite of strenuous measures. February 8 the right base was full of moist râles obscuring the respiratory sounds. A marked transmission of cardiac murmurs suggested consolidation at both bases. That afternoon he died.

*Clinical Diagnosis (from Hospital Record).*—Rheumatic heart disease with mitral stenosis and regurgitation and aortic regurgitation.

Adhesive pericarditis?

Auricular fibrillation.

Congestive failure.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the mitral and aortic valves with mitral stenosis.

Acute endocarditis.

Chronic adherent pericarditis?

Hypertrophy and dilatation of the heart.

Cerebral embolism.

Chronic passive congestion.

Terminal pneumonia?

*Anatomical Diagnosis.*—Chronic endocarditis of the aortic and mitral valves.

Chronic adhesive pericarditis.

Thrombi in the right auricular appendix.

Area of softening in the left cerebral hemisphere.

Hypertrophy and dilatation of the heart.

Chronic passive congestion, general.

Infarcts of left lung.

Wet brain.

Edema of the ankles.

Anasarca.

Chronic pleuritis.

Old infarcts of spleen and kidneys.

DR. RICHARDSON: This man was poorly nourished. The pia showed marked edema and a slight excess of fluid in the ventricles. The brain tissue was negative except in the places to be mentioned, but generally wet. In the left cerebral hemisphere beginning in the region of the outer margin of the optic thalamus and extending along

the basal ganglia for three cm. where at its anterior tip it touched the outer end of the internal capsule, there was a very pale circumscribed area of frank softening and disintegration about 2½ × 2 cm. in the other dimensions reaching to within 2½ cm. of the cortex. The vessels of Willis were all normal as far as made out, but the vessel that contained the embolus was lost in the area of degeneration. The pineal and pituitary glands were negative.

The ankles were slightly swollen and pitted on pressure. There was no marked evidence of any edema elsewhere in the body. Much brownish granular fluid material run from the nose and mouth. There were brown-red spots in the left cubital space and some beneath the left pectoral.

The peritoneal cavity and appendix were negative. There was no ascites, no anasarca. The only edema was in the ankles. The stomach showed some passive congestion. The intestinal mucosa was rather pale.

The mesenteric and retroperitoneal glands were negative. The liver at the time of necropsy was one finger below the costal margin. The diaphragm was at the fifth interspace on the right, on the left at the sixth.

The right pleural cavity was obliterated by old adhesions infiltrated by much thin pale brownish fluid. That is, of course, the cavity was obliterated, but the fluid saturated the adhesions. Still on the left there were only a few c.c. of thin brownish fluid and a little fibrin. Both lungs showed a few old adhesions, but were otherwise free. The trachea and bronchi showed purulent bronchitis. There was typical chronic passive congestion of the lungs, and in addition to that, areas of bronchopneumonia at the time of necropsy, best marked on the right. The lower lobe on the other side was negative for areas of bronchopneumonia, but curiously enough on that side there was an infarct.

The pericardial cavity was obliterated by thin membraneous adhesions; consequently there was no space for fluid. The heart weighed 917 grams. I think if I say that is an enormous heart I shall be excused. The myocardium was of good consistence, pale brown-red. The right ventricle measured four mm., the left thirteen; that is, of course, rather thickened. The foramen ovale was closed, consequently nothing could pass from one side of the heart to the other. The cavities were greatly dilated. The mitral valve measured 11 cm., the aortic 7½ cm., the tricuspid 14 cm., the pulmonary 9 cm. These are all full-sized circumferences, the mitral measuring eleven

cm. although there were changes on it. The mitral curtain showed a moderate amount of diffuse fibrosis with thickening and shortening of the chordae tendineae. The free margin was thickened and a little nodular. In the region of the junction of the cusps there was a large fibrocalcareous plaque. On the anterior aortic cusp there was a small fibrocalcareous mass, and elsewhere fibrocalcareous change and deformity of the valve. No recent endocarditis was made out. The pulmonary and tricuspid valves were negative except that their circumferences were increased. In the right auricular appendage there were two small thrombi.

So that we have here chronic endocarditis and chronic adhesive pericarditis. This combination of lesions gives us our largest hearts. The aorta and great branches were perfectly good. It rules out any question of arteriosclerosis playing a rôle in the condition.

The liver showed nutmeg markings. The bile-ducts, gall-bladder, pancreas, negative. The spleen weighed 190 grams, was plump, dark red and elastic,—chronic passive congestion. The surface showed an area of infarction.

The kidneys weighed 485 grams. The pelves, ureters, bladder, prostate, seminal vesicles, testes, were negative. The kidneys were large but negative except for small infarcts.

### Necropsy 3736

A metal worker of thirty-three entered March 27, 1917, for relief of vomiting and loss of weight. He had always been strong. For

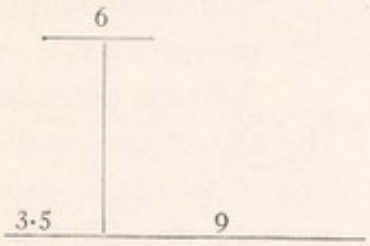



FIG. 152.—Dimensions by percussion.

seven years he had had painful hemorrhoids. For four or five years his skin had been yellow. In 1913 he had rheumatism involving several joints and later was told by his doctor that he had a "leaky heart." A year before admission he was ill in a hospital two months with a severe attack of gastric ulcer with delirium and much abdominal pain. While convalescent he had an acute attack of articular rheumatism and was in the hospital again for a month. He had done

no work for a year. Since an attack of gonorrhea eight months before admission he had been unable to retain his urine. In 1915 he weighed 134 pounds, his best weight. In the autumn of 1916 he weighed 128 pounds.

Since his illness in 1916 he had felt ill and tired all the time. His bowels had been irregular. A month before admission he was in bed for two days with sharp pain across the entire front chest. He had vomiting, which persisted. His appetite was poor. For a month he had had frequent chills, shaking all over, and felt cold all the time.

Examination showed an emaciated man 5 feet 5⅞ inches in height, weighing 97½ pounds. The skin and mucous membranes were pale, the former dry. There were hard, shot-like axillary, inguinal and epitrochlear glands. The apex impulse of the heart was felt in the fourth space 9 cm. from midsternum. The dimensions by percussion and by X-ray are shown in Figs. 152 and 153. The action was regular, slightly rapid. The aortic second sound was slightly accentuated. There was a blowing systolic murmur transmitted to the axilla and the base, with a slightly musical quality over the lower sternum. There was a split second sound followed by a thrill and a short soft diastolic not transmitted to the axilla or the base but heard well over the body of the heart. The artery walls were slightly palpable. The blood pressure was 95/45 to 105/50. The liver dullness extended from the fourth space to the costal margin. The rest of the examination was negative.

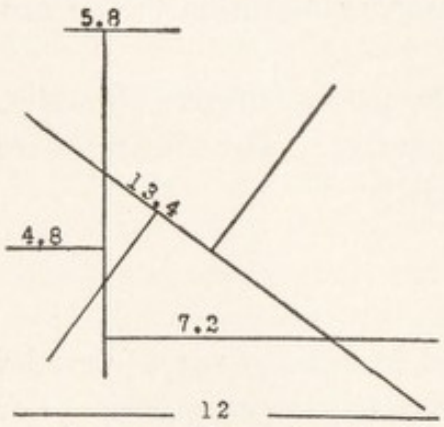



FIG. 153.—Dimensions by X-ray. Diameter at base 11.2. Questionable slight enlargement of the right side of the heart.

The temperature was 94.3° to 104.8° usually elevated, the pulse 70 to 160, the respiration 20 to 36. The amount of urine was 9 to 35 ounces, the specific gravity 1.022 to 1.012. There was the slightest possible trace of albumin at the last of seven examinations. One specimen showed cellular and granular casts, two showed pus. The hemoglobin was 85%. There were 18,800–5200–52,700 leucocytes, 89 to 70% polynuclears, 3,840,000 reds, slight variation in size and shape, no stippling. A Wassermann and a Widal were negative, a gonococcus fixation test strongly positive.

For nearly a month the patient showed little change. The temperature oscillated between 98° and 104.8°. Lead was reported

in the urine and stools April 8.* Vomiting and chills persisted. April 23 he had an attack of sharp substernal pain with marked pallor. There was no change in the heart murmurs.

From this time on he complained of pain in the apical region. May 3 a pericardial rub was heard. (See Fig. 154.) Thoracentesis yielded no fluid. Pericardicentesis was done next day in the fourth left and right spaces next to the sternum, and in the third space 3 cm. to the right of the sternal border. Blood was obtained in the third

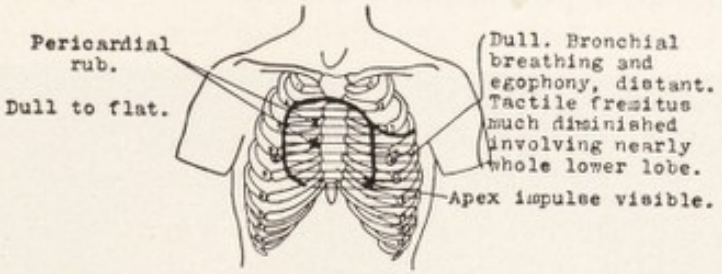



FIG. 154.—Physical signs in Case 3736.

and fourth right spaces, nothing at the fourth left space. The heart was felt at the end of the needle at each puncture, and the patient complained of pain at each.

May 3 X-ray showed the chest as in the illustration. A Widal was negative. May 12 the pericardial rub was faint, obscured by a




FIG. 155.

loud systolic murmur at the base. There was much pain in the side and over the precordia. The measurements by percussion are shown in Fig. 155. May 21 there was a systolic murmur at the apex and the aortic area, dullness at both bases, more on the right, with diminished breath sounds and coarse râles. That day he died.

*Clinical Diagnosis (from Hospital Record).*—Ulcerative endocarditis.

Cardiac failure.

*Dr. William H. Smith's Diagnosis.*—Chronic endocarditis of the mitral valve.

*The finding of lead in the urine and stools may explain his epigastric pain. This emphasizes the importance of associating occupation (metal worker) with abdominal pain. W. H. Smith.

Presumably acute endocarditis of the mitral and aortic valves.
Dilatation of the heart.
Pericarditis, with little if any effusion.
Passive congestion.
Possibly acute glomerulo-nephritis.

*Anatomical Diagnosis.*—Ulcerative endocarditis of the aortic valve.
Chronic endocarditis of the mitral valve (stenosis).
Chronic adhesive pericarditis.

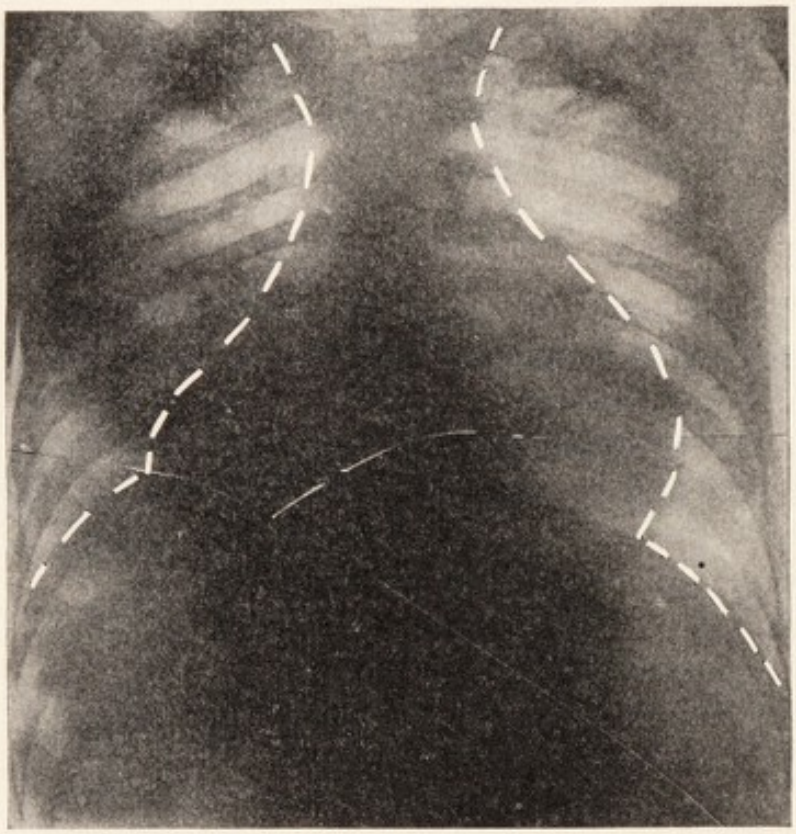


FIG. 156.—May 3. Shows a shadow over the lower two-thirds of the left base, higher in the axillary than in the median line. The shadow of the diaphragm is obliterated. The heart shadow is very much enlarged. Its shape suggests fluid in the pericardium. Pulsation is diminished. Conclusion: fluid in the left chest and probably fluid in the pericardium.

Septic aortitis with thrombus formation.
Hypertrophy and dilatation of the heart.
Septicemia, streptococcus.
Suppurative infarcts of the spleen with abscess formation.
Infectious nephritis.
Acute glomerulo-nephritis.
Foci of pneumonia.
Fibrinopurulent pleuritis, left.

Chronic passive congestion, general.

Decubitus.

Slight chronic pleuritis.

DR. RICHARDSON: There was not a luetic but a septic aortitis with thrombus formation in this case. The heart weighed 315 grams. There was some dilatation of the right cavities. The man was slender and rather short. A normal heart for him would weigh about 260 grams, so that the hypertrophy and dilatation was slight.

The condition at the mitral valve was a chronic one that occurred years ago. There was fibrosis of the curtain and a shortening of the chordae tendineae, with decrease in the circumference of the valve. The endocarditis of the aortic valve was an acute process. Then an area on the wall of the aorta was attacked and a septic aortitis was set up, with formation over it of a large thrombus mass. The interesting point about this is that it shows that some of the cases of aortitis may not be luetic but may be due to some of the other micro-organisms.

There was a thick layer consisting of fibrous strands in the pericardium. In the anatomical record I described it as a thick, edematous layer. That means there is fluid there, and some coagulation of the fluid, at least at the time of death. Sometimes with transudates we get a fibrin clot; that may be the case here. There was a layer of fibrous adhesions there, but much of the thickness was due to the edema.

There was fibropurulent pleuritis on the left, perhaps 100 c.c. of purulent fluid material. The right side was negative.

The infarct in the spleen came from the masses washed out either from the aortic valve or else from that thrombotic material in the situation of the septic aortitis. The spleen in this case was represented by a large ovoid mass about eight inches in diameter. That is a pretty good-sized spleen. There was little splenic tissue left, and that was wrapped around this large abscess filled with broken down, necrotic material and pus.

In the kidneys there were some foci of infectious nephritis.

The cultures were taken from the blood of the inferior vena cava because of the mass of thrombotic material on the aortic wall. We obtained a profuse growth of a fine streptococcus which is more closely related to the pyogenic than it is to the hemolytic streptococcus. From the spleen we obtained a few streptococci and a profuse growth of the staphylococcus aureus. There was probably a double infection which would account for the large abscess in the

spleen and possibly for the small areas of infectious nephritis present in the kidneys.

Here again we have the end result of an infection of years ago illustrated by the chronic endocarditis of the mitral valve and the chronic adhesive pericarditis. Besides that we have acute endocarditis at the aortic valve, septic aortitis, the condition in the kidney, pneumonia, and abscess of the spleen. One very interesting thing was the septic aortitis.

# CHAPTER XI

## THYROCARDIAC DISEASE

### 1. THE OVER-DEMONSTRATIVE HEART

One of the earliest cardiac manifestations of thyrotoxicosis and one which, if we are on the watch for it, may give us the signal to look deeper and so to identify the disease itself, is the *extraordinary demonstrativeness of the heart.* It makes its presence strikingly, to the patient distressingly, obvious to sight, to touch and to hearing. There seems to be a great deal of the heart in contact with the chest wall, so that (for example) a heart weighing only 365 grams bangs against the ribs in the anterior axillary line and shakes the whole thoracic cage. When we feel of this shock, we are amazed at its violence and prone (mistakenly) to believe that we are feeling a systolic or pre-systolic thrill. When we listen over it, the sounds are almost painfully loud, louder than in any other condition except one, presently to be mentioned. These *surprisingly loud heart sounds,* with or without a systolic murmur, *should always make us suspect thyroid poisoning.*

All this sound and fury is due presumably to the irritation of the cardiac mechanism by the perverted thyroid secretion, combined with the lowered vascular tension. These influences are shown in the large pulse pressure (often a true "Corrigan pulse") the capillary pulsation, jumping arteries and loud arterial sounds, as well as in the flushing and sweating from which some patients suffer much.

The only condition, so far as I know, that simulates the over-demonstrative heart of thyrotoxicosis, is the so-called "effort syndrome" (soldiers' heart, neuro-circulatory asthenia) which is the cardiac manifestation of great nervousness. By cardiovascular examination alone the two diseases may be indistinguishable, but luckily the history and the metabolism test—high in thyroid poisoning, normal in effort syndrome—make it easy to distinguish them in almost every case.

## 2. EARLY CONGESTIVE HEART FAILURE IN THYROID DISEASE

In spite of the seemingly desperate exertion of the racing thyroid heart, cardiac failure is rare in the acute exophthalmic goitre of young persons. Even when the organ seems actually driven to death, there is usually no sign of passive congestion before death or at necrospy. It is usually a toxic, not a congestive death. There are but three cases of our series to illustrate congestive failure.

In necropsy 425 we were studying the organs of a patient who three years earlier (1896) had been treated in our clinic for "rheumatism" and for some supposed cardiac result of it. The thyroid disease, if it existed, passed altogether unnoticed until six months later when after her second period of hospital treatment (2½ years before her death) the diagnosis was changed to "exophthalmic goiter with chronic endocarditis." She gave the history of an illness interpreted as "rheumatic fever" which had occurred a few months previously and stated that every August for as long as she could remember she had had a peritonsillar abscess. With this history, with an extension of cardiac dullness to one inch outside the nipple line and with a loud apical systolic murmur, it was natural enough to consider the case one of rheumatic endocarditis with valvular deformity. But one point should have made us suspect that we were wrong. The record states that "the heart sounds were very strong, sharp, quick and regular."

Six months later (January, 1897) when the thyrotoxicosis had been recognized, the record reads: "Cardiac impulse in the 5th interspace, 3 fingers' breadth outside the nipple, the right border of dullness one finger to the right of the sternum. Pulse rapid, easily compressed, regular. Rough blowing systolic murmur at the apex, transmitted to the axilla. Pulmonic 2nd accentuated. Bruit de galop in the 2nd right interspace."

Two and a half years after this—at her 3rd and last hospital entry—the cardiac apex was in the anterior axillary line (5th interspace). Otherwise the heart was as before. *Necropsy, however, showed no hypertrophy or dilatation of the organ* (weight 365 grams) although there were a few old pericardial adhesions in addition to the thyroid poisoning.

In this case—and in only one other of our series (No. 3189, page 729) there were signs of passive congestion before death and after it. Ascites had developed and the abdomen was tapped three times for its relief during the year preceding her death. The legs were edematous during this period. Post mortem the spleen and kidneys showed the

passive congestion especially well. The heart showed no myocardial or valvular lesions and if the "rheumatism" of 1896 affected her heart at all, it was in producing the pericarditis still recorded in the localized adhesions found at necropsy.

No metabolism measurements are recorded in this case but the continued fever without evidence of infection post mortem, the exophthalmos, tachycardia, goitre, tremor, muscular twitchings, insomnia and vomiting, leave little doubt as to the diagnosis.

### 3. TOXIC DEATH

**Toxic death** is much commoner than congestive failure in acute, primary, exophthalmic goiter. In Hamilton's* series there were but seven congestive deaths among 18 fatal cases. The rest were toxic. In our series there were 6 toxic to 2 congestive failures—almost the same proportion.

In these patients who died a toxic death, *the appearance of cardiac enlargement during life, though necropsy showed none*, was sometimes as striking as in those ending by congestive failure. The records contain such data as this:

1. (No. 2077) A woman of 24. Apex 6th interspace, 1 finger's breadth outside the nipple. Dullness 2 fingers' breadth to the right of the sternum.

Necropsy: Heart weight 260 grams. No enlargement. Normal myocardium and valves. Persistent thymus. Bronchopneumonia. Streptococcus sepsis.

2. (No. 3468) A woman of 29. Transverse dullness 27 cm. Supracardiac dullness 9 cm.

Necropsy: Heart weight 220 grams. Myocardium and valves normal. No enlargement. Status lymphaticus (with persistent thymus). Purulent pleuritis. Purulent pericarditis. Pigmented skin.

3. (No. 3731) A woman of 23. "*Heart greatly dilated*" just before death (from lobar pneumonia).

Necropsy: Heart weight 270 grams. Myocardium and valves normal. Hypoplasia of the aorta.

4. (No. 3825) A woman of 20. Impulse diffuse and forceful. Sounds loud and snapping. "Blowing systolic murmur and *systolic thrill* at apex."

Necropsy: Heart weight 315 (considered to be slightly enlarged, as the patient was a girl of 20). Valves and myocardium negative

* Hamilton: Journ. of the A.M.A., Aug. 9, 1924.

(note the mistake about the "thrill"). Status lymphaticus (with persistent thymus).

In two other patients of this group (exophthalmic goitre with toxic death) there was no apparent cardiac enlargement in life and none post mortem. All these six patients had the typical manifestations of fever, vomiting, diarrhoea, sweating and extreme tachycardia. In one, the motor restlessness was so extreme that the skin had become hyperaemic and morphia was needed to prevent her wearing it through and forming bed sores.

Status lymphaticus with persistent thymus was present in five out of six cases, a finding noted by many writers but ignored by many clinicians.

The characteristically high pulse pressure was well shown in one of this group, a woman of 38, with very acute toxaemia leading to a toxic, non-congestive death, four weeks from the first symptoms noticed. The pulse averaged 166 but was regular. The blood pressures, recorded three times in the 12 days of her life in the ward, showed: 160/60, 170/65, 155/85.

5. (No. 3705) An American woman of 58 had noticed exophthalmos ever since her first pregnancy 20 years ago. Last summer she noticed goiter, three months ago her daughter noted that her pulse was usually above 120. Four weeks ago she had a sudden attack of tachycardia, dyspnea and vomiting. Two weeks ago a fine tremor of the hands appeared. She had been very nervous.

Examination showed moderate exophthalmos with lid lag and diminished reflex winking. Moderate general thyroid enlargement with a systolic bruit. The pulse was rapid (120–150) and absolutely irregular, with sharp heart sounds and a soft systolic at the apex. Slight edema of the legs with coldness and no pulse in the dorsalis pedis. Fibrillation persisted despite rest and digitalis and she died in 12 days.

Necropsy showed a sclerosis of the left coronary with marked decrease in its lumen, slight fibrous myocarditis, general arteriosclerosis, thrombosis of the left auricular appendage and embolic occlusion of the abdominal aorta. Streptococcus sepsis. No general passive congestion.

This is probably a purely toxic case without heart failure though the cardiovascular system (through thrombosis and embolism) was immediately responsible for her death.

## 4. LATE CONGESTIVE FAILURE IN TOXIC ADENOMA AND EXOPHTHALMIC GOITER

Plummer, Boothby, Hamilton,* Coller,† and others, have recently aroused a much needed interest in another clinical picture long ago described by Kraus‡ as "*Goiter heart.*" This term means the development of cardiac failure in persons (usually women) who have long had a goiter (or a small hyperfunctioning thyroid without visible goiter) but no considerable symptoms until cardiac manifestations appear. In Hamilton's 50 cases (34 of primary hyperthyroidism and 16 of adenomatous thyroid with secondary hyperthyroidism) 42 were women with an average age of 50 (but looking much older). 41 of the 50 had a history of *severe prolonged heart failure*, confining them to bed or to a chair for months or years and *resisting treatment.* The heart rate was usually high, often 180–200, falling below 100 with rest and digitalis in only 14 cases. *Stubborn tachycardia* was thus a feature of 36 cases. Goiter and exophthalmos were noticeable in only a few. *Emaciation* was notable in 43 (average weight 109 pounds despite edema) and even in the other seven, there had been a loss of 30 pounds or more. *Pigmentation* of the skin local or general was present in 48 of 50 cases, *mental apathy or exhaustion* in 28. 48 out of 50 had auricular fibrillation. The metabolism tests, which one might expect to be quite characteristic, are not so in fact for, though they show marked increase (16+ to 125+, average 61.8+), similarly high figures can be obtained in non-thyroid heart failure.

We have here a clinical picture, the great importance of which (despite its rarity in non-goitrous districts§) is obvious because operation seems to offer some possibilities of relief and even of cure. I venture to say that by most clinicians it is still overlooked; yet in goitrous districts it is not rare, as the studies of Coller make apparent. In 300 cases of endemic goiter without toxic symptoms, occurring in persons past the 20th year and with normal metabolism,‖ Coller records the following valuable observations:

* Hamilton: Op. cit.

† Coller: Journal of the A.M.A., May 31, 1924.

‡ Kraus, F.: "Uber Kropfherz: Wien Klin. Woch., 1890, XII, 416.

§ Hamilton found but 50 cases of this type among 900 patients with thyrotoxicosis at a Boston clinic.

‖ Not over 15+ or 15−. These are selected non-toxic cases. About ⅓ of Coller's hospital cases of thyroid adenoma, between 30 and 50 years of age, show increased metabolism.

TABLE 158

| | Pulse rate* over 100 | Cardiac enlargement | Auricular fibrillation | Palpitation | Dyspnea | Signs of tracheal pressure |
|---|---|---|---|---|---|---|
| Age 20–30..... | 18% | 18% | 0 | 53% | 47% | 18% |
| Age 31–40..... | 14% | 43% | 0 | 48% | 60% | 36% |
| Age 41–50..... | 37% | 45% | 4% | 65% | 58% | 24% |
| Age 51–60..... | 26.6% | 50% | 30% | 63% | 76% | 26% |

* Counted at the heart's apex during sleep.

In view of Hamilton and Coller's findings it certainly behooves us all to study more closely our cases of unexplained cardiac failure in elderly women, especially when they are of the apathetic and pigmented type, not improving as one would expect under rest and digitalis and maintaining a persistent tachycardia with fibrillation.

In our series there are a few which, though not recognized in life as belonging to the thyrocardiac group, may perhaps retrospectively be placed there. So far as I know, very few post mortem examinations are yet recorded in cases of this type; therefore it seems worth while to give our results in some detail.

1. (No. 2067) A woman of 47, seen in 1908, had noticed a goiter since she was 11 years old. It enlarged with each of nine pregnancies and grew somewhat smaller again between them, but on the whole gradually increased in size. For the past two years she had been growing "nervous" and felt too warm much of the time, so that she preferred cold weather. Tremor of the hands had been noticed for six months.

Examination showed slight exophthalmos, considerable emaciation, marked tremor and goiter (16½ inches circumference), a very rapid heart with a presystolic roll at the apex, a congested liver and swollen legs. The urine three times examined showed a gravity of 1010 to 1012, traces of albumen and, in one specimen alone, a few hyalin casts. She died in five days, the heart rather suddenly giving out a few hours before the end. Necropsy showed a heart weighing only 405 grams but considered the seat of hypertrophy and dilatation. There was also hydrothorax, hydropericardium, anasarca and general passive congestion, with a thyroid adenoma and a chronic glomerulo-nephritis (the latter an astonishing feature in view of the negative urine). Possibly the congestive failure in this case

may be attributable to the nephritis and its effects on the heart, rather than to the thyroid.

2. The other congestive failure of this series was in a woman of 32; heart No. 493 grams. In 3189 the heart was not at all enlarged (285 grams). But the *clinical* picture was characteristically one of *very considerable* cardiac enlargement. Though the heart was not enlarged *post mortem* the apex impulse was "heaving" and extended 3.5 cm. outside the nipple line in the 5th interspace. There was also visible systolic pulsation along the left sternal margin and in the 2nd right interspace, as is often the case in exophthalmic goiter. This is due in part, perhaps, to the lack of normal pulmonary expansion, the heart's surface being less covered by lung than normal. This patient had auricular fibrillation during the three days of her life in the hospital ward. There was also a loud rough apical systolic murmur and a very loud 1st sound, with great accentuation of the pulmonic 2nd. Yet the valves were negative at necropsy and there was no evidence of the cardiac dilatation and hypertrophy which had seemed so obvious in life. This patient was 45 years old, had had goiter for 17 months, exophthalmos and "nervousness" for two years, tremor for 7 months. In 2½ years her weight had fallen from 140 pounds to 86 pounds. Her skin was generally pigmented. Palpitation had been noticed for two months and diarrhoea for the same period.

Histologically the thyroid showed tubular elements of varying size formed of cylindrical cells, sometimes with short papillary ingrowths. No colloid.

3. (No. 3739) A negress of 50 was seen in May, 1918, complaining that for years she had had a bad heart and at times been disabled by it, but never so severely as in the past six months when her dyspnoea increased to orthopnoea. Dropsy appeared a few weeks ago.

On examination she was found to be poorly nourished, cyanotic, with evidence of much edema in the lungs, legs, liver and subcutaneous tissues generally. There was moderate symmetrical thyroid enlargement. The heart's dullness reached 10.5 cm. to the left and 5.5 cm. to the right of mid-sternum; its action rapid and regular. A rough systolic murmur and a suggestion of a presystolic was heard at the apex. The pulses and artery walls normal. The pulse ranged from 110 to 200 during 10 days' observation, usually above 150. The systolic blood pressure 170, diastolic 80. At times there was fibrillation.

Necropsy showed a heart weighing 280 grams but as the woman was but 4 feet, 10¾ inches high, this weight was considered

about 60 grams beyond her normal, i.e., moderate hypertrophy and dilatation. The aorta was capacious, fibrous and inelastic and in its abdominal portion there were some fibrous and fibrocalcareous areas. The myocardium and valves were normal except for slight fatty degeneration in the myocardium, such as is often associated with infection or anemia (both of which were absent). The rest of the body showed nothing, except general passive congestion, to explain the death, although there was a slight degree of chronic interstitial hepatitis, obsolete tuberculosis in the bronchial glands and a few small foci of fibrosis in the kidneys—not sufficient to affect their function.

Mitral stenosis and regurgitation and possibly a similar lesion at the aortic was the diagnosis in life, with chronic hyperthyroidism and auricular fibrillation.

Possibly an operation on the thyroid early in this patient's illness might have saved her from the fatal decompensative attack.

4. (No. 3961) A woman of 46 gave in 1919 the history of a goiter which she had noticed for 15 years, increasing and decreasing in size. Her eyes, she said, "had always been prominent." Two years ago she began to notice dyspnea and palpitation; later edema of the legs and loose cough. For six months she had had diarrhoea (3–5 movements daily). For nine weeks she had been orthopneic and for four weeks vomiting had occurred almost every day. Recently tremor of the hands, increased circumference of the neck, increased edema of the feet, nervousness and restlessness had been noticed. In five weeks her weight had fallen from 145 pounds to 100 pounds.

Examination showed poor nutrition, nervous restlessness, a warm moist skin, a questionable slight exophthalmos and lid-lag, a smooth elastic enlargement of the thyroid, more marked on the left but without thrill or bruit, and coarse tremor of the fingers. The heart showed no enlargement or arrhythmia; rate 77–131; a snappy first sound and a slight systolic murmur. At this time there was no evidence of passive congestion in the lungs, liver or legs. Two weeks after entrance she became drowsy and ate but little. Next day auricular fibrillation began and persisted until death a week later. There is no record of a metabolism test.

At necropsy the left ventricle showed slight hypertrophy. There was a suppurative nephritis (of which there was no evidence in life), regarded by the pathologist as a terminal infection. *No signs of passive congestion* so that the death in this case probably

belongs in the toxic-infectious group, though earlier in the disease swollen legs and loose cough suggested congestive failure.

These necropsies suggest:

1. That in the toxic deaths of fulminating or untreated thyrotoxicosis, the toxemia is apt to be associated with infection, as is the case of most of the "toxic" death in diabetes, in uremia, and cholemia. Terminal acute pericarditis (two cases), acute endocarditis and suppurative nephritis and streptococcus sepsis were among the infections found.

2. That chronic passive congestion is a relatively rare cause of death in thyrotoxicosis.

3. That characteristic, cardiovascular change is not here demonstrable either in the toxic or the congestive deaths. The heart is very slightly, or not at all, hypertrophied or dilated, the valve orifices are seldom relaxed. The myocarditis demonstrable in one case was in all probability due to coronary sclerosis rather than to the thyrotoxicosis.

4. Persistent thymus, with or without status lymphaticus (found in six out of our ten necropsies), is doubtless a factor in the symptomatology.

5. Two cases were markedly pigmented at the time of necropsy.

## SUMMARY AND CONCLUSIONS

1. In exophthalmic goiter the characteristic cardiovascular changes are an over-demonstrative heart, which seems enlarged but usually is not, and a low peripheral blood pressure.

2. Congestive heart failure is rarely the cause of death. Most deaths are toxic, infectious, or mechanical.

3. In long standing quiescent goiters of the adenomatous type heart symptoms of the congestive type may gradually develop and though resistant to treatment by rest and digitalis may be greatly benefited by operation. Post-mortem no definite pathology is established.

## ILLUSTRATIVE CASES

### Autopsy 3189

A widowed American book-keeper of forty-five entered May 3 for relief of diarrhea, edema of the legs, and skin eruption.

Her husband had died of tuberculosis.

She had been well except for malaria ten years ago.

Until the present illness her micturition and catamenia were normal, her bowels somewhat constipated. Her weight was 140 two and a half years ago, 115 a year ago, 86 two weeks ago.

Two years ago she began to feel nervous, her neck began to swell, her eyes to become prominent, and her heart to beat so that she could feel it. She developed a great desire for cold fluids, and still has it. She began to have frequency, sometimes $\frac{\text{Day } 7\text{–}10}{\text{Night } 9\text{–}10}$. A year and a half ago she began to have tremor. Two months later she entered a hospital and had an operation. After it the tremor increased, her heart became more rapid, and she developed edema of the legs and feet. After four months in the hospital she went home to the country for five months. All the symptoms persisted. In September she began to have dull to sharp pains, first in the right arm, then in both, worse on lying down. She entered the hospital again. By December the pains left, and had not returned. In February she went to a convalescent home. Her eyes, twitching, and nervousness improved, but the tachycardia remained the same. The middle of February she had a "relapse," developed a cold in the head, and had ever since had cough with sputum, several times blood-tinged. She had also had persistent diarrhea, at first 12–15 watery stools a day, more recently 7–8. Swelling of the legs and feet also developed and persisted. For two months she had had brown spots on her legs. She had been dyspneic for a month. Ten days ago she left the hospital, feeling no better. The night before admission her legs began to get red.

The patient was a wild-eyed, fluttering, emaciated wreck of humanity with a brownish pallor. The skin of the legs, chin, etc., showed scattered shallow brown blister-like lesions the size of a dime, several showing scratch-marks. Over the lower abdomen and the inner aspect of both thighs, most marked over the veins, were numerous uniform red-blue areas of capillary stasis. The general nutrition of the skin seemed impaired; a subcutaneous injection of morphia gr. $\frac{1}{6}$ caused a wheal to form rapidly. Sclerae were injected, the mucosae pale. Tongue dry, rough, and pale, with a white coat. Marked exophthalmos, loss of convergence and lagging of the lids. Brownish circles of pigmentation about the eyes. Thyroid very much enlarged, the right lobe more than the left. Marked thrill. The apex impulse of the heart was heaving, in the 5th space. There was visible impulse, systolic, in the 2nd space at the right sternal margin. There was absolute irregularity. The first sound was loud,

$P_2$ accentuated. There was a rough high-pitched double murmur at the apex, probably not endocardial; the action was too rapid to differentiate. The pulses were rapid, thready, irregular in force and rhythm. The lungs were negative. The abdomen was tympanitic except for induration in the wall of the left lower quadrant at the margin of the discoloration. There was some tenderness over this area. The liver dullness extended from the 5th rib to two cm. below the costal margin, where a smooth, rounded, insensitive edge was felt. The genitals were not examined. Extremities: The hands showed nodular enlargement of the finger-joints and fine tremor. The legs showed a good deal of edema and induration, with the discoloration described. The pupils were normal. The reflexes were not attempted.

T. 104.4°, falling to 100.2°. P. 132–160. R. 28–40. Urine: Amount normal. Sp. gr. 1010. A very slight trace of albumin at both of the examinations, a very rare red blood corpuscle and hyalin

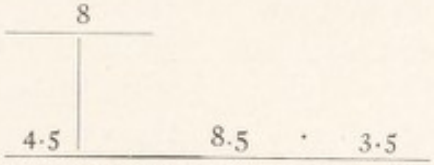



FIG. 157.—Percussion dulness in cm., Case 3189.

cast at the first. Blood: Hgb. 65%, leucocytes 5700, polynuclears 87%, reds 2,824,000, showed considerable achromia. Wassermann negative. Stools, (two examinations): All elements undigested. No ova or parasites. Guaiac negative. Report of skin consultant: "The lesions on the legs might be specific or traumatic. I cannot make a positive diagnosis or suggest treatment. The left thigh shows subcutaneous extravasation of blood. This might also be traumatic." Report of surgical consultant: "I am inclined to think the skin picture is part of the circulatory disturbance associated with the goiter, possibly posterior mediastinal in location."

Following the application of ice-bags to the heart and cold compresses to the left leg and lower abdomen there was a fall in temperature and rapid improvement. The edema of the legs decreased. The diarrhea was less evident. Morphia had, however, been given to the physiological limit (gr. $\frac{1}{6}$ two doses; gr. $\frac{1}{4}$ one dose). In spite of sedatives the patient grew more and more excited and had ideas of persecution, until finally she required restraint with sheets. She was given another dose of morphia gr. $\frac{1}{6}$, and triple bromides gr. 30, hyoscin gr. $\frac{1}{100}$, atropin gr. $\frac{1}{100}$, caffein gr. ii without effect.

She went into coma, the bronchi filled with numerous coarse audible râles, and May 6 she died.

*Clinical Diagnosis (from Hospital Record).*—Exophthalmic goiter. Thrombosis of the pelvic veins.

*Dr. Richard C. Cabot's Diagnosis.*—Thyrotoxicosis.

Hypertrophy and dilatation of the heart.

*Anatomical Diagnosis.*—

| | |
|---|---|
| 1. Chemical or physical origin of fatal illness. | Exophthalmic goiter. |
| 2. Secondary or terminal lesions. | Septicemia, pneumococcus.<br>Amyloid nephritis.<br>Slight chronic interstitial hepatitis, with fatty metamorphosis.<br>Serous atrophy of the fat tissue of the pancreas.<br>Hypertrophy of the spleen.<br>Double hydrothorax and slight ascites and anasarca.<br>Hypoplasia of the adrenals.<br>Foci of pigmentation of the legs.<br>Punctate hemorrhages in the skin of the left thigh. |
| 3. Historical landmarks. | Old salpingitis.<br>Cicatrices of operation wounds.<br>Myomata of the uterus. |

The heart weighed 285 grams and showed no hypertrophy or dilatation, no myocarditis.

The eyeballs protruded.

On the anterior aspect of the neck and extending from the larynx to the suprasternal notch and to the sternocleidomastoid muscles on each side was a bulging firm tumor mass beneath the skin.

The thymus was absent.

The tumor mass beneath the skin showed on section of its inferior portion near the suprasternal notch a grayish translucent firm tissue as well as some hard calcareous matter. A piece of the former was taken for microscopical examination.

Microscopical examination. Thyroid. The tissue was composed chiefly of tubular elements of varying size and formed of cylindrical cells. Sometimes a tubule showed short papillary ingrowths. There was no colloid.

**Necropsy 3468**

A married American woman of twenty-three entered November 28, 1908, for the relief of recurrent attacks of tonsillitis for several years. Her tonsils and adenoids were removed. She became very cyanotic during the operation, and her pulse was of very poor quality, so that shock treatment had to be given. The temperature was 98.4° to 100°, at discharge 97°. The pulse was 109–145–120. She was discharged relieved December 4. The Out-Patient Department records show at this time she had tremor and swelling of the neck.

In February two years later she had had a "cold" all winter with slight expectoration, occasionally blood-tinged. The sputum was negative.

May 8, 1915, she reentered for the relief of pain and swelling in the neck and nervousness. She now gave a history of the diseases of childhood, including scarlet fever. Ten years ago she had "malaria," and "rheumatism" in one joint of the hand. She had tonsillitis every winter. She had one child, in good health. She had had one miscarriage. Her best weight was 110 pounds, her usual weight 100, her present weight 76½.

She had always been nervous and weak, and had tired easily. Since girlhood she had had "sour stomach," sour eructations, and a slight burning sensation in the epigastrium after eating. For ten years she had had paresthesia, a numb "creepy" feeling starting in her left thumb and in the course of two or three hours extending over the hand and arm and the left side of the face and sometimes down the other side. The arm felt dead, and when lifted dropped heavily. After an attack she had a dull, heavy feeling in her head. For eight years she had at times been so nervous that she shook all over and had to take to her bed. For the same period she had had a bilaterial swelling in the front of the neck, varying slightly in size and hardness. Occasionally she had had sharp, severe pain starting in this swelling and running up into her head, beginning and ending suddenly, apparently brought on by excitement or over-fatigue. For seven or eight years she had had attacks of diarrhea brought on by fatigue, excitement or chill and lasting from a few days to four weeks. For seven years she had had rapid heart on emotional disturbance, with sometimes the dropping of a beat; fine tremor of the hands; and when very nervous, tremor of the lips and lower jaw; at times a little protrusion of the eyes. She occasionally had sick headaches, rarely vertigo. For four years her hair had been coming out. She urinated every fifteen minutes by day and three or four

times at night. For two months her skin had been growing browner all over the body. During the past month she had lost twenty pounds. She perspired a great deal. Lately she had had some itching.

Examination showed a poorly developed woman weighing 76½ pounds, yellowish-pale, with brown pigmentation around the eyes, which showed marked exophthalmos, imperfect convergence, and Von Graefe's sign. Except over the face the skin was markedly pigmented, most deeply on the lower abdomen. The tongue protruded with coarse tremor. There was a large pulsating thyroid tumor measuring 33.5 cm., moving on deglutition and showing marked thrill and a loud venous systolic hum. The apex impulse of the heart was in the fourth space. The left border of dullness was 13.5 cm. from midsternum, the right border 3.5 cm. to the right. The supracardiac dullness was 9 cm. The first sound was sharp and ringing. The rate was 126. A roughened systolic murmur was heard at the apex, along the left border of the sternum, and over the pulmonic area. The blood pressure was 130/70. The lungs, abdomen, rectal and vaginal examinations were negative. The arms showed evidence of muscular atrophy, and the fingers fine tremor. The pupils were slightly irregular. Their reactions and the other reflexes were normal.

Until operation the temperature was usually normal, with occasional rises to 99°–99.8°. The pulse was 80–125. The respiration was normal. The output of urine was normal. The urine was cloudy, alkaline at entrance, acid later. The specific gravity was 1.016–1.010. There was the slightest possible trace of albumin at one of three examinations. The hemoglobin was 70 to 80%, the leucocytes 7600, the polynuclears 45%, the reds 4,560,000–5,348,000. The smear showed slight variation in size and shape, an occasional large stippled cell, an increased number of plates, and one normoblast. A Wassermann was negative. The stools were negative to guaiac at three examinations. Bile was present at three of eight examinations, many fatty acid crystals at seven.

The patient was given hydrobromate of quinine gr. v t.i.d. from entrance, changed May 21 to tincture of opium gr. v t.i.d. p.c. She grew quieter, but had marked diarrhea, and by May 21 had lost three pounds in spite of a tremendous appetite and extra diet. May 21 X-ray treatment was started. May 23 she took 6550 calories, though she weighed only seventy-four pounds. Dr. Means found the basal metabolism + 63. The diarrhea and the pigmentation persisted. Nothing was found to account for the latter except

the thyroid. June 2 tannalbin gr. v and fel bovis gr. iv were given t.i.d. They stopped the diarrhea so effectually that the tannalbin had to be omitted after two days because of constipation.

June 14 the left superior thyroid vessels were ligated under novocain anesthesia. The patient was much excited after the operation, but was quieted by bromides. Next day she was very nervous, but in fair general condition. The temperature was 99.5° the evening of operation, then normal until the second operation. The pulse was 120 June 14, 86 June 19, 120 June 21. By June 21 the general condition was good.

June 22 the right superior thyroid vessels were ligated under gas and oxygen. The breathing stopped during the early part of the operation, but was restored after a little artificial respiration. The patient became cyanotic after the operation and could not be aroused. The pulse was rapid and irregular. She was given morphia freely. Her color gradually cleared up, and the pulse became of better quality, but she was very restless. The next morning she seemed a little better, but by night was worse, very restless and weak, with a pulse of 208. She was given digitalis intravenously. Next morning she was found very cyanotic, with feeble and irregular pulse. The right border of the heart was out. After being bled ten ounces her color cleared up and the pulse became of better quality. June 24 she was semiconscious and died.

*Clinical Diagnosis.*—Exophthalmic goiter.

Mitral disease.

Acute dilatation of the heart.

Ligation of the superior thyroid vessels.

*Dr. Richard C. Cabot's Diagnosis.*—Thyrotoxicosis.

Hypertrophy and dilatation of the heart.

*Anatomical Diagnosis.*—(Exophthalmic goiter and ligation of the superior thyroid vessels.)

Status lymphaticus.

Purulent pericarditis and pleuritis.

Pigmentation of the skin.

Chronic appendicitis.

The heart weighed 200 grams and showed no hypertrophy or dilatation and no myocarditis.

There is no pathological report on the thyroid. The neck was covered with a surgical dressing which was not removed.

The thymus gland was present in the form of an elongated flattened mass about 8 cm. in greatest length. On section it was found to be composed of a grayish, translucent, fairly firm tissue.

## CHAPTER XII

### CONGENITAL HEART DISEASE

In our four thousand necropsies which include a good many in children, there were but seven cases of congenital defects or lesions such as seemed of actual or possible damage to the circulation. These comprise three cases of pulmonary stenosis, one complicated by an interventricular septal defect and another by an apparently independent post-natal endocarditis on the tricuspid. There were four cases of septum-defect alone, three between the ventricles, one between the auricles.*

The diagnosis of *some* congenital lesion was suggested by the presence of clubbed fingers in one case and by a deep, unexplained cyanosis in another. The rest were unrecognized. The details are given in the case-reports to follow.

For this book the chief importance of these scattered data is to show how rare congenital lesions are in a twenty-three-year hospital experience. Only seven in a series that comprises 1906 cases were of the congenital type. Unless seen in infancy or childhood, these lesions then are naturally very rare.

Their ages, seven, nine, nineteen, twenty-two, twenty-four, thirty, forty-nine, are what one might expect. But these early deaths are not due wholly to the severity of the cardiac lesion since four of the seven patients died of disease unconnected with the heart, and passive congestion was found at necropsy in only three cases, two of which showed post-natal (rheumatic?) valve lesions as well. So that in only one case of the seven was death due clearly to the congenital lesion.

The cases are too few in number to justify any conclusion except the rarity of congenital lesions after early infancy.

Detailed histories follow.

* Beside the seven cases there were five with harmless anomalies of valve-cusps: In two cases the aortic had but two cusps, in one case it had four. In another case the cusps were slightly fenestrated. In a fifth case the pulmonary valve had four cusps.

I may also mention here the 19 cases of hypoplastic or small-sized aorta. Ten of these apparently produced no effect on the heart. In nine others there was cardiac hypertrophy, occasionally very considerable.

## ILLUSTRATIVE CASES

### Necropsy 106

A mentally defective girl of nine was brought to the Accident Room March 17. She had had measles and chickenpox, and had always been nervous and easily excited. She did not walk well until she was four. She had always complained much of pains in the legs. For the past three weeks she had had cough. Five days before admission she began to feel stupid and to complain of pain in the stomach. March 15 she had a little vomiting of watery material with the cough, which was very severe. The night before admission there were several blood spots on her pillow. The night of admission she had a hemorrhage of liquid and clotted blood and some fecal material from the bowels, fully a quart altogether. An hour later she had another movement of about the same amount.

Examination showed a pale, fairly well nourished child looking older than her years, tossing about on the bed with sighing respiration and convulsive movements of the hands, head and facial muscles but no suggestion of exhaustion. She was said to do this commonly, especially when excited. There was a loud systolic murmur at the apex of the heart transmitted to the axilla, and heard also all over the back and at the aortic area transmitted upward. At the apex was a presystolic murmur. The pulmonic second sound was accentuated. The abdomen, pupils and reflexes were normal. The output of urine was not recorded; the findings were normal.

The child had two bowel hemorrhages in the first night and early the next morning, perhaps five ounces in all, with a few clots and some loose fecal material. She continued to have slight hemorrhages for the next three days. She was very nervous, and screamed without apparent cause. She had troublesome cough at first which improved. April 10 she was discharged relieved.

After leaving the hospital she was well until the morning of June 6, when she had another intestinal hemorrhage (amount?). She was brought to the hospital that day, pale, restless, and frequently crying out, with scarcely perceptible pulse. The heart action was rapid. There was a loud systolic murmur heard everywhere over the chest and back. The day of admission she had a hemorrhage of about sixteen ounces, during the night another of eight ounces, and next day one of five ounces. Just before each hemorrhage she cried out and threw herself about on the bed. Her respiration varied greatly. Sometimes it was quick and short, sometimes sighing. Restlessness

was the most marked feature of her condition. June 7 her pallor was much greater than at admission. That afternoon she had two movements of the bowels containing considerable fecal material mixed with blood. Her pulse was very poor. Her respirations grew more rapid and the pallor extreme. She became, however, less restless. The evening of June 8 she was much weaker, with Cheyne-Stokes respiration. Just before the infusion of eight ounces of salt solution the heart sounds were short and no murmurs could be heard. Twenty minutes later the general condition seemed better and the murmur was heard again. She seemed better during the rest of the night, with fair pulse and quiet respiration. Early the following morning when apparenly in good condition she suddenly collapsed and died.

*Clinical Diagnosis.*—Intestinal hemorrhage.

*Anatomical Diagnosis.*—Congenital malformation of the heart (partially defective interventricular septum and defective mitral valve).

Chronic mitral endocarditis?

Slight cardiac hypertophy.

Fatty degeneration of the myocardium.

Bronchopneumonia and atelectasis of the inferior lobe of the right lung.

Purulent bronchitis.

Meckel's diverticulum with localized dilatation of the ileum leading to

Intestinal hemorrhage.

General anemia.

Old pleural adhesions.

The heart was somewhat larger than normal, weighing 163 grams. The left ventricle had a capacious cavity with rather thick pectinate muscles. In the ventricular septum just below the insertion of the mitral valve and under one of the cusps of the aortic valve was a semicircular defect in the muscle tissue, its convexity pointing downward toward the apex and measuring 18 by 12 mm. This defect was filled up by an irregular membrane of yellow fibrous tissue which in places was diaphanous and separated the cavities of the ventricles. This membrane of fibrous tissue seemed to represent and to be continuous with one of the curtains of the tricuspid valve. The mitral valve opposite the defect in the ventricle over a length of 3.5 cm showed a marked shortening and almost obliteration of its curtain in an irregularly crescentic form with the convexity upward. The

edges of the valve along this defective portion were markedly thickened but smooth. The median portions of what remained of the valve were attached to the margin of the defect in the septum by short branching chordae tendineae. The remainder of the valve seemed normal and was not thickened. At the extremities of the crescentic shortening of the valve were long rather thick chordae tendineae. The mitral valve measured 9 cm. in circumference, the tricuspid 9 cm. The wall of the left ventricle was 1 cm. thick, the right 4–5 mm. The myocardium generally was pale yellowish brown with fine mottling of the pectinate muscles of the left ventricle. The endocardium of the left auricle showed yellowish irregular fibrous

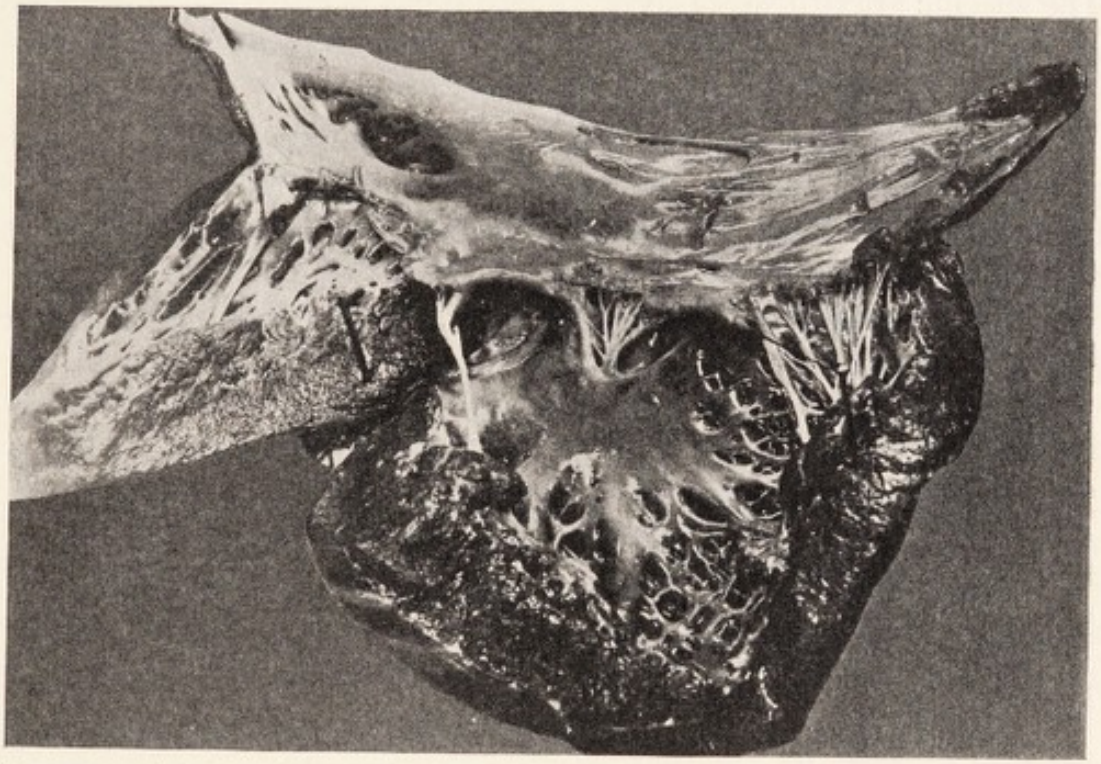


FIG. 158.—Congenital, defective interventricular septum and defective mitral valve. (Photograph by Lewis S. Brown. Dr. Oscar Richardson.)

patches. The foramen ovale was closed. In the pericardium there was some clear fluid with fibrinous clots. The bowel bleeding which was the main cause of death, is not clearly explained but was apparently connected with the Meckel's diverticulum.

### Necropsy 238

A Swedish cook of nineteen entered March 15, 1898. She said she had never been ill before. The friend who came with her however said that at twelve years of age she was in bed a year with bloody vomiting and great epigastric pain. For the past two months she had felt weak, had often vomited after eating, and had gradually

become pale, losing appetite and some weight. Two weeks before admission she had severe sore throat. After being in bed a week she was up for four days. March 12 her legs began to swell and were so painful that she could not walk. Her hands also swelled and were painful. She still vomited after eating and had some epigastric pain. Her bowels were very constipated.

Examination was negative except for extreme pallor, an occasional faint systolic murmur at the apex of the heart and over the pulmonary area, and swelling, heat, and tenderness of both ankles.

The temperature was 102.8° falling steadily to 97.7°, the pulse 115–88, the respiration 28–32. The output of urine is not recorded. The specific gravity was 1.037. There was a slight trace of albumin at the single examination. The hemoglobin was 45%, the leucocytes 20,000, the reds 3,248,000.

The patient vomited everything taken by mouth. She complained of great pain in the arms. On examination nothing was found but tenderness. The evening of March 16 she started to vomit and very suddenly died.

*Clinical Diagnosis (from Hospital Record).*—Acute articular rheumatism.

*Anatomical Diagnosis.*—Ulcer of the stomach.

Anemia.

Verrucose endocarditis of the mitral valve.

Acute degeneration of the myocardium.

Partial defect of the ventricular septum.

Slight acute hyperplasia of the spleen.

Small cysts of the right ovary.

Dr. Richardson: The skin seems to have been noticeably pale and waxen. The brain and meninges were frankly negative. There were some old adhesions in the peritoneal cavity and some of these were about the stomach.

The heart showed slight hypertrophy. There was a partial defect in the interventricular septum just below the aortic cusp and some acute degeneration of the myocardium. On the mitral valve there was a row of minute granulations. Other than that the heart, the aorta and great branches were negative.

The liver, spleen, and kidneys called for no mention, and the microscopic examination of the kidney was negative.

That brings us to the gastro-intestinal tract. About midway between the cardia and the pylorus in the posterior wall was an ulcer, rather deep, five to six mm., and the other dimensions two by three

cm. On the peritoneal aspect there were adhesions, and at the base of the ulcer the pancreas was visible. There was no evidence of new growth. It was frankly an ulcer of the stomach.

DR. CABOT: Of course she died of the gastric ulcer. Those mitral granulations were terminal things and not the cause of all this anemia?

DR. RICHARDSON: I should think so.

### Necropsy 303

An Italian housewife of twenty-two entered April 20. She had had two children and one miscarriage. She had had no catamenia for four months, and during that period had had dyspnea and cough. For the past two weeks the cough had been worse and she had had bad color, pains in the joints and very little urine.

Examination showed cyanosis of the whole body, marked about the lips, cheeks, tongue, ears and nails. The hands and feet were cold. There was slight glandular enlargement. The lungs were normal except for slightly harsh respiration at the left apex. The apex of the heart was in the fifth space in the mammillary line. The left border of dullness was just to the left of the nipple line, the right border three finger-breadths to the right of the sternum. There was no thrill, but a duplicated impulse was felt. At the apex was a harsh systolic murmur transmitted to the axilla and heard over most of the cardiac area. Just at the end of the murmur two short sharp sounds were heard faintly. At the base the sounds were very feeble, with accentuation of the pulmonic second. The liver extended to a hand's breadth below the costal margin and was somewhat tender. No enlargement of the uterus was made out. There was slight edema of both legs.

During her thirteen days in the hospital she ran an irregular temperature, 97–101.5° with one drop to 95.6°, followed by two days of subnormal temperature. The pulse was 83–113, the respirations 22–36. The output of urine was 13–70 ounces, the specific gravity 1.013–1.028. All of three examinations showed albumin $\frac{1}{20}$–$\frac{1}{6}$%. The sediment at one examination showed a few abnormal red blood cells. There is no record of the blood except "leucocytes 8200."

Under rest and purgation the patient made marked improvement. The edema decreased, her color improved, and the liver became smaller. May 2 she was discharged relieved.

After leaving the hospital she was fairly well until early in June, when edema of the legs and dyspnea returned. At her readmission

to the hospital June 22 she was unable to use the right half of the body.

Examination showed her very cyanotic, with right hemiplegia. The whole surface of the body was cool and the extremities cold. The left pupil was larger than the right and the left eyelid opened wider than the right. The face was drawn slightly to the right, and in laughing the muscles of the right side of the face were more active. Over the back and the sides of the chest was a brownish maculo-papular eruption. The bases of the lungs showed edematous râles and the left apex slightly harsh respiration. The apex impulse of the heart was felt in the fifth space two inches outside the mamillary line, forcible and very diffuse, coinciding with the left border of dullness. The right border was three finger breadths to the right. The systolic murmur was heard all over the cardiac area except in the aortic region. Over the tricuspid area it seemed to have a higher pitch. The heart sounds were confused, rapid, and at long intervals intermittent. The pulse was rapid and very slightly intermittent. The liver dullness extended to a hand's breadth below the costal margin. The right leg was much more edematous than the left. No knee-jerks were obtained.

The temperature was 97.7°–99.7°, the pulse 80–107 until July 3, then July 4 and 5 swinging from 53 to 108, dropping to 67 the day of death. The urinary output was 10–33 ounces except one day when it reached 45 ounces; cloudy, specific gravity 1.025–1.009. The sediment showed leucocytes at both examinations and a few red blood corpuscles at one.

The edema and hemiplegia gradually decreased and the cyanosis cleared a little. The cardiac condition remained unchanged. July 4 the patient's sister died of heart trouble. July 7 the patient insisted upon going home.

After leaving the hospital she grew more cyanotic and became blind except for recognizing light. Pain developed about the liver and she became unable to lie down.

Upon examination at her third admission, July 29, she showed external strabismus of the right eye and internal strabismus of the left. She could bring the eyes to the median line but not to the left side. She appeared wholly blind. The pupils reacted to light. The lungs were negative. The cardiac impulse was very weak, scarcely felt. The percussion measurements were as before. The systolic murmur was faint at the apex, transmitted into the axilla. At the tricuspid area was a faint systolic blow of higher pitch than the apex systolic.

The pulmonic second sound was slightly accentuated. The sounds were much fainter than at her last stay in the hospital. The base sounds were very weak. The liver was three finger breadths below the costal margin, hard, somewhat tender. The legs showed no edema. The left upper thigh was larger than the right, tender along the femoral vein, hard, resistant. The patient cried out with pain upon pressure over Scarpa's triangle. No paralysis was made out, merely equal sided weakness. The rash was generalized over the body. No urine could be obtained for examination. The blood is not recorded.

During her three days in the hospital she ran a temperature of 101.3°–103.4°, the pulse 120–130, the respirations 30–15. The day after admission the tenderness and swelling over the left femoral vein had largely cleared up. The general condition however was worse. There was much more cyanosis. The systolic murmur was large at the apex. She gradually failed, became purple, yet showed no edema of the lungs, abdomen or legs. July 31 she died.

*Clinical Diagnosis.*—Mitral regurgitation.

Congenital heart lesion.

*Dr. Richard C. Cabot's Diagnosis.*—Chronic endocarditis of the mitral and tricuspid valves.

Mitral stenosis.

Embolism and thrombosis.

*Anatomical Diagnosis.*—Stenosis of the pulmonary valve of the heart (congenital malformation?).

Verrucose endocarditis of the tricuspid valve.

Hypertrophy and dilatation of the right ventricle and the right auricle.

Partially patent foramen ovale.

Thrombosis of the right auricular appendix, of the right ventricle, and of the left ventricle.

Infarctions of the kidneys.

General chronic passive congestion.

DR. RICHARDSON: There was no edema of either upper or lower extremities. There was some ascites, moderate hydropericardium, no fluid in the pleural cavities. There was some congestion of the lungs, and old pleural adhesions on the right side.

The liver was one hand down.

The heart weighed 631 grams,—marked hypertrophy. The right ventricular wall measured from twelve to fifteen mm., the left

ventricular wall ten mm. There was slight dilatation of the right ventricular cavity, much dilatation of the right auricle.

DR. CABOT: That is, the right ventricle was actually thicker than the left? That is extraordinary!

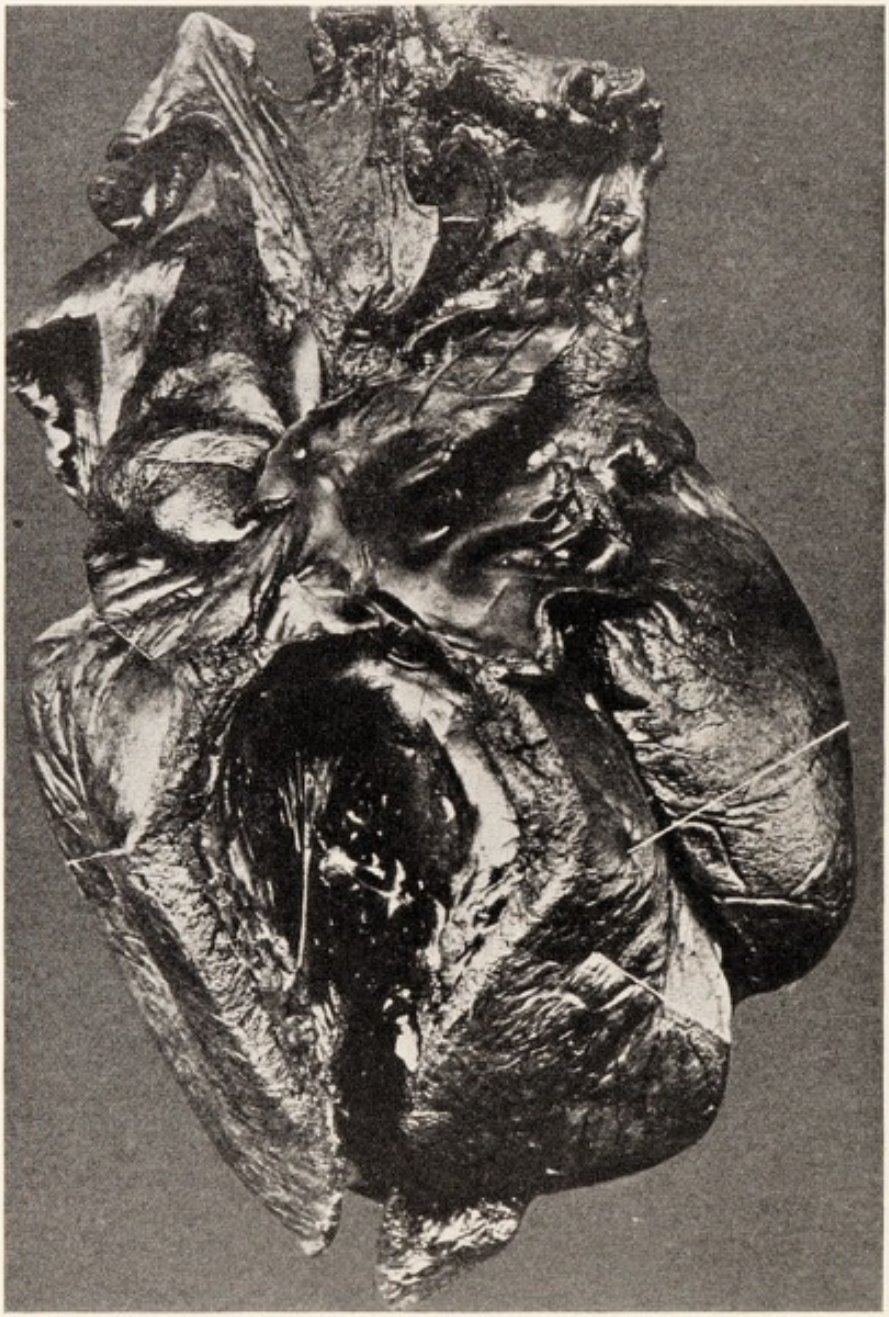


FIG. 159.—Stenosis of pulmonary valve (congenital?). (Photograph by Lewis S. Brown. Dr. Oscar Richardson.)

DR. RICHARDSON: The wall of the right auricle was four to five mm. thick; the left negative. There was slight dilatation of the left cavities.

The aortic and mitral valves were negative. There were mural thrombi in the left ventricle. On the tricuspid valve there were a

few small firm fibrous granules,—the so-called verrucose endocarditis The cusps of the pulmonary valve generally were fused, thickened, a smooth fibrosis, and from them three bands extended on the pulmonary artery, the whole process producing pulmonary stenosis, congenital. There was also a partially patent foramen ovale. There were mural thrombi in the right ventricle and right auricular appendix.

The liver, spleen, and kidneys showed chronic passive congestion. From the mural thrombi on the left side there were infarcts in the kidneys.

The mucous membranes were markedly purple.

DR. CABOT: Pulmonary stenosis is one of the severe congenital lesions. I do not believe for a moment that that child had failed to show cyanosis and heart murmurs much earlier. I entirely believe that if we had got a decent history here I should not have gone so entirely wild as I did.

### Necropsy 2453

A boy of seven who had been found deserted and starving in a tenement was brought to the hospital October 5. Nothing was known of his history except that he had been ill about a week.

Examination showed a fairly well developed, poorly nourished, sick looking child with marked rosary and prominence of the superficial veins of the chest and abdomen. The tonsils were somewhat enlarged. There were slightly enlarged glands in the neck and groins.

The precordia showed slight prominence. The apex impulse of the heart was barely palpable in the fifth space just outside the nipple line, 2¼ inches from midsternum, corresponding to the left border of dullness. There was no enlargement to the right. The action was regular and slow. A loud harsh blowing systolic murmur was heard all over the precordia entirely replacing the first sound, loudest in the third and fourth spaces to the left of the sternum, transmitted deep into the axilla and into the vessels of the neck. The pulmonic second sound was accentuated and louder than the aortic second, which was also accentuated. The pulses were poor in quality. Both testes were undescended. The fingers were somewhat clubbed, the nails cyanotic. Examination was otherwise negative.

The child lived only a day in the hospital. His temperature rose from 98.4° to 101.9°. The pulse and respirations were not remarkable. The urine was normal except for a trace of albumin. The

hemoglobin was 100%, the leucocyte count 26,000. Early the morning after admission he vomited and immediately stopped breathing. After fifteen minutes of artificial respiration the breathing began again, deep, slow, stertorous, of the air hunger type. During the artificial respiration vomitus was expressed through the mouth and nose. The type of breathing suggested the possibility of thymus —though no thymic enlargement could be determined—or of some metabolic or some central nervous affection. Air hunger breathing continued for about three hours. The respirations ceased before the heart.

*Clinical Diagnosis.*—Congenital heart disease.

Aspiration of vomitus.

Persistent thymus?

*Anatomical Diagnosis.*—Abscess of brain (streptococcus).

Leptomeningitis (streptococcus).

Stenosis of the pulmonary valve (congenital).

Incomplete closure of the interventricular septum.

Dilatation of the heart.

Right ventricular hypertrophy of heart.

Chronic pericarditis.

Chronic perihepatitis.

Chronic tuberculosis of the mesenteric lymph glands.

Dr. Richardson: The age is given here as five. At the base of the brain there was frank meningitis. On the left side—the absolute boundary is not given, but I should think about in the left parietal region—there was a large abscess which extended into the left lateral ventricle,—frank abscess of the brain and leptomeningitis at the base, the organism streptococcus.

The skin and mucous membranes were purplish, the fingers slightly clubbed.

The heart weighed 135 grams. The organ was not remarkable except for a congenital stenosis of the pulmonary valve and a perforation, incomplete closure of the interventricular septum, rather a common type of congenital condition.

Culture from the heart blood yielded streptococcus.

There was some slight chronic tuberculosis of the mesenteric lymph glands.

Dr. Cabot: How do you feel about the air-hunger? What caused it?

Dr. Fremont-Smith: I think he had increasing intravenous pressure.

DR. CABOT: That is, he did not have any air-hunger, he had breathing mistaken for that.

DR. RICHARDSON: The thymus gland was present and weighed four and a half grams,—negative.

### Necropsy 2599

A Nova Scotian housewife of forty-nine entered April 25. She remembered no serious illnesses or injuries. She passed the menopause six years before admission. Three weeks and a half before admission while working in a factory her hair was caught in some machinery and her scalp torn almost completely off. Twenty minutes after the injury she was taken to a hospital where the wound was dressed without ether. The pain had increased somewhat but had not been unendurable.

Examination was negative except for obesity and the injured area, which was covered with healthy looking granulation tissue with fresh epithelium growing in about its edge. There was no pus and not even a red angry look.

The *heart* was not enlarged. The action was regular, the sounds clear and of good quality. No murmurs were heard. The pulses were of fair volume and tension.

The patient slept fairly well and complained of nothing. April 30 a Tiersch skin graft was done under spinal anesthesia, skin being taken from the thighs. The patient stood the operation well and returned to the ward in good condition. Except that some of the grafts at the back of the head floated off with pus she seemed to be doing very well until May 7, when she suddenly became very cyanotic, with extreme air hunger and thready pulse. She failed to respond to stimulation or artificial respiration and died that day.

*Clinical Diagnosis.*—Avulsion of scalp.

Pulmonary thrombosis.

*Anatomical Diagnosis.*—Absence of the scalp (avulsion) with granulation tissue formation and skin graft.

Thrombosis of the posterior tibial veins, left and right.

Embolism of the right auricle and the pulmonary arteries, right and left.

Perforate interventricular septum.

Open foramen ovale.

Fatty metamorphosis of the liver.

Fibromyoma of the uterus.

Heart: Weight 314 grams. The epicardium contains abundant fat. On section the cavities of the left side empty. Myocardium brownish red and limp. Wall of left ventricle 1.4, of right 0.4 cm. in thickness. Mitral valve 9.6, aortic valve 6, pulmonary valve 6, tricuspid valve 10.5 cm. in circumference. Depth of left ventricle 9.3 cm. The aortic curtain of the mitral valve contains several small, smooth, yellowish patches; the endocardium and valve curtains otherwise normal. The interventricular septum at a point 3 cm. below the anterior curtain of the aortic valve shows an oval depression about 1 cm. across, at the bottom of which is an opening 0.2 cm. across; a probe introduced into this passes obliquely downward and to the right, emerging in the cavity of the left ventricle at a point 5 cm. above the apex. Coronary arteries contain small, soft, yellowish patches. At the site of the foramen ovale, along the anterior border of the membrane, is a slit-like opening about 1 cm. long, well guarded.*

DR. RICHARDSON: This case was examined by the Medical Examiner, and we have the anatomical diagnosis and a note through the courtesy of Dr. Magrath.

There was a thrombosis of the posterior tibial veins, left and right. That of course is the important statement, because that is the probable source for the pulmonary embolism which was present.

DR. CABOT: How much did the congenital heart lesion enter in? Do you think it is of any importance?

DR. RICHARDSON: Not as a cause for death.

DR. CABOT: She would have got along all right with that?

DR. RICHARDSON: Yes. It is not a thing we like to have in a heart, but we should not expect death to result from it. It was an additional load, but the death here was due to frank pulmonary embolism, and the only source we have for it is in the thrombosis of the posterior tibial veins.

DR. CHURCHILL: It would look as though the skin-grafting might have been the cause of death rather than the avulsion. They took the skin from the thighs according to the record.

DR. CABOT: Why should that bring about a thrombosis in the leg veins?

DR. CHURCHILL: There is no reason whatsoever. There is no mention made of sepsis, and the wounds of a skin graft are really intradermal.

* The anatomical data in this case are furnished through the courtesy of Dr. George Burgess Magrath, Medical Examiner of Suffolk County, Massachusetts.

DR. RICHARDSON: Except as mention of the vein is made. The thrombosis is due to something.

DR. CABOT: It is hard to see how a skin-graft in the thigh could do that.

DR. RICHARDSON: Unless there was some infection. There certainly was infection higher up.

DR. CHURCHILL: Is it possible that in a patient with a congenital heart lesion there exists some mechanical situation which would favor thrombosis?

DR. CABOT: I do not know any reason to think so.

### Necropsy 2753

An American of thirty, who had been working intermittently as a salesman, entered December 22. His father died of kidney trouble. Since infancy the patient had at times been blue, especially when he was chilled, and had always had dyspnea on exertion. At four he had measles followed by "dropsy." Until the age of twenty-one he had frequent mild nosebleeds. At twenty-three he was confined to bed for seven months with rheumatism—fever and pain in the joints.

For six months he thought his dyspnea on exertion had been worse. He had had a slight cough. A month before admission he had a cold for a week, with nasal discharge and chilly feelings. Four days before entrance he had chills followed by severe headache and pains all over the body, especially in the right chest upon deep inspiration, with cough, and for three days a good deal of white frothy sputum, now blood-tinged. Although feeling feverish and unwell he had tried to do a little business. For a few nights he had urinated twice. There had been some general pain low in the abdomen. At entrance from the Accident Room his hands, face, and ears were very cyanotic, the pulse only moderately rapid. He complained of being chilly.

Examination showed a fairly developed, poorly nourished man with rapid breathing frequently interrupted by a short cough, and with the cyanosis mentioned. The throat was very red. His nervousness and garrulity suggested toxic delirium. The apex impulse of the heart was seen and felt over the outer region of the precordium and as far as 3 cm. outside the nipple line in the 5th space, with a very slight suggestion of impulse in the sixth space, nipple line. The upper border of dullness was at the 4th rib, the lower in the sixth space. No enlargement to the right was made out.

The action was somewhat rapid, occasionally intermittent, forceful. The first sound at the apex was replaced by a low-pitched blowing systolic murmur transmitted towards the axilla, heard best in the 3rd and 4th left spaces. The second sound at the apex was rather weak. At the base $P_2$ was indistinct, fairly forcible but not loud, rather obscured by systolic murmurs (*sic*). $A_2$ was not heard. At the aortic area was a higher-pitched systolic murmur transmitted to the region of the arch, the subclavian and carotid arteries on both sides. A systolic murmur was heard also in the axilla and back. The pulses were of fair volume and tension, regular except for occasional intermissions. The artery walls were palpable. Lungs: There were sharp crackles and a few moist râles at the base of both axillae, slightly exaggerated bronchovesicular breathing with increased whisper and voice but no râles, at the anterior right upper lobe. The voice from the posterior apex to the spine of the scapula was slightly increased. Expansion was slightly restricted. The abdomen was held rigid. The pupils and reflexes were normal.

Temperature 104°–104.5°, pulse 89–101, respiration 22–25. Systolic blood pressure 105–85, diastolic not recorded. Urine: Amount and sp. gr. not recorded. Cloudy. A trace of albumin; many brown granular casts with occasional red blood corpuscles attached; a few free red blood corpuscles; leucocytes. Blood: Hgb. 90%. Leucocytes 17,700–20,300. Reds 5,800,000, slight achromia,, considerable variation in size, slight poikilocytosis. Sputum: Prune juice; predominating organism pneumococcus in pairs and in chains, occasionally intracellular.

The day of admission dullness, bronchial breathing, bronchophony and numerous coarse moist râles developed over the right upper and middle lobes. The heart remained as at admission except for a sharp and reduplicated first sound at the apex suggesting a presystolic murmur. A fairly definite presystolic thrill was now felt at the apex. At the base was a loud systolic murmur apparently of different pitch and quality from the apical one, heard equally well over the aortic and pulmonic areas. During the day the patient became very delirious, at six p.m. showed a pulse of poor quality, and at eight p.m. began to hiccough spasmodically, the pulse becoming very weak and intermittent during the attacks. The patient grew steadily worse, the chief complaint being irregular and superficial breathing. Next morning he died.

*Clinical Diagnosis (from Hospital Record).*—Lobar pneumonia. Chronic mitral endocarditis.

Congenital heart disease.
*Dr. Richard C. Cabot's Diagnosis.*—Mitral stenosis.
Aortic stenosis.
Lobar pneumonia.
Hypertrophy and dilatation of the heart.
Possibly some congenital heart lesion.
General passive congestion.
*Anatomical Diagnosis.*—

| | |
|---|---|
| 1. Primary fatal lesions. | Lobar pneumonia, right lung.<br>Stenosis of the pulmonary valve, congenital (?).<br>Fibrous endocarditis of the tricuspid valve; slight stenosis. |
| 2. Secondary or terminal lesions. | Hypertrophy and dilatation of the heart.<br>Chronic passive congestion, general. |
| 3. Historical landmarks. | Chronic pleuritis, right.<br>Defective closure of the foramen ovale. |

The heart weighed 430 grams. The myocardium on section was of good consistence, firm and pale red. The wall of the right ventricle generally was markedly thickened measuring from 10 to 15 mm. The right auricular wall was thickened The left ventricle wall was 14 mm. The columnae carneae were markedly thickened in the right ventricle and fairly well marked in the left. The cavity of the right auricle was enlarged. The other cavities of the heart showed no definite enlargement. The mitral valve measured 10 cm. in circumference, not remarkable. The aortic valve measured 5¼ cm. The cusps were slightly thicker than usual but otherwise the valve was not remarkable. The tricuspid valve measured 10 cm. The circumference was decreased. The curtain showed a moderate amount of diffuse fibrous thickening which in places was slightly nodular. The pulmonary valve was about 5½ cm. in diameter and about 17 mm. in circumference. The orifice of this valve just about admitted the passage of an ordinary lead pencil. There were three cusps. The cusps were small but rather deep. The walls of the cusps showed generally a moderate amount of diffuse fibrous thickening which was slightly nodular in places. There was some fusion of the contiguous cusp margins. The pulmonary artery and its primary branches were small but the branches in the lung were of good size.

The coronary arteries were free. Each coronary was rather capacious, the right showing more increase in size. The intima of the coronary showed a few small plaques. The foramen ovale presented an oval defective closure about 6 mm. in greatest diameter. The aorta and its great branches were small. The intima of the aorta showed a few yellowish fibrous plaques here and there. Vena cava not remarkable.

*Note by Dr. Oscar Richardson.*—The stenosis of the pulmonary valve in this case is regarded as being probably congenital. The cusps were small and the character of the fibrosis present was not definitely that of endocarditis. In support of the hypothesis that the lesion was congenital are the facts that the pulmonary artery and its primary branches were small and that there was an oval defective closure of the foramen ovale 6 mm. in diameter. Against this hypothesis is the mentioned fibrous endocarditis of the tricuspid valve. This however was slight in amount and apparently bore no relation to the process in the pulmonary cusps.

### Necropsy 2997

*First Entry.*—An unmarried American working-girl of twenty-four entered December 13. Her father had died of "heart trouble." She had had measles in infancy, "dropsy" at eight, and pneumonia at ten. As a child she was never able to run and play because of dyspnea.

For the last four years she had been unable to do much work on account of dyspnea and occasional edema of the legs. She had also had a slight hacking cough with occasional white sputum. She slept with three or four pillows. For six weeks she had been especially dyspneic and orthopneic.

The physical examination was as at the second entry, except for a less marked general anasarca. X-ray showed a mass which did not pulsate at the right of the heart. The pulsations of the heart itself were very indistinct. The outline of the heart shadow with the poor pulsation suggested pericardial disease. There was extensive mottling extending out from both lung roots. The apices were slightly involved on the left.

Temperature, 96° to 100°. Pulsations, 61 to 129. Respirations 23 to 33. Urine: Specific gravity 1013–1019. Slight traces of albumin at three of five examinations. Blood: Hemoglobin 95%. Leucocytes 17,900 to 9000. Two Wassermanns positive, one strongly. Gonococcus fixation test positive.

The patient improved in general condition. The heart condition did not change. The vaginitis cleared up under douches and stock vaccines. The sputum was negative for tubercle bacilli. January 10 she was discharged.

*Second Entry.*—February 3 she returned, having been in bed since her discharge with much dyspnea, oliguria, orthopnea, increasing edema of the legs and abdomen, and a troublesome cough. Her physician reported that twice lately he had found her very cyanotic, gasping for breath, frothing at the mouth, with relief from morphia and nitroglycerin.

Physical examination showed a poorly nourished girl with yellow and cyanosed skin, cyanotic mucosae and yellow sclerae, breathing heavily. The lungs, and abdomen showed evidence of passive

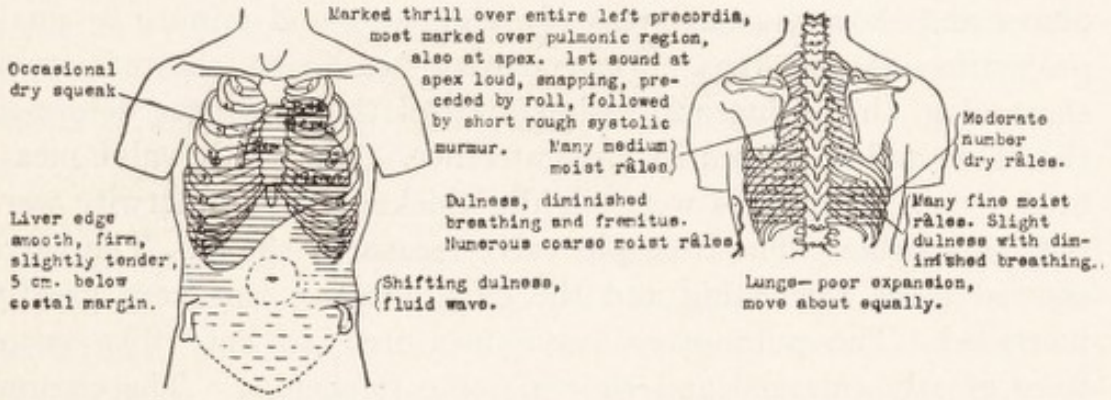



FIG. 160.—Necropsy 2997. Clinical signs in a case of mitral stenosis, congenital defect in the interauricular septum, defective closure of the foramen ovale. hypertrophy and dilatation of the heart.

congestion. Marked systolic thrill over the precordia, most marked over pulmonic region, also at apex. 1st sound at apex loud, snapping, preceded by a roll, and followed by short rough systolic murmur. The pulses were of fair volume and tension. The artery walls were not felt. There was marked edema of the legs and ankles, and some over the sacrum. The fingers and toes were clubbed. The pupils and reflexes were normal.

Temperature 96.4° to 97.8°. Pulse, 120° to° 125. Respirations, 30 to 44. Systolic blood pressure 100. Urine not recorded. Blood: hemoglobin 90%. Leucocytes 11,000, polynuclears 77%.

The day after entrance the patient was more cyanotic and complained of more precordial distress. The right border of the heart had moved out slightly. The heart sounds were very irregular and poor in quality. After digipuratum gr. iss had been given intravenously and again six hours later intramuscularly, there was

some improvement in the character of the sounds. That night ℥vi of blood were withdrawn. After this the sounds became more regular and better in quality, and the right border came in a little. That night the patient died.

The heart weighed 665 grams, enlarged. On section the myocardium was of good consistence and pale brown red. The right ventricle wall measured 6 mm., the left ventricle wall 9 mm. The columnae carneae, the papillary muscles and the pectinati on the right side were enormously thickened. The columnae carneae on the left side were fairly well marked. The cavities of the right side were enormously enlarged. The cavities of the left side were only slightly enlarged. The mitral valve circumference was 7 cm. The curtain showed much diffuse fibrous thickening with irregular ridge-like thickening of its free margin which was somewhat nodular in places and showed small areas of roughening and minute to small projecting fibrous tags. The chordae tendineae showed much shortening, thickening and fusion. All of these changes deformed the valve and decreased its circumference. The aortic valve measured 5 cm. The cusps were slightly thickened but otherwise were not remarkable. The tricuspid valve measured 18 cm. The valve showed some thickening and the circumference was very greatly increased. The pulmonary valve measured 10 cm. The cusps were greatly enlarged and showed some thickening. The circumference was increased. The foramen ovale presented an oval shaped defective closure 1 cm. in greatest diameter. The upper border of the foramen ovale appeared as a strongly-marked, smooth-surfaced, rounded ridge above which there was a defect in the interauricular septum which easily admitted the passage of the middle finger. The endocardium in the region of the defect was perfectly smooth and shining. The superior and inferior cavae were as usual, but only two pulmonary veins were made out and they were much larger than usual. One of the veins emptied into the left auricle near the region of the defect in the interauricular septum. The auricular appendices were not remarkable. The right side of the heart was engorged with current jelly-like blood clot. The coronary arteries were free and the intima was smooth. The aorta was rather small but otherwise not remarkable. The great branches were not remarkable. The venae cavae were not remarkable.

The other features of the necropsy beside the *mitral stenosis and the defect in the interauricular septum* were:

Chronic passive congestion, general.
Infarct of the left lung.
Hydropericardium.
Hydrothorax.
Ascites.
Anasarca.
Slight acute glomerulo-nephritis.
Scoliosis.
Old infarct in one kidney.

*Note by Dr. Oscar Richardson.*—In this case there was a defective closure of the foramen ovale 1 cm. in greatest diameter and just above the foramen border there was a defect in the interauricular septum which easily admitted the passage of the middle finger. The margins of the defect were smooth and shining. Taking the open foramen ovale and the defect in the auricular septum together there was a rather large channel of communication between the two auricles, sufficient to interfere with the circulation.

### Case LXIX

An unoccupied Bavarian-American of twenty-one entered September 2 for study. His mother had one child stillborn. An uncle died of tuberculosis. The patient had measles and pertussis in childhood. For six years he had had regularly intermittent periods of coughing. The present one was of two months' duration. He raised sputum, occasionally slightly bloody, chiefly on getting up. Two years before admission he had tonsillitis. The spring before admission he had severe nosebleeds lasting three or four hours. Altogether he bled about three glassfuls. He had had two or three heartburns and occasional indigestion and headaches. He was deaf in the left ear. He sometimes urinated once at night. Three years before admission he weighed 138 pounds, his best weight. His usual weight was 130. He had not worked for two years and a half, but had lived quietly out-of-doors all day.

His mother said that except for slight dyspnea on exertion he seemed normal in every way until he had pertussis at four years. Then for the first time after an attack of coughing he became cyanotic. The cyanosis had persisted, with remissions of a few hours. It became more marked on exertion or excitement, and sometimes when he was quiet, usually in such cases after meals. He had dyspnea on slight exertion. Medicine never helped him. His condition was about the same as it always had been.

Examination showed a well-nourished man with dusky, moist skin and prominent veins. The mucous membranes were very cyanotic, the sclerae injected. The chest showed an anomaly of the fourth and fifth ribs in the left nipple line. The apex impulse of the heart was not found. The percussion borders were 9 cm. to the left of midsternum, 3 cm. to the right, the supracardiac dullness 5.5 cm. The sounds, action, and pulses were normal. A rough systolic murmur was heard over the whole precordia, loudest at the pulmonic area. Electrocardiogram showed marked right ventricular prepon-

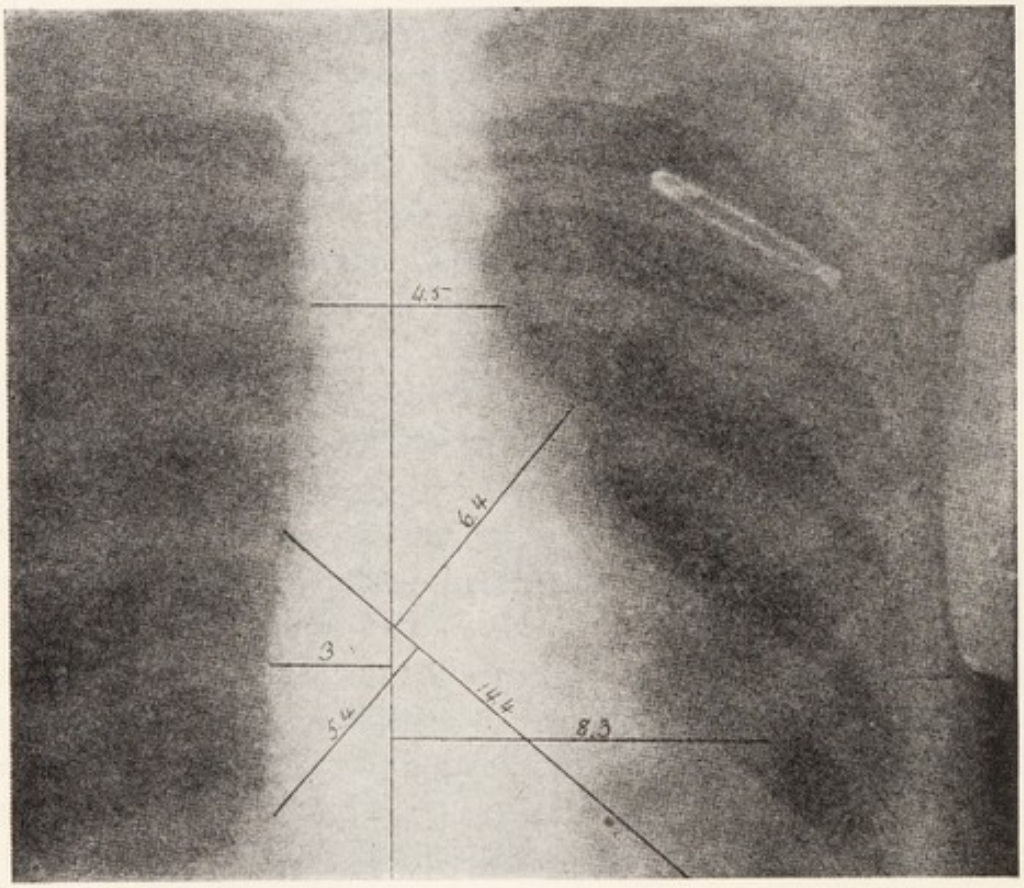



FIG. 161.—Cardiac shadow in Case LXIX. (Roentgenological Department, Massachusetts General Hospital.)

derance. The blood pressure was 118/80 to 105/75. The lungs were negative. The liver edge was just palpable on inspiration. There was marked clubbing of the fingers and toes. The genitals, pupils and reflexes were normal.

The temperature was 96.2° to 99.6°, below 98° every morning but three. The pulse was 63 to 105, usually 80 to 100. The respirations were normal except for two rises to 29–31. The venous pressure was 21 cm. water. The amount of urine was 27 to 63 ounces, the specific gravity 1.010 to 1.020. There was a slight trace of

albumin at three of four examinations occasional leucocytes at one. Tests for urine albumin before 8 a.m. on two days showed 0; between 10 a.m. and 1 p.m., two tests on two days, the slightest possible trace;

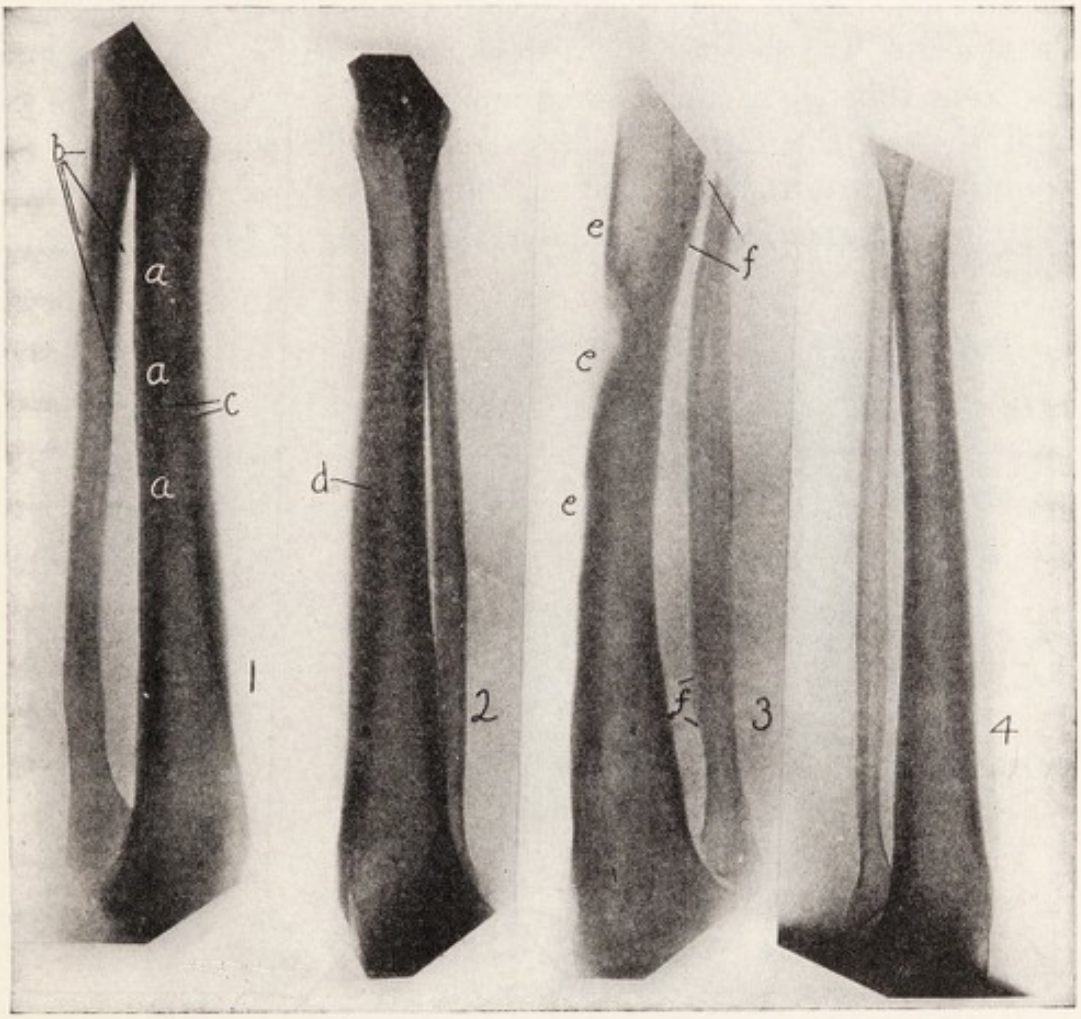



FIG. 162.—1. Shin in Case LXIX. 2 and 3. Shins of syphilitic patients. 4. Normal shin.—No. 1 shows distinct thickening of the cortex of the bone (*c*). There is also some waviness of outline (*a*), and a periosteal proliferation (*b*) at the lower end of the tibia, signs seen also in syphilis, as in No. 3 (*e*, *f*; the spot near the middle *e*, however, is a defect in the plate). No. 1 differs from No. 2 in that the thickening of the cortex in No. 1 is equally distributed over *both* the posterior and anterior surfaces, (*c*, *c*) while No. 2 shows marked thickening in the anterior surface *only* (*d*). The thickening in No. 2, as in No. 1, is at the expense of the marrow cavity. This equal distribution of thickening on both anterior and posterior surfaces has been observed at the Massachusetts General Hospital in only four cases out of about 100 syphilitic tibiae. (Dr. G. W. Holmes.)

Dr. Edwin A. Locke, whose studies of pulmonary osteoarthropathy have made him an authority on the subject,* agrees that it is not possible at present to say that equal distribution of the thickening of the cortex is indicative of this condition. In his opinion it would be impossible from the X-ray alone to distinguish No. 1 from a syphilitic bone. (Roentgenological Department, Massachusetts General Hospital.)

at 3.30 p.m. 0. On a fourth day the morning urine had a shade less than the evening. The renal function was 40 per cent. The hemo-

*See his article, Secondary hypertrophic osteoarthropathy, etc. Arch. Int. Med., May, 1916, Vol. XV, Part 1, p. 659.

globin was 150 to 145% (Sahli). There were 10,000 to 8000 leucocytes, 70 to 58% polynuclears, 30 to 42%, mononuclears, 9,390,000 to 8,688,000 reds, appearing normal at two examinations, universally large and massed at the third. The blood nitrogen was 61 mg. per 100 gm. blood. The coagulation time was 8½ minutes. A Wassermann was negative. A stool was negative. The sputum showed no tubercle bacilli at two examinations. Examination of the fundi showed the retinal veins extremely dilated, tortuous, and cyanotic, the arteries somewhat so, the discs very red. X-ray of the tibiae (see Fig. 162) showed notable thickening of the cortex at the

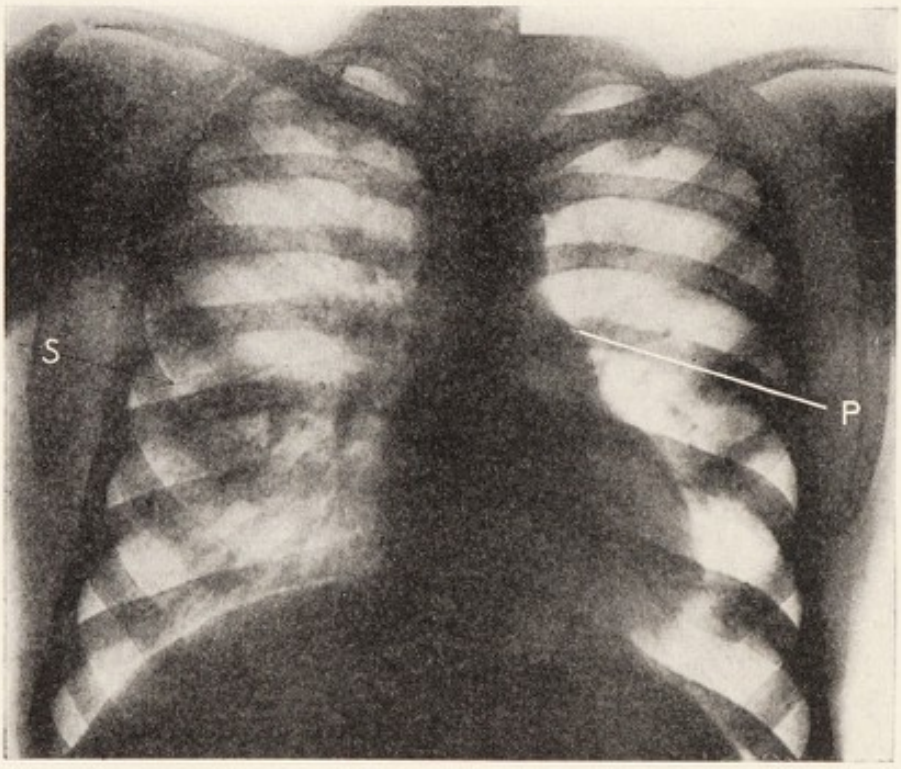



FIG. 163.—Case LXIX. P.—Projection often seen in congenital heart disease. S—Thickened interlobar septum. (Roentgenological Department, Massachusetts General Hospital.)

expense of the medullary portion, and well-marked periosteal proliferation. The skull, hands, and feet were not remarkable. The lungs (see Fig. 163) showed an area of mottled opacity between the fourth and fifth right ribs in the nipple line. The shadow of the lung root was much thickened on this side. There was also evidence of thickening of the interlobar septum.

The patient was kept in bed, with the usual house diet and no medicine. He showed no change during his stay. The visiting physician noted a systolic thrill, felt with difficulty, and found the

murmur late systolic and the pulmonic second sound diminished. September 16 the patient was discharged relieved.

*Dr. Richard C. Cabot's Diagnosis.*—Congenital deformity of the heart. (Probably pulmonary stenosis and interventricular septum defect.)

Polycythemia (symptomatic).

Osteoarthropathy affecting the long bones.

*Interpretations of X-rays.*—Tibiae: The process is quite definitely specific.

Lung: Lesion is pneumonic in type; may be a localized infection. Syphilis should be considered.

Heart: Slight enlargement of the left side of the heart; some bulging in the region of the left auricle.

## SUMMARY OF THE BOOK

### I

### AS REGARDS HEART DISEASE AS A WHOLE

1. Over ninety per cent of all cases of genuine* heart disease fall under one of the three types here called "*rheumatic,*" *syphilitic, and hypertensive.* This knowledge is of value because by narrowing the field, it focusses our attention.

2. The only other type occurring with any considerable frequency is *acute and subacute endocarditis* ordinarily due to a streptococcus. This made up a little less than 10% of the present series, if one includes the terminal and probably insignificant cases and those quite overshadowed by a primary septic malady, such as perforating appendicitis with peritonitis. Without these, the primary and recurrent types of the disease amount only to 102 cases in 1906 or 5%.

3. Of the three main types of heart disease, the "rheumatic" usually begins in early youth, and the hypertensive after the fiftieth year, while syphilitic heart is commonest between thirty-five and forty-five. Heart disease in a young woman is almost always rheumatic (mitral stenosis). In a middle-aged man without rheumatic history a valvular lesion is apt to be due to syphilitic aortitis, producing regurgitation at the aortic valve. In an elderly man previously free from heart trouble, circulatory disease is usually of the hypertensive type.

4. *Each* of these three diseases has not only its own characteristic time of life, but *attacks a particular part of the heart.*

(*a*) Rheumatism usually affects the mitral valve.

(*b*) Syphilis falls upon the aortic arch (aneurism, angina pectoris) or the aortic valve.

(*c*) Hypertensive disease enlarges the whole heart, leaving the valves intact.

Thus one gets from the physical examination hints as to the type of disease and from the type of disease, as revealed in the history, hints as to what physical examination will show. But the history is of value chiefly in this way and not by delineating a characteristic

* Most diagnoses of heart disease, whether made by physicians or suspected by the patient, are in my experience wrong. In such cases the heart is usually sound.

symptomatology. "Heart symptoms" are mostly those of passive congestion (lungs, legs, liver especially), whatever type of heart disease produces it. Only the age, the sex, and the negative or positive history of rheumatism or syphilis give us much help.

5. *Affections of the aortic valve are usually in males.* This applies to syphilitic aortitis, to rheumatic aortic stenosis and regurgitation (with or without mitral trouble as well), to acute and subacute endocarditis, and to the non-deforming aortic scars.

6. Within the field of the three main types in which our diagnostic inquiries move, *hypertensive heart disease occupies by far the largest area.* It is so much commoner than any other type that if we know that we have a case of heart disease to study, we know at the same time that it is probably of the hypertensive type, that we shall probably find a chronic elevation of blood pressure and that there will be nothing else very notable in our cardiovascular examination.

This relative predominance of hypertensive trouble will presumably increase in proportion as the measurably preventable types—rheumatic and syphilitic—are diminished by public or by private effort.

7. *Most of the other types of heart trouble* (acute endocarditis, acute and chronic pericarditis, congenital defects, thyrotoxic disease) *are for one reason or another practically unimportant* as well as relatively rare. The congenital effects are almost never seen beyond childhood; in childhood they are usually obvious ("blue baby") or very difficult to identify. Pericarditis in either of its forms we usually cannot diagnose and its effect in weakening the heart is in most cases slight. The remaining deleterious and occasionally diagnosable cases can probably be classed as rheumatic.

Non-deforming chronic endocarditis does not produce symptoms or signs and has therefore no clinical significance.

*Acute and subacute endocarditis* is by no means unimportant but runs most of its course with infectious rather than with cardio-vascular symptoms. Its unsusceptibility to treatment is notorious, so that its recognition is important mainly for prognostic ends.

8. The focussing of attention on three main types of heart trouble is aided both by realizing the rarity of the remaining types and by clearing the ground of *lesions not clinically recognizable, such as myocarditis and mitral regurgitation.* This is especially important with fashionable diagnoses which, like the two last mentioned, do much to blur our knowledge.

There is no justification for making these diagnoses any longer.

9. *Each of the three types—rheumatic, syphilitic, hypertensive*

*usually occurs alone.* Rheumatism and syphilis almost never affect the same heart and a combination of hypertensive disease with either of these infections is rare. The practical importance of this fact is that if we have satisfactorily identified one of them—e.g. syphilitic aortitis, with aortic regurgitation—we can be practically certain that the others are absent and the other valves sound.

## II

### AS REGARDS RHEUMATIC VALVULAR DISEASE

1. Rheumatic fever (with Sydenham's chorea and tonsillitis) is the only infection clearly related to deforming lesions of the mitral valve. When the aortic is diseased, syphilis is the only other cause to be considered. (I am excluding here the cases of acute and subacute endocarditis.) Our series contains no good evidence that scarlet fever, pneumonia, tuberculosis, typhoid, influenza, gonorrhea or any other infection, except rheumatism and syphilis, can deform the heart valves.

2. The non-deforming scars or scleroses—often called "chronic endocarditis" in necropsy protocols—are harmless landmarks and should not be confused with the deforming lesions.

3. Rheumatic disease affecting the mitral valve alone (almost invariably stenosis) is commoner in women than in men, 64 to 50. But when the aortic and the mitral are both affected, males strongly predominate and when the disease is confined to the aortic, not women but elderly men are the sufferers in eight-ninths of the cases.

4. Rheumatic heart disease is predominantly a malady of *young* girls and *young* women. Somewhere between three-fourths and nine-tenths of all cases begin before the thirtieth year.

5. Mitral stenosis is the usual rheumatic lesion. In over one-half the cases it occurs without any other valve lesion. On the other hand no other valve is often affected alone. Not once in our whole experience has the pulmonary or the tricuspid been affected alone by a clearly rheumatic endocarditis and the aortic alone has been the seat of rheumatic disease only 28 times as compared with 107 solitary lesions of the mitral. 25 of these 28 cases occurred in men.

6. The only notable peculiarity in the symptomatology of mitral rheumatic disease is the relative frequency of an early and prolonged palpitation, corresponding presumably to auricular fibrillation. This type of arrhythmia seems to be twice as common in solitary stenosis of the mitral as in cases complicated by aortic disease and shows a tendency to appear "early or never," i.e. the patients who are aware

of any palpitation at all have usually been aware of it among their earliest and most constant symptoms.

7. Possibly the greater frequency of palpitation in "pure" mitral disease may be connected with another peculiarity of that affection, namely, that *in the "pure" cases* uncomplicated by any other valve lesion, the *stenosis is ordinarily of greater duration and of greater degree* than in the cases with aortic or other valve lesions in addition to the mitral lesion. The more prolonged and more *stenosing* endocarditis perhaps involves the auricular walls more extensively and thus sets up the fibrillation which thereafter is usually constant because it does not stop without treatment.

8. But it is notable that the degree of narrowing at the mitral orifice bears no other relation to the kind or severity of the complaints or to the physical signs. The most extreme stenoses are not correlated with the severest symptoms of passive congestion.

9. When fever and leucocytosis occur they are usually due either to a complicating acute endocarditis or to an auricular thrombus.

10. *Over half the cases of mitral disease do not die of passive congestion*, but of some intercurrent infections, of trauma, operation, etc. This is especially true of the patients who get by the second decade. In them the disease remains latent for many years and is compatible with long life.

11. In the minority of cases when death *is* due indirectly to the valvular disease itself, it may be produced not by heart failure (passive congestion) but by an implanted acute endocarditis with septicemia or by an embolism of leg, arm or brain from a loosened bit of auricular thrombus which our statistics show is present in a quarter of the decompensated fibrillating cases. Hence the clinical picture of the final illness may be not at all that of heart disease, but rather of a peripheral gangrene, a hemiplegia or a septicemia. In such cases heart disease may be never suspected and the heart never carefully examined.

12. These facts and also the fact that death may be very sudden account, in part, for the small number of correct diagnoses made before death in the cases of this series. *Over half the cases of mitral disease were not recognized in life.* I doubt if the percentage of success is any greater in other institutions or in private practice.

13. Recognition would be more frequent if physicians realized that the characteristic mid-diastolic or presystolic murmur is not always to be heard unless one takes special measures (exercise, amyl-nitrite inhalation, special positions of the body, auscultation at differ-

ent times of day) which may *bring out* the important evidence. Well directed search for mitral stenosis will be undertaken oftener if we remember that it is a latent possibility, especially under the following conditions:

(*a*) In cases of cerebral or peripheral embolism occurring in relatively young persons and so presumably not due to arteriosclerosis.

(*b*) In arrhythmic decompensating heart disease without evidence of syphilis or hypertensive cardiovascular trouble, and especially if there is a rheumatic history.

(*c*) With evidence of an acute endocarditis (since a chronic endocarditis usually underlies this lesion).

(*d*) In the presence of a sharp first sound and a dull or feeble second sound at the apex; of a doubling of the second sound (or a third heart sound) along the left sternal margin; of absolute arrhythmia without known cause in a young person.

14. Mitral regurgitation without stenosis was apparently present in seven out of the 1906 necropsies of this series, three of them doubtful. It is obviously, therefore, one of the rarest of lesions discoverable post-mortem. During life there are no signs by which it can be recognized. It is not a clinical entity.

### III. AORTIC STENOSIS

1. On the whole it seems most probable that our twenty-eight cases of solitary aortic stenosis belong with the rheumatic group. Their history and the post-mortem findings incline us to this belief.

On the other hand the age and sex distribution is a strange one if the lesion is a rheumatic one, for whereas in most rheumatic heart disease the typical patient is a girl or a young woman, in aortic stenosis it is an elderly man. These facts make us think first of that disease most characteristic of elderly men—arteriosclerosis; but there is strong evidence against this in the pathological anatomy. Only in one doubtful case out of this whole series did arteriosclerosis appear to the pathologist to have produced an aortic lesion.

Some stray considerations make me consider the question whether the cases of "pure" aortic stenosis (the other valves remaining untouched) may represent healed cases of subacute streptococcic endocarditis. These considerations are:

(*a*) The presence in the necropsies on cases of aortic stenosis of an unusual number of *old* renal infarcts, possibly thrown off during an earlier attack of vegetative endocarditis.

(*b*) The fact that while in most rheumatic lesions an old complicating pericarditis is often shown by the presence of pericardial adhesions, these adhesions are rarely found in aortic stenosis (one case in twenty-eight) or in subacute endocarditis.

(*c*) The fact that the male sex is the commoner in both these diseases.

Admittedly these stray facts are not important evidence; they only suggest a possibility which is not negatived by the excessively fatal nature of acute and subacute endocarditis, since it is demonstrable that some of these attacks do get well.

2. In its clinical picture aortic stenosis is peculiar not only in its proneness to occur in elderly men, but also in the frequency of precordial pain, of faintness on exertion, and of angina pectoris.

3. The contour of the rigid valves, fixed in a position neither open nor shut, seems to compel us to believe that there is regurgitation as well as obstruction of the blood stream. Yet in nearly half the cases no diastolic murmur is detected.

4. Another strange fact is that even in a case with rigid and immovable valves the aortic second may be not only audible but may be actually accentuated and the systolic blood pressure high. How physiologists would account for this I do not know.

5. The classical quartette of signs (systolic murmur and thrill in the second right interspace, with a diminished or absent aortic second sound and a flat-topped pulse wave) were not often present as a complete group in this series. Indeed Corrigan's pulse, the opposite of which we expect, is recorded in six out of twenty-eight cases, and a characteristic thrill is often wanting. Yet it can be said that it is still by these four signs (some or all of them) that we can recognize the lesion if we recognize it by any physical signs at all.

But reasoning helps. When a diastolic murmur and other evidence of regurgitation are found in a case with a rheumatic history, it is safe to assume the presence of aortic stenosis also, since only in rare cases does rheumatic disease produce regurgitation, alone and with stenosis. In this way we can often reason ourselves to a correct (though not very important) diagnosis, which direct observation could not make.

6. The average amount of cardiac hypertrophy is greater in aortic stenosis than in any other valve lesion except syphilitic aortitis with aortic regurgitation.

7. Even when the aortic orifice is reduced to a mere slit, an abnormally high systolic and diastolic blood pressure may in rare cases

be maintained in connection with the complicating nephritis such as was present in over one-third of the cases of this series.

8. If the disease is rheumatic and begins like most rheumatic heart disease in youth, the advanced age attained by most patients would imply that they live longer than in any other type of heart disease.

9. Death in six out of twenty-eight cases was notably sudden. In most, however, it was due to chronic passive congestion.

## IV

### SYPHILITIC HEART DISEASE

1. Aside from a single case of myocardial gumma, the lesions here studied were all in the aorta.

2. Here *manifest* syphilitic aortitis shows itself clinically as aneurism, as aortic regurgitation, as angina pectoris or as some combination of these three troubles. The manifest and harmful cases number about one-fifth as many as those of rheumatic valvular disease and about one-ninth as many as those of hypertensive heart disease (see page 419).

3. So far as the aorta is concerned, the disease probably remains latent and harmless for fifteen to twenty years after the original syphilitic infection. In many, perhaps most, cases it is merely a historical landmark in the post-mortem examination.

4. It is six times as common in men as in women and usually makes itself felt between the thirty-fifth and the fiftieth year, a little later than most rheumatic and a little earlier than most hypertensive cases. An isolated aortic-regurgitant lesion in a man under forty-five is usually syphilitic.

5. Though it may be latent and symptomless for many years, it usually leads to death within two years of its first clinical manifestation.

6. There is no evidence that the disease attacks any valve but the aortic. There it produces in about one-half the cases a regurgitation. The larger portion of the valve remains flexible and capable of good function, but at the contiguous margins where two valves touch each other, a fibrous process binds them back against the aortic wall so that they cannot spread out to support the column of blood during diastole. Hence the regurgitation.

The heart walls, especially the left ventricle, undergo so great an hypertrophy that the heart's weight is greater in this type of disease than in any other except in adhesive pericarditis with mediastinitis.

7. When the syphilitic process attacks the aorta higher up, above the valves, and leaves the latter normal, no hypertrophy and ordinarily no weakening of the heart occurs even though the aortic wall may be weakened so that aneurism results. Aneurism does not produce cardiac hypertrophy.

8. Angina pectoris is a common feature of the disease. It may occur (*a*) in association either with narrowing of the coronary orifices by the syphilitic process in the aorta, or (*b*) without any change either in these orifices or in any part of the coronary artery. Angina pectoris before the forty-fifth years should arouse a suspicion of syphilitic aortitis.

9. The diagnosis of syphilitic aortitis should be relatively easy when angina pectoris or an isolated aortic-regurgitant lesion appears in a young or middle-aged man with a history of syphilitic infection fifteen to twenty years earlier and without any rheumatic history. The Wasserman reaction—which is positive in at least 80% of cases—may assist the diagnosis.

10. When the disease produces aneurism without angina or aortic regurgitation, it may not be discovered or suspected for a good while unless accidentally shown up by an X-ray taken for some other purpose. For it is only when symptoms of pressure on adjacent structures, (symptoms such as pain, hoarseness, abnormal pulsation of the chest wall) happen to occur that the patient is led to seek an examination. All such pressure symptoms may be absent.

11. On the other hand when definite evidence of an aneurismal tumor is obtained, it may be safely assumed to be of syphilitic origin. There is no good evidence that aneurismal tumors are due to any other cause. The so-called "dissecting aneurisms" and "mycotic aneurisms" do not produce a tumor or any other recognizable signs. Clinically they are negligible. Arteriosclerosis often enlarges the whole aortic arch but does not weaken it, does not produce a tumor, and does no harm of any sort, so far as our cases show.

## V

### HYPERTENSIVE HEART DISEASE

1. The commonest of all types of heart disease with decompensation is that here called hypertensive. In fact it is commoner than all the other types put together.

2. Under this heading are grouped in the present study all cases of enlarged heart* without valve lesions or chronic pericarditis, even

*(But see no. 12, page 771.)

though the proof of hypertension was not always obtained, the cases being studied only at the end of life.

3. Whether nephritis and arteriosclerosis are to be regarded as causes, as results, or as diseases independent of hypertension, is not clear. The impressions derived from the cases studied in this book are (*a*) that arteriosclerosis has little or no tendency to enlarge the heart, (*b*) that nephritis *does* exert such a tendency, but (*c*) that hypertension (with its results in cardiac enlargement and final cardiac failure) is a common disease independent of any nephritis or arteriosclerosis.

4. Hypertension and the associated cardiac hypertrophy last in many cases for years without producing any considerable discomforts or limitations in the individual's activity. It is discovered ordinarily in the course of a physical examination undertaken for some other purpose (e.g. life insurance). High blood pressure is then the substance of the finding, for the cardiac enlargement is often difficult to make out unless with the aid of the X-ray, and murmurs and arrhythmias are usually absent at this relatively early stage of the disease.

5. In many, perhaps most, cases, cardiac failure never occurs at all and the patient dies of some other disease or of some extra-cardiac manifestation of hypertension, such as cerebral hemorrhage.

6. As the disease progresses it may show itself in slight dyspnea on exertion, in a "pounding" of the arteries of the neck and head and in an awareness of the cardiac impulse. Still, it bothers the patient little unless he is alarmed by what he is told about the disease.

7. At this latent stage it may, nevertheless, contribute to death in case the patient is prostrated by infection, trauma, toxemia or surgical operation.

8. Less often—in about one-third of our cases—the heart itself weakens and death occurs from passive congestion. In such cases the hypertension may continue to the end or may disappear.

9. In these cases death usually follows within a year from the first serious evidences of stasis, shown as in other types of heart trouble by arrhythmia, tachycardia, pulse-deficit and passive congestion. Systolic (and occasionally diastolic) murmurs may be present at the base or at the apex of the heart, but are of no diagnostic value; the same is true of an accented aortic second sound.

Diagnosis rests on the presence or history of a chronic hypertension, on the cardiac enlargement, and on the lack of evidence pointing to valvular disease, chronic pericarditis or thyrotoxicosis. Hypertensive

disease is often wrongly diagnosed as myocarditis, mitral regurgitation, "cardiorenal disease" or "the senile heart."

10. The degree of cardiac enlargement discoverable *post-mortem* is usually moderate; the extreme hypertrophies resulting from chronic pericarditis or from disease of the aortic valve were not seen in hypertensive cases of this series. 910 grams was the weight of the largest heart.

11. The disease is more than twice as common in men as in women and appears usually about the fiftieth year, though no decade of life, except the first, is exempt from it.

12. The disease is to be distinguished from the cardiac hypertrophies which occur almost constantly in pernicious anemia and in leukemia. These diseases are associated with low or normal blood pressure.

## VI. MYOCARDITIS

1. Fibrous myocarditis is an item of *post-mortem* anatomy usually associated with other and more important cardio-vascular lesions, such as arteriosclerosis, nephritis and hypertension in elderly men.

2. It is not recognizable clinically and should be left out of diagnostic terminology, although its pressure may be vaguely suspected when evidence of cardiac infarction is present.

## VII. ACUTE AND SUBACUTE ENDOCARDITIS

1. The labors of Libman and others have gone far to separate out a recognizable group of cases to which the name of "subacute bacterial endocarditis" is given. Very possibly this is a separable disease entity. In our series, however, its distinction from rheumatic, septic and terminal cases of endocarditis is not clear. It has seemed better, therefore, to group together here all cases showing *soft* vegetations on the valves and then to attempt clinical-pathological sub-groupings as follows:

(*a*) Those in which the soft endocarditis and its results constituted apparently the whole of the disease, before and after death (the so-called "*primary*" *cases*, 36 out of 180).

(*b*) Those identical with the above except that an old hard endocarditis accompanies the soft acute process ("*recurrent primary*" cases, 66 in 180).

(*c*) Those in which a septic process (such as purulent peritonitis) outside the circulatory system seemed at necropsy to be the starting

point and main body of the disease (*pyemic cases* with or without an old endocarditis, 44 in 180).

(*d*) Those in which some non-septic disease—such as cancer or nephritis appeared to be the main cause of death, ("*terminal endocarditis*," 34 in 180).

In each of these groups there have been cases with bacteria found in the circulation (usually some type of streptococcus) and cases in which none were found. In each there have been cases clinically acute and others clinically subacute. On a histological basis alone, it is doubtful whether one has a right to make time-distinctions.

The relation of the soft endocarditis to the hard valve lesions which were also present in 102 of 180 cases is problematic. Are we correct in speaking of the soft process here as *recurrent*, i.e., a return of the *same* disease which produced the hard lesions? Or is it another disease which has supervened, as pneumonia may invade a previously tuberculous lung? I do not see how an answer can as yet be given. In the "primary" groups of cases (*a* and *b* above) the coincidence of hard and soft lesions attains 64%.

2. It is a disease prone to attack both sexes equally and especially young adults (twentieth to fortieth year). But possibly in children it gets well or becomes arrested and so is not reckoned with in statistics which like these deal only with fatal cases.

3. The mitral valve is affected in over three-fourths of the cases. It is the only valve attacked in nearly one-half. In general the frequency with which the different valves are attacked is about the same as in chronic ("rheumatic") endocarditis with deformity. The tricuspid is rarely attacked (13 in 180).

4. We have never found the gonococcus or the influenza bacillus in the blood or in the lesions.

5. Clinically the disease is recognized when symptoms of sepsis (fever, leucocytosis, chills, anemia, jaundice, bacteriemia) are combined with evidence of a valve lesion and of embolism. But the latter is discovered clinically in only one-third of the cases, though at necropsy embolism is shown in two-thirds of the primary cases (*a* and *b* above). The skin, spleen, kidneys, brain and lungs are oftenest hit by emboli.

6. A palpable thrill and an increasing anemia have been to me the most helpful diagnostic signals.

7. Nephritis occurred in thirty to fourty per cent of the cases, but acute pericarditis, which one would suppose would be a common complication, was found here in only nine cases out of 180 or five per cent.

8. In many instances the diagnosis is masked by evidences of passive congestion, associated in nearly 40% of our cases with cardiac enlargement. This enlargement is usually explainable by chronic lesions (valve deformities or hypertension), but occasionally seems to be produced by the acute endocarditis itself.

9. The duration is usually weeks or months, sometimes years.

10. Evidence of a healed (perforating) lesion is sometimes found.

## VIII. ACUTE PERICARDITIS

1. As already said, we can recognize this disease in only one-fifth of the cases and then can usually do nothing to cure it. Its clinical importance is therefore small.

2. This clinical insignificance is the more striking if we realize that it is the main cause of death in less than 5% of the cases in which it occurs. Usually it is only one localization of a general septicemia, or is a terminal event in nephritis, neoplastic disease, etc.

3. Other cardiovascular lesions (except hypertension and its results) are rarely found with an acute pericarditis. Hence if we are sure of that lesion, we can usually exclude endocarditis, acute or chronic.

4. Organisms of the streptococcus-pneumococcus group can usually be found in the blood or in the pericardial exudate; in children the staphylococcus once freed from a local lesion, such as osteomyelitis, is prone to attack the pericardium as well as to produce myocardial abscess.

5. Enlargement of the heart is present in two-thirds of the cases and passive congestion in one-third. Over one-half of the enlargements are not explained post-mortem and while a previous hypertension probably accounts for some, it seems possible that an acute or subacute pericarditis can itself produce cardiac enlargement.

6. Effusion in considerable amount is found in about one-third of the cases, only 13% of which were recognized in life either by the X-ray or by the presence of an extraordinarily wide percussion area.

7. Like chronic pericarditis, the acute form is "*a diagnostic marplot*" in that it produces sounds indistinguishable from intracardiac murmurs. But since (as above said) endocarditis and pericarditis seldom occur together, the former should usually be excluded if we are sure of the latter.

## IX. CHRONIC PERICARDITIS

1. When extensive and associated with mediastinitis in young rheumatic subjects, this disease produces the largest hearts known (1328, 1158, 1150 grams in uncomplicated cases) and leads to death from passive congestion (28 cases out of 112).

2. In only 20 cases of 112 was the disease associated with a chronic endocarditis. Most cases are "vestigial," that is, are evidence of a previous acute pericarditis but produce no passive congestion; the patients (mostly elderly men) die of some non-circulatory disease.

3. As in acute pericarditis males overwhelmingly preponderate.

4. Diagnosis is rarely possible (6 of 112). Thus far X-ray has given us no help.

5. Diastolic and presystolic murmurs are often heard, especially in cases with large hearts, and lead to false diagnoses of endocarditis.

## X. THYROCARDIAC DISEASE

1. In most thyroid intoxications the heart is noisy and demonstrative; it is apparently but not actually enlarged and if death occurs it is from the self-poisoning, not from passive congestion.

2. In old non-toxic goitres and in toxic adenomata, congestive heart failure with fibrillation may after many years supervene. Operation appears to be of benefit in some cases.

No constant cardiac lesions are found *post-mortem*.

## XI. ANGINA PECTORIS AND CARDIAC INFARCT

1. Angina pectoris if distinguished from the symptoms accompanying cardiac infarct has no constant anatomical basis.

2. Coronary disease and macroscopic changes in the aortic arch are often absent in cases of angina pectoris, and still more often present without angina.

3. Angina may cease when passive congestion is established, but in most of our cases this is not true.

4. When a coronary is blocked by clot or sclerotic changes, and cardiac infarction follows, we may find a characteristic clinical picture corresponding: intense, usually epigastric pain lasting for hours, often with pulse failure and fainting. The symptoms are not produced by exertion or emotion and are not relieved by rest or nitrites.

5. Coronary occlusion, acute or chronic, with or without cardiac infarction, may be symptomless.

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