

**Clinical observations on diseases of the heart and thoracic aorta / by
Peyton Blakiston.**

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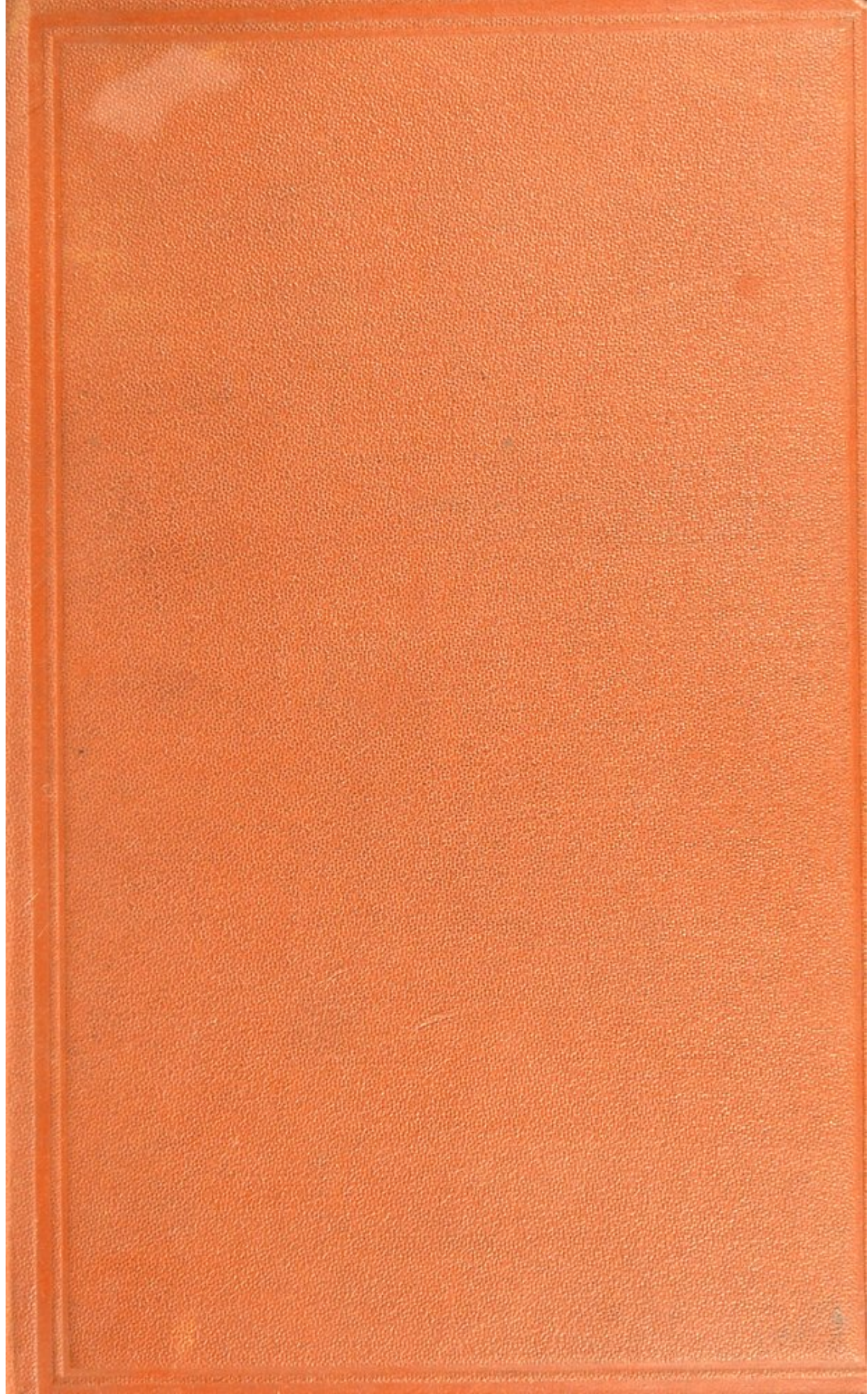
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ON
DISEASES OF THE HEART.



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CLINICAL OBSERVATIONS

ON

DISEASES OF THE HEART

AND

THORACIC AORTA,

BY

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PREFACE.

IT is now upwards of thirty years since the observation of facts here recorded first commenced. During this period many new views have arisen concerning the nature, origin, diagnosis, and treatment of diseases in general. This may help to account for the differences that will be observed between the reports of the earlier cases, many of which have been already inserted in the author's work on Diseases of the Chest, published in 1848, and those more recently reported; especially as regards the treatment of acute diseases.

In a work like the present, recording the results of personal experience by the bedside, extensive reference to the writings of others would hardly be looked for. Should the author, however, have failed to acknowledge the labours and discoveries of others, he trusts that such omissions will not be attributed to any desire upon his part to underrate the labours of fellow-workers more valuable and important than his own.

An arrangement has been adopted differing from that which usually prevails in works of this descrip-

tion; pathology, diagnosis, and treatment having been kept distinct the one from the other. By this means some repetition has been avoided, and the connection existing between the different diseases has been better preserved; whilst, for reference or study, it is believed the arrangement will be found equally convenient.

Whatever may be thought of the deductions from the observations here recorded, the author has the satisfaction of knowing that a certain number of cases will have been published, which may furnish material for the further elucidation of truth, and in some measure promote the advancement of practical medicine.

ST. LEONARD'S-ON-SEA,

November, 1864.

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CLINICAL OBSERVATIONS
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THORACIC AORTA.

INTRODUCTION.

DURING the period over which the observations recorded in the following pages have extended, many changes have occurred in the aspect of medical science. Facilities for the diffusion of medical knowledge have been greatly extended, and an immense increase has taken place in the number of well-informed and thoughtful practitioners, who are now found scattered throughout the length and breadth of the land.

Although no striking discoveries have been made by them, such as that of the circulation by Harvey, or auscultation by Laennec, yet numerous additions have been made to our practical knowledge of disease. A slight glance at some of the changes which have thus been brought about in our times, concerning our knowledge of the *nature, mode of discovery, and treatment* of many diseases, may possibly tend to throw some light upon the particular subjects proposed for consideration in this work.

PATHOLOGY.—In no instance, perhaps, has a greater change occurred, than in our views of the nature and causes of dropsy. Many practitioners of the present day can remember the time when it was considered a primary disease arising from deficient action of the absorbents. Dr. Blackall noticed, that in certain cases of dropsy the urine coagulated by heat, but it was reserved for Dr. Bright to connect dropsy, in which the urine was albuminous, with a diseased state of the kidneys. This led to a definite distinction being drawn between dropsies depending upon morbid changes in the kidneys, and those arising simply from obstruction to the circulation as occurs not uncommonly in the liver, in the heart, or in the lungs.

In the meantime, the microscope was coming into more general use; and by its aid much light was thrown upon the nature of the insoluble substances found in the urine in different diseases, as well as upon the minute structure, and the physiological action of the kidneys.* The way being thus cleared, microscopic examination of *diseased* kidneys revealed the existence of *different* affections, in which the common feature of albuminous urine was present. The principal of these were *acute nephritis*, including the inflammatory dropsy of old time, induced by exposure to cold and wet, and those which appeared as the sequelæ of scarlatina and other eruptive fevers.—*Chronic nephritis*, sometimes called *gouty* or *contracted kidney*. *Fatty degeneration*.† Chemistry also lent its aid; Dr. Christ^{so}tiⁿ demonstrating, that in albuminaria the quality of the blood was altered, being deficient in albumen, whilst a large proportion of urea was present. Thus, step by step have data been furnished for determining the nature, causes, and progress of dropsies.

Let us endeavour to trace them out in one of the simplest forms, that of acute dropsy resulting from exposure to cold

* See Mr. Bowman's paper in the Phil. Trans. for 1842.

† "On Diseases of the Kidneys," by Dr. George Johnson.

and wet. The first result of this is a chilling of the surface of the body, and diminution in the quantity of blood in the capillaries, whereby perspiration is notably diminished. The next effect seems to be produced in the kidneys, the action of which is well known to be vicarious to that of the skin, cold weather diminishing the cutaneous excretion, and increasing the flow of urine, and warm weather producing exactly a contrary effect. The kidneys, therefore, having more work thrown upon them, by a well-known law, receive more blood than usual, and hence, become congested. This leads to the escape of a certain proportion of the serum of the blood, from the Malpighian capillaries into the uriniferous tubes, in which case *albuminous urine* results.

But more than this takes place in the kidneys, for the secretory apparatus has also thrown upon it the task of eliminating from the blood those ingredients which should have been removed by cutaneous secretion; hence, the secreting cells become clogged and disintegrated, many of them passing into the urine by a process of desquamation. The blood, therefore, soon becomes more and more impure, in consequence of a portion of the urinary constituents being retained. Thus altered in quality, it is less fitted than in its normal state, freely to traverse the capillary vessels of the system generally. The blood itself gradually becomes poor in red corpuscles, and the proportion of the solids generally becomes diminished; the permeating power of the serum is increased; exudation takes place into the areolar tissue, and sometimes into serous cavities, and so general dropsy results.

Other forms of dropsy, not accompanied with albuminous urine, have been found to result from congestion of the capillary vessels, induced by impediments offered to the return of the blood to the heart, or to its free circulation through the pulmonary capillaries. When the liver is in a shrunken and contracted state, as commonly results from spirit-drinking, the portal blood cannot flow freely through

the capillaries of the organ to the inferior cava and right side of the heart. Although in many cases much of the blood may return by the hæmorrhoidal veins and other channels of communication between the portal system and inferior cava, great congestion of the intestinal veins usually occurs, serum transudes through their coats, and ascites results. The morbid state of kidney, known as chronic nephritis, in which the organ is very small and hard, is closely allied to contracted liver, and it is probable that albuminous urine bears precisely the same relation to that disease, as ascites to cirrhosis, or contraction of the liver.

The origin of *cardiac dropsy* was formerly supposed to be threefold:—1. From the capillaries being congested by an unusually large supply of arterial blood propelled by an hypertrophied left ventricle. 2. From a weak and dilated condition of the heart producing an engorgement of its cavities, and thereby an obstacle to the free return of the venous blood. 3. From obstruction offered by diseased valves on the *left* side of the heart to the blood returning from the lungs, and then acting secondarily through them on the systemic circulation.

It will be seen, that neither of the first two supposed causes are instrumental in the production of dropsy, and that the third only acts remotely in doing so; but that its constant proximate cause is regurgitation of the blood through the tricuspid foramen, rendered incomplete either by disease of its valves, or much more frequently by simple dilatation to such an extent that the auriculo-ventricular valves are no longer able to close it.

Here, then, through the joint labours of the physiologist and the clinical observer, a group of diseases, the nature and causes of which were profoundly hidden from us, have in our own day, and under our own eyes, been brought into the clearest light; with what effect on their successful treatment will appear in the sequel.

The same may be said of those cases in which *blood* is

effused, as *Cerebral Apoplexy*, *Pulmonary Apoplexy*, and *Aneurism*. The commonest cause of all these affections was supposed formerly to be congestion produced by a large and powerful stream of blood thrown into the capillaries, very frequently in consequence of hypertrophy of the left ventricle of the heart. It has now, however, been discovered that the most frequent cause of *cerebral apoplexy* is the rupture of an artery of the brain, which has become affected with atheromatous and calcareous degeneration; and that when it arises from congestion, it is venous and not arterial; not resulting from hypertrophy of the left ventricle, but more frequently from that of the right ventricle driving back the venous blood through an incomplete tricuspid foramen. The same holds good as regards *Pulmonary Apoplexy*, which will be seen to have no connection with hypertrophy of the right ventricle, but to be caused by mitral regurgitation, increased in some instances by hypertrophy of the *left* ventricle. *Aneurism*, too, of the aorta, has been found almost invariably to result from atheromatous and calcareous degeneration of the arterial coats, whereby they are rendered more brittle, and less capable of resisting the distending force of the heart acting upon the contained blood. Of course, when the coats are already diseased, hypertrophy of the left ventricle may increase the tendency to dilatation or rupture.

It has been distinctly shewn by microscopical observation, that atheromatous disease of arteries consists in a form of fatty degeneration of their coats; and that in such degenerations the fat is not of the ordinary kind existing in adipose tissue, but is very rich in cholesterine. It results from changes occurring in the elastic and contractile elements of the arterial tissue. The "brown paper" appearance of the muscle of the heart is due to a similar cause, but the fat in this condition is in a free granular state.

Rheumatism, supposed formerly to result from exposure

to wet and cold, has been found by the clinical observer to be associated with a *morbid state of the blood*, which is often hereditary; and the chemist has shown that the rheumatic state probably depends upon the presence of a large quantity of lactic or some allied acid in the blood. Great excess of uric acid has been demonstrated in the blood of gouty persons.*

Many more instances might be adduced. Enough, however, has been brought forward to illustrate the manner in which the clinical observer and the physiologist, aided by chemistry and the microscope, are working, and gradually adding to our knowledge of disease. But although much has thus been done and is still doing, every step we take in advance serves but to show us how very far we are from the end. Our investigations have carried us up to a certain point. Some morbid alterations in the different organs of the body have been attributed to inflammation, and others to impaired nutrition; but now we must ask what we mean by these terms? in what consists inflammation? and how is nutrition effected? Something has been done of late towards answering these important questions. Dr. Lionel Beale, by means of an improved method of preparing specimens for examination, and the use of very high magnifying powers, has succeeded in partially lifting up the veil, and shewing us something of the process of nutrition; the conversion of nutrient pabulum into living matter, and that of the latter into formed tissue, by a process very different to that which had been supposed by the propounders and supporters of the cell theories; but still the *cause* of the formation of different tissues out of the same pabulum remains unexplained, and is referred by this observer in part to peculiar inherent powers. By his researches in another direction, Dr. Beale has shown that nerves are distributed to the most minute vessels, even to capillaries, and an explanation has been offered of the

* See Dr. Garrod's work "On Gout."

precise manner in which the process of nutrition is influenced by the nervous system. These results also throw some light on the proximate causes of inflammation; which seems to consist in a morbid excess of nutrition, according to Dr. Beale, in consequence of increased facilities being afforded for the access of pabulum to the living matter. For instance, when the living matter in the interior of a cell becomes exposed to the full access of nutrient pabulum, as in cases of rupture or softening of the cell wall, it grows faster; no time is allowed for the conversion of the living matter into cell wall, and hence little else but soft, rapidly increasing but short lived living matter results. In a short time the so-called "nucleus" of a cell (Dr. Beale's living or germinal matter) may increase, divide, and subdivide, and the bodies resulting from this process are pus corpuscles.*

Thus although it is not long since we hardly saw or knew anything, now we "see through a glass," albeit "darkly;" and now we know, although "only in part."

DIAGNOSIS.—Nearly half a century has elapsed since Laennec's discovery of auscultation of the chest threw such a brilliant light on the diagnosis of certain of its diseases, previously buried in the most profound darkness. His quickness of perception and persevering industry enabled him to work out the details of auscultation in such a manner as to leave little for his successors to do. Almost the only instance in which any discovery of *practical* importance has been made since his time,† is that of the late Dr. Jackson, of Philadelphia, who whilst a student at Paris, about 1831, pointed out that a prolongation of the respiratory sound during expiration, indicated commencement of solidification of the lung, and thus furnished a most valuable sign by which the earliest approaches of

* Lectures at the Royal College of Physicians, 1861.

† "Life of Dr. Jackson," p. 129.

tuberculisation could frequently be detected. Laennec, however, failed in several instances to make out the *manner* in which sounds were engendered both in health and disease, and many years elapsed before the mode* in which the respiratory sound was produced in health, and modified in disease, had been satisfactorily made out.

It is remarkable, too, that up to within a short period the exact manner in which some of the motions of the heart take place, and its sounds are engendered have been disputed points. It may be well shortly to inquire how this matter now stands, without, however, attempting to go through all the discussion of former years, or dilating on the principles and practice of auscultation.

About seventeen years ago the question stood thus: The heart's impulse was generally considered to be produced by its apex being tilted forward by the systolic contraction of the ventricles, and striking the walls of the chest. The second, or diastolic sound, was admitted to arise from the tightening of the arterial valves by the rebounding of the column of blood during diastole. Concerning the origin of the first or systolic sound, two opposite opinions chiefly prevailed. Dr. Billing and others argued that it was caused by the tightening of the auriculo-ventricular valves alone, whilst the London Committee of the British Association for the advancement of Science, consisting of Drs. Williams, Todd, and Clendinning, maintained that it was *solely* caused by the sound of muscular contraction taking place in the walls of the ventricles. The latter observers based their conclusions on certain experiments made on young asses, in which they asserted that a considerable amount of systolic sound was heard when the blood was prevented from entering the heart and unfolding the valves; indeed, when the heart was removed from the body. This fact was so prominently put forward, that assent could hardly be refused to the deduction that a

* "Practical Obs.," Dr. B., p. 18 and following pages.

portion of the systolic sound must have been caused by muscular contraction; but it was contended* that this sound was not solely produced in this manner. Objections were raised to the argument by which the Committee endeavoured to account for the diminution in the intensity of the first sound when the blood was prevented from acting on the valves, and it was found "difficult to understand on what grounds the mitral and tricuspid valves should be excluded from taking any part in the formation of the systolic sound of the heart, when the whole of the diastolic sound had been attributed to a similar action of the aortic valves." It was also shown that "on the supposition that the systolic sound was solely formed by that of muscular contraction, it was impossible to explain in what manner it was modified in certain states of disease."

It was therefore concluded that "the systolic sound was caused by the friction of the muscular fibres of the ventricles *inter se*, and the tightening of the auriculo-ventricular valves; strengthened in certain cases by the impulse of the heart against the ribs, and by the collision of the blood against the orifices of the aorta and pulmonary artery."

In consequence, however, of subsequent observations of muscles in action, and of certain modifications of sound heard in disease, a suspicion gradually arose that there must have been some source of error in the experiments of the London Committee; and that possibly the sound heard when the heart was removed from the body, was caused by the ventricle during its contraction rubbing against the stethoscope. This conjecture received confirmation from a series of experiments admirably planned and carried out by Dr. Halford about five years since.† It was found that on preventing the venous blood from entering the cavities of the heart, without removing the pericardium, or in any other way disturbing its action, *all sound ceased*.

* "Diseases of the Chest," Dr. B., p. 40.

† "The Action and Sounds of the Heart." By G. B. Halford, M.D.

As it appears from the concurrent testimony of several unbiassed and competent witnesses of these experiments, that they were conducted with great skill and accuracy, the conclusions drawn from them must be admitted to be established, viz.: That the systolic sound under ordinary circumstances is caused by the tension of the auriculo-ventricular valves, and the diastolic sound (as has been generally held) by that of the semilunar valves.

In the same experiments it was seen that the apex of the heart was not tilted forward against the walls of the chest by the contraction of the ventricles, as had been generally supposed, but was, on the contrary, "pressed downwards, backwards, and from right to left," and "that any part of the ventricles in contact with the walls of the chest, can give an impulse during this contraction."

It was also demonstrated that as long as the pericardium was preserved intact, the action of the heart was remarkably easy, smooth, and gliding; but that "when the pericardium was removed, and its fluid consequently lost, the heart's action became immediately tumultuous." "The pericardium, therefore, regulates the heart's action."

Subsequent experiments made by Messrs. Chaveau and Moray confirm the accuracy of the conclusions arrived at by Dr. Halford, and render it unnecessary to carry out an intention which had been formed once more to repeat the experiments.

As the cause of the sounds of the heart in health may now be considered as determined beyond dispute, the confusion that has attended the interpretation of their modifications by disease must cease also, and their diagnostic value be greatly enhanced.

Some advances towards an accurate knowledge of chest diseases have also been made in other directions. Thus Dr. Hutchinson, by means of his spirometer, has devised an ingenious method of measuring the capacity of the lungs for air. By this it can be ascertained whether any part of the air cells have been supplanted by liquid or

solid, and thus, in some cases, the first traces of disease may be detected.

The application, too, of the plan of illuminating deep seated cavities by reflected light, as in the case of the speculum vaginae, has been applied to the exploration of the larynx by a method first suggested by Dr. Babington, and perfected by Czermac. The laryngoscope consists of a mirror so arranged as to throw a strong light from a lamp down the larynx. By this means the action of the vocal chords is laid bare, and the formation of the voice can be studied with great care, and certain diseases of the larynx can be recognised. Whether any large number of practitioners will acquire the amount of dexterity necessary for the successful employment of this method of investigation, or whether many persons will be able to undergo the process without great distress, remains to be seen.

Soon after the introduction of auscultation, chemistry, in the hands of Dr. Prout, Dr. Cristison and others, was brought to bear both on pathology and diagnosis, and in their hands the examination of the urine in health and in disease was made to furnish signs of considerable value in the diagnosis of renal diseases, diabetes, and other constitutional maladies. Since that time, however, neither chemical nor microscopical researches, although they have brought to light many new and important facts in minute anatomy, physiology, and pathology, have in any remarkable degree contributed to the advancement of diagnosis.

In comparing the present time with the commencement of the last half century, we cannot but be struck with the great advance that has been made by our profession at large in the practical knowledge of disease. Hundreds of country practitioners are now good auscultators, and are fully competent to make a chemical and microscopical examination of the urinary and other secretions sufficient for all practical purposes, and are able at once to recognise the true nature and the existence of diseases formerly known only to a small minority.

TREATMENT.—Changes in the treatment of disease have not been less remarkable than those which have occurred in the mode of their discovery. Thus, of late years, a gradual but decided change has taken place in the treatment of acute diseases. It consists not merely in the discontinuance of copious venesection and violent purging, but in the employment of stimulants, sometimes to a great extent.

This change has resulted not so much from an increased knowledge of the nature of disease, and of the chemical changes that take place within the body, or from the researches of any one person, as from the careful study of the progress and termination of various diseases by many clinical observers.

The employment of stimulants in typhus and enteric fevers can hardly be styled a *change*, because it was practised a long time back, but had been over-ridden, to a certain extent, by the disciples of a school that flourished about 50 years ago. Up to a comparatively recent period, however, inflammation has been almost universally treated by depletion; as if it were what its name would denote, a burning flame, which would be lit up and fed by meat and alcohol, and could only be extinguished by venesection, purging, and low diet. Thus about 30 years since the attention of the senior surgeon of a large provincial hospital having been directed to a bleeding from the stump of a youth whose leg he had just removed. "Let it bleed," said he, "it will prevent inflammation." This remark was received with no surprise by the majority of the bystanders, but it rather startled some, who thought that the poor lad, with his pale, anxious face, and form wasted by strumous disease of the knee, had no blood to spare, and did not seem a subject for violent inflammation. From that time every patient in that hospital who had been the subject of a severe accident or operation was closely watched and examined, and a few years afterwards a paper* was pub-

* Medical Gazette.

lished, in which it was shown that inflammation occurring under these circumstances in the organs of the chest had, more or less, of an asthenic character, resembling those which appear after parturition; and that, consequently, it was the duty of the surgeon to prevent all unnecessary loss of blood during operations, and to sustain his patient by every possible means. This proposition might safely have been extended to every organ of the body. These views respecting depletion were not, however, held by *all* the older practitioners, for the senior physician remarked one day, "When I see so many cripples in the streets my conscience sometimes smites me, and I ask myself how many of them have been reduced to this state by my having bled them in rheumatic fever."

At this time it was the custom of some practitioners to deplete, even in those cases wherein the patient had been much enfeebled previous to the inflammatory attack; so that it was not unfrequent to see a very large number of leeches applied to the abdomen of a female labouring under puerperal peritonitis. Others, however, recognised an asthenic state of the system as requiring support rather than depletion. Thus Sir C. Mansfield Clark and some other physicians treated erysipelas with bark and wine; but when it occurred after operations and accidents, some surgeons employed depletion, others gave salines, but few, if any, stimulants.

Such was the state of things when towards the close of 1836 Influenza attacked a large number of persons in Birmingham. In a short* treatise, which embodied the results observed in one hundred cases, this disease was characterised as "an affection of the nervous system, with its concomitant derangements of digestion, circulation, &c., commonly known under the name of nervous fever, accompanied throughout its whole course by irritation

* "Treatise on the Influenza of 1837," by Peyton Blakiston, p. 59.

of the pulmonary mucous membrane, which not unfrequently amounted to congestion and even inflammation," and in which it was stated that "it was often found necessary to have recourse to diffusible stimulants at the commencement, and to administer tonic medicines in an early stage of the disease;" a definition of the nature of the disease, and an outline of its treatment, which was accepted and adopted by Dr. Graves.*

Whilst pursuing this line of treatment, the use of stimulants was occasionally suspended when inflammatory complications seemed to run high; but it was soon found that this suspension made matters worse, and that, on the other hand, when stimulants were persisted in, the inflammation, to say the least, was not increased by the treatment. Here, then, the field was widened, and a strong presumption was raised that stimulants might be advantageously administered, not only in fever, but in inflammation occurring in an asthenic form, or in a debilitated state of the system, and from that time to this it has been extensively acted upon in practice. As, however, the debility for which stimulants were given was usually associated with *irritability*, more or less of sedative drugs were conjoined with them, particularly opium.

A clinical lecture given at the Birmingham General Hospital, five years afterwards, concluded thus: "You will not fail to perceive that the backbone of the treatment which has been pursued in these cases is the employment of *stimulant* and *sedative* remedies in a comparatively early stage of the disease; indeed, the necessity of 'keeping up the strength during the period that nature is exerting herself to effect a cure cannot be too strongly impressed upon you. In all acute diseases, not excluding the exanthemata, and inflammations of an important organ, whenever you have good reason to believe that your patient's strength is less than it was at your previous visit, com-

* Graves' "Clinical Medicine," p. 558.

mence supporting it by ammonia, wine, brandy, bark, &c., as each case may seem to demand, and, depend upon it, you will not be too soon. Do not wait for complete prostration, but *anticipate* it. Neither delirium, nor great frequency of pulse need deter you, as in the majority of cases both will subside in some degree under the influence of stimulants. It is true that indications of great congestion may induce you to relieve vascular tension by the abstraction of a few ounces of blood, or the application of some leeches, or the cupping-glass; but this should not prevent your supporting your patient at the same time, for it is wonderful how quickly an apparent sthenic plethoric state of the system may be superseded by fearful prostration."

It is highly probable that a large number of clinical observers drew similar conclusions from their study of the influenza of 1837. Of these, the late Dr. Todd was one, and the subject was freely discussed with him in 1838, and subsequent years.

These views have now been carried out in public and in private practice for nearly thirty years, with what results may perhaps be made to appear by slight sketches of a variety of cases, in the treatment of which the administration of stimulants formed a leading feature.

No attempt will be made to solve the question so often mooted, as to whether the state of the air or the constitution of man is different from what it was in former times; as it would be impossible to arrive at a satisfactory solution of it by the numerical system, there being no fixed standard by which to measure the amount of depression of the vital forces in different outbreaks of diseases, or in different periods and individuals. Few persons, however, who have practised many years will, it is thought, hesitate to admit that diseases, in every way apparently similar, are now treated very differently from what they would themselves have treated them thirty years since.

Nor will the nature of inflammation or the physiological

action of alcohol on the system be discussed. These subjects have recently been investigated by Dr. L. Beale,* whose papers can be consulted by those who are desirous of going into these questions. It may, however, be remarked that in the treatment of the cases here brought forward alcohol has not been given as *nutriment* or *food*. This has been furnished by the juices of muscle, milk, farina, &c. Alcohol has been administered as a *medicine*, chiefly to keep up the heart's action, and with it the strength of the patient, and latterly under the belief that it tends to diminish and cut short inflammatory action in the manner pointed out by Dr. Beale in the paper above referred to.

This line of treatment was commenced cautiously, more particularly when employed in inflammation. About the year 1845, however, such an impression had been made on the consumption of wine and spirits at the Birmingham General Hospital, that it excited the attention of one of the Managing Committee, who was a stanch teetotaller, and induced him to inquire the cause. He was requested to repeat the question at the next board-day. In the meantime, the secretary was directed to make out a list of the number of entries of fevers and of their terminations that had taken place during the last two years, and also a similar return for two consecutive years, five or six years back. When these returns were examined by the Chairman of the Board, it was found that the proportion of deaths to entries was much less in the two last years than in those of an earlier date. It is not pretended that this return could furnish data sufficiently numerous for generalisation; let it be taken for what it was worth. At any rate it made out a *prima facie* case, and encouraged those medical officers who had employed this line of treatment steadily to persevere in it. Nor in the present instance are cases adduced for

* "On Deficiency of Vital Power in Disease, and on Support; with Observations upon the Action of Alcohol in serious cases of Acute Disease." 1863.

statistical purposes, but simply as illustrating what has occurred in the practice of one person.

There are three questions, the answers to which will be anxiously sought by thoughtful and practical men:—

1. At what stage of disease and under what circumstances should the administration of stimulants be commenced?

2. When and wherefore should they be discontinued?

3. Have any injurious effects from their use been observed during the course of the disease, or in after-life?

The particular form in which they should be given is a matter of minor importance, and has sometimes to be determined more by the sensations of the patient than the choice of the medical attendant. Of late years, brandy has been more used than wine.

It is not easy to fix a maximum quantity beyond which it is not safe to proceed. A great amount has been sometimes given in the London hospitals, much larger in fact, than was ever given in any of the cases here adduced; but it is well known that a great number of patients labouring under acute diseases are admitted into them in a state of extreme prostration, and that very many are habitual spirit-drinkers. The wonder is that any of such cases should terminate in recovery. It is but fair to state that a very large majority of the patients in the Birmingham General Hospital, and nearly all those attended in private practice were placed in circumstances much more favourable to recovery.

Let us now proceed to discuss the above questions *seriatim*—

1. At what stage of the diseases under treatment, and under what circumstances should the administration of stimulants be commenced?

There are some cases in which there is but little perceptible depression, at least in the early stages of the attack, and consequently stimulants are not called for; the treatment consisting for the most part of a due regulation

of the secretions and attention to diet. Thus, in certain mild cases of fever and the exanthemata, the specific poison may be presumed to have attacked the system in a very diluted state, and to have had but little effect in depressing the vital powers. But this absence of debility is sometimes more apparent than real.

CASE 1.—*Scarlatina—Death.*

A gentleman, æt. 40, was attacked with scarlatina. He progressed favourably for some days, taking meat-tea, and light nourishment, with effervescing draughts containing a slight excess of ammonia. He was seen one evening by his medical attendant at seven o'clock, and seemed as well as usual. Towards one o'clock in the morning the nurse perceived what she called "a change," in him, and proposed sending for the doctor; but this was declined by his aged father, as being unnecessary. At eight o'clock in the morning he was visited, and found in a state of extreme exhaustion; and in spite of a large amount of stimulants and every other remedy that could be suggested, he died before night.

This occurred many years ago, and was ever afterwards regarded as a warning. It has indeed been a source of deep regret that the evidences of failing strength were not more carefully looked for, as it has been felt that they must have existed in a more or less degree; and that, had they been discovered and a small dose of stimulant administered, a fatal issue might possibly have been prevented, although it was doubtless mainly due to the neglect of the nurse's warning. Ever since that time, the tendency of all acute diseases to reduce strength has been assumed, and the way has been felt, as it were, by giving at the onset very small doses of stimulant, in the shape of brandy, wine, bark, or ammonia, whenever the patient has shown the slightest indication of debility, gradually increasing the dose when necessary. By adopting this line of treatment in *anticipation* of exhaustion, it has seldom been necessary to have recourse to a large amount of stimulant.

The importance of discovering the *first* indications of de-

bility, and at once commencing to give stimulants, is well illustrated in the following:—

CASES 2.—*Enteric Fever—One death—Two recoveries.*

Three unmarried sisters of middle age were residing at the sea-side. Two of them were attacked with enteric fever. When visited, in consultation with Mr. Gardiner, it was found that the one who was first seized had been treated homœopathically; that violent diarrhœa had existed during the last four or five days, during which time her diet had consisted of rice-water. Her prostration was extreme. An attempt was made to revive her by stimulants, meat-tea, &c, whilst astringent, sedative, and nutritive enemata were administered. The diarrhœa was arrested, but no impression was made on the system, and she sank in three days. Another sister had been ill three or four days. Beef-tea and ounce doses of wine were given alternately at short intervals. She eventually recovered, but her convalescence was rather protracted, and from first to last she took a considerable amount of stimulant. In a few days the third sister sickened, and to her was given one teaspoonful of brandy or one tablespoonful of wine whenever she was faint or exhausted; and it was never found necessary materially to increase the dose; so that on the whole she took a much less quantity than the second sister; the disease ran its course much more quickly, and the convalescence was less protracted; in fact, she was well some time before the second sister.

The advantage of giving stimulants in an early stage of the disease, particularly as contrasting with other cases in which its administration had been postponed, is strikingly seen in the following:—

CASES 3.—*Enteric Fever—Recoveries.*

Two unmarried sisters, rather under middle age, were both seized about the same time with symptoms which ushered in an attack of enteric fever; and in both of them there was a frequent tendency to fainting. One of them had the greatest repugnance to stimulants, in consequence of which they were withheld until the approach of collapse became imminent. Large doses were then required and given. No material amendment took place for 24 or 25 days, and the convalescence was very protracted; and when last heard of, this patient remained weakly and delicate. The other sister took from the first a

teaspoonful of brandy in a little soda-water whenever she felt faint. This dose was never increased. In 10 days' time this patient was better than her sister was at the end of 30 days, and in three weeks she was well, not having taken a fourth part of the brandy consumed by her sister. When last heard of she was in excellent health.

Thus not only has the *early* administration of stimulants rendered it unnecessary to have recourse to large doses, and has kept down the quantity consumed throughout the illness, but it has seemed to lessen its duration and greatly to hasten the recovery.

CASE 4.—*Enteric Fever—Recovery.*

A little girl, æt. 9, had enteric fever without any rose-coloured spots. From the first she lost her appetite and flesh, and her strength rapidly failed. There was a tendency to diarrhœa. A teaspoonful of brandy was given frequently when she was seen to be most exhausted; but the quantity never exceeded two ounces in 24 hours. Strong beef and chicken tea were also given, with sedative enemata. A decided turn for the better took place in 14 days from her first sickening, and in another week she was enabled to return home to London.

Here, as in Case 3, the course of the disease seemed to be shortened by about seven days, and the convalescence was extremely rapid and sound.

CASE 5.—*Scarlatina—Recovery.*

Two girls, æt. 7 and 9, and one boy, æt. 8, were taken with scarlatina. The boy and one girl sickened within one day of each other, the second girl some days afterwards. To both girls a teaspoonful of brandy was given occasionally from the first, and the quantity in each case never exceeded three ounces in 24 hours. Both were occasionally delirious, and whenever they were so a little additional brandy brought them to themselves and lowered the pulse. The disease ran a very short course, and convalescence was rapid and sound in both cases.

The boy was treated without any stimulant. The disease was very severe, and ran a slow course; and at times he was thought to be at death's door. During convalescence anasarca came on extensively; and although he eventually recovered, he was long in a very weak and sickly state.

The same plan of treatment was pursued in certain cases of *Pneumonia*.

CASE 6.—*Pneumonia—Death.*

A single lady, æt. 48, had been very much reduced in health and strength by over-anxiety and fatigue in nursing a sick relative, when she was attacked with pneumonia. After she had been ill some time she was seen in consultation. The lower half of the left side of the posterior part of the thorax furnished signs of pneumonic condensation. No traces of crepitation could be discovered. She was greatly prostrated, and stimulants were immediately and freely administered, but she never rallied, and sank without the disease having extended itself.

The fatal termination of this case can only be accounted for by the low state of the system when the attack came on, and from no attempt having been made to overcome it by the prompt administration of stimulants.

CASE 7.—*Pneumonia—Death.*

A traveller, æt. 40, had been ailing for some days, and at length, having taken to his bed, he was visited.

The lower half of the right side of the thorax, both before and behind, gave signs of pneumonic condensation. On inquiry, it was found that he was in the habit of drinking spirits to excess, and was the subject of delirium tremens, and that since the commencement of his illness all stimulants had been withheld from him. They were at once freely given in conjunction with opium, &c., but they had not the slightest effect on him, and he sank rapidly.

In this case also the organic lesion was comparatively slight; but there were three circumstances which contributed to bring about a fatal issue. The constitution was injured by habitual drinking and attacks of delirium tremens; stimulants had been withheld during the early stages of the attack; when given they produced a comparatively feeble effect on organs which had been accustomed to large quantities of raw spirit.

In the following cases the symptoms indicated a greater extent of disease; but stimulants were given in a much earlier stage of the attack:—

CASE 8.—*Pneumonia—Recovery.*

A lady, æt. 50, was attacked with cough, dyspnœa, rusty expectoration, and other signs of pneumonia. Her medical attendant, knowing that she was easily prostrated, gave her ounce doses of wine from the first. When seen in consultation with Mr. Ticehurst, there were signs of

pneumonia in the first and second stages over the lower two-thirds of the right side. At the back of the left side there was a mixture of large and fine crepitation, but there was but slight dulness on percussion. It was considered not improbable that at the back of the left side there was some emphysema, which, joined with congestion, or at any rate with small patches of pneumonia, gave rise to the signs observed. Two or three days after this she was seen by her brother, an eminent physician in London. He was of opinion that she was labouring under pneumonia in both lungs, and that she would not live 24 hours. Dyspnœa was very distressing, and there was much lividity of countenance. Stimulants were pushed to a bottle of sherry and six ounces of brandy daily for some days, with counter-irritation, &c. She gradually recovered, and has enjoyed excellent health for some years.

CASE 9.—*Double Pneumonia—Recovery.*

A farmer, æt. 28, of temperate habits, after exposure to wet and cold, was seized with shivering, succeeded by dyspnœa, cough, and rusty-coloured expectoration. Within three days he was seen in consultation with Mr. Wilkins. There were then signs of pneumonia in both lungs, but condensation had not taken place. The case was looked upon as hopeless, owing to the extent of lung invaded. Tartar emetic and opium were prescribed, with turpentine stupes, and half an ounce of brandy, to be rapidly increased to a larger one if required, and given every hour. In about 10 days a letter was received from the medical attendant, which caused no little surprise, because it stated that the patient was progressing towards convalescence, when it was supposed that he must have been dead some days. He has enjoyed good health ever since.

Here were two cases of infinitely graver character than either of those which terminated fatally. They were, however, treated very differently at the onset, and ended in complete but certainly unexpected recovery. No cases could more forcibly illustrate the importance of an early administration of stimulants, and the danger of postponing them.

The success that attended this plan of treatment in the following case of pleurisy cannot but appear very extraordinary to those who remember how this disease was treated in former years, when it was not uncommon to

hear it said that a case had been lost because venesection had not been carried to a sufficient extent. How far it was often actually carried might seem almost fabulous to mention now.

CASE 10.—*Pleuro-Pneumonia—Recovery.*

A man-servant, out of place, of temperate habits, æt. 50, was seized with severe pains in the lower and front part of the right side of the chest, accompanied by great dyspnœa and "a catching in his breath." He was sleepless and had lost his appetite. His wife, considering his pains to be spasmodic, had given him from time to time small quantities of brandy and water. When he was seen about three days after the commencement of his illness, he was sitting up in bed, in great pain, with almost a sardonic grin on his countenance.

An examination revealed the existence of pleuro-pneumonia, of which the pleurisy formed the principal part, and chiefly affected the diaphragmatic portion of the right pleura. His general appearance exhibited signs of great prostration. He was perspiring freely. From half an ounce to an ounce of brandy was given every two or three hours, according as he seemed to require support, alternating with strong beef-tea. One grain of gum opium was given at bed-time, and half a grain in the morning, and the lower part of the left side was rubbed every four hours with an ointment containing mercury and opium. After each inunction a very large linseed-meal poultice was applied over the right side of the chest. In less than 48 hours the most acute symptoms disappeared. It was never found necessary to give more than from six to eight ounces of brandy in the 24 hours. In about 10 days from his first attack he was convalescent, made a most rapid and complete recovery, and has been in good health ever since.

2. When and why should the administration of stimulants be discontinued?

As a general rule, it has been found that as they should be first given when the *earliest* indications of weakness are perceived, so they should be gradually discontinued from the moment that evidences of returning strength make their appearance.

Occasionally, however, certain symptoms appear which would seem to call for their discontinuance. Such have

been represented as being furnished by the state of the skin and the countenance, the tongue, the breathing, the pulse, &c., &c. These, doubtless, demand the attention of the physician. Thus a dry, hot skin, and a flushed countenance, may call for strong doses of citrate or acetate of ammonia, or tartar emetic, as the case may be. A very full pulse may require the tension of the vessels to be relieved by the abstraction of a few ounces of blood or by some leeches. Dyspnœa may require counter-irritation, and occasionally local depletion; restlessness and sleeplessness in some cases opiates; and headache cold lotions; whilst delirium, frequency of pulse, and dryness of tongue, are often benefited by an *increase* in the dose of stimulants. But none of these symptoms have been ameliorated by their total discontinuance, although the experiment has often been tried. There is, however, one occurrence which does call for a diminution in the dose of stimulant, and that is where signs of intoxication suddenly appear.

CASE 11.—*Enteric Fever—Intoxication—Recovery.*

A single lady, æt. 25, was attacked with influenza as it appeared to her medical attendant, Mr. Gardiner; but it soon became apparent that she was labouring under enteric fever. Finding her much prostrated at the onset, and knowing that her constitution was such as required support, he at once kept her well up with wine; first with champagne, and subsequently with port. Several of her family had at different times died of the same disease. When seen in consultation she was very weak, and lay apparently in an unconscious state, refusing all nourishment; but it was said that, at times she was very restless and rambling, muttering lowly to herself. On being roused up, she was told that her life depended upon her taking what was offered to her, and the nurse was ordered quietly, but peremptorily, to press it upon her. After that she took what was given to her. The dose of wine was increased up to such a point, that for three successive days four bottles of port wine were said to have been consumed in twenty-four hours. Half a grain of opium was given night and morning, with liq. cinchonæ. One day she was seized in a manner that greatly alarmed her friends. When visited, she was found talking loudly and

rapidly, and her eyes were brilliant and sparkling. It was found that owing to a mistake of a new nurse, a dose of brandy had been given instead of wine. She was evidently intoxicated. The dose of wine was at once greatly reduced, and from that moment she rapidly improved, and made a splendid recovery. She has enjoyed excellent health ever since.

This case is remarkable on account of the quantity of wine consumed. It was, however, very old, and consequently had lost much of its strength; and it may be doubted whether the whole amount which was taken into the sick room was consumed by the patient. It proves an exception to the rule, that when stimulants are given in an early stage of disease, it is seldom necessary materially to increase the dose.

CASE 12.—*Enteric Fever—Intoxication—Recovery.*

A young lady at school, æt. 15, was seen in consultation with Mr. Gardiner, labouring under enteric fever. She was left doing well and taking about 20 ounces of wine daily. In a day or two afterwards a consultation was urgently asked for by her medical attendant, who had found her furiously delirious, and thought her dying. She was found talking and laughing incessantly and loudly, with her eyes brilliant and dilated, and her breath smelling strongly of wine. She was in fact intoxicated. The amount of wine was at once greatly diminished, and she mended rapidly and soundly.

With this exception, it was often very difficult to know when to diminish the amount of stimulant; but it was always found necessary to continue to give a certain quantity until convalescence was well established. The danger of leaving it off too suddenly and too soon is shown in the following cases:—

CASE 13.—*Enteric Fever—Death.*

A tradesman, æt. 50, of full habit of body, was attacked with enteric fever, which soon prostrated him. He was well supported with wine and bark, and having apparently got over the worst, he was left under the care of his ordinary medical attendant; taking from 20 to 40 ounces of port wine daily. In three days another consultation was requested. He was found rapidly sinking. It appeared that about eighteen hours before, his medical attendant, who was old and timid, seeing his face flushed and his skin rather hot, had entirely suspended

the wine and bark, and had substituted saline mixture. He never rallied, and died in eight hours more.

CASE 14.—*Enteric Fever—Death.*

A young man who had drank water from a well, which had been poisoned by leakage from a drain, was attacked with enteric fever. He was treated with moderate doses of stimulants, and was left on the high road to recovery. After three days he was found in a dying state. The stimulant had been suddenly and totally withdrawn by his ordinary medical attendant, who had always been averse to giving it, and in four hours he was dead.

CASE 15.—*Asiatic Cholera—Death.*

A young woman was left recovering slowly, but steadily, from a severe attack of Asiatic cholera, and taking small doses of brandy and good beef-tea every two hours. Two days afterwards she was found dead in her bed in the morning, and the nurse fast asleep in an arm chair, intoxicated.

3. Have any injurious effects been observed?

It has been seen that in some cases signs of intoxication were noticed, but so far from their proving injurious, they were of the greatest value, foreshadowing the decline of the disease and returning strength, and indicating the propriety of rapidly diminishing the dose of stimulants.

Those persons who have drawn a picture of their fellow-creatures being hurried off into the presence of their Maker in a state of intoxication, must, it is thought, have been prompted more by the suggestions of a fertile imagination than by the truthful observance of facts.

Of course it is almost impossible to trace the subsequent career of hospital patients, but in private practice it is comparatively easy to do so.

No habits of drinking have been traced in those persons who had been treated with stimulants. Those who were temperate previous to their illness have been so since, at least, not one instance to the contrary has come under my notice; nor have they been found to be more prone to

chronic diseases; whilst, on the other hand, cases of pulmonary consumption are very often seen, the apparent origin of which can be traced to attacks of fever, bronchitis, or pneumonia treated without stimulants, and succeeded by a long and lingering convalescence.

The following are the results observed:—

1. The administration of stimulants was found advantageous in the great majority of acute diseases.

2. The earlier they were given, the less was the amount required in the course of the illness.

3. When given in an early stage of the disease, they seemed to shorten its duration in some cases, and in all to induce a speedy convalescence.

4. On the contrary, when the administration of stimulants was deferred, the course of the disease and the convalescence were more protracted, and much larger doses were required.

5. When the slightest sign of intoxication appeared, it was taken to point to the necessity of an immediate and considerable reduction in the amount.

6. No other symptoms seemed to call for their discontinuance.

7. When suddenly and totally discontinued during the course of the illness, serious, and in some cases, fatal effects ensued.

8. In no case was a habit of drinking known to be induced, and very rarely was any chronic disease seen to arise out of the acute attack.

Whilst this important change in the treatment of disease has resulted from the labours of the *clinical observer*, others have arisen from those of the *pathological anatomist*.

Thus, depletion has been in a great measure discontinued in *cerebral apoplexy*, because it has been found to arise in very many cases from rupture of the coats of an artery affected with atheromatous degeneration and calcareous deposit, or else from congestion of the *veins*; and the latter remark applies to pulmonary apoplexy and congestion.

So, too, as the immediate cause of cardiac dropsy has been found to be regurgitation through the tricuspid foramen, produced in the majority of cases by dilatation of the right side of the heart, the means now adopted for its prevention or cure consist in employing such remedies as we can command to strengthen the walls of the heart, and in the avoidance of everything that tends to weaken them, in conjunction with such measures as are adapted to remove the dropsy, when it has taken place. Again, in consequence of the discovery that the "acute inflammatory dropsy" of former days, as well as that which occurs after scarlatina, measles, and other eruptive fevers, depends on an inflammatory affection of the kidneys, their irritation by strong diuretics is avoided, and the attention is directed to produce relief through the action of the skin and the bowels, thus leaving the kidneys as much as possible *at rest*.

Formerly, violent purgatives were often employed in fevers of all kinds; but of late they have been excluded from the treatment of enteric fever, because the irritation and ulceration of the intestinal glands which exist in this disease, call for soothing rather than exciting remedies.

The use of the *microscope* has also led to changes in treatment. For instance, the microscopic examination of the urine has enabled us to distinguish between fatty degeneration, and chronic contraction of the kidney; in the former of which diseases, diuretics are inadmissible, whilst in the latter they are often of great use.

It also enables us to decide whether acid or alkaline medicines are most suitable in certain states of the system.

Chemistry, too, has lent its aid. The analysis of the urine and of the blood, has revealed the existence of certain acids, as associated with gout and rheumatism. We are thus led to give alkalies, in order to make these acids form more soluble compounds; for instance, converting uric acid into urates, which are more easily eliminated from the body.

It has assisted us in our choice of wines suitable to the different forms of diseases we are called upon to treat, such as gout, dyspepsia, &c. Grape sugar having been found as unsuitable as free acid, a particular wine is not selected for its freedom from acid, or from sugar separately, but from the product of the two. Thus port wine has but little acid and a large amount of sugar, the product is, therefore, very high. Claret, on the contrary, has some acid, but no sugar, and the product is very low; therefore, in certain forms of dyspepsia, gout, &c., claret would be chosen in preference to port.

Certain *new medicines* have been introduced of late years. The only two which will be touched on here, are *cod-liver oil* and *pig's pepsine*.

Cod-liver oil may almost be considered as a new form of an old remedy, *suet* and *milk*, so strongly advocated some years back; they both contain animal oil. In 1848, the effects of cod-liver oil on 100 cases of phthisis pulmonalis, were recorded and published. In 20 of these it could not be borne, because it caused either vomiting or purging. But in those days, the oil was usually more nauseous than it is now, and disturbed the stomach and bowels much more than at the present day. It has certainly proved most valuable in strumous cases, particularly when there is great loss of flesh. Sometimes, when it cannot be borne, glycerine will be retained, and act very beneficially, and afterwards, on cod-liver oil being again administered, it can be tolerated.

The *pig's pepsine* was not the form in which the gastric juice of an animal was first made use of to assist the digestion of man. Calf's pepsine was employed in France, and introduced from thence into this country. When fresh and pure, it was found a very good remedy for indigestion, in doses of from 15 to 20 grains. But the mode of its preparation made it so unstable, that the greatest care was required to prevent its losing its strength; and being costly, it was often greatly adulterated, so that many practitioners

ceased to employ it. Thus the Professor of Physiology in King's College, London, sometimes found it fail when it was used for the purpose of producing and demonstrating the artificial digestion of different articles of food. This led him to turn his attention to the process of manufacture, with a view of making it more stable, and consequently more uniform and trustworthy in its action*. Having succeeded in this object, he bethought him that the pig's gastric juice must be much stronger than that of the calf, as it has so much harder work to accomplish in digesting the variety of food that frequently finds its way into the pig's stomach. It proved to be so, and pig's pepsine is three or four times stronger than that of the calf, and is, certainly, one of the most valuable medicines that has ever been discovered. It is perfectly stable, and if kept dry, will preserve its properties unchanged for years. Given in doses of from three to six grains, it not only wonderfully assists digestion, but provokes appetite. It is, therefore, a particularly valuable remedy when the vital powers have been weakened by exhausting fits of illness. Some persons deny its utility on chemical grounds, stating that it only dissolves the albuminous part of the food; but here practice may be allowed to outweigh theory, and it may be confidently asserted, that none who have tried it extensively, will for a moment deny its great value. The following case affords a most striking instance:—

CASE 16.—*Marasmus.*

A young lady was abruptly informed of the death of a gentleman to whom she was affianced. She was completely prostrated, and in a short time reduced to a most fearful state of marasmus. She took no notice of any one or anything, could just crawl about, but was generally carried in her father's arms. Her sole food during the 24 hours was a piece of raw mutton suet about the size of a thumb. After taking five grains of pig's pepsine thrice daily for 12 days, she ate

* Archives of Medicine, vol. 1, p. 269 and 316. 1858.

three meals a day with ravenous appetite, and was able to walk a quarter of a mile. She rapidly recovered her flesh and strength.

It has also a great effect in lessening the secondary affections that result from dyspepsia, more especially palpitation and certain forms of asthma.

CASE 17.—*Spasmodic Asthma, &c.*

A gentleman, æt. 70, had been out of health for some time, suffering, it was stated, from some permanent disease of the heart, and occasional violent and distressing attacks of dyspnœa and exhaustion.

In passing through London on his way from Liverpool, he was detained several days by a very severe attack of this kind.

He travelled down with difficulty, and in the night was very ill. Seen the next morning, he was found sitting up in bed labouring for breath. The action of the heart was very tumultuous and irregular, and the sounds were very weak and confused. The pulse very intermittent. Half an ounce of brandy was directed to be given every hour, or oftener if required, with chloric ether and ammonia. The region of the heart was rubbed with belladonna liniment. As his appetite had entirely failed him for many days, as soon as he had slightly rallied from this state, five grains of pig's pepsine were ordered every four hours, to be followed in fifteen minutes by an offer of some nutritious food. In less than a week he was enabled to take a fair amount of food, and he then mended rapidly; in a few weeks his pulse became much more regular, and he was enabled to walk more than he had done for years. He had an occasional attack of dyspnœa, with intermitting pulse, but of a much less severe character. He gained strength and flesh, and was altogether better than he had been for many years, and much more free from palpitations.

Pepsine was given in this case for the following reasons. The attacks of palpitation and dyspnœa seemed to be exaggerated, if not mainly caused, by extreme debility. The physical examination of the chest revealed signs of chronic bronchitis and emphysema of the lungs; no traces of valvular disease of the heart, but indications of very feeble action, probably the result of fatty degeneration. Tonics in a variety of forms had been exhibited for a long time, and had failed to improve the appetite or increase the strength. Pepsine was therefore given, in the hope that it might improve both appetite and digestion, and thus favour nutrition and restore strength in an easy and natural manner.

CASE 18.—*Spasmodic Asthma.*

A gentleman, æt. 55, many years resident in India, settled in this country on his return. He was a martyr to spasmodic asthma, which seemed intimately connected with the state of his digestion, which was very bad. He was ordered to leave off mercurials and brown sherry, and to take brandy or dry sherry, and four grains of pig's pepsine as a pill before his two principal meals.

In 14 days his digestion had greatly improved, and the asthmatic attacks diminished in strength and frequency. In about six weeks he was better than he had been for years, and only suffered from asthma in a modified form when he had been unusually excited and fatigued, or had been incautious in diet. He now enjoys excellent health.

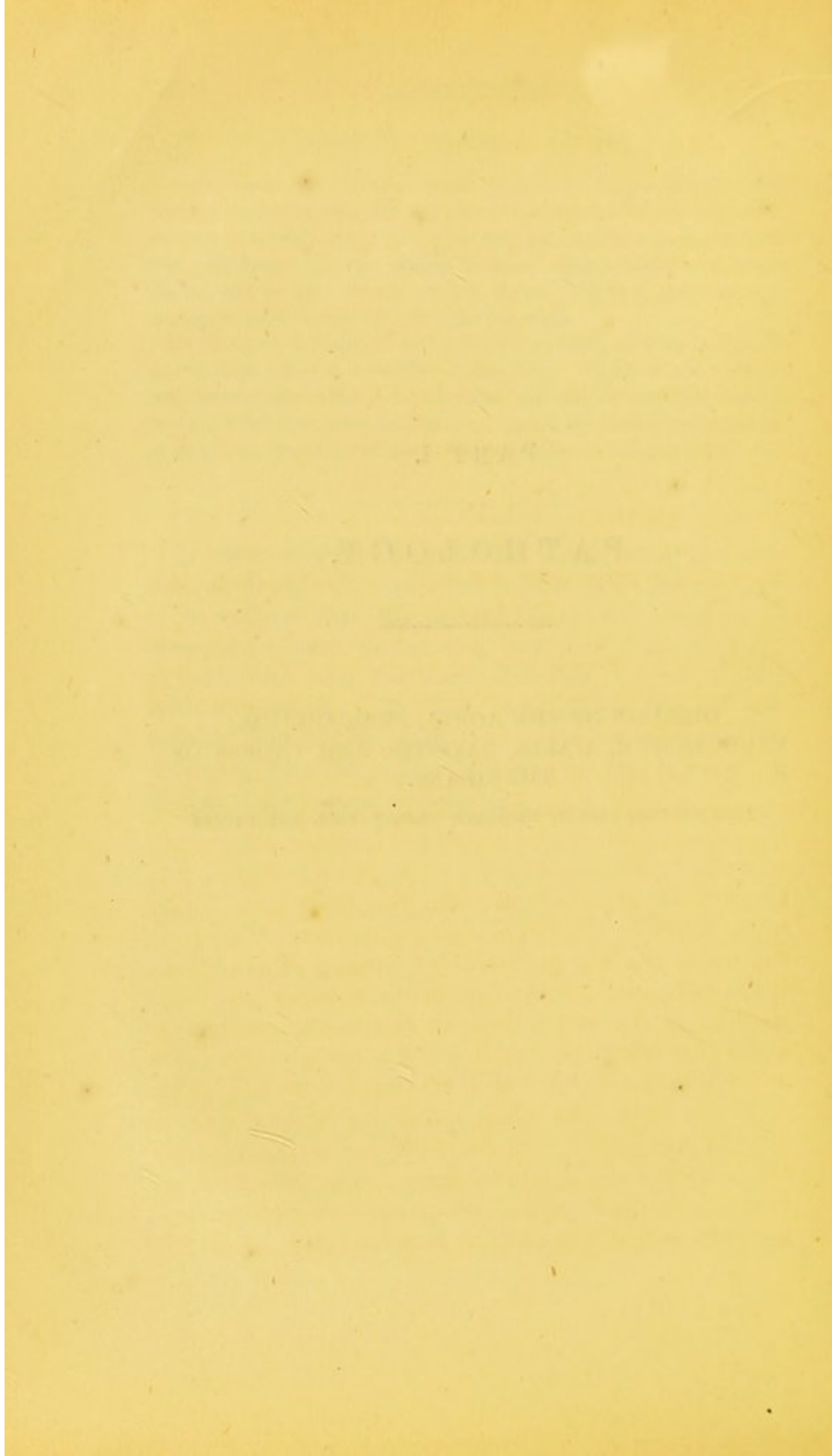
In contrasting the practical knowledge which has now been attained, with that which existed half a century ago, it is evident that the nature, causes, and progress of diseases are better understood, that their existence is discovered with more certainty, and that they are treated more successfully. In short, although we are still profoundly ignorant on many points, yet we are gradually diverging from the path of the empiric, and approaching to that of the natural philosopher.

PART I.

P A T H O L O G Y.

DISEASES OF THE AORTA, PERICARDIUM,
ENDOCARDIUM, WALLS, CAVITIES, AND VALVES OF
THE HEART.

THEIR NATURE, ORIGIN, PROGRESS, TERMINATION, AND CAUSES.



PATHOLOGY.

CHAPTER I.

NATURE AND FORMATION OF DISEASES OF THE THORACIC AORTA.

ARTERIAL COATS.—*Horny Deposits*—*Atheromatous Degeneration* — *Calcification*. ANEURISM.—*Dilated* — *Sacculated* — *Mixed*.

SINCE it has been of late years most conclusively demonstrated that non-vascular tissues, such as the cornea, tendon, and other fibrous tissues and cartilage, may be the seat of inflammation, as well as the vascular tissues, it is by no means impossible that the two inner coats of the aorta should be affected in a similar manner. Where inflammation has been produced by ligature or other violence in the outer areolar coat of an artery, changes have been shown to extend even to the inner coat, and in consequence a clot (thrombus) is formed which adheres to the coats and obliterates the calibre of the smaller vessels. This does not often take place unless the areolar coat is brought into contact with the inner one or the blood, by the laceration of the middle fibrous coat. It is doubtful, however, if arteritis of the thoracic aorta ever occurs *spontaneously*, so that this condition possesses little interest in

practice. Chronic *disease of the coats of the aorta*, and *aneurism* demand especial notice.

ARTERIAL COATS.—The most important diseases of the arterial coats are *horny deposits*, *atheromatous degeneration*, and *calcification*.

Horny deposits are sometimes called semi-cartilaginous patches, but they can be said to resemble cartilage in appearance only, not in structure. The substance of which they are composed bears a very close resemblance to softened horn, and causes the interior of arteries to present a rugged and uneven appearance, being disposed either in patches, or around the whole circumference of the vessel; and appearing in some places opaque, in others more or less transparent.

Andral considers these patches to arise from a deposit between the inner and middle coats of the artery; and Hodgson represents them as resulting from a thickened state of the inner membrane itself, comparing the latter to the peritoneum of an old hernial sac. Bizot, however, has shown that they are, for the most part, deposits on the free surface of the inner coat. He has shown that the substance deposited passes by insensible degrees from the consistence and transparency of jelly, to the opacity, and almost to the hardness, of cartilage; and this in an exact ratio with the age of the subject examined. In three cases in which the patients were suddenly seized and quickly carried off by an acute attack, he saw it in its softer state, and of a rosy colour, lining a great part of the principal arteries. He found that the patches in an early stage of their development, before they had acquired hardness and opacity, might be removed with the inner membrane, which could be thus traced *under* them. They are often covered by a delicate film, closely resembling the inner coat itself, and this, doubtless, has given rise to the supposition that the deposit takes place between the inner and middle coats. Microscopic examination shows that this is only a very thin fibrinous film, similar to that which is found lining aneurismal

sacs and investing fibrinous clots. Bizot considers this deposit to be a secretion from the inner coat of the artery, acted on by inflammation, in fact, coagulated fibrin, resulting from what Vogel calls fibrinous dropsy. Patches of a similar kind are sometimes found on the pericardium covering the heart, and on the lining membrane of the left auricle, in which situations they are generally regarded as the result of inflammation. On the other hand, no traces of inflammatory action are usually found in the lining membrane near them, and hence it has been argued that they may possibly be deposited direct from the blood in certain abnormal conditions of that fluid. Simon thinks it probable that the same process sometimes takes place in granular disease of the kidney. Bizot states that they never ossify, or become the seat of calcareous deposits, as asserted by Morgagni, Halle, Bèclard, and Andral. In confirmation of this view it may be remarked that these horny patches are not a form of cartilage, nor are calcareous depositions true bone, and, therefore, the transformation of one into the other cannot be assumed. Viewed under the microscope they are seen to consist of an amorphous mass, more or less transparent, and rendered still more so by acetic acid, which contains some granules and pale cells without nuclei. In some cases no cells can be discovered, and in others there is a trace of fusiform cells, showing their fibrinous nature, and perhaps a tendency to the formation of a low form of connective tissue.

As repeated examinations have shown the accuracy of Bizot's researches, it may be concluded that the horny patches are *imperfectly organised false-membranes*, deposited on the free surface of the inner coat of arteries.

Atheromatous Degeneration.—Atheroma has been described by some writers as a substance deposited between the internal and middle arterial coats; by others between the fibres of the latter; being thus considered as a secretion independent of the coats. Recently, however, it has been represented by Virchow and others as resulting from fatty

degeneration of the inner coat itself. It is probably a compound of the two processes, for at an advanced stage the atheromatous mass is often thicker than all the coats of the artery; in the smaller vessels, in fact, obliterating the canal. Now, this could hardly be produced by the swelling and disintegration of the fibrous tissue alone; but when this process has taken place, fibrinous material might be deposited amongst it from the blood, as in the case of false membranes. Thus the mass would be considerably increased in bulk. The general appearance varies with the stage of development. At first very minute specks are seen on the interior of the artery; these gradually increase to roundish patches, at first flat, then a little raised. These patches appear more homogeneous in their nature than the arterial coats, are of a whiter colour, and a softer consistence. Arrived at this stage, in middle or old age, the internal coat gives way, and the atheromatous mass often projects into the artery, varying in consistence from that of cheese to pus. In some instances its consistence is rendered more firm by the presence of connective tissue, in which case it has been regarded as one of the forms of steatoma. It has been found by Gluge to consist chiefly of fat; and Cruvellier, Hope, and Gulliver have discovered cholesterine crystals in it. Viewed under the microscope it has been described by Vogel as consisting of "(1) many tabular, colourless crystals of cholesterine, with rhomboidal tablets; (2) of irregular granular masses which did not dissolve in water, but were soluble in alcohol, after which they again thickened into amorphous brownish clots, probably fat. Besides these elements and some fat globules, nothing was present." Virchow describes it as "showing cholesterine crystals, granule cells, fat cells, and lumps of half softened substance, which are fragments of the undegenerated tissue of the inner coat." It may, therefore, be considered to result from fatty degeneration of the vascular coats, similar to that observed in other parts of the body.

Calcareous concretions are frequently deposited in the

atheromatous patches, which coalescing form crusts and bony-looking scales. They are chiefly composed of phosphates, with some little carbonate of lime. Valentin* describes them as "organised calcareous deposits, the earthy bodies being deposited as round, granular, or irregular compact bodies (with prolongations radiating in all directions) within a pellucid, more or less lamellated, and finely granular, organic texture." Virchow,† however, considers that although most of the calcification thus seen arises from mere deposit of lime, that in some cases true bone is formed.

THORACIC ANEURISM.—Several varieties of aneurism were formerly described by systematic writers, but they will here be referred to *two* divisions only, an arrangement which will be found to be of practical utility. These are *dilated aneurism* and *sacculated aneurism*.

Dilated aneurism consists in an enlargement of the artery, all the three coats being preserved; sometimes taking place in an uniform manner, and assuming a globular or cylindrical form, at others bulging out from one side of the vessel like a hemisphere. Such an aneurism, as long as it retains its distinctive character, never reaches a very large size, its extent being limited by inextensibility of the internal coat.

Hodgson having seen the aorta thus morbidly dilated "without disease in its structure," asks whether this "may not be owing to a state of the fibrous or middle coat not unlike paralysis." But it might be remarked, that were the vessel otherwise healthy, paralysis would only affect its insensible contractility, by which means it might be prevented from contracting on a diminished column of blood. Its permanent dilatation could only be occasioned by a loss of the elasticity by which it returns to its usual size after distension. In truth, however, the healthiness of the coats of the artery under those circum-

* Hasse's Pathological Anatomy, Syd. Soc., p. 79.

† Chance's Virchow. Cellular Pathology, p. 365.

stances is only apparent; for since attention has been drawn to this subject by Mr. Gulliver, the tissues have repeatedly been examined under the microscope, and have invariably been seen to show traces of atheromatous degeneration.

So that it may be inferred that the aorta is never morbidly dilated unless its coats are more or less diseased; sometimes to a very slight extent not discoverable by the naked eye, more frequently, however, shewing unmistakably the existence of atheromatous degeneration and calcareous deposit.

It may also be asserted that in its healthy state the aorta does not admit of dilatation, for cases have been recorded in which the most violent action of a largely hypertrophied heart has been observed for a long time, and no dilatation whatever discovered after death. This was particularly observable in Case 33. Nor again will constriction of the aorta produce dilatation to any extent; for in two cases great constriction existed; in one just below the origin of the subclavian artery, and in the other at the point where the ductus arteriosus is given off; and there was no dilatation, although in one case there was atheromatous degeneration.

The form of the dilated artery is necessarily such that the current of blood sweeps freely through it, and therefore cannot deposit coagula on its inner surface. Sometimes, however, through extensive disease, the walls crack and present fissures, in which coagula are deposited, but not to any great extent.

Sacculated Aneurism arises from ulceration or rupture of the internal and middle coats of the artery. The blood, escaping through the aperture, expands the external areolar coat into a sac. Although such rupture was artificially produced by Hodgson with a ligature, yet he considers that no pressure from within could bring about such a result, nor any violence from without that did not at the same time rupture the neighbouring parts. The two fol-

lowing cases prove that the walls of the heart may be ruptured while the pericardium remains unbroken; and that the inner and middle coats of the aorta and of the auricles of the heart may be lacerated without the outer coat being injured.

CASE 19.—*Laceration of the Endocardium and Muscular Fibres of the left Auricle of the Heart.*

A female, æt. 90, was run over by a cart within a few yards of the Birmingham General Hospital, to which she was immediately carried, and died in half an hour.

Inspection.—The sternum was transversely fractured an inch and a half above the ensiform cartilage, without the periosteum being lacerated. The fourth, fifth, sixth, and seventh true ribs on the right side were fractured about an inch from their junction with their respective cartilages. The exterior surface of the pericardium had a healthy appearance, but on opening its sac, a patch of ecchymosis was observed on the posterior surface of the right auricle. At the interior of the auricle which corresponded to this patch, a laceration was observed an inch and a half in length, having irregular edges and extending quite through the endocardium and muscular coat. Through this wound a small quantity of blood had passed, and thus gave rise to the ecchymosis observed. A small ecchymosis was also perceived under the reflected pericardium, opposite to that on the auricle.

CASE 20.—*Laceration of the Inner and Middle Coats of the Aorta.*

A groom, æt. 54, was running out a horse for sale, when he was suddenly struck on the chest by the shaft of a cart. He was immediately taken to the Birmingham General Hospital, a distance of one mile, and on his arrival there he was found to be dead.

Inspection.—The sternum was transversely fractured between the second and third ribs. The second, third, and fourth, left costal cartilages were cracked through. The pericardium contained two pounds of blood, which had escaped from the heart through an aperture in the appendix of the left auricle half an inch in length. A laceration an inch and a half long was found in the aorta between the orifices of the left common carotid and left subclavian arteries. It extended through the inner and middle coats of the vessel. A small quantity of blood was extravasated for some distance around it under the cellular coat, which was uninjured.

Supposing such laceration not to have been immediately followed by death, it is not probable that, even in that case, aneurism would have resulted ; for the experiments of Dr. Jones and Mr. Hodgson render it likely that lymph would have been thrown out, and that the edges of the wound would have been reunited. This point, however, is of no great practical value, because it will be seen that more or less of structural change is always found in the coats of the aorta when it is the subject of aneurism.

When a sac is thus formed in the case of an artery previously dilated, the sides of the pouch in part consist of all three coats, and partly of the outer one only ; the sacculated being, as it were, engrafted upon the dilated form, thus constituting a *mixed aneurism*. There is little practical difference between these two forms of disease, except that in the mixed aneurism the communication with the aorta is necessarily somewhat large, and allows a free passage to the blood ; whilst in the sacculated aneurism it is often, at first at least, small ; so that the interior of the sac is not exposed to the action of a large current of blood. It is often very difficult to determine whether such an aneurism has arisen from simple perforation, or from previous dilatation of the two inner coats, and hence much difference of opinion and controversy has arisen, which it would be useless to revive. Cases of mixed aneurism are by no means uncommon. Such were Cases 25, 60, and 72. On the other hand, many which on first inspection appeared to be such, have, on a closer examination, turned out to be simple sacculated aneurisms, the mouth of the sac being lined for a certain distance only by the middle coat, terminating with an abrupt edge, and corresponding exactly in size with the opening into the sac. Now had the pouch been formed in the first instance by dilatation of all the coats, the extent of the middle coat in the sac must have exceeded the area of the orifice. Supposing, however, the formation of the sac to have been preceded by perforation of the inner and middle coats, if the latter adhered closely to the areolar coat, it would be dragged into the sac by its extension and dilatation. But if the two were not closely

united, the areolar coat would for some distance be dissected off the middle coat, which for a time would stand as a kind of diaphragm between the aorta and the pouch; sooner or later it would yield to the outward pressure of the blood, and be forced against the inside of the mouth of the sac, and probably become agglutinated to its walls by lymph. In either case the result would be a sacculated aneurism, having its orifice lined for a certain distance proportioned to its size by the middle coat of the aorta. It may be argued that the inner and middle coats might have originally extended further into the sac, but had been subsequently destroyed. In that case the raised edge of the middle coat would not have been so distinctly marked, nor would its extent have been so exactly proportioned to the size of the orifice in so many cases.

Sometimes a thin delicate membrane, apparently continuous with the inner coat of the aorta, may be seen entering the aneurismal pouch, and lining it for a considerable distance. The extent of surface it thus covers in certain cases proves that in them it cannot be the inner coat, which was long since proved by Hunter and Sir E. Home to be never very extensible. Bizot* has shewn that this membrane is not a continuation of that which lines the aorta, although its point of union with it cannot be detected with the naked eye. It was seen in Cases 35, 52 and 62. The following case, however, furnishes a decided proof of the nature of the membrane in question, for it not only lined a portion of the sac, but completely invested a large laminated coagulum, shewing that it was formed subsequent to the deposition of fibrin in the sac.

CASE 21.—*Mixed Aneurism springing from the Concavity of the Arch of the Aorta.*

A stonemason, æt. 48, applied for advice. He complained of pains flying about him, particularly in the chest, and of dyspnœa. Signs of

* *Memoires de la Société Médicale d'Observation*, vol. i, p. 339.

slight bronchitis were detected in the chest, but nothing beyond this. He was relieved, and applied again in six months. He then complained of a troublesome cough, and occasional severe fits of palpitation. The respiratory sound was natural, but was absent to the left of the upper part of the sternum. The impulse of the heart was strong. The pulse at the right wrist was decidedly more feeble and much smaller than that at the left wrist.

Between the second and third left costal cartilages, a loud hollow systolic rasp-sound was heard, the intensity of which rapidly decreased in all directions, but still could be heard at the precordial region. Over this spot there was a slight dullness of sound on percussion, and absence of respiratory sound, as has been stated, and it was thought there was slight fulness of the space between the cartilages. He was relieved by small bleedings, &c., &c.

Six months after this he was visited, and found in bed. He was thin, and was excessively troubled with cough, accompanied by clear expectoration, and by pain under the sternum, shooting both backwards and down the right arm, and great constriction across the chest. The pulse in the right wrist was almost imperceptible. The action of the heart was strong and heaving. Between the cartilages of the second and third left ribs, close to the sternum, a dull sound was given out on percussion, and an elevation as large as a half-crown was perceived. A pulsation not very liquid, but no purring thrill, was felt over this spot. No pulsation was felt over the top of the sternum, or over either clavicle. The respiratory sound was accompanied by a little cooing in spots. A short raspish systolic sound was heard over the left nipple, increasing in intensity as the ear approached the elevation between the second and third cartilages of the left side, where it was louder and more hollow. Mild sedatives were ordered, and cold applications to the elevated spot, but he did not bear this well, his pain being much increased by it. One day he felt something burst under his breast-bone, and the blood seemed to rush up to his head and ears, after which the pain and constriction of the chest and dyspnoea became much less. In another six months the pulse in the right wrist was still extremely small, and his right arm had for some time felt rather benumbed. This he attributed to its being much out of bed, as he could only lie on his left arm, with his right arm crossed over him, without urgent dyspnoea. He had also twitchings in his right arm, and thought his hearing was impaired.

Between the cartilages of the fourth and sixth ribs on the left side there was an elevated cone, with a base as large as a shilling, a slight elevation spreading for an inch or two around it. There was dullness for some inches around this spot, and pulsations were felt all over it,

not very liquid. A systolic rasp-sound was heard very loud near the centre, decreasing as the ear receded from it, but still heard above the left nipple. Behind, the respiratory sound was accompanied by much muco-crepitant rattle, and in front it was whistling, as if the trachea was compressed. The expectoration was usually clear and tenacious, but he had once or twice spat a little blood.

He sank gradually ; greatly emaciated.

Inspection.—The external prominence had nearly subsided. On opening the chest, a large tumour was seen lying under the sternum, and projecting into the left side more than an inch beyond the point of the union of the second, third, and fourth ribs with their respective cartilages, and lying on the base of the heart, which was thus slightly pushed downwards. The left lung was compressed by it, and, being cut into, was found carnified of a dark slate colour. The heart was large, firm, and red, but its valves, though a little thickened, acted quite well. The aorta, from within half an inch of the semi-lunar valves up to the origin of the left subclavian artery, was found to communicate with an enormous pouch, which thus involved the greater part of the concavity of the arch. On careful dissection, it appeared that the middle coat of the aorta did not cover more space than it would have done had it been laid open in a healthy undilated state. The natural channel of the aorta was rendered rugged and uneven by the deposit of atheroma, and by semi-cartilaginous ridges. The greater part of the sac to the left was formed of the outer coats of the vessel, and near the ribs these had apparently given away. At this spot a very large, thick, yellowish-brown, fibrinous clot adhered ; its base was as large as a shilling, and it extended more than an inch into the sac. It had abrupt edges, and was *covered with a smooth membrane, which was reflected from it on the walls of the sac, where it soon became rough and granulated, as if coagula had been detached from it.* To the right of the sternum was a similar clot, partly detached from that bone, and hanging loosely into the sac. The origin of the right subclavian artery was completely plugged up by firm coagulum.

On the first visit of this patient it is probable that only a slight examination of the chest was made. On his second visit, the state of the pulse in the right wrist led to a very careful exploration of the chest, when signs of solid matter were found to the left of the upper part of the sternum, and as in the same spot a very hollow rasp-sound was heard, much louder than at the precordial region, it was concluded that the arch of the aorta was dilated, and perhaps also ruptured at its left angle ; and the mouth of the arteria innominata partially closed by atheroma or coagulum. The quality of the rasp-sound was the same as that heard in Cases 26, 55, and 103, and was more hollow than I

have ever heard when it was formed at the orifices of the heart. In a short time the nature of the affection was made manifest, by the appearance of pulsation. After the sensation of bursting, the aneurism pointed in a spot two or three inches lower than where it first appeared, owing to the rupture of the coats, or the spreading of the sac downwards.

In Case 26 a membrane of this kind closed the orifice of a small aneurismal sac of the size of a hazel nut, which sprang from the arch of the aorta, and was completely filled with layers of dense fibrin. Viewed from the inside of the aorta, the orifice looked like a small grey-coloured depression over which the inner coat was continued.

When these membranes are submitted to microscopical examination, their true nature is at once revealed. They are found to be totally devoid of epithelial cells which exist on the inner membrane of arteries, and to possess all the characteristic appearances of fibrinous films, being, in fact, a species of delicate false-membrane, which, in the first instance, consists simply of fibrin deposited from the blood. This undergoes subsequent changes, becoming fibrous, and often approaching connective tissue in its characters. The existence of such a membrane as this lining an aneurismal sac, cannot therefore be advanced as a fact in favour of the view that the walls of the sac were originally formed by dilatation of all the coats of the vessel from which it sprang.

For the same reasons we cannot allow the correctness of the inference drawn by Dupuytren, Breschet, and others from certain cases, that the inner membrane is sometimes forced through a perforation in the middle coat, and thus made to form an aneurismal sac by a kind of hernia. It is probable that the false-membrane above described has been mistaken for the inner coat of the artery, as pointed out by Bizot.

Hodgson has inferred the previous existence of the inner coats in aneurismal sacs from the fact of atheromatous and

calcareous deposits having been found in them.* "This circumstance," he remarks, "appears to me to prove that the coats of the artery compose the disease, for it is the internal coat in which calcareous matter is deposited, and if we find that substance in all parts of the sac it appears fair to infer that the internal coat enters into its composition, inasmuch as identity of disease indicates identity of structure." This observation of Bichat, quoted by Hodgson, does not apply to the case in point. For calcareous deposit is not confined to the arterial tunics, but is often found in false-membranes and imperfectly organised formations on the pleura and pericardium, and even in fibrinous concretions in the cavities of the heart, and in tubercular matter. So as regards atheroma, it is probable that a deposit takes place sometimes very much resembling atheromatous degeneration of the arterial coat, but altogether independently of this pathological change. Thus Bizot relates a case in which a moderately sized sac was completely lined with such a substance, over which was spread a delicate false-membrane, in which the middle coat terminated with short raised edges around the orifice.

It may be inferred therefore that mixed aneurisms are not of such common occurrence as some authors have supposed, and that the formation of an aneurismal sac by the inner coat of the artery passing through a perforation of the middle coat is not proved.

* Diseases of Arteries and Veins, p. 46.

CHAPTER II.

PROGRESS AND TERMINATION OF DISEASES OF THE THORACIC AORTA.

ARTERIAL COATS.—*Horny Deposit—Atheromatous Degeneration—Calcification.* ANEURISM.—*Termination by Perforation—State of Walls—Nature of Contents—Quality of Coagulum—Compression of Neighbouring Organs—Aneurism within the Pericardium—Aneurism below the Bifurcation of the Trachea—Compression of Trachea and Bronchi—Of Recurrent Nerve—Of Æsophagus—Of Vena Cava—Of Heart—Contact with the Vertebrae—With the Integuments.*

ARTERIAL COATS.—*Horny deposit* has been traced from a soft gelatinous substance to one of cartilaginous hardness. It does not pass beyond this, nor ever become the seat of calcareous concretions. When it is of cartilaginous consistence and in large masses, it tends to thicken and harden the aorta, and to destroy its elasticity.

The progress of *atheromatous degeneration* is two-fold, in the one instance being *moist* and *soft*, and in the other *hard* and *dry*, and forming the seat of extensive calcification. The former state occurs chiefly in youth and middle age, the latter at a more advanced period of life. When moist, after a certain time the inner coat gives way, and the diseased mass protrudes into the channel of the artery, even blocking up some of the smaller ones, and thus giving rise to various morbid phenomena by interrupting the circulation. In such cases ulcers are sometimes formed in the coats of the aorta, having a chronic, indolent aspect, with-

out any traces of inflammatory action around them. They are said sometimes to not heal up and leave scars, but such appearances are common.

Occasionally the inner coat is eaten away, as it were, and perforated, and the outer coat either gives way, and the blood is poured out with a fatal issue, or else it becomes distended and forms a sacculated aneurism.

At other times, instead of softening, an atheromatous patch, even in the same artery, becomes invaded by *calcareous deposit*, presenting the appearance of ragged, uneven, bony-looking, brittle scales. This may be carried to a great extent, so much so as to obliterate the smallest arteries, and convert others into hard tubes like tobacco-pipes, seriously interfering with the circulation in all. When this occurs in the coronary arteries, the heart being deprived of a portion of its ordinary supply of blood, becomes imperfectly nourished, and its muscular tissue wastes and degenerates in a remarkable manner. Two or three of such cases are recorded by Hodgson. Under these circumstances spasmodic attacks of angina pectoris are apt to take place. In the following case, however, although the coronary arteries were extensively calcified, the heart was firm and red, and there were no attacks of angina pectoris.

CASE 22.—*Calcification of the Coronary Arteries.*

A light porter applied to Mr. Wilson for advice, in consequence of his suffering from a sense of constriction and occasional darting pain at the præcordial region. He suffered much from habitual dyspnoea and frequent fits of palpitation. The chest was unusually sonorous on percussion, particularly under the sternum. The respiratory sound was unequal, being inaudible in some spots, and mixed with cooing rattles, and large-bubbled crackling. The first sound of the heart seemed double. The pain soon subsided under soothing treatment and rest, but in other respects his state did not much alter for three or four years. Latterly cedema of the ankles and venous pulsations of the neck appeared. The peculiar double sound at the heart was less marked; anasarca gained on him; and he died exhausted by dyspnoea.

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Inspection.—The lungs were extensively emphysematous. The heart was large and dilated. The walls of the left ventricle were firm, red, and slightly hypertrophied. The carneæ columnæ were firm, red, and hypertrophied, most so on the right side. There were a few patches of mottled fatty degeneration in the walls of the right ventricle. The tricuspid foramen measured six inches. One of its valves had short chordæ tendiniæ, the others were normal. The aortic and mitral valves were in their natural state. The coronary arteries were converted into hard calcareous pipes, that of the right side being the more ossified of the two. A milky patch, about the size of a sixpence, was seen on the surface of the right ventricle, the greater part of which could be dissected off from the pericardial covering of the heart. Beads of a similar character followed the tract of the coronary veins. The coats of the aorta were but slightly atheromatous.

Considering the state in which the coronary arteries were found, it is surprising how well this heart would seem to have been nourished. The peculiar modification of the systolic sound remained unaccounted for.

When the aorta is thus invaded by atheromatous degeneration and calcareous concretions, the elasticity of its coats, by which it returns to its normal size and position after any temporary distension by an unusual pressure of blood into it, is much impaired, and hence the artery becomes predisposed to permanent dilatation, general or circumscribed, according to the seat and extent of the degeneration.

The termination, therefore, of these diseases of the coats of the thoracic aorta is either *perforation* of all the coats, and sudden death by outpouring of blood; *dilatation* of the three coats, general or partial; *sacculated aneurism* from rupture of the inner and middle, and distension of the outer coat; or else the artery remains in a comparatively rigid state, which must tend, more or less, to interfere with the general circulation, although, possibly, not to such a degree as to prove fatal.

THORACIC ANEURISM generally terminates fatally by escape of blood through a *perforation* of its walls, or from *compression* or irritation of some important structure near it.

Perforation.—Effusion of blood may take place into the pericardium, mediastinum, pleural cavities, trachea and bronchial tubes, and œsophagus; or externally through the thoracic walls.

The progress of an aneurismal tumour towards one or other of these terminations, is much influenced by several circumstances connected with the *state of its walls*, and the nature of its *contents*.

State of the walls.—There is a constant tendency to attenuation, or rupture of the walls of an aneurism, whether dilated or sacculated, which is not unfrequently counteracted to a certain degree by an inflammatory process, in which lymph is thrown out, and the walls are thereby thickened. Unfortunately whilst the walls are thus strengthened, they are at the same time made more solid and unyielding, so that they are less able to accommodate themselves to a neighbouring part, when they reach one by extension; striking instances of which will be alluded to shortly. Again, adhesive inflammation is not unfrequently set up between an aneurism and the parts with which it comes into contact, in the same manner as the stomach and intestines are sometimes glued together in chronic peritonitis, and thus perforation is prevented, and death is averted.

An evil, however, may result from the aneurism becoming firmly tied to some vital organ, instead of being allowed to extend itself in some other direction, where less mischief might be produced by it. This was observed in cases 60 and 72, where the sac was fastened down to the trachea; in cases 26 and 60, where it adhered closely to the vena cava; and in case 25, where it was bound down to the spine, and erosion of the vertebræ followed.

Contents.—As long as the internal membrane remains entire, it has been seen that an aneurism contains nothing more than the blood circulating through it. As soon, however, as it becomes *sacculated*, there is a tendency for the blood to coagulate within it; and this has in some

instances a very remarkable effect on the progress and termination of the disease, as was first shown by Hodgson, whose researches on this point evince an accuracy of observation and soundness of deduction rarely met with. He has well described the method by which an aneurismal cavity becomes obliterated. Being filled with coagula, generally deposited in successive layers, the tendency to dilate from the outward pressure of the blood is arrested; the clot then becomes smaller and drier by absorption of its fluid parts, and the walls of the sac contract upon it; till at length only a solid fibrous tumour remains. Two such cases are given in his work. In both of these death resulted from pressure on the trachea; so that the solidification of the tumour, although it prevented perforation, caused increased and unyielding pressure on the trachea, and death.

The coagulation of the blood within the sac in some measure depends on the degree of inflammation and roughening of the walls, as it is well known that inflamed surfaces, perhaps from the inequalities of surface resulting, cause the blood in contact with them to coagulate; subsequent changes occurring upon the lining membrane cause the clot to become agglutinated to the walls, and thus an additional security against perforation results. For want of this, perforation of a sac has taken place when it was actually filled with coagula, the blood having insinuated itself between the coagula and the walls of the sac, which at length burst externally; so that Hodgson's remark, that a sac filled with coagulum cannot prove fatal by rupture, although perhaps generally true, is not universally so.

CASE 23.—*Sacculated Aneurism springing from the summit of the Arch of the Aorta—Rupture of external integuments.*

A japanner, æt. 39, applied for advice, and stated that he had experienced more or less pain in his head for the last six months, from exposure to cold; and that at the time of application it was very severe and constant. The pulse was moderate. The edges of the tongue were red, and its middle was covered with a thick, yellowish brown

coat. The appetite was impaired; he could not sleep. His bowels were very much confined. The respiratory sounds and those of the heart were natural. In ten days he found himself greatly relieved.

Four months after this he again applied for advice, stating that during the whole of the previous month he had felt sharp cutting pains between the shoulders, and that towards its close he perceived a swelling above the sternum, inclining towards the left side, in which he occasionally felt severe pain. It was about the size of a hen's egg, and irregular in shape, owing to the cicatrix of an old abscess in the middle of it, at which point the skin was of a purple colour. The tumour rose up from behind the top of the sternum, and bent forwards over it. Fluctuation was very manifest in it, and strong and liquid pulsations were felt all over it. The pulse was natural, and the pulsations of the carotids very moderate. The action of the heart was also rather feeble than otherwise. The chest sounded clear on percussion, the precordial dullness being more limited than usual. The respiratory sounds and those of the heart were quite natural, nor was there any unusual resonance of voice, or any sound heard over the tumour. He was much relieved by sedatives, and by the application of cold lotion to the tumour. Still it rapidly increased, until it was larger than the fist.

In six weeks' time blood began to ooze from the purple spot upon it, which ceased on the application of mucilage and creosote, which was applied by Mr. Bindley, who saw him at this time. It continued to ooze occasionally, and he became much emaciated, was sometimes troubled with dyspnoea, and was very feeble.

A month later the tumour seemed harder, fluctuation was less marked in it, and the pulsation over it was less liquid, but strong. Soon after this a gush of blood burst forth, and he died.

Inspection.—The tumour was found firmly adherent to the top of the sternum, which was removed with it. The heart was very small, but healthy, as also were the lungs. The inner surface of the aorta was smooth; it was uniformly dilated. At the anterior and upper part of the arch was an oval orifice, the edges of which were smooth and rounded; it was as wide as an half-crown piece in the direction of the long axis, which was from side to side, and opened into an aneurismal sac, nearly spheroidal, the external circumference of which measured twelve inches. A smooth membrane, that seemed continuous with the inner membrane of the aorta, entered the sac for about half an inch all around the orifice, and beyond this layers of red coagulum adhered to the walls. This kind of clot completely filled up the pouch, but a circuitous channel from the aorta to the external ulcerated opening could readily be traced.

The study of such aneurisms as this, springing from the upper part of the arch of the aorta, and rising above the sternum or clavicles, is extremely interesting in a surgical point of view, because they have sometimes been mistaken for aneurisms of the subclavian and carotid arteries; a mistake of the highest moment, since it might be the means of inducing a surgeon to commence an operation which, to say the least, would soon be found impracticable, and might hasten the end of the patient.

A case of this kind, recorded by Burns in his work on the Surgical Anatomy of the Head and Neck, was mistaken for aneurism of the subclavian artery, "by several of the most distinguished practitioners in Edinburgh, and almost every surgeon in Glasgow," some of whom strongly advocated an operation. After death, the aneurism was found to arise from the aorta, and included a large portion of the *arteria innominata*. It mounted considerably above the sternum, pressing in its ascent the descending vena cava to the right, and the trachea to the left side.

A somewhat similar case is recorded by Sir Astley Cooper, who mistook the disease for aneurism of the carotid artery. The tumour had the shape of a Florence flask inverted, and arose by a very narrow neck from the arch of the aorta, between the roots of the left subclavian and the left carotid arteries. It rose up between these vessels, and appeared at the root of the neck, so that it resembled an aneurism of the carotid artery more than one of the aorta.

The aorta in Case 23 not having been submitted to microscopic examination, the existence of atheroma was not proved. The sac might have originated either in a partial dilatation, or rupture of the coats, which were traceable for about the distance of half an inch into it. The aneurism once formed, would rapidly gain the surface above the neck, from the circumstance of the deep-seated fascia which extends from the sternum and clavicles to the lower edge of the thyroid gland, having been partially

destroyed by the abscess which had been formed in boyhood, and the cicatrix of which was seen in the centre of the tumour; and this accords with the history of the case. Before, however, it gained the surface, it must have interfered with the nerves distributed to the shoulders and arms, and thus have produced the pains described, which pressure would be diminished in proportion as a free passage was opened in an upward direction. So also, while yet within the cavity of the thorax, the veins of the head must have been more or less compressed, and thus the violent headaches of which the patient first complained may have been produced. The tumour was at once recognized as a sacculated aneurism of the aorta, being traceable by pulsation a considerable distance behind the sternum with the finger, and being evidently filled with fluid blood. It was some time before cold applications were successful in causing coagulation of blood within the sac. Before death, however, this had taken place to a considerable extent, and the life of the patient might have been protracted for some time, had it not been partly for the attenuation of the integuments by the abscess in his younger days, and partly from the soft nature of the clot, which prevented its adhesion to the sac.

None but the ordinary sounds of the heart were heard over or about the tumour. The heart was small, and its action feeble, and therefore not such as would easily generate an abnormal murmur. Nor was the uniform dilatation of the aorta, which was smooth, likely to give rise to any murmur, although it might have impressed a particular character on one originating in diseased aortic valves. These, however, were healthy.

Coagulation within the sac is further influenced by the force of the circulation, and is consequently favoured by feebleness of the heart's action, but not when such an effect is the result of repeated bleedings, which increase its irritability.

Again, the circulation of blood within the sac is to some

extent influenced by the degree of elasticity of its walls. Thus the thicker and more inelastic they are, the less will they yield to the systole of the heart, and the smaller will be the amount, and the less the force of the current of blood which will enter and leave the sac.

The same result will take place when the sac having been gradually enlarged, meets with and at length becomes united to a firm resisting substance, such as the sternum and ribs, whereby its power of expansion is limited to a considerable extent.

Coagulation is also materially affected by the *quality* of the blood; if it be impoverished and deficient in solid constituents, the clot formed from it will be soft and gelatinous, and little likely to adhere to and strengthen the sac.

So that the most favourable circumstances for preventing a fatal issue by perforation of the sac, are when the blood is tolerably rich in solid constituents, and when a moderate amount of inflammatory action takes place in the walls, which favours the coagulation of the blood, agglutinates the clot to them, and thickens them by the deposition of lymph. It need hardly be mentioned that the smaller the orifice of communication between the aorta and the sac, the greater is the chance of coagulation taking place in it.

Not only is rupture thus delayed for a considerable time, but death is actually in some cases warded off after an aneurismal sac has burst into a cavity of the body, by the orifice becoming plugged up by a portion of the coagulum from within the sac, as in the following:—

CASE 24.—*Sacculated Aneurism of the Descending Aorta—
Death from Rupture into the Trachea.*

A pig-killer and night-constable, æt. 46, had been for some months addicted to spirit-drinking. Seven months since he had a pain in the lower part of the front of the chest. Thirteen weeks ago, having one night drank a great deal of raw brandy, towards morning he was seized with dyspnoea and a partial loss of voice, which has never regained its power, but has remained little more than a whisper. Soon

after this, one night whilst on duty he suddenly fell down, and on coming to himself felt very giddy, and his limbs were numbed, from which he recovered in the course of the next day, but has ever since felt a severe pain between the shoulder-blades shooting up and down the back. A fortnight since he expectorated nearly half a pint of clotted, dark, mottled, coloured blood, which (to use his wife's words) looked as if it came from an old ulcer. The day before he was seen he expectorated a little more of a similar kind.

When visited as a patient of the Birmingham General Dispensary, he complained of great dyspnoea and severe pain between his shoulders. The pulse was natural.

The chest sounded well on percussion. Over the right side of the chest the pulmonary sound was pure but very intense. Not a trace of it could be heard on the left side. The action and sounds of the heart were natural. When the patient spoke, a slight vibration was felt by the hand placed on the right side of the chest, but none on the left. The pain was relieved by the application of leeches.

He was suddenly choked eight days after he was visited at home.

Inspection.—The lungs were quite healthy, as also the heart, except that there was opacity of the lining membrane of the left auricle and some patches of atheroma under it. The inside of the aorta was covered with such patches.

A tumour rather less than a hen's egg was found firmly attached to the commencement of the descending aorta, and also to the trachea just above its bifurcation, and to the left bronchus, which was compressed and flattened by downward and backward pressure. On cutting into it, its walls were found to be thin, and it was seen to be a sacculated aneurism of the aorta, with which it communicated by an opening commencing close to the transverse portion of the arch, and extending one inch downwards, and half an inch in width. Its shape was thus oval. It was filled with layers of solid, tough, discoloured fibrin, and for some distance around the opening into the aorta was a smooth lining membrane apparently continuous with that of the aorta, but the middle coat seemed to stop abruptly. It had ulcerated, and perforation had taken place into the trachea at the point of adhesion just above its bifurcation. Through this opening a piece of firm old coagulum had passed into the trachea. A slight quantity of recently coagulated blood was found in the left bronchus and trachea.

The only physical signs observed in this case were loss of voice, and a complete absence of respiratory sound, with persistence of clearness on percussion over the left side.

evidently caused by the pressure of some small tumour on the left bronchus, which did not compress the trachea to any sensible extent. The blood which the patient had expectorated the day before he was visited having been preserved was found to contain fragments of tough discoloured fibrin. The tumour was, therefore, suspected to be a sacculated aneurism of the descending aorta, which, as in Case 70, communicated with the bronchus or trachea. Unfortunately the connection between the tumour and the recurrent nerve was not investigated. The membrane which lined the sac for a certain distance was, doubtless, the fine false membrane described by Bizot.

Nor does a fatal hemorrhage necessarily at once take place when the parenchyma of the lung itself becomes the boundary of a portion of the sac, because it has been prepared to act as a barrier by the congestion of blood and its coagulation caused by the irritation of the tumour, as in the following :—

CASE 25.—*Mixed Aneurism of the Thoracic Aorta communicating with the Left Lung.*

A boatman, æt. 30, eight months ago was attacked with a dry cough, and pains flying about his chest, more especially his left shoulder; two months ago his cough became violent and convulsive, and he expectorated a viscous fluid, often streaked with blood.

On his admission into the Birmingham General Hospital, his face was tumid and his lips purple. He preferred a sitting to a recumbent posture, as the latter, more especially when he lay on his right side, produced severe headaches. He had great dyspnoea. His cough came on in fits, and resembled the barking of a dog. The expectoration was thick, scanty, and clear. His voice was hoarse. He complained of a dull, aching pain in his chest, shooting backwards. He perspired greatly during the night. His pulse was 72; soft.

A shade of dullness was observed at the left of the top of the sternum. The pulmonary sound was accompanied by cooing and hissing sounds. The impulse of the heart was weak, and the sounds feeble but distinct. Soon after this he took cold, and his bronchitis became much aggravated; this, however, was relieved, and he left the hospital.

A week after this, having exerted himself, he expectorated half a pint of blood, and in another fortnight a pint. He was readmitted, labouring under troublesome cough and dyspnoea. He had lost flesh, and his lips and cheeks appeared to be slightly congested. He complained of violent pains in the left temple. The dyspnoea and cough were both increased by exertion, and also by his assuming the recumbent position. The voice was hoarse and unequal, the cough barking, sometimes followed by palpitations. The expectoration was aqueous and frothy. He had intermittent pain darting from the right scapula through to the front of the chest. The trachea seemed pushed a little towards the right side. On a level with the nipple, the left side measured a little less than the right. A pulsation, but no purring thrill was felt between the cartilages of the first and second left ribs, close to the sternum. In that spot there was dullness on percussion, and a tracheal whistling was heard very close to the ear. The sounds of the heart were rather extended. Soon after this, he stated that he experienced a difficulty in swallowing solids, owing, as he supposed, to some obstacle at the bottom of the neck; but that when one morsel had passed, he could swallow his meal with ease. One day he perceived a tickling in the throat, which was followed by the expectoration of half a pint of blood, at first solid and black, then red and fluid. At this time a greater dullness than usual was perceived on percussing the supra-spinal region of the left scapula. The tracheal whistling was very strongly marked over the dull spot to the left of the top of the sternum, where the sounds of the heart were very audible, the diastolic being louder than the systolic, which was feeble. After another expectoration of two pints of blood, the dullness of sound and pulsation to the left of the top of the sternum were much less marked, but reappeared on his raising himself up. Dullness was more marked behind, where a fine, moist rattle was heard. About this time, it was thought that the pulmonary sound on the left side of the chest was jerking, as if interrupted for a moment every now and then. The diastolic sound of the heart became raspish, between the second and third left costal cartilages, and slightly so above the left nipple; this was never observed before. Great oppression of the chest succeeded, and he died from hemoptysis.

Inspection.—All dullness had disappeared from the front of the chest. The lungs did not collapse, being retained in their position by adhesions, chiefly at their summits and on the left side.

On dissecting down towards the vertebral column, a tumour was perceived, and found to be a dilatation of the descending aorta; which commenced immediately below the origin of the left subclavian artery,

on a level with the upper edge of the third dorsal vertebra, and extended to the upper edge of the sixth. It adhered closely to the vertebræ included between these two points, from a little to the right of the mesian line, to the bases of the left articular processes. On its right side lay the trachea and œsophagus, a little pushed out of their proper places. Its anterior surface was crossed by the left bronchus, and by the recurrent nerve; the former of which was much compressed, and the latter was stretched, but not altered in structure. The sac also adhered closely to the left lung. Its cavity might have contained a moderately-sized pear. The lining membrane was much thickened and puckered up with atheroma. The middle coat was traceable throughout the pouch, with the exception of the place where the bodies of the vertebræ were involved, and also a spot of the size of a half-crown-piece, which was an aperture opening into a cavity in the back part of the substance of the apex of the left lung as large as a chestnut, which was lined by a smooth membrane in some parts; in others the lining was roughened by irregular tufts of coagulated fibrin, and was surrounded by a dense uncrepitating substance. The bodies of the vertebræ implicated were deeply excavated, leaving the cartilages between them projecting into the pouch; they were, however, covered by a smooth membrane which appeared in all respects as if it were a continuation of the lining membrane of the aorta. Below the dilatation the coats of the aorta were much thickened and diseased. The heart was very small and pale. On cutting into the left lung a great portion of its apex around the cavity above mentioned was carnified, and contained some small groups of grey granulations. Immediately around the cavity was a mass of laminated fibrin, one inch thick in the middle, but tapering off on all sides towards the edges of the opening into the aorta.

Several bronchial tubes were traced down from the trachea, and were found to open on the fibrinous clot, their lining membrane being stained of a purple red colour as they approached it. In one spot a secondary branch was dilated into a cavity of the size and figure of an almond without its shell.

The abdominal viscera were healthy.

There was abundant evidence of the existence of a tumour compressing the trachea, œsophagus, and recurrent nerve, and in contact with the aorta; and it was further probable that it was connected with the left lung and the spine. Aneurism was of course suspected, but not predicted with confidence. Further consideration has con-

vinced me that the diminution of the extent of dullness on percussion after copious hemoptysis, and which was observed more than once, could only be accounted for on the supposition that the tumour was an aneurism imbedded in the left lung.

Hodgson has shown that when perforation takes place through the integuments or into a cavity lined by a *mucous* membrane, such as the œsophagus or trachea, it is preceded by *sloughing ulceration*, whereas when it takes place into a cavity lined by a *serous* membrane such as the pleura or pericardium, it is formed by *laceration*.

Compression of the neighbouring parts depends very much on the situation of the tumour, and the point from which it springs. Thus aneurisms which arise either from that portion of the aorta which is within the sac of the pericardium, or from that which lies between the left bronchus and the diaphragm, usually run their course without materially affecting the neighbouring organs. Those which spring from the root of the aorta seldom attain a size larger than an egg before they burst into the pericardium, in consequence of that membrane, which forms the outer coat of the vessel in this spot, being very thin and inextensible, as in case 71. A case is recorded in the Dublin Journal, in which an aneurism in this situation had attained a considerable size, but this is quite an exceptional case. Those observed by Cooper, Morgagni, and others, besides myself, have never exceeded the size mentioned, and therefore may be considered too small to allow of their pressing injuriously on neighbouring parts. When they occur at the other extremity of the thoracic aorta, below the left bronchus, they sometimes attain a very large size; but in that situation they only encounter the bases of the lungs, the apex of the heart, and the œsophagus, parts which allow of considerable displacement without fatal consequences.

The intermediate portion of the aorta, however, is surrounded by many important parts, so that aneurisms arising from it can hardly fail to interfere with the func-

tions of some of them. Those that spring from the *anterior* side of the vessel can attain a tolerable size before they reach the ribs and cartilages, whilst a very slight dilatation, or a small sac from the *posterior* side speedily interferes with the trachea. In some cases this effect varies with the position of the patient, before the tumour has become fixed to the ribs, and whilst it retains a certain amount of mobility. Thus a remarkable case (72) occurred, in which a mixed aneurism springing from the upper right angle of the aorta, and involving the orifice of the arteria innominata, was in front of a large branch of the right bronchus, and fell back on it when the patient was in an upright or recumbent position, and stopped the ingress of the air to the upper third of the right lung; but gravitated away from it and set it free when he bent forwards. Here death resulted from excessive irritation, and the exhaustion produced by incessant coughing. Hence it is that small aneurisms tied down to an organ so irritable as the trachea, produce more distress than larger ones, which are free to spread in other directions, as has been noted by Dr. Law. The œsophagus being more yielding, pressure against it does not give rise to so much uneasiness. When the recurrent nerve is involved, the voice is in some cases weakened very much; in case 25 it was only rendered hoarse, although the nerve was much stretched.

Sometimes the attention is first directed to the seat of disease, by the appearance of œdema of the head, neck, and arms, indicating pressure on the *vena cava*, the trunk which receives the veins coming from these parts. It is curious that as the disease advances, the œdema in some cases subsides; this depends on the state in which the walls of the pouch happen to be; when they are thin and yielding, no pressure is exerted, and œdema does not take place; when thick and unyielding, œdema takes place and persists. When, however, the walls are in an intermediate state, the vessel is compressed, and œdema is produced; but after a time the engorgement of the veins exerts a pressure in its

turn on the coats of the tumour, which yield, and a groove is made in which the cava is lodged, and the œdema gradually passes off. This was well shown in case 105, and in

CASE 26.—*Dilated Aneurism of the Arch of the Aorta, compressing the superior Vena Cava.*

A bargeman, æt. 40, of a spare habit of body, and addicted to hard drinking, had been asthmatic, and had at different times felt palpitation of the heart for the last ten years. He was intoxicated for a week, during which time his head and neck swelled, but more particularly during the last night of the week, and he awoke early in the morning with great stiffness and pain in his throat, and in the epigastric region. His head, neck, and arms were found to be amazingly swelled, and his face was almost black.

When visited as a patient of the Birmingham General Dispensary, his head, neck, arms, and the upper part of his chest were greatly swelled, of a purple colour, and pitted on pressure. His eyes were injected and watery. He suffered much from dyspnœa. The pulse was feeble and resilient. The action of the heart was strong and heaving.

The natural sounds of the heart were distinctly heard in the precordial region. The chest sounded rather dull on percussion throughout, owing to the œdema of its walls; but no one part sounded duller than another. The breathing was rather stridulous, even when heard at some little distance off. The respiratory sound was natural, but slightly modified by the stridulous breathing. About four inches from the top of the sternum, and one inch to the right of it, an extremely hollow and rather prolonged systolic rasp-sound was heard, and was quickly lost on the ear receding from this point. Ineffectual attempts were made to bleed him in both arms. Erysipelas came on in each arm around the incision. In a few days the œdema of the head, neck, and arms was much diminished, but the erysipelas progressed, and he died.

Inspection.—The tumefaction of the head, neck, and arms had nearly disappeared. The ravages of the erysipelas were confined to the subcutaneous cellular tissue. The lungs were healthy. The heart was firm, red, and hypertrophied, being half as large again as the fist; but the size of its cavities was but slightly increased, nor were its valves diseased.

A tumour occupied the upper part of the mediastinum, and projected to the right of the sternum. This was the arch of the aorta dilated into a pouch capable of containing a small orange. The arteria innominata was also slightly dilated at its origin, like a funnel. The

coats of the aorta could be traced throughout every part of the pouch but one, and this was a small spot at the back of it, where a tough old fibrinous clot adhered to a bronchial gland without the intervention of any of the arterial coats. A more recently formed clot adhered to the summit of the pouch, and sent off branches into the arteria innominata and left carotid artery. This pouch had overlapped the superior vena cava, which adhered closely to its posterior surface, lying in a shallow groove. By this means the vein was flattened, and contained a firm, flat, and somewhat discoloured clot. The left subclavian vein was twisted, its insertion into the vena cava being drawn down under the pouch.

The existence of a tumour compressing the superior vena cava and the trachea was at once apparent. A hollow rasp sound being then found confined to the spot where such a tumour might be supposed to lie, sufficed to reveal the nature of the tumour, and to determine its being an aneurism of the arch of the aorta. The hollow character of the sound rendered it probable that the aneurism was the result of dilatation, a supposition which was to a certain extent confirmed by the resilient character of the pulse.

Sometimes an aneurismal pouch commencing with dilatation, attains such a size, that it encroaches on the heart and impedes its action; at the same time the heart has to force the current of blood through the distended pouch, and consequently its action may be suspended, and fatal syncope result.

When the pouch reaches the vertebral column, or the ribs and sternum, the cartilage with which it comes in contact being elastic, is not easily disintegrated; and being but scantily supplied with vessels, is little susceptible of absorption, and therefore remains for the most part uninjured; but the bone of the vertebræ, ribs, and sternum is more or less destroyed, partly by absorption caused by pressure, and partly by attrition. Perforation of the walls by sloughing may then take place, or another pouch may be formed by the blood insinuating itself between the ribs and the skin, as in Case 62.

CHAPTER III.

CAUSES OF DISEASES OF THE THORACIC AORTA.

ARTERIAL COATS.—*Sex—Age—Rheumatic Diathesis—Gouty Diathesis—Uric Acid—Mal-assimilation.* ANEURISM.—*Diminution of Elasticity—Atheromatous Degeneration—Inflammation—Passion—Violent Exertion—Age—Sex.*

ARTERIAL COATS.—It matters little whether *horny deposit* be considered to arise from a morbid secretion from the internal coat of the artery, acted on by unhealthy blood, or from a deposition of fibrin from the blood itself. In either case the cause must be looked for in the state of the blood, and not in that of the artery.

So also *atheromatous degeneration*, and *calcification*, occurring as they do all through the arterial system, must have a *constitutional* rather than a *local* origin.

Sex, it would appear from the researches of Bizot and others, does not exert any influence either way.

Age, however, has a marked influence; one and all of these three affections increasing in frequency and extent with the age of the subject. Hence the constitutional taint cannot partake of the *tubercular* character, which is at its maximum in youth, and decreases as age advances. The *cancerous*, *rheumatic*, and *gouty diatheses* all become more developed with age, and so far fulfil the conditions required. No connection, however, has been traced between these affections and diseases of the arterial coats.

It will be seen further on that *horny deposit* is of the same

nature as the fibrous growths from the valves of the heart, and is closely allied to the rheumatic diathesis. In accordance with this view it may be mentioned that Bizot has shown that the younger the subject, the more acute is the attack in which gelatinous matter, the earlier form of horny deposit, is thrown out; just as in youth attacks of acute rheumatism are more common than in advanced age.

But the rheumatic diathesis does not seem to have any influence in producing *atheromatous degeneration*. Dr. George Johnson has found this affection very frequently associated with fatty degeneration both of the kidneys and the liver; and considers all three to be related to each other, as joint effects of one common constitutional cause. What is this cause? Andral and Lobstein consider it to be identical with that of gout; and Dr. George Johnson finds gout to be frequently associated with this state of the arteries. Becquerel and Rodier have found cholesterine, one of the constituents of atheromatous degeneration, to exist in the blood in much larger quantities after forty years of age, than at earlier periods of life; upon which Vogel remarks that "the augmentation of this substance in the blood is probably connected with an increased separation of it in the various parts of the body; and that a similar increase of it may take place in young persons in consequence of a morbid process." Hence the exceptional occurrence of atheromatous degeneration in youth. The remarkable fact that atheromatous degeneration is in a great measure confined to the arteries springing from the left side of the heart, containing arterial blood, except when the two bloods are mixed in the right side of the heart by an abnormal communication between the two sides, would, it might be thought, throw great light on the origin of the evil. But chemists have failed in discovering any difference between the blood of the aorta and that of the vena cava, sufficiently marked to account for this fact, although a decided difference has been found between the blood of the renal artery and vein, and between that of the portal

and hepatic veins. But it by no means follows that this should be so in *disease*. Simon* gives reasons for believing that "*urea, uric acid, and bilin* are formed as a consequence of the consumption of blood corpuscles; that they must necessarily be formed as products of the changes which the constituents of the blood undergo in the circulation, and are not (as observations on starved and emaciated individuals show) a consequence of the changes which the circulating fluid undergoes during the nutrition of the tissues, but are dependent on the metamorphic action that is produced by the respiratory process." Dr. Garrod has also found uric acid to exist in small quantities in healthy blood. If, therefore, in gout a large quantity of uric acid exists in the arterial blood, is separated by the kidneys, and passes off in the urine, it follows that in such cases the arterial blood must be much more highly charged with it than the venous blood. Hence arises a palpable difference between the two, and herein is a strong argument in favour of the view that atheromatous degeneration takes place under the influence of the gouty diathesis.

The tendency to *calcification* would seem to depend upon a low state of the system generally, whether arising from mal-assimilation or the progress of age, rather than upon any special diathesis; for it takes place in every part of the body, showing itself more particularly in the midst of previous morbid deposition or degeneration, as in false-membrane, atheromatous degeneration, tubercular masses, &c., &c.

It is probable therefore that *horny deposit* on the inner coat of arteries takes place in some measure under the influence of the *rheumatic diathesis*, and that *atheromatous degeneration* of the coats, in common with some not dissimilar forms of disease, is more or less under the influence of the *gouty diathesis*.

This, however, does not bring us to the root of the

* Simon's Animal Chemistry; translated by Dr. Day; vol. i., p. 219.

matter, or divulge the essence of these constitutional disturbances. We know it is true that rheumatism is characterised by an excess of *lactic* or some nearly related *acid*, and gout by that of *uric acid* in the blood, and thus we have advanced a step, and that by no means an unimportant one, as regards treatment; but of the causes or exact nature of the metamorphic changes which give rise to these products in excess we know absolutely nothing; no more than we do of the nature of those subtle and invisible poisons which produce small-pox, measles, typhus, scarlatina, &c.

There is every reason, however, for believing that the abnormal metamorphic changes which lead to these morbid states of the blood are in a great measure favoured, if not induced, by imperfect nutrition and mal-assimilation, causes of which may be found in faulty diet, whether too high or too low, including spirit-drinking and its accompanying evils, want of good air and exercise, mental depression, over-work of the brain and nervous system, &c.

But in considering the causes of such structural changes as the above, we must not pass over the numerous facts which seem to justify the conclusion that they are to a great extent due to some peculiarities of constitution, which are inherent, and which may be derived from one or other parent. It would appear that in certain individuals the yellow elastic tissue had retained its integrity up to a certain period of life, but that at length it underwent changes resulting in degeneration, loss of elasticity, and complete alteration in structure. These alterations are in their essential nature very similar to those which take place, so to say, naturally in the same tissue in old age; and there are not wanting examples of arteries taken from men of 35 which closely resemble corresponding vessels taken from patients of very advanced age—vessels which have, in fact, grown old before their time. This premature old age probably depends, at least in many instances, upon the development of an abnormal or not perfectly healthy tissue

in the first instance, which in its turn may be referred partly to the effects of improper pabulum, but mainly to imperfect action of those forces or powers which are directly instrumental in the production and development of the tissue.

ANEURISM.—Dilatation or perforation of the aorta can arise only from very great increase in the force with which the blood is impelled into it from the heart, or from a diminution of elasticity or loss of substance of its inner coats. The middle coat, however, is so strong, tough, and elastic that it is impossible to conceive that its permanent dilatation or rupture can be produced by any effort of the heart, *as long as the vessel continues in a healthy state*. Accordingly we have seen that neither excessive hypertrophy of the left ventricle, nor contraction of the aorta at particular points, will produce such effects. Two remarkable cases (19 and 20) have been adduced where the inner and middle coats were ruptured by violence, and the blood was effused under the outer coat, which was uninjured; but these are exceptional cases; and even had such a blow not been fatal, it is probable that congluable lymph would have been thrown out, and would have repaired the rent before aneurism had been produced, in accordance with the experiments of Hodgson and Dr. Jones.

Diminution of elasticity and loss of substance therefore alone remain as the proximate causes of aneurism. These can only result from such a morbid alteration of the arterial tissues as shall lead to a partial or total displacement of their proper substance by matter possessing less of elasticity and cohesion. Consequently alterations of this kind are invariably found in aneurism, resulting from *atheromatous degeneration* and *calcareous deposits*.

Horny deposits, when they reach an advanced stage, may cause the destruction of the inner coat; but as they do not invade the middle coat, they cannot be considered materially to diminish the elasticity of the artery, unless in conjunction with some other morbid change. Accordingly

they are not found in *all* cases of aneurism. *Atheromatous degeneration* must therefore be considered to be the chief *proximate cause* of aneurism.

When this has once commenced, its progress may be modified by a variety of circumstances tending to hasten or retard the rupture or dilatation of the artery. Much must of course depend on the amount of constitutional derangement, but something also on the *quantity* as well as the *quality* of the general nutrient fluid, and the consequent moisture or dryness of the exudation. This must bear some proportion to the state of the system generally, and to the activity of the circulation. Moisture of the exudation would tend to produce softening and ulceration, whilst dryness would probably give rise to a cheesy state and calcification. It is possible that a mixed plasma may be thrown out, as was supposed by Vogel,* who remarks:—"In this exudation two formative processes are simultaneously going on, an organisation of the fibrin and the formation of concretions, generally of earthy salts. The product of this formation consists chemically of two distinct steps, one relating to the conversion of the fibrin and its modifications into areolar tissue, granular cells, typhic deposits, and tubercular matter; the other to the constituents of concretions, as the salts of lime and magnesia, the urates, fat, &c. The individual constituents of either group may assume a vicarious position; moreover, the whole of the first group may assume the place of the second, and conversely the second may replace the first, so that the one is subordinate in the same degree as the other predominates." When therefore rapid alteration takes place, the former process takes the lead, and when the exudation remains dry, or is succeeded by calcareous deposit, the latter predominates.

Other causes also may concur with atheromatous degeneration to favour dilatation and rupture. *Inflammation* when it reaches the middle coat, may possibly diminish the

* Dr. Day's Translation, p. 387.

elasticity and cohesion of its fibres, but, when occurring in arteries, is generally attended with effusion of plastic lymph, as has been proved by the experiments of Dr. Jones and Mr. Hodgson before alluded to. Whilst therefore it might on the one hand diminish the elasticity of the middle coat to a certain extent, the tendency to dilatation or rupture would on the other hand be counteracted by the effusion of lymph between the outer and middle coats, binding them together and strengthening them.

Again, the force with which the blood is propelled into the aorta may be increased by nervous palpitations under the influence of the *passions* (as in Case 62), or from sympathy with other diseased organs, or else by excessive hypertrophy of the left ventricle. A violent shock might also cause the rupture of a vessel previously diseased, and probably did so in Cases 62 and 103.

Such are some of the causes which may be supposed to favour the formation of aneurism when atheromatous degeneration has taken place in an artery. The influence of two further causes has been ascertained by actual observation. These are *age* and *sex*.

Age.—An examination of cases published by different writers in conjunction with those here recorded, proves that aneurism especially prevails during the ages of forty and fifty, whilst it has been seen that the atheromatous diathesis is seldom marked under forty years, but increases with age afterwards. It would appear therefore that above fifty aneurism decreases and atheromatous degeneration increases. The following observations may perhaps tend to explain how this occurs. Assuming atheromatous degeneration to be the constant proximate cause of aneurism, its rarity under 40 accounts for the rarity of aneurism under that age. When, however, it has once commenced, the younger the subject the greater would be the tendency to the formation of aneurism. For this depends not so much on the extent of atheromatous degeneration as on its tendency to soften. Now the younger the subject the more active

would be the circulation, the more moist the exudation, and the greater the tendency to softening and ulceration. In old age the reverse of this takes place; the circulation is retarded, the quantity of nutrient fluid is probably diminished, and the atheromatous patches are dry, or are soon invaded by calcareous deposition. By the same law the softening of tubercular matter takes place with less and less rapidity as age advances, so that the older the subject the slower is the march of the disease. Again, the nerves are more excitable and the muscular power is greater in earlier life than in old age; hence nervous palpitations are more easily excited, and the action of the heart is more energetic during the former period of life. More violent exertions are usually engaged in, and falls and shocks of different kinds are of more common occurrence than in later life. On the other hand, however, Bizot has shewn that the size and thickness of the heart gradually increase up to extreme old age, but not probably in a greater degree than is necessary to compensate for the diminished elasticity in the arterial system.

Sex.—The influence of sex is most marked, aneurism only occurring in one female to nine males or thereabouts. Now as the amount of atheromatous degeneration has been found to be much the same in the two sexes, it follows that the only difference which can be supposed to have any influence on the formation of aneurism, is that which exists in respect to habits of life and modes of employment. In this point of view it would be very interesting to know the ratio in which aneurism is found in the two sexes in those countries where their modes of life are in one important respect assimilated by the women engaging in laborious employment. The effects of violent exertion in the production of this disease, when the vessels are predisposed to it, must be considerable, and may be in some degree estimated by the fact ascertained by Bizot, that the popliteal artery, although very little invaded by atheroma in proportion to some other arteries,

is far more frequently the seat of aneurism than other vessels.

We are not in possession of sufficient data to enable us to institute a comparison between the effects of different modes of employment. Some writers have hazarded generalisations on this point, but they evidently rest on very slender foundations.

The causes on which *atheromatous degeneration* depend have already been investigated, and have been seen to have their origin in a peculiar state of the blood, very similar to that which is found in the gouty diathesis. This state of the blood, therefore, may be considered one of the chief *remote* causes of aneurism. Rokitansky states that out of 108 cases of aneurism, tubercle existed in five cases only, restricted to a small portion of the lung, and was either undergoing the process of retrogression, or was altogether extinct. He traces an affinity between aneurism and cancer. This has not fallen under observation in these cases; possibly if research had extended to the smaller arteries, the result might have been different.

CHAPTER IV.

NATURE OF DISEASES OF THE PERICARDIUM.

CHANGES IN THE PERICARDIUM.—*Colour — Consistence — Milky Patches.* EFFUSION IN ITS CAVITY.—*Serous — Plastic — Gelatiniform — Tubercle — Cancer.*

The morbid changes connected with the pericardium are more or less the result of inflammatory action (pericarditis), the effect of which is to produce an alteration in the *membrane* itself, and *effusion* of variable nature within its cavity.

CHANGES IN THE PERICARDIUM.—When the pericardium is examined after death arising from a violent attack of pericarditis, particularly when combined with pleuritis, and when lymph is found, not only is a bluish redness observed in larger or smaller streaks and patches on the surface, but it is distinctly seen to be injected, even when the attack has been protracted many days; an appearance not to be confounded with the more uniform staining produced by cadaveric imbibition, or blood within the cavity. In other cases, generally of longer standing, in which serum or sero-purulent fluid is found in the cavity, no traces of injection are discoverable; but the pericardium is, on the contrary, paler than usual, is no longer bright and glistening, but opalescent; not unfrequently it has become soft and pulpy, being easily torn, and sometimes raised here and there from the subjacent tissue by serum.

In some chronic cases of long standing, the membrane is even considerably thickened and hardened, and rarely calcareous deposition has taken place in it, so as to make it almost of bony consistence. All these changes may occur throughout the whole surface, or merely in isolated patches.

Thus partial thickening occurs, and presents itself under the appearance of *milky patches*, which are found to penetrate, and are inseparable from the membrane itself; occurring usually towards the close of life, and generally formed on the right side of the heart only. The common form of milky patch, however, varies in size from a pin's head to a shilling, or larger; at one time following the tract of the coronary vessels like a string of beads, and at another appearing in irregularly rounded patches; sometimes separable from the membrane by proper manipulation; being, in fact, fibrinous exudation; but more commonly inseparable from, and forming a portion of the fibrous pericardium. These white patches in many cases seem to result from exudation into the pericardial membrane, and the subsequent conversion of the exuded matter into a very hard, firm, fibrous tissue.

EFFUSION WITHIN THE PERICARDIUM.—In a practical point of view, the nature of the effusion that takes place in pericarditis, is much more important than the state of the membrane itself. Like that from the pleura, it has a twofold character, being *serous* and *plastic*. It is seldom, however, that either form of effusion occurs without a trace at least of the other. In the simple form of serous effusion, a pale yellowish or reddish serum is poured out. Sometimes it is limpid, but in such cases some flocculi of yellow lymph are seen, or else very delicate films are here and there spread over the pericardium; and at other times a transparent jelly is found gravitating towards the lowest part of the cavity. Oftener, however, the serum is clouded by holding some of the lymph in solution or suspension.

In some cases, to which Laennec has given the name of hemorrhagic pericarditis, blood is mixed with the serum, and this usually occurs when the system has been reduced to a low asthenic state, as in typhus, &c. In other cases genuine pus is found mixed with the serum.

Plastic effusion occurs in two forms—the one *gelatiniform*, the other of *solid*, but soft consistence.

Gelatiniform effusion is variable in colour, being yellow, red, or even brown, and is clear and limpid. It is highly organizable, and for this reason, being rapidly filled with blood-vessels, it soon forms a connection, partial or general, between the visceral and parietal layers of the pericardium. This form of plastic effusion is usually found in the milder attacks of inflammation affecting the pericardium and the pleura, and is revealed by death occurring from some other cause, such, for instance, as occurred to Hope, when the patient died from an attack of apoplexy during his convalescence; and it was seen in a lad who died after serious injury to the lower extremities, and in whom the only symptom of mischief about the heart had been a very slight pain in the precordial region, described by himself as a stitch. It was seen also in Cases 84 and 42. The similarity between this effusion and that observed by Bizot as the forerunner of horny deposit on the lining membrane of the arteries, is striking.

The *solid* form of *plastic effusion*, soft and yellowish, is found, for the most part, in concentric layers over parts or the whole of the pericardium, usually with a rough, uneven surface, often resembling a honeycomb, or a piece of tripe or sponge; at other times hanging in flocculent shreds or masses in much serous effusion. It is mostly found after violent inflammation, particularly in connection with that of the neighbouring organs, as occurs in several forms of acute rheumatism. In smaller amount, and confined to isolated spots, it is found in attacks of a milder character, which arise in the course of rheumatism and renal disease.

No blood-vessels or distinct traces of organization are to

be discovered in this form of plastic effusion, or rather at this stage of the process ; but it must not be inferred from this that it does not become organized, because the cases in which it is observed are for the most part those in which the vital powers have been suddenly so reduced that death has resulted before there has been time for the organization of the lymph.

It is quite certain that in cases in which the metamorphosis of tissue is more active, and the patients' strength is sustained, these fibrinous effusions, if excessive, are converted into areolar tissue, while if very small in quantity, they gradually undergo disintegration without previous organization, and are dissolved in the surrounding serum and absorbed. Clinical observation would incline us to take the view that the latter favourable result is not uncommon in many instances. Cases have come under treatment where the auscultatory signs have clearly indicated the existence of such exudations on both layers of the pericardium. Such signs, after a certain time, have disappeared, and a moderate amount of health has been regained, or complete recovery taken place ; thus leading to the inference that either the lymph had been absorbed, or, at the worst, that a few unimportant adhesions had occurred.

Hope and others suppose that both forms of effusion have a common origin, a liquid secretion from which the solid is formed by coagulation. There are strong reasons, however, for believing that the quality of the effusion varies with the degree of the inflammatory action that gives rise to it, and with the state of the system. Thus the essential difference between the highly organizable gelatinous effusion, and the so-called solid non-organizable plastic lymph, and again between the clear serous or sero-gelatinous effusion in low chronic pleuritis and the flaky sero-plastic matter found when the attack has commenced in the acute sthenic form, alike point to a difference in the system, and in the degree of inflammatory action as exist-

ing at the time of the effusion, and impressing each with its distinctive character. Further than this, it may be inferred that after effusion has taken place, it may be modified, and its condition influenced by the state of the system; just as it has been shown elsewhere in respect to grey matter deposited in the air-cells of the lungs, which has been seen to rise in the scale of organisation and approach to the nature of areolar tissue, or to descend in the scale and become granular and tubercular, according as the metamorphosis of tissue has increased or decreased with the general health.

Accordingly, although the greater number of cases of pericarditis terminate in absorption of the effusion, or in the establishment of adhesions, partial or general, a certain number pass into a chronic state, in which false membranous bands, more or less perfect, are found, and in them, according to the state of the system, various morbid deposits take place. Thus says Hope (p. 117): "Either a turbid grey liquor, more or less thin, is at the outset discharged into the sac, whilst the free surfaces of the pericardium become the seat of plastic deposit, that is to say, of an aggregate of grayish white or faint yellowish, flat, and tolerably firm granules, denoting, both in number and character, their tubercular character; or else a number of greyish white granules, as large as a pin's head, congregate in the midst of plastic effusion, go on accumulating, and whilst the organizable portion of the effused matter is effecting an adhesion between the heart and pericardium, form into greater or smaller masses of tubercle, which, after absorption of the fluid parts, may assume a mealy consistence. In all such cases, tubercles of old or recent date are found in other organs." This, of course, takes place under the influence of tuberculosis of the system, which thus modifies the effused matter and impresses its own character upon it.

So, again, when the cancerous diathesis is developed, medullary fungus is found in the effusion. Kollestcha de-

scribes "the transformation as a gradual one, and at the same period that one portion of the exudation is already in the condition of medullary cancer, an adjacent one is still engaged in the act of transition from the plastic exudation to the fungoid form, whilst a further portion contiguous to the second betrays no such evidence of degeneration. During the act of medullary transition, the plastic mass becomes paler, white, or light-grey, possesses delicate blood-vessels, and acquires a cellular structure, the walls becoming filled with medullary substance."

CHAPTER V.

PROGRESS AND TERMINATION OF DISEASES OF THE PERICARDIUM.

Resolution and Absorption—General Adhesion—Partial Adhesion—Duration—False Membranous and Non-organisable Products—Fatality.

The TERMINATIONS of pericarditis are three-fold. By *resolution or absorption*. By *adhesion*. By *formation of false membrane and unorganizable products*.

1. *Resolution and Absorption*.—It is seldom, if ever, that the effect of a mild attack of inflammation can be seen on the pericardium at the time, but it is unquestionable that even a considerable amount may take place and be speedily resolved, with the restoration of tolerable health; for it is no uncommon occurrence to find the pericardium thickened and opaque, showing that it had been much inflamed, when the patient has died of another affection—as in Cases 93, 46, and 48. *A fortiori* it may be assumed that in milder attacks, complete resolution may take place without leaving any marked traces behind it. That fluid is often absorbed, we know, as well as if we could see the interior of the sac, for we can by percussion frequently trace its disappearance, as well as its appearance, when we are on the look-out for it. So, too, we can detect the presence of lymph by the friction sound to which it gives rise; but when that sound has dis-

appeared it is difficult to say whether this is the result of absorption, or of the adhesion of the two opposite pericardial surfaces. Post-mortem examinations, however, shew that it is no uncommon occurrence for patches of lymph to be, in a great measure, absorbed or formed into innocuous false membrane (milky patches), closely attached to the pericardium—Cases 93, 89, 46, 95.

Although a fatal issue is thus prevented, and the health is more or less apparently re-established, it is probable that the seeds of future disease are sown in some of these cases. Inspection of the body proves that not unfrequently the endo-cardium has been affected at the same time, and hence we may be well assured that the muscular walls of the heart, placed between these two inflamed membranes, could not have escaped free. Traces of inflammation may not be found in them, but we can hardly conceive it possible that their tone and contractile power should not have been in some degree and for a time impaired. The first effect of the proximity of the inflamed tissues would doubtless be to irritate the walls and increase the force of their muscular contractions, and hence the palpitations and irregularity of the action of the heart so often witnessed. This, however, would soon be followed by re-action, and decrease of power from impaired vitality. The effect thus produced on muscular fibre may be seen in a marked degree on the intercostals and diaphragm after pleuritis. The muscle is, in fact, paralysed, and its contractility is impaired; as may also be seen when the muscular coat of the intestines is affected by its proximity to an inflamed peritoneum, in consequence of which the intestine became greatly distended.

This state of things must, in a similar manner, favour the *dilatation* of the cavities of the heart. It will be seen in the sequel that this is one of the most serious accidents that can happen, and that it can very often be traced to an attack of pericarditis as its starting point, as in the following case :

CASE 27.—*Pericarditis—Mitral Contraction—Tricuspid Dilatation—Dropsy.*

An iron-caster, æt. 26, had an attack of rheumatism between six and seven years ago, from which he quite recovered. Eighteen months since he first expectorated blood, after carrying a heavy load, and has occasionally done so ever since, under similar circumstances. He has been in the habit of drinking much ale. Sixteen months since he caught cold, and suffered from severe pains about his left nipple, cough, dyspnœa, and palpitation. These symptoms have more or less persisted, being all aggravated by exertion.

On admission into the Birmingham General Hospital he was very pale; his feet and ankles were slightly œdematous, the tongue was of a natural colour, rather excoriated in spots. His pulse was 104, small, and quick. He complained of great thirst, and pain under the left nipple shooting backwards, occasionally with much severity. He had cough, with expectoration of clear mucus, and occasional vomiting. Palpitation and dyspnœa were both distressing on exertion.

A pulsation was seen to the left of the xiphoid cartilage, and a purring thrill was felt below the left nipple, and there alone; and the veins of the neck were turgid. The diastolic sound of the heart was strong and sharp. A double rubbing sound was heard below the left nipple, diminishing in intensity with the distance from this point in every direction. He improved slightly for nearly a month, under tonic and expectorant medicines, and anodyne frictions over the heart; when the cardiac pain became rather suddenly aggravated, and with it dyspnœa, palpitation, and cough. The legs and scrotum became rapidly anasarcaous. The pulse soon reached 140, and was irregular. The veins of the neck undulated. The expression of the countenance became most anxious. The action of the heart was fluttering and tumultuous. Its sounds were marked by a double rubbing sound close to the ear. The precordial dullness was extended. Mercury was actively rubbed in over the heart. But, in spite of all treatment, he sank in a week's time.

Inspection.—The right lung adhered firmly to the chest. Both lungs were engorged, but healthy in structure. The outer surface of the pericardium was congested and thickened, and on it were several patches of rough lymph. It contained 12 oz. of straw-coloured serum. Its internal surface was highly injected, and its posterior portion adhered to the heart, and on the other part there were rough patches of lymph, and also on the corresponding parts of the heart.

The heart was enlarged by dilatation of its right side, which con-

tained a large coagulum. The tricuspid orifice was six inches in circumference, the valves were thickened. The mitral ring was cartilaginous and calcareous, as also were the valves, so that the point of the little finger could not pass through it. The calibre of the aorta was small. The coronary vessels were dilated.

The liver was large and congested. The kidneys were also congested, and appeared slightly granular.

Two distinct attacks of pericarditis may be clearly traced in this case, the second of which hurried on death, both by directly interfering with the heart's action, and also by producing softening of its walls, and thereby favouring their dilatation. For by this means regurgitation through the tricuspid orifice, which must already have taken place from the corrugation of its valves, would be greatly increased, the circumference of the aperture being six inches.

The progress of the disease is well marked; at first the symptoms of pulmonary congestion appearing in consequence of mitral obstruction, and then those of impeded general circulation, as the right side became dilated, and with it the tricuspid orifice.

Adhesion may be general or partial. In the former case it has usually been seen to take place rather rapidly, although the adhesion may not become intimate for some time, and it has been formed by the organizable gelatiniform fluid described above. In cases 42 and 84 this was seen; where nature was, as it were, surprised at her work, whilst adhesion was yet incomplete. This gelatiniform highly-organizable fluid can hardly be considered a pure product of inflammation, although it may be caused by its presence, and may take place under its influence. It is rather a peculiar form of fibrinous dropsy, the fibrin of the blood being separated from it, partly in consequence of the proximity of an inflamed membrane, and partly from its existing in excess in the blood. It is possible that the large amount of lactic, or some similar acid so frequently found in these cases, may have something to do with the result.

When the *adhesion* is only *partial*, the opposite pericardial surfaces become either closely adherent to each other in *patches*, or else are joined by bands of connecting cellular tissue. In this case the exudation is usually sero-plastic. The patches of lymph deposited on the opposite surfaces are kept asunder for some time by the intervening serum; at length by its absorption they are enabled to approach each other, and either become firmly and closely adherent, or else by the action of the heart are drawn out into gradually elongating bands, just as occurs between the two surfaces of the pleura.

Adhesion not only thus takes place in the interior of the pericardial sac, but the exterior of the membrane sometimes becomes attached to the contiguous parts, such as the pulmonary or thoracic pleura, either closely or by connecting bands, as in Case 28.

Such is the *progress* of adhesion; much difference of opinion exists in regard to its *terminations*. Some authors have maintained that its injurious effects on the health are inappreciable; others, on the contrary, that it invariably leads to hypertrophy and dilatation of the heart, and sooner or later produces fatal results. Implicit assent cannot be given to either of these views. No cases have come under notice in which a post-mortem examination revealed the existence of adhesion of long standing, without their history having afforded a great probability that some disturbance of the health had resulted from it; but it by no means follows that because the health has been notably disturbed, a fatal issue should necessarily take place.

As regards the effects of adhesion in producing hypertrophy and dilatation, although in Case 28 the heart retained its natural size, yet in almost every other case considerable *dilatation* was found, but in very few *hypertrophy*. If, indeed, the authors in question intended to denote *hypertrophy by extent*, then their accuracy could not be called in question; but no increase in the thickness of the walls of auricles or ventricles was generally observed. It would,

indeed, be extraordinary if dilatation were not a sequence of adhesion, seeing that it had been observed to follow pericardial inflammation of a milder type than that which gives rise to adhesion; and as it very frequently leads to fatal results, as will be seen in the sequel, adhesion may also be held *indirectly* to do so. The following case, however, proves that it may exist a considerable time without destroying life:—

CASE 28.—*Adherent Pericardium—Mitral Obstruction.*

A dressmaker, æt. 29, single, had rheumatic fever, attended with violent pain in the left side, at the age of 17, and ever since that time has had palpitations and dyspnœa, which, on exerting herself, were most urgent and distressing. She was also subject to a teasing cough, with clear mucous expectoration.

Outside the left nipple, between the seventh and eighth ribs, undulations were seen, corresponding with the impulse of the heart. Some cooing and bass-viol sounds, with traces of fine muco-crepitating rattles, were detected in different parts of the chest. A shrill rasp-sound was heard with the systole of the heart; being at its maximum under the left nipple, and at the angle of the left scapula. She was relieved by sedative and expectorant medicines, and by belladonna frictions over the heart. Soon afterwards she had an attack of left pleurisy,—this was at the age of 27. Two years after this she was suddenly seized with a violent pain under the left nipple. Her pulse was sharp, small, and frequent, 120. The eyes and face were injected.

There was no dullness on percussion. The respiratory sound was natural, but there was a creaking at the outer part of the left side. A systolic and rather prolonged bellows-sound was heard at the left nipple, but did not extend far from it. She soon became delirious, and died in a few days.

Inspection.—The outer and lower part of the left lung was connected with the side by bunches of white threadlike cords, from one to four inches in length. The free edge of this lung adhered to the pericardium. The lungs were healthy, but much congested.

The pericardium was closely and universally adherent to the heart, and also to the chest at the seventh and eighth ribs, by means of *bands* similar to those which connected the pleural surfaces. The heart was of a natural size. The mitral valves were thickened, and at the base of one of them was a patch of fibro-cartilaginous matter, as large and as

thick as a bean. The lining membrane of the left auricle was of a deep violet colour, and was rough and granulated with whitish raised grains, over a space as large as a crown-piece, running into the mitral orifice.

The history of this case renders it probable that the adhesion of the pericardium took place at the period of the first attack, when this patient was 17 years of age. The bands which connected it with the outer side of the chest were certainly not of recent formation; and the undulations in the side to which the adhesion gave rise had been seen two or three years before her death. The uneven thickened edges of the mitral valves must, for some time, have prevented their perfectly closing the mitral orifice, and hence there was regurgitation through a narrow slit, giving rise to sharp, shrill, systolic sounds. The fatal termination was hastened by an attack of endocarditis.

It is true that this patient, ever since the time when adhesion of the pericardium took place, had palpitations and distressing dyspnoea on exertion, and was also subject to a teasing cough; but then it must be remembered that there was also extensive mitral disease. And yet, in spite of this serious complication, life was protracted through twelve years, and it was at last terminated by an attack of acute inflammation, which left unmistakeable marks on the lining membrane of the heart.

It may be concluded, therefore, that adhesion of the pericardium, although it may materially affect the health and comfort of those attacked by it, is occasionally borne with for many years, and has no *direct* tendency to shorten life; but that in the majority of cases it sooner or later gives rise to dilatation of the cavities of the heart, and thus *indirectly* leads to a fatal result.

When pericarditis terminates neither by resolution, absorption, or adhesion, its progress may be very rapid, or it may run into a chronic form. In the former instance it will usually be found that the attack has either taken

place in the course of rheumatic fever, or that inflammation has occurred simultaneously in the adjacent parts—pleura, lungs, or peritonium. In either case it is severe. Andral relates a case in which death occurred only twenty-seven hours after the onset of the pericarditis, which appeared on the sudden cessation of pain in the joints in the course of a rheumatic fever. The pericardial sac was found lined with a whitish exudation, membraniform and reticulated, containing only about one ounce of greenish coloured serum.

Here death could not have resulted from the effects of pericarditis alone. There was no amount of fluid to compress the heart and control its action, and the lymph on it was soft and recent. The pericarditis was, in fact, the finishing stroke of the acute attack of rheumatic fever. Indeed, no instance has been noticed in which a pure, uncomplicated attack of pericarditis resulted in speedy death.

When it occurs in the course of rheumatic fever, its severity will generally be found to be proportioned to that of the inflammatory action around the joints, and of the general fever; both depending on a highly deranged state of the blood.

When this disease takes on a *chronic* intractable form, although it may not have been ushered in by pain and other symptoms of high inflammation, yet the exudation is of the same character as in the more acute form, and contains, so to speak, the elements of life and death—organizable and unorganizable matter. It is seldom even then that it produces a fatal result by itself; for it is surprising what an amount of fluid may be effused into the pericardium without fatally oppressing the heart.

But, in fact, as in the acute form, so here, other causes are at work, although not altogether of the same kind. For it almost always happens that this form of disease makes its appearance after the constitution has been impaired; when, for instance, a diseased state of the blood

has given rise to rheumatism or gout, renal degeneration, tuberculosis, cancer, &c.; and when these diseases have existed for a considerable time. In the latter cases it has been seen that tubercular or cancerous matter have been sometimes found in the false-membranous exudation, and in others a cheesy matter closely resembling in appearance atheromatous degeneration of the arteries has been observed; and also calcareous concretion, sometimes to a great extent, investing the heart with a bony crust. These of course act as foreign irritating bodies, and may tend to shorten life.

When the attack has followed on some depressing causes, as after serious operations and accidents, such as in Cases 79 and 80, a low granular matter (typhic deposit) has been found. It matters little whether this be considered to have been secreted from vitiated blood, or to have descended in the scale of organization, after having been secreted, by increasing feebleness in the metamorphosis of tissue. Such cases occasionally, after remaining stationary for a time, improve; when it may be presumed that the nature of the exudation has been altered by the improvement that has taken place in the patient's general health.

Some discrepancy appears to exist between the various statistics brought forward relative to the comparative mortality arising from pericarditis as occurring in connection with rheumatic fever, and with other diseases; more especially renal degeneration. But in truth a comparison cannot be fairly instituted in these cases, for renal degeneration is of itself fatal, whilst rheumatism is not so. When asthenic pericarditis occurs in the progress of curable diseases, as for instance after serious accidents, it will be usually found *not* to be fatal.

CHAPTER VI.

CAUSES OF DISEASES OF THE PERICARDIUM.

*Inflammation — Pyæmia — Rheumatic Diathesis — Depletion —
Renal Degeneration — Age — Sex.*

It is useless to search for a special direct cause of simple uncomplicated pericarditis, because such a disease is almost unknown, unless induced by some local injury. One very interesting case, however, of what might fairly be termed idiopathic pericarditis, is reported by Dr. Beale in vol. ii. of the "Archives of Medicine." The patient was a young man, age 20, and appeared to be in robust health before the attack. He had never had rheumatism or other disease.

In the acute sthenic form it has usually been seen to occur either simultaneously with inflammation of a neighbouring organ, such as pleuritis or pneumonia, or in the course of rheumatic fever.

In a more asthenic and chronic form it has been seen to supervene on different diseases which have a tendency to lower the vital power, such as pyæmia, renal degeneration, serious accidents, &c. Dr. Kirkes* considers it probable that in certain cases of pyæmia, pericarditis is excited by secondary deposits in the muscular coats of the heart, either by their mere proximity, or by their bursting into the cavity of the pericardium, and gives two very interesting illustrative cases.

Medical Times and Gazette, vol. ii, 1862, p. 431.

When pericarditis occurs jointly with pleuritis or pneumonia, neither of them can be said to be the cause of the other; but one and all must be referred to the common causes of inflammatory action, which need not be discussed here. Nor when it occurs in connection with rheumatic fever, can the latter condition be said to be its cause, for in fact, pericarditis sometimes precedes the rheumatismal inflammation of the joints. That which gives rise to the peculiar state of the blood is doubtless the cause of both.

But are there no special causes at work tending to determine the inflammatory action to the heart? It will presently be seen that anything which, by lowering vitality, makes the heart weak and irritable, renders it prone to pericarditis; and as it is not long since most active depletory measures were employed in rheumatic fever, it is open to inquiry how far such treatment might have acted as a cause of pericarditis. Bouillaud and Hope, who both carried venesection and mercurialisation to a great extent, have shown that pericarditis occurred in one-half of their cases of rheumatic fever. Subsequently, Drs. Latham, Taylor, and Budd, whose treatment was much less depletory, state the proportion to have been only one-eighth in their practice; and the proportion has been still less in the practice of Dr. Fuller, and in that on which these observations are grounded. These are facts of much significance.

When pericarditis supervenes on chronic diseases, such as renal degeneration, it does not generally appear until the disease has existed for a considerable time; a proof that debility joins with the original diathetic mischief in bringing on pericarditis. In renal disease another special cause *may* exist. For when in such cases a notable diminution in the secretion of urine takes place, the blood becomes completely altered in character. Formerly this, and many other pathological phenomena were supposed to result simply from the accumulation of urea in the blood, but this opinion, and also that of Frerich's, who maintained that

in those cases in which there was no excess of urea, this substance had undergone decomposition into carbonate of ammonia, which latter material was supposed to produce fatal coma and other conditions, has been shown to be incorrect. It is more probable that the tendency to pericarditis in such cases is due to some general changes occurring in the blood.

Pericarditis occurs almost as frequently in renal disease as in rheumatic fever; but not more so than in pneumonia, pleuritis, and bronchitis; indeed not so frequently as in the last-named disease, according to Dr. Prout.

The influence of *age* and *sex* cannot be disputed. Thus among the young not only is acute rheumatism more prevalent than in advanced age, but during its course pericarditis makes its appearance in a larger proportion of the persons attacked. The contrary holds good as regards *chronic* rheumatism, and the non-rheumatic attacks. So also in respect to *sex*; the young female is perhaps more subject to acute rheumatism and pericarditis than the male.

CHAPTER VII.

NATURE AND FORMATION OF DISEASES OF THE HEART.

WALLS AND CAVITIES.—*Inflammation—Degeneration, Fatty; Mottled; Brown-stained—Hypertrophy—Dilatation.*
ENDOCARDIUM.—*Colour—Texture—Milky Patches—*
VALVES.—*Adhesion—Thickening and Shrivelling—Rupture—Shortening of Chordæ Tendinæ, &c.—Vegetative Growths—Atheromatous Degeneration—Calcification—* MORBID PRODUCTS.—*Coagula—Polypoid—Globulosis.*

WALLS.—The walls of the heart being muscular are for the most part similarly affected with other muscles, and are subject to *inflammation, morbid degeneration, and increase and extension of substance.*

INFLAMMATION rarely occurs in muscle unless the neighbouring tissues are at the same time attacked. Thus acute carditis, or myocarditis affecting the whole organ, and unconnected with pericarditis and endocarditis, is so rare an affection as to require no notice in a practical work of this kind. A certain amount of *partial* myocarditis is not unfrequently observed accompanying inflammation of the membranes, when portions of the muscles are found infiltrated with serosity, varying in appearance from red-

dish brown to yellowish white colour, the fibre being softer than natural, or even reduced to a pultaceous mass in which all traces of the normal appearance are lost. In some few cases pure *pus* is found between the fasciculi, and in others in small abscesses, which are not however to be confounded with the purulent deposits occurring in phlebitis. Hope once found a gray granular matter interspersed amongst the fibres, resembling probably the typhic deposit of Vogel.

In *chronic inflammation* the muscular fibre is sometimes replaced by "yellow knots, chords, and callosities," and these coalescing, form a tough substance resembling condensed cellular tissue, without a trace of the appearance of healthy muscle.

Occasionally also *ulcers*, evidently of inflammatory origin, are seen to penetrate the muscle; but oftener ulcers in this situation arise from atheromatous degeneration at the base of the valves.

DEGENERATION of the muscular walls of the heart may be seen under three aspects, presenting a *yellow fatty*, a *mottled*, and a *brown-stained* appearance, more or less accompanied by softening.

In the *fatty heart* the cellular tissue immediately under the serous membrane is seen to be loaded with a very yellow wax-like fat, of rather oily consistence, more especially on the right side. This fat dips down between the fibres and in some cases accumulates to such an extent as to appear to take their place; the walls of the heart become soft and flabby, and the red colour of health is replaced by that of a dirty yellow, owing to the large quantity of fat being deposited amongst the muscular fibres. The fat in this case is of the ordinary kind found in adipose tissue, and does not invade the texture of the muscle itself, but may from its undue accumulation cause wasting of the latter.

In the *mottled heart*, the whole organ is rarely affected, but patches of a mottled yellow and brown colour are

seen, in which the consistence of the muscle is notably diminished; so much so that occasionally, unless great care be taken, the finger passes through the walls of the right auricle or ventricle in removing the heart from the body.

By the aid of the microscope this state is seen to be one of *fatty degeneration* of the muscle itself. The fat globules are very minute, not occurring in the form of adipose tissue, as in the yellow fat heart; they are found in the interior of the muscular fasciculi, and like strings of beads, lie between some of the primitive fibres, and even sometimes take their place. The fat in these cases is rich in cholesterine.

Brown Heart.—More than twenty years since a peculiar appearance was pointed out and styled *brown paper heart*, from its colour closely resembling that material. It was often seen to extend over the whole organ, sometimes joined with hypertrophy; affecting the left more than the right side when it was not general. With it were associated very frequently milky spots on the pericardium, and slight adhesion between its opposite surfaces; and the endocardium, especially that portion of it which lines the left auricle, was almost always more or less thickened and opaque; it was therefore considered to be the result of inflammatory action, or at any rate to have taken place under its influence. Some time afterwards a description of a similar state of the heart was found in "Rokitansky's Pathology," who stated it to be caused by very minute *fat granules*, interspersed amongst the primitive fibres. Microscopic examination shows that the muscular fibre is invaded, and not merely displaced; that, in fact, there is a *fatty degeneration* of the muscular fibres. Probably some examples of fatty degeneration may result from prior changes in the muscular tissue, but the very frequent association of this condition with fatty degeneration in other tissues, as the cornea (*arcus senilis*), cartilage, fibrous tissue, &c., as has been shown more particularly by Mr. Edwin

Canton⁽¹⁾ clearly proves that the local alteration is due to constitutional disturbance, and, most probably, to an altered state of the blood. It must also be borne in mind, that this change occurs in almost any organ which is about to waste and cease to discharge its function, as in the placenta towards the termination of utero-gestation.⁽²⁾ For an account of the various forms of fatty degeneration, I would refer the reader to the paper of Dr. Richard Quain, in the 33rd volume of the Transactions of the Medico-Chirurgical Society. It is probable that the fatty matter results from changes occurring in the contractile tissue itself. Indeed it is almost certain that normally a certain quantity of fatty matter results in the disintegration and waste which accompanies muscular action, but in health these, with other products of decay, are removed as fast as they are produced, but under certain altered conditions they accumulate and very soon interfere with the due action of the tissue.⁽³⁾ Dr. Ormerod, of Brighton, has recently added some interesting observations to what is known of the pathology of fatty degeneration. He has shown that the change is not due to a chemical alteration such as may be produced artificially by maceration of the fibres. In the latter case adipocere, which is merely a soap of lime combined with the fatty acids pre-existing in the tissue is produced; while in true fatty degeneration a fatty acid is formed, of which only a small quantity is present in health. The oleates greatly preponderate over the margarates in fatty degeneration, while the fats present in the normal tissue consist almost entirely of margarates. Dr. Beale has detected cholesterine in considerable proportion in the fatty matter obtained from hearts in a state of

(1) On "The Arcus Senilis."

(2) See a paper in the "Med. Chir. Trans.," 1857, on "Fatty Degeneration of the Vessels of the Placenta," by Dr. Druitt.

(3) "The Address in Medicine," delivered at the meeting of the British Medical Association, at Cambridge, 1864.—"Brit. Med. Journ.," August 6th, 1864, page 153.

fatty degeneration in several instances, and he states that this substance is always to be found in the fat which is extracted from various tissues in a state of fatty degeneration.

In some cases of pyœmia small *purulent deposits* are formed in the muscular walls of the heart, as occurs in the lungs, liver, and other organs.

HYPERTROPHY AND DILATATION.—The *increase and extension* of the walls and cavities of the heart are commonly denominated *hypertrophy* and *dilatation*; and these terms alone will be used; the phrases “excentric and concentric,” “active and passive aneurism of the heart,” being found frequently to mislead and confuse.

The walls of the heart may be increased in *thickness* and *in extent*, and in *both* respects. In order to ascertain whether such increase has taken place or not, it is necessary to have a natural standard of health, by which to compare the heart we examine. The accuracy of Bizot's tables of the weight and size of the heart, and of the thickness of its walls and the width of its orifices, having been accurately tested, they will be taken as the standard of reference. In accordance with these tables, the left ventricle will be considered hypertrophied in *thickness* when it measures more than 6 lines in the male and 5 in the female, midway between the base and the apex; and the right when its measurement exceeds 3 and $2\frac{1}{2}$ lines respectively. Increase of the *extent* of the walls can only be measured by taking into consideration the dimensions of the cavity they enclose, or the weight of the whole heart, and necessarily involves the *dilatation* of the cavity or cavities surrounded by the muscle. In fact hypertrophy is always accompanied by dilatation, although the converse does not hold good; for in 155 cases of fatal heart disease, not one is recorded of simple hypertrophy of the left ventricle without co-existing dilatation, and only one of the right ventricle without it; and whenever both were hypertrophied both were also dilated. The remarks which follow apply to the ventricles alone, as, unless in some very extreme

and exceptional cases, the alteration of the auricular walls do not exercise any important influence on the circulation and the health. On referring to the tables on page 109, it will be seen that on the left side the proportion between the cases of hypertrophy with dilatation, and dilatation alone, was about equal, and that on the right side of the heart one-third consisted of hypertrophy with dilatation, and two-thirds of dilatation alone. It would be needless here to trace further the connection between hypertrophy and dilatation, and the order of sequence in which they appear, as this will be done in the chapter on the progress and terminations, and the causes of these affections.

It is a very interesting question, and one of the highest importance to determine, whether hypertrophy consists, as the word denotes, of an excess of healthy nutrition, or whether any diseased or depraved action is at work. That this latter may sometimes occur in connection with hypertrophy, or rather be engrafted on it, is certain, because hypertrophied walls are frequently found invaded by fatty and granular degeneration. When hypertrophied muscle which retained its *natural colour and appearance* has been submitted to microscopic examination, no traces of disease have been discovered. Vogel concludes from his own researches and those of Henle, that the "volume of muscle is increased without the single fasciculi gaining in thickness, whence he considers it must follow that their number is increased, and that new ones have arisen amongst those previously existing." Others consider that the single fasciculi do increase in size. In either case, however, the increase is one of simple nutrition, and does not arise from the invasion of new morbid products. When magnified 500 diameters, the average breadth of the fasciculi, both in hypertrophy and health, was seen to be $\frac{1}{400}$ th of an inch. Hence it may be inferred that the *quality* of the muscle is unaltered; and this view will be found to accord with the facts to be detailed in regard to the manner of its increase under certain circumstances.

When one of the cavities becomes dilated without any increase taking place in the number or size of the muscular fibres of its walls, they become stretched and *attenuated*; but it is very doubtful whether the muscular walls ever become *atrophied*, in the proper sense of the word, in the same manner as the muscles in other parts of the body, when they are paralysed, or disused from any cause; a state of things which cannot occur in the constantly working heart. Again, with the heart in never-ceasing action, it is almost impossible to conceive that any one of its cavities into which and from which the blood is constantly flowing, could be notably contracted by the walls closing in upon it; because the increase from hypertrophy must naturally be *outside*, where no pressure from the blood opposes it; unless, indeed, it happened that this was prevented in a particular case by the heart being bound down by adhesion with a thickened and unyielding pericardium; or when again there was a congenital malformation by which a communication existed between the two sides of the heart. Then, of course, one of the cavities might diminish in size, because the blood instead of being forced during its expulsion against the muscle so as to distend it, would rush through the opening of communication. Consequently it is in such circumstances that well-authenticated cases of diminution in the cavities have been observed; and also after sudden death, when strong contraction has taken place during the last agony, and the muscle has remained contracted. In such cases, Cruvellier was able by dilatation or maceration to restore the heart to its normal state. Hope and others, however, think that contraction sometimes takes place from the enlargement of the fleshy columns and papillary muscles.

It might be supposed that dilatation of the orifices of the heart would naturally take place with that of its cavities; but this by no means follows. Both the pulmonary and aortic orifices, more especially the latter, are sometimes seen to be thus dilated, but not frequently. The fibrous

zone surrounding them, in fact, seems little prone to distension. It is far otherwise, however, with the foramina between the auricles and ventricles. The mitral foramen appears to be surrounded by a stronger and more unyielding ring than the tricuspid foramen, but still is frequently found dilated along with the left auricle and ventricle; whilst the tricuspid foramen may be said invariably to dilate, sometimes to a great extent, when the right ventricle and auricle are similarly affected. The evils arising from this will be seen in the sequel.

ENDOCARDIUM.—The diseases which affect the endocardium produce *alterations in the membrane* itself, and give rise to *morbid products* within the cavities it bounds. Some of them are of an inflammatory, others of a constitutional character. *Inflammation* may produce alteration in the *colour* and in the *texture* of the *membrane*, or it may be covered with *vegetative growths*.

Colour.—Nothing is more common than to find the endocardium and valves of a deep violet or scarlet colour, with their intermediate tints. As it has been shown that such colour is by no means indicative of inflammation of the pericardium, *à fortiori*, it may be taken to be even less so in respect to the endocardium, which is constantly in contact with scarlet or purple blood. Accordingly, it is generally allowed that in the majority of cases such colour is derived from the blood; sometimes during lifetime, when the blood is impure and imperfectly de-carbonized, and the colouring matter dissolved in the serum, as in typhoid pneumonia, and in a low condition resulting from long-continued spirit drinking; at other times by imbibition after death. In the former case the darkest tint is found in the right auricle, and diminishes in intensity successively in the right ventricle and the left auricle, until the colour is almost lost in the left ventricle. In cadaveric imbibition it is usually seen solely on the most dependent parts of the membrane. A distinction is said to be caused by the difference in the character of the colorization; that result-

ing from inflammation existing in streaks of varied colour, whilst that from imbibition is a uniform even staining. But if the endocardium remains smooth and glistening, it is doubtful whether the inflammatory origin of the colour can safely be inferred; in fact, as so few persons die in an early stage of endocarditis, we can hardly expect to have opportunities of witnessing the first inflammatory blush, although we know, from Dr. Richardson's experiments on animals, that it is produced. When, however, inflammatory action has persisted for some time, certain characteristic changes are produced in the endocardium. Thus its colour may become even paler than when in its normal state, and its bright lustre may be changed for an opaque, dull velvety appearance, often seen in the left auricle, and running into the mitral valves.

Texture.—Alterations of texture, however, afford the most conclusive proof of the inflammation of the endocardium. Thus it is sometimes both thickened and softened, being easily torn, particularly where it forms the valves; more frequently it has become harder and of denser consistence. The whole lining of the left auricle, and the mitral valves are not unfrequently found in this state, presenting an appearance of leather.

VALVES.—The arterial valves are occasionally much thinner than usual, and are pierced here and there with small holes, giving them a net-like appearance when held up to the light. *Milky patches*, too, are seen penetrating the membrane, as on the pericardium. It is probable that neither of these affections are in every case to be attributed to inflammatory action; at any rate, as they are frequently observed in the bodies of persons who have never presented any marked symptoms of heart disease during lifetime, they cannot be looked upon as of great importance.

There are other changes in the valves, which impede their action, and thus tend materially to derange the health. Such are *adhesion* of one or more *valves* to the walls of the heart or aorta, or to each other; *thickening* and *shrivelling* of

the valves; *rupture*, *thickening*, or *shrivelling* of the *chordæ tendinæ*.

Adhesion of one or more flaps of a valve to the walls of the ventricle or aorta, is a very frequent occurrence, except in the pulmonary artery. At the tricuspid orifice this affection must have been very much overlooked in former years. It was by observing a case of this kind after death from cardiac dropsy, that special attention was drawn to tricuspid regurgitation, as the proximate and constant cause of such dropsy, and led to accurate measurement of the tricuspid foramen and its valves, and testing of their action, in nearly all cases of cardiac disease subsequently examined. It was seen in Cases 39, 40, and 93.

Cohesion of the different flaps of the valves with one another chiefly takes place in the semilunar valves of the aorta, more rarely in the mitral valves, and still more rarely in the valves on the right side of the heart. Sometimes it occurs to such an extent that only a small circular opening, or a slit, is left, through which regurgitation of the blood takes place during the systole of the ventricle. The effects of this state of the valve on the circulation are most serious, and are often aggravated by the addition of considerable thickening, and in some cases, calcification. It was seen in Cases 46, 88, 36, 27, 41, 93, and 58.

Chordæ Tendinæ.—Sometimes there is a *rupture* of one of the *chordæ tendinæ*, which then shrivels up into a small nodule, but more frequently they are *thickened* and *shortened*, so as to prevent the valvular flap to which they are attached from rising up into the plane of closure, whereby the action of the valve is impaired. This was seen in Cases 28, 38, 42, 91, 100, 58, 29, 93, and 95.

Vegetative growths, varying in size from that of a fig seed to that of a date, sometimes opaque, at others of semi-transparent horn-like appearance, are not unfrequently seen attached to the endocardium; more particularly to the surfaces and free edges of the left auriculo-ventricular valves. These are the warty excrescences of Laennec, in

their larger form denominated by some condylomata. Their microscopical appearances are identical with those of the horny deposits on the lining membrane of the aorta previously described. Like them, too, they are invested with a smooth, shining, delicate membrane, resembling the endocardium, but which, in fact, is a fibrous film.

Virchow considers these vegetative growths to arise from a kind of hypertrophy of the membrane itself occurring under the influence of inflammatory irritation. He remarks:—"There is no exudation, but the cellular elements take up a greater quantity of material, and the spot becomes rugged and uneven. Then we see, when the process runs its course rather slowly, either that an excrescence, or condyloma arises, or that the swelling assumes a more mamillated form." Others conceive these substances to be simply coagula formed from the blood, under the influence of inflammation. In a practical point of view, this difference is of little importance. They were seen in Cases 87, 84, 45, 28, 37, 27, 58, 92, 39, and 47.

Besides these direct effects of inflammatory action on the endocardium and valves, there are others produced by *atheromatous degeneration* and *calcification*. As these occur in the arteries, and not in the veins, so are they limited to the arterial or left side of the heart, except when arterial blood is admitted to the right side by an abnormal communication between the two sides. Their nature, origin, and mode of development in the coats of the aorta have been already shown.

In the valves of the heart, however, they present a very different appearance from that in the aorta, the difference being in their not lying in thin scaly patches, but in rough globular masses, which present uneven and rugged projections, often as hard as stone. In these situations, associated with, or without, induration, thickening and adhesion of the valvular flaps, they frequently offer great obstacles to the blood flowing through the orifices where they exist, or

else allow of its regurgitation, both evils of a very formidable nature, of which the effects have to be considered.

Ulceration, Rupture, and Aneurism.—Analogy would lead us to expect that atheromatous deposit would, in some cases, be followed by *ulceration, rupture, and aneurism* of the walls of the heart, as in the aorta, and accordingly all these accidents are known to take place on the arterial side of the heart. As they have been fully described when occurring in the aorta, and as they can neither be prevented, discovered, nor remedied during lifetime, they will not be dwelt upon here.

MORBID PRODUCTS.—No visible analogy can be established between the morbid products found within the heart, and those within close serous sacs, such as the pericardium, pleura, and peritoneum, because, being constantly washed by the current of the blood, secreted fluids are at once swept away; and on the other hand, under certain circumstances, the blood itself furnishes morbid products which are not found in serous cavities. There can be no doubt that when the endocardium is inflamed, the usual morbid products take place, as has been shown in the experiments of Dr. Richardson; and sometimes, besides the effect produced on the membrane itself, traces of coaguable lymph may be found in parts least exposed to the action of the current of the blood, as under the valves and betwixt the *carneæ columnæ*. For the most part, however, they are carried away, and may give rise to abnormal deposits in the capillaries, chiefly of the *spleen* and the *kidneys*. But the most visible effect is that which is produced on the blood itself. It is well known that blood in contact with an inflamed surface is rendered more prone to coagulate, and the same tendency is produced by the presence of certain substances such as tubercle, and more especially pus. Coagulation, however, takes place without any such exciting cause, particularly after death, or during a protracted struggle, when the force of the heart's action has been much diminished. Hence coagula of inflammatory, as well as non-inflammatory

origin, must be recognised, the latter either taking place from diminished action, or induced by the presence of actual pus, or some morbid product, transferred from some distant part of the body, as in certain cases of phlebitis.

There are two forms in which coagula present themselves within the heart's cavities—*polypoid* and *vermiform* or *globular coagula*.

Polypoid Coagula.—That the greater number of polypoid coagula found within the heart have been formed after death, or during the last moments of life, is now universally admitted. They are of a yellowish colour, transparent, gelatinous, homogeneous, moulded to, but not entirely filling the cavities in which they are found, often prolonged into the vessels, particularly the pulmonary artery, and *not adhering to the walls*. There are others, however, which bear evident traces of having been formed during lifetime. They are of a dull, whitish-grey, opaline appearance, are composed of concentric layers or rings, and are more or less attached to the valves and carneæ columnæ. Rarely they contain in the middle a dirty purulent-looking fluid. Sometimes not a trace of inflammation can be detected on the endocardium, but then it will be found that there is some more or less distant source from which pus has been absorbed into the circulation, and thus caused the blood to coagulate in the cavities of the heart, more particularly in cases of phlebitis and suppurating pneumonia, and sores giving rise to purulent deposits in other organs. When no such source is discoverable, the evidence of inflammation having occurred in the endocardium itself will generally be found. It is evident that the formation of these polypi during lifetime must be attended with the greatest possible danger to life, interfering, as they must do, with the circulation and producing a kind of suffocation.

Globular Coagula.—A much rarer form of coagula is that denominated globular vegetations. They are said to vary from the size of a pin's head to that of a pigeon's egg,

but in general do not exceed that of a pea. They are of a cream colour, the smaller ones solid, with a speck of red coagulated blood in the centre; the larger ones containing a dirty, purulent-looking fluid. They are composed of concentric rings or layers, and they are attached to the heart by soft pedicles, which interlace among the *carnea columnæ*. In a case where these globular vegetations were found in large numbers within the right ventricle, there were no traces of endocarditis, nor had symptoms of it been observed during lifetime. It is probable that they sometimes arise from coagulation, induced by portions of tubercle carried into the heart by the current of the blood. Sometimes, like false membranes, they become calcified.

Vermiform Coagula.—These coagula, instead of taking the form of isolated globular bodies, are sometimes grouped together, resembling raspberries, or in strings taking the form of worms. They are generally, but not always, easily detached, and the portions of the valves to which they are attached are observed to be more or less diseased and rugged. In this form they are called *vermiform coagula*.

These various affections of the walls, cavities, and orifices of the heart will be seen to have a great effect in interfering with the pulmonic, and systemic circulations; and the evils thus induced will be found, more or less, to arise from the retardation of the flow of blood through the *veins*. Of late, however, Dr. Kirkes, and Virchow of Berlin, have drawn attention to a derangement of the *arterial* circulation, caused by small fragments being detached from fibrinous exudation on the valves or vegetative growths, and carried into the smaller arteries, more particularly those of brain, spleen, kidneys, and liver, thereby plugging them up, and giving rise to serious, even fatal consequences.

This affection has received the name of *embolism*. A remarkable case of it is recorded in the "British Medical Journal" of November 25, 1863, on which Dr. Kirkes has remarked: "Surely the pathology of this case is quite clear—

ordinary rheumatism in the first instance; then acute ulcerative inflammation of the mitral valve; then contamination of the arterial blood by lymph, pus, and other inflammatory products from the valve; then the signs of general blood poisoning, namely, febrile disturbance of a low typhoid form, nausea, vomiting, profuse diarrhoea, and erythematous eruption; then local suppuration of the parotids; lastly, obstruction of the cerebral vessels, with consequent softening of the brain substance, and hemiplegia; all terminating in death, and revealing proofs of blood-poisoning in various parts of the body."

CHAPTER VIII.

CAUSES OF DISEASES OF THE HEART.

WALLS AND CAVITIES.—HYPERTROPHY.—*Inflammation—Obstacles without the Heart—Obstacles within—DILATATION—Inflammation—Degeneration—Obstacles without—Obstacles within—Age—FATTY DEGENERATION.*
ENDOCARDIUM AND VALVES.—*Inflammation—Rheumatism—Gout.*

WALLS AND CAVITIES.—HYPERTROPHY.—It has been seen that hypertrophy consists of an increase both in the number and size of the muscular fibres. This can only take place in consequence of their receiving a larger amount of nutrient fluid than usual, which by a well-known law is promoted by increased action of the muscle itself. Anything, therefore, which tends to augment the heart's action, may be a cause of hypertrophy. Such exist *without* and *within* the heart.

Of those *without* the heart, pericarditis, especially when terminating in adhesion, has been supposed to be a cause of hypertrophy by Hope and others, as they imagine that the heart, being oppressed by the exudation around it, or cramped in its motion by the closely adhering membrane,

must be forced to unusual exertion to carry on the circulation. Rokitsansky and others deny this, as regards adhesion, and an examination of these cases certainly leads to the conclusion, that practically, inflammation and adhesion of the pericardium do not cause hypertrophy, although they might theoretically be supposed to do so. It was only observed in one case out of nineteen in which there was adhesion of the pericardium. Traces of pericarditis or endocarditis, however, were found in about half the cases of hypertrophy. Increased action, as evidenced by violent palpitations, is frequently excited by moral emotions, as well as by sympathy with diseased organs, more particularly the uterus in cases of hysteria; but we have no means of determining whether this temporary increase of action is sufficient to induce hypertrophy. In some of these cases it certainly did not. In Case 35, "violent palpitations," and pericardial adhesion had both existed for twelve years, at the end of which time, the "heart was found of a natural size." The main cause is undoubtedly furnished by the existence of an obstacle to the onward course of the blood, which it is the office of the muscular walls to promote.

On the *left* side, such an obstacle may be offered by a congenital constriction, or an aneurismal dilatation of the aorta, by a sacculated aneurism or mediastinal tumour compressing it, or by a diminution of its elasticity produced by atheromatous degeneration and calcification; and also by an engorgement of the capillaries, such as exists in anæmia; and experience shews that this is very frequently the case.

An obstacle to the course of the blood flowing from the *right* side of the heart, may exist in the lungs, in consequence of their becoming the seat of emphysema, chronic bronchitis, tubercular or cancerous degeneration, of their being compressed by pleuritic effusion, or congested by impediments to the circulation existing on the *left* side of the heart.

Obstacles *within* the heart may arise on the *left* side from narrowing of the aortic orifice. In this case, the left

ventricle is excited to unusual exertion, in order to push a larger quantity of blood through the diminished aortic aperture, than would otherwise pass through it.

Mitral obstruction and regurgitation tend, of course, to induce hypertrophy of the left auricle, but it is doubtful whether they act similarly on the ventricle.

Obstacles on the *right* side of the heart arise from contraction of the pulmonary aortic orifice, which has been seen to be very rare.

Regurgitation through the right auriculo-ventricular orifice must necessarily induce hypertrophy of the right auricle, but does not similarly affect the ventricle; for amongst 106 cases of tricuspid regurgitation, only two of simple hypertrophy of the ventricle were found.

	<i>Cases.</i>
Hypertrophy of the left ventricle alone existed in. .	20
Combined with disease of aortic valves ..	0
" " mitral and aortic ..	8
" " mitral ..	8
" " tricuspid ..	4
" no valvular disease ..	4
Hypertrophy of both ventricles ..	68
Combined with disease of aortic valve ..	16
" " mitral and aortic ..	8
" " mitral ..	16
" " tricuspid ..	20
" no valvular disease ..	28

DILATATION.—The causes of dilatation of the cavities of the heart must be sought for in *alterations of structure* in their *walls*, and in the varying amount of outward pressure exerted on them by the blood from within.

Walls.—The effect of inflammation in diminishing the contracting force of muscle has been seen; and, therefore, pericarditis and endocarditis must almost necessarily tend to induce dilatation. Accordingly, amongst 19 cases of adhesion of the pericardium, dilatation was found in 10;

and amongst 108 cases of dilatation, evident traces of endocarditis or pericarditis were seen in about 50.

Fatty degeneration of the walls in which healthy fibre is replaced by fat, must impair the contractile power, and thus act as a direct cause of dilatation, more especially when any extra pressure is exerted by the blood from within. Dilatation was found to exist in twenty of the twenty-five cases in which there was fatty degeneration of the walls of the heart.

The same *obstacles to the circulation*, both *without* and *within* the heart, which have been seen to produce hypertrophy, would seem to have an equal tendency to induce dilatation, inasmuch as the blood must exert an increased pressure outwards on the walls of its cavities, in proportion as its onward flow is impeded, and additional power is required to propel it. A careful examination, however, of the cases here recorded, will show that diseases of the valves have a much less direct influence in promoting dilatation than has been supposed.

Thus the cases in which dilatation of the <i>left</i>			<i>Cases.</i>
ventricle alone existed were	28
Combined with mitral disease in	24
„ aortic disease in	4
„ tricuspid valves in	4
„ traces of pericarditis or endo-			
carditis	24
The cases in which the <i>right</i> ventricle alone was			
dilated, were	20
Combined with mitral disease in	8
„ tricuspid „	8
„ aortic	0
Cases in which both ventricles were dilated	92
Combined with aortic disease	28
„ mitral „	20
„ tricuspid „	32
„ no disease of valves	56
Remains of pericarditis or endocarditis	36

So that, taking account of the dilatation of each ventricle, whether occurring singly or conjoined with the others, we have:—

Dilatation of left ventricle occurring in	..	120
Combined with mitral disease in	..	48
„ tricuspid disease in	..	36
„ aortic „	..	32
„ results of pericarditis or endo- carditis	60
Dilatation of right ventricle occurring in	..	112
Combined with mitral disease	..	28
„ tricuspid disease	..	44
„ results of pericarditis or endocarditis		44

Thus whilst there was aortic valvular disease in only one-fourth of the cases of dilatation of the left ventricle alone, the mitral valve was inefficient in nearly all of them. From this it might, at first sight, be supposed that mitral disease was a direct cause of dilatation of the left ventricle, unaccountable as such a supposition would be; but on looking closer we find that in all these cases there were evident traces of inflammatory action having taken place in the pericardium and endocardium, more especially near the mitral valves; and thus we see the true connection between dilatation and mitral disease, and recognize each as having arisen from this common cause, or, at least, having been favoured by it.

A different state of things reveals itself when we examine the statistics or the simultaneous dilatation of both ventricles. There the proportion of aortic valvular disease remains the same, but mitral diseases fall to *one-half*, tricuspid disease rises from one-sixth to *one-third*, and the traces of inflammation fall to *one-half*.

In one-half, therefore, of all cases of dilatation of the *left* ventricle, inflammation may be considered the cause; in the remaining half, obstacles to the circulation without the heart, more especially capillary congestion, induced by tricuspid regurgitation from *diseased* valves; or, again, in

CHAPTER IX.

PROGRESS AND TERMINATIONS OF HEART DISEASES.

Former views of the effects produced by Disease on the Motive Power and the Valvular Machinery of the Heart, separately and combined. WALLS.—Effects on Motive Power — Hypertrophy — Dilatation — Attenuation and Softening. VALVES.—Aortic — Pulmonary — Mitral — Tricuspid — Safety Valve Function. Combined Effects of these Lesions on the Heart's Action, on the Pulmonic, and on the Systemic Circulation.

WHILST examining into the nature of various affections of the walls and cavities of the heart and its valvular apparatus, alterations have been seen to take place of such a nature as to affect its *motive power*, and the *working of its machinery*.

It is seldom that any one of these lesions is alone concerned in producing derangements of the heart's action. Oftener several of them are found in combination, and not unfrequently some produce effects antagonistic to those caused by others.

It is proposed, therefore, first to trace the effect supposed to be produced on the circulation by each lesion separately, and next to examine in what manner they act in combination with each other; subsequently comparing the views generally entertained on these points, with the

- results of a large number of cases observed in hospital and private practice. We shall by this means be enabled to see how the health is affected by the various derangements in the heart's action thus induced, and to trace the steps by which a fatal termination is approached.

WALLS.—*Hypertrophy, Dilatation, Attenuation, and Softening* are Lesions of the Walls of the heart, which may be supposed to affect its *motive* power.

Hypertrophy of the ventricles is one of the most frequent of these affections. This may act favourably or unfavourably on the circulation. When occurring on the left side it has been supposed by many to give rise to arterial congestion of the capillaries of the systemic circulation, and thus induce anasarca and cerebral apoplexy; and when joined with mitral regurgitation, to assist in producing congestion of the lungs, with its concomitant evils, such as hydrothorax. On the right side it has been held to promote active pulmonary congestion; and when joined with tricuspid regurgitation, venous congestion in the systemic circulation. When, however, there exist obstructions to the arterial circulation, caused by disease of the aortic valves and inelasticity of the coats of the aorta on the left side, or by emphysema of the lungs, condensation, &c., on the right side; then hypertrophy has been considered to act beneficially by enabling the heart to overcome in some degree the obstacles opposed to the onward course of the blood.

Dilatation always, more or less, accompanying hypertrophy, somewhat increases its effects on the circulation; as a larger quantity of blood is thereby put in motion. When, however, dilatation is accompanied by *attenuation* of the walls of the ventricles, a contrary effect is produced; the weakened state of the walls preventing their propelling the blood with their ordinary vigour, *passive* congestion results; on the left side giving rise to pulmonary engorgement, and on the right to anasarca, &c. Indeed, Hope considers dilatation with attenuation to be the *immediate*

impaired elasticity of the walls of the aorta, the result of atheromatous degeneration and calcification.

Looking at the right side of the heart in the same manner, and from the same point of view, we recognize the influence of inflammatory action in about one-third of the cases of dilatation of the right ventricle, the remainder arising from pulmonary congestion, whether induced by disease of the lungs, or by impediments to the circulation existing at the mitral valves; such congestion, often amounting to pulmonary apoplexy, having been found in more than half of these cases.

Attenuation, being the result of simple dilatation without hypertrophy, must arise from similar causes.

FATTY DEGENERATION.—It is to be regretted that so little light can be thrown on the causes of fatty deposit and degeneration. It existed in about one-sixth of the cases here recorded, but the different forms it assumes were not always noted. In about half of them there were traces of inflammatory action in the pericardium and endocardium, and as the muscle lying between these two membranes could hardly have escaped some amount of inflammation, it is possible that in some cases, at least, its degeneration may have originated in this cause. Virchow is of opinion that fatty degeneration has often an inflammatory origin. The influence of age was marked, fifty being the average of all the ages taken together, of which only three were under thirty years of age.

ENDOCARDIUM AND VALVES.—The cause of those affections of the valves, which originate in atheromatous degeneration, have already been investigated. Those which spring directly or indirectly from inflammation remain. In this point of view it matters little whether vegetative growths are considered to have been formed by abnormal growth of the valves themselves, or by the addition of fibrinous deposits. In either case inflammation is the starting point, and the real question for our consideration is, "What is the cause of this inflammation?"

In twenty-four cases of vegetative growth rheumatism in a marked degree had existed, and in four of them spirit-drinking, which poisons the blood equally with the rheumatic diathesis; and as the same has been observed by other writers, rheumatism must be considered the main cause, as we have seen it to be that of the horny deposits in the aorta. Like atheromatous degeneration, these growths are mainly found on the left side of the heart, so that it may be that lactic acid plays the same part in their production as uric acid does in that of the former; and that the inflammatory process to which they give rise does not take place under the same circumstances that causes the valves to adhere to the sides of the ventricle or aorta, a state which is found equally on both sides of the heart.

cause of cardiac dropsy, &c., whatever might be its *remote* causes.

Fatty Degeneration of the muscles of the heart must necessarily be attended with similar results. It is also considered by some to be a frequent cause of syncope and sudden death.

VALVES.—*Disease of Aortic Valves*.—It has been long known that extensive disease of the valves at the orifice of the aorta may exist for a considerable time without causing death, or even seriously injuring the health. Dr. Hope has detailed a case in which the orifice was reduced to the size of a pea without having given rise to any disturbance of the circulation*. Dr. Law exhibited to the Sheffield Pathological Society a heart “in which the aortic orifice was nearly closed by a mass of calcification, there being left only a small cleft through which the blood could pass. Slight attacks of dyspnoea and occasional faintings were the only symptoms of heart disease observed in lifetime.” Andral relates a case of extreme narrowing of the aorta by calcareous deposits, and states it to be “certain that many aged persons are similarly affected, without being attacked by dyspnoea and dropsy, in consequence,” he argues, “of the circulation of the aged being slow.” But the circulation in old age is not always slow, nor is this affection of the aorta confined to old age, although it is found more frequently in advanced than in early life.

When aortic regurgitation exists, a greater effect is produced on the flow of the blood from the lungs than when merely obstruction is present, because no increased power in the force of ventricular contraction can prevent the reflux of the blood through a patulous orifice during diastole, which, meeting the blood that is flowing in from the auricle, offers a great obstacle to it, and must tend to produce engorgement of the pulmonary veins.

Disease of the Pulmonary Orifice being so rare, and occurring, for the most part, only in conjunction with incom-

* Lancet, 1846. Page 566.

plete closure of the foramen ovale, has hardly been taken into consideration as productive of injurious effects on the health; for any obstacle caused by regurgitation or obstruction would, by means of the open foramen, be divided between the two auricles, and so would less affect the general venous circulation.

Disease of the Mitral Valves has always been regarded as the most frequent cause of pulmonary obstruction, in the first place; and, secondarily, of that of the systemic circulation, acting through the lungs. A great amount, however, of *obstructive disease* may exist without causing death.* Mr. Prescott Hewitt has described a case in which "the mitral orifice was reduced to the size of a quill," and during lifetime no signs of diseased heart were exhibited. This state of the mitral aperture is often overlooked during life, because, as has been observed by Dr. Hope, "if the contraction is extreme, the current is too small to engender sound, and if it is slight, sufficient space is left for the blood to pass from the auricle into the ventricle, without such an amount of friction as to occasion murmur."

Regurgitative disease is much more formidable, and as in this situation it arises more from calcareous concretions and vegetative growths, than from dilatation, obstruction generally exists in conjunction with regurgitation. The two together, therefore, particularly when joined with hypertrophy of the left ventricle, must necessarily give rise to engorgement of the pulmonary veins, and of the lungs.

Disease of the Tricuspid Valves was formerly considered to be very rare. There being in this situation hardly any atheromatous degeneration or calcification, nor any vegetative growths, except when arterial blood is admitted by a morbid communication with the left side, of course little or no obstructive disease could be expected. Nor had the existence of such an amount of inflammation been recognized as could lead to regurgitation by shortening,

* Lancet, 1846. Page 5.

thickening, or adhesion of the valves to the walls of the ventricle. The fibrous zone of the tricuspid foramen, however, was known to be much more capable of dilatation than that of the mitral, and thus the orifice might be enlarged to such an extent that its valves could no longer effect its closure.

In fact, Hunter, and after him Dr. Adams and Mr. T. Wilkinson King, have asserted that the tricuspid valves seldom completely close the orifice to which they are attached, so that a certain amount of regurgitation takes place during health; from which Mr. King argued that in this manner the tricuspid orifice acted as a kind of safety valve, and prevented too much blood being thrown into the pulmonary artery. This hypothesis, however, assumes the truth of two propositions that will presently engage our attention: that serious, if not fatal, active congestion is produced by a too vigorous ventricular contraction,—and that blood regurgitating through the auriculo-ventricular foramen, and meeting the current flowing into the heart from the veins, produces no injurious effects. Besides which, if the experiment of injecting fluid into the ventricle is carefully performed, and unnatural dilatation be prevented, no regurgitation will take place.

On the whole, writers in general have attributed very little of the ill effects seen in the course of organic diseases of the heart, to defects of the tricuspid valves and foramen. Thus, Hope states that “tricuspid regurgitation is very rare.” Bouillaud, it is true, remarks that “the dilatation of the orifices of the heart is not less common than that of the cavities themselves,” and recommends “diligent observers not to neglect examining the orifices of the heart in all organic diseases; for if the dimensions are such that the valves cannot close them, the circulation of the blood through the heart must be more or less seriously disturbed.” Throughout the whole of his work, however, he attributes the disturbance of the

systemic circulation to disease of the valves of the *left* side of the heart, and gives to the tricuspid orifice little or no share in producing them.

Such being the mechanical effects on the circulation which each of these organic changes has been considered to produce, it remains to examine the manner in which they combine to bring about a fatal result, or serious derangement of the health; whether by a direct *stoppage of the heart's action*, or by *impeding one or both of the circulations*.

Stoppage of the action of the Heart may of course be produced by a penetrating wound or a crushing blow, whether it be diseased or not. Syncope, too, may occur in health, but is more likely to happen when the walls of the heart are enfeebled by degeneration or attenuation, or when the working of its valves is incomplete. When it does thus occur, restoration of the action is rendered more difficult; most so, when the obstruction lies at the valves on the left side of the heart.

Derangement of the Pulmonic Circulation, giving rise to congestion and œdema of the lungs, has been considered to arise from an over-supply of blood thrown into them by an hypertrophied right ventricle; in other cases from obstruction offered to the free return of blood to the left side of the heart by diseased mitral and aortic valves, and also by feebleness of circulation caused by general attenuation or softening of the ventricles. The cases of most frequent occurrence have been supposed to be those in which the pulmonary engorgement arises from obstruction, or regurgitation through the mitral foramen, with or without a similar state of the aortic orifice. The right ventricle, in its endeavour to overcome the obstacle thus offered to the circulation by the engorged state of the lungs, becoming hypertrophied, acts more strongly, and as the obstacle at the left side of the heart is invincible, the evil is aggravated, and either death ensues from dyspnœa, or the obstacle is propagated further back to

the systemic circulation before the fatal catastrophe takes place.

Derangement of the Systemic Circulation, more particularly that part of it which passes through the brain, has been considered by some to arise from an over-supply of blood forced into the capillaries by an hypertrophied *left* ventricle; also from attenuation and softening of the walls; but more particularly subsequent to pulmonary congestion produced by valvular disease on the left side of the heart, as alluded to in the last paragraph. It has not been denied that tricuspid regurgitation, if *strongly* marked, might cause it, but this affection having been considered to be of very rare occurrence, it has hardly been taken into consideration as a cause of cardiac dropsy.

Dr. Hope, however, considered dilatation of the heart to be a more direct and efficient cause of derangement of the systemic circulation than valvular disease. He remarks, "I have repeatedly witnessed cases in which a well marked, if not a considerable obstacle, as a contracted valve, a regurgitation, or a dilatation or aneurism of the aorta had existed for a long period, even for years, without producing any material symptoms of an obstructed circulation; but the moment that dilatation of the heart supervened, the symptoms made their appearance in an aggravated form." The truth of this remark will be attested by all those who have had extensive opportunities of examining this class of cases. Dr. Hope considers the attenuation and consequent weakness of the walls of the ventricles, whether dilated by the pressure of obstructed blood, or by the debilitating effects of fever, chlorosis, inflammation, &c., to be the cause of engorgement of the systemic circulation. This explanation is satisfactory as far as it goes, but it does not apply to those cases of frequent occurrence in which hypertrophy is joined with dilatation. Andral endeavours to meet this case by the supposition that "the impediment arises from the excess of the capacity of the heart relative to that which has been preserved in the

blood-vessels." Cases, however, were known in which hypertrophy and dilatation had existed for many years without giving rise to any symptoms of impeded systemic circulation. It seemed probable, therefore, that there was either some cause of obstruction connected with dilatation undiscovered, or that one of its known causes had been overlooked.

CHAPTER X.

PROGRESS AND TERMINATION OF HEART DISEASES.

Analysis of Cases of Heart Disease. — DISTURBANCE OF NEITHER CIRCULATION. — PULMONIC CONGESTION. — *Hypertrophy—Dilatation—Attenuation—Fatty Degeneration—Aortic Valvular Disease—Mitral Valvular Disease.* SYSTEMIC CIRCULATION. — *Hypertrophy — Dilatation — Aortic Valvular Disease—Mitral—Pulmonary Artery—Tricuspid—Inflammatory Affections of Tricuspid Valves —Adhesion—Shortening and Thickening—Dilatation of Tricuspid Orifice —Regurgitation the constant Cause of Dropsy—Not necessarily Fatal—General Conclusions.*

SUCH were the views generally held twenty years ago concerning the progress and termination of organic diseases of the heart; some of them purely theoretical, and on the whole leaving unanswered several questions of great practical importance; amongst them the three following:—
1. The effect of hypertrophy on the circulation. 2. The mode by which the circulation is successfully carried on when there is excessive narrowing of the aortic orifice. 3. The direct cause of cardiac dropsy, when dilatation with or without hypertrophy is the only apparent lesion.

Hence the necessity for re-opening the subject by examining the results of a long course of clinical observations. Accordingly 155 fatal cases of heart disease were submitted to analysis, and the results were embodied in a Table (II) here republished. Of these 37, in a condensed

form, are scattered through these pages, being brought forward in illustration of various points in pathology, diagnosis, and treatment. Although they are included in Table (II), yet they are also placed by themselves in Table (I), in order that all the various affections that took place in each case may be seen at a glance. For the purpose of facilitating the inquiry into the cause of cardiac dropsy, which is a point of the greatest practical importance, the cases have been divided into two groups, respectively styled the *pulmonic* and the *systemic* group, the former excluding, and the latter including, cardiac dropsy.

TABLE I.
THIRTY-SEVEN CASES OF FATAL HEART DISEASE.

Number of Case	(1) PULMONIC GROUP.													(2) SYSTEMIC GROUP.																								
	87	90	84	46	28	88	33	56	44	36	37	38	27	41	42	43	93	99	91	100	58	89	92	46	29	31	39	40	93	94	95	47	48	49	85	35	34	
Rheumatism
(3) Pericarditis
(4) Pulmonic congestion
(5) { Systemic congestion (anasarca)	
{ Systemic congestion (cerebral)	
{ Aortic regurgitation	
{ Aortic obstruction	
{ Mitral regurgitation (disease)	
{ Mitral regurgitation (dilatation)	
{ Mitral obstruction	
{ Pulmonary artery regurgitation	
{ Pulmonary artery obstruction	
{ Tricuspid regurgitation (disease)	
{ Tricuspid regurgitation (dilatation)	
{ Tricuspid obstruction	
{ Dilatation of left ventricle	
{ Dilatation of right ventricle...	
{ Hypertrophy of left ventricle	
{ Hypertrophy of right ventricle	

(1) The *Pulmonic Group* includes all the cases in which there was neither *anasarca* nor *cerebral congestion*.

(2) The *Systemic Group* includes those in which *one* or *both* of these states existed.

(3) AD denotes *Adhesion of the Pericardium*.

(4) In this column PA denotes *Pulmonary Apoplexy*. E denotes *Emphysema*.

(5) In this, CA denotes *Cerebral Apoplexy*.

TABLE II.

One Hundred and Fifty-five Cases of Fatal Heart Disease.

	Pulmonic group.		Systemic group.	
Rheumatism		8		46
Pericarditis (partial)	6	12	17	30
" with adhesion of pericardium ..	6		13	
Pulmonic congestion	24	31	34	53
Pulmonary apoplexy	7		19	
Systemic congestion (anasarca)	102	109
" (with cerebral apoplexy	7	
" (cerebral apoplexy alone)	3	3	7	7
Incomplete aortic orifice (alone)	9	17	16	22
" aortic and mitral orifices	8		6	
" mitral orifice (alone)	16	24	37	43
" pulmonary orifice	*4		1	
Tricuspid regurgitation from disease alone ..	1	5	5	106
" from disease and dilatation	4		41	
" dilatation alone	0		60	
Dilatation of left ventricle alone	10	25	9	92
" of left and right ventricles	15		83	
" right ventricle alone	3	†18	22	105
Hypertrophy of left ventricle alone	7	19	13	45
" left and right ventricle	12		32	
" right ventricle alone	1	13	2	34

An examination of these Tables gives a tolerably clear idea of the manner in which various affections of the heart combined with each other to produce derangements of the health, and eventually death; and will be found to furnish satisfactory answers to the question above stated.

Bearing in mind, then, the steps by which these results are reached, let us successively look for the lesions which took place when *death occurred without disturbance of either circu-*

* In three of these cases there was open foramen ovale.

† In 15 of these cases there was also dilatation of the tricuspid orifice; but in 12 of them the valves were so large that they closed the orifice and prevented regurgitation, and in the remaining three the foramen ovale was open.

lation, and when it resulted from *pulmonary* congestion, and from *systemic* congestion.

DISTURBANCE OF NEITHER CIRCULATION.—Cases in which death has occurred without any marked signs of disturbance of either circulation having been observed, are not likely to be found in these tables; because they are almost exclusively drawn up from the records of hospital and private consultation practice, in which sudden death would very seldom come under notice. In some of these here recorded death was caused by cerebral apoplexy, in others by syncope, but in none without some symptoms of derangement of one or other of the circulations having been observed. In two or three cases resembling (88), however, they were very slight. No attempt, therefore, will be made to draw any conclusion in respect to this class of cases.

PULMONARY CONGESTION occurred in 31 cases in the *pulmonic* group of 39, and in 53 cases of the *systemic* group of 116, in all 84.

Hypertrophy and Dilatation.—In 23 of these cases pulmonary congestion was probably the immediate cause of death, and amongst them hypertrophy of both ventricles was found in 3, of separate ventricles in *none*. In the 31 cases of the *pulmonic* group, there was double hypertrophy in 10, of the left alone in 5. In the 10 cases there was disease of the aortic valves in 3, and of aortic and mitral joined in 4, leaving only 3 of uncomplicated hypertrophy of the right ventricle.

Amongst the 53 cases of pulmonary congestion occurring in the *systemic* group, there was double hypertrophy in 29, and of the left alone in 9. In the 29 of double hypertrophy there was mitral disease in 4, aortic in 9—*tricuspid regurgitation in all*. As this last affection would divide the force of the right ventricular systole, and so divert from the lungs any extra pressure which its hypertrophy might have exerted, hypertrophy of the right ventricle cannot be considered to have been in these cases a cause of active pulmonary congestion.

The form of congestion known as *pulmonary apoplexy* was found in 26 out of the whole 155 cases. In 4 of them there was hypertrophy of the left ventricle alone, in 20 of both ventricles, joined with aortic valvular disease in 3, mitral in 4, and tricuspid regurgitation in 17.

So that hypertrophy of the right ventricle was hardly ever found, unless complicated with some valvular defect on the left side of the heart, which acted as a direct cause of pulmonary venous congestion; or on the right side, which diverted the force of the ventricular systole from acting on the lungs; and *hence in these cases it could not be recognised as a direct cause of active pulmonary congestion.*

Attenuation.—In the whole 84 cases of pulmonary congestion, there was attenuation of the right ventricle in 16, of the left in 17, of both in 8, total 41. In the 23 cases of fatal pulmonary congestion there was attenuation of the left ventricle in 10; of the right in 5—total 15. In all these latter there was also mitral disease, so that in all probability the attenuation of the right ventricle was the *result* of pulmonary congestion caused by mitral disease; that of the left ventricle being joined with a more powerful and direct cause (mitral disease), can only be considered as auxiliary.

Fatty Degeneration.—This was found in 17 of the 84 cases of pulmonary congestion; in 9 accompanied by attenuation of the left ventricle, and in 4 by that of both; and with hypertrophy of both ventricles in 4. In all the 13 cases in which there was both degeneration and attenuation of the left ventricle, mitral disease existed.

Valvular Derangements.—The condition of the *aortic* and *mitral* orifices is that which demands our chief attention in connection with pulmonary congestion.

Aortic Orifice.—In the 84 cases of pulmonary congestion the aortic valves were diseased in 13, and also in 17 more in conjunction with mitral disease. Not one of these 13 cases occurred amongst the 23 cases of fatal congestion, and only 3 amongst the 30 cases of pulmonary apoplexy.

The proportion of 13 to 84 is probably not greater than that which would be found to exist in any given number of bodies examined promiscuously. So that *aortic valvular disease must be excluded from the direct causes of pulmonary congestion.*

Indeed, it is remarkable to what an extent aortic valvular disease may exist, and for how long a time, without destroying life.

CASE 29.—*Aortic Obstruction—Tricuspid Regurgitation.*

An iron-caster, æt. 61, had oppression of the chest and indigestion for twelve months. He complained of troublesome cough and distressing dyspnœa. The pulse was very small and thread-like. An obscure thrill was felt to the right of the upper part of the sternum. A single prolonged rasp-sound was heard, its maximum being over the same spot where the thrill was felt.

Four months after this, the pulse in each wrist was reduced to a mere flutter; and in nine months more, the intensity of the rasp-sound was much diminished. The whole of the right side of the chest sounded dull, and the respiratory murmur of that side could only be heard up the spine. He could not lie on the left side. There was extensive dullness around the heart.

In six months the veins of the neck were almost varicose; the ankles became œdematous, and in another month, general anasarca appeared. The urine was not coagulable by heat. A little before his death, which took place nearly two years after my first visit, a slight fluctuation could be occasionally detected in the jugular veins.

Inspection.—The right side of the chest contained a gallon, the left a pint of fluid, and there was a large quantity in the abdomen. The size of the heart was truly astonishing; it projected for some distance under the sternum into the right side of the chest. All the cavities were uncommonly dilated, and the walls of the right ventricle were attenuated. There were only two aortic valves which were hard and calcareous, and stretched tightly across the orifice of the vessel. The free edge of the smaller valve was cartilaginous, so that it could slightly recede, and allow a slit for the passage of the blood from the heart. These valves had long calcareous bases, which extended some distance into the ventricle. The tricuspid foramen measured six inches in circumference; its valves were small, and totally incompetent to close it. There was atheroma in all the great arteries.

The other organs were healthy.

The nature of the disease of the aortic orifice in this case proves that it must have been of long standing, and yet but little inconvenience had been felt until within a short period of death.

It is unfortunate that the size of the mitral orifice should not have been noted and accurately compared with that of the valves; for it is hardly possible to conceive that any valves could have effectually closed orifices so much dilated as these were; and yet they appeared to do so on viewing them. As long as the disease was confined to the left side, as in Case 29, there was no œdema; but when the tricuspid orifice began to dilate, as proved by the turgescence of the external jugular veins, it soon appeared. In Case 46 the progress cannot be thus traced, as these veins were dilated when the patient was first seen. It is very remarkable, that with this extreme venous distension but little anasarca should have been present, so that after death the traces of it had disappeared from the body. It was on this account difficult to refrain from placing this case in the Pulmonic Group.

The adhesion of one of the tricuspid valves must have been of inflammatory origin. Other similar cases will be given, in which no other traces of inflammation beyond the adhesion could be detected. This case tends to prove that strong regurgitation may occasionally take place through the tricuspid orifice, without for a time giving rise to any considerable amount of anasarca.

A very lengthened systolic rasp-sound was heard, which proved that the contraction of the ventricle was prolonged. By this means the blood was squeezed through the contracted aortic orifice, and thus the circulation was kept up, and the stagnation of the blood in the heart and lungs to a certain extent prevented. It will be remembered that a similar sound was heard in Case 38, and was produced by the passage of blood through the narrow mitral orifice.

The same state of things is not unfrequently found in

aged persons, who yet live on without experiencing any great amount of dyspnœa, and who display no symptoms of anasarca or cerebral congestion. The following case affords an instance of this, and is, perhaps, the most remarkable on record.

CASE 30.—*Aortic Obstruction, without Dyspnœa or Dropsy.*

A lady, æt. 65, was thrown from a carriage, and struck the ground with her left shoulder, in the year 1824. Considerable inflammation ensued. She was attended by Sir P. Crampton, and recovered; but from that time she was subject to violent fits of palpitation, which lasted generally twenty-four hours, in one of which I saw her nine years after the accident. The pulse was intermittent, and there was very *lengthened* rasp-sound running up the aorta. The diastolic sound was strong, but muffled. On the fit of palpitation subsiding, the intensity of the sound decreased, but its quality remained the same. I saw her again in 1837, when the fits of palpitation were less frequent and less severe, and the pulse less intermittent.

In 1846, I again attended her at Torquay, in consultation with Dr. Battersby and Dr. Toogood. The palpitations had entirely ceased for five years. She was then in her 85th year. The sound was the same as on the two former occasions.

She remained free from dyspnœa, palpitation and œdema, and died in 1862, aged 101 years, retaining the full possession of her faculties!

Many other instances might be adduced of old persons presenting all the signs of obstruction at the orifice of the aorta, and who are either still living without dyspnœa or dropsy, or else who have died of some other complaint unconnected with the lesion in question, which had hardly been suspected before death, but which was revealed at an inspection of the body.

When the valves are so shortened, or the orifice is so dilated that *regurgitation* takes place during the systole of the heart, a greater effect is produced on the venous circulation than when obstruction alone is opposed to a current of blood through the aorta. For in this case no increased power in the ventricular walls can prevent the reflux of the blood from the aorta during their diastole,

which meeting the current that is flowing from the auricle, offers a great obstacle to it, and thus produces engorgement of the pulmonary veins. But the aperture through which regurgitation takes place from the aorta must be large materially to affect the circulation, because the auriculo-ventricular foramen, through which the blood enters the ventricle during its diastole, is naturally so ample. The following cases illustrate the truth of this remark:—

CASE 31.—*Aortic Regurgitation—Dilatation of Aorta.*

A miner, of sallow complexion, æt. 27, had articular rheumatism six years ago, after exposure to wet and cold, but the joints did not swell. He was confined fourteen days by this illness, and has ever since felt a dull, aching pain at the left of the epigastrium, from which he has sometimes been relieved by leeches. He had palpitations for twelve months. His breath was good, and he had walked up to within a day or two of his admission into the Birmingham General Hospital; but for the last fourteen days his epigastric pain and palpitation had increased. There was not the slightest dyspnœa or cough. The impulse of the heart was strong and heaving from a point two inches below the left nipple to the xiphoid cartilage, and even below it and around it. Strong pulsation and purring thrill were felt up the carotid arteries, over each clavicle, and more especially over the top of the sternum, where the aorta seemed to rise up within reach of the finger. Pulsation was also felt under the humeral end of the clavicles, but not under the sternal ends. The pulse was 76, sharp, quick and strong, generally resilient and double, the second stroke being very feeble. It was very visible at the wrists.

There was no dullness over the usual precordial space; but it commenced two inches below the left nipple, and, like the impulse, extended obliquely inwards and downwards towards the xiphoid cartilage. The respiratory sound was rather feeble, particularly behind the left side. A loud systolic rasp-sound was heard in the course of the carotid arteries, and on the sternum. To the right of the top of the sternum a double rasp-sound was heard, the systolic being the loudest; at the bottom of the right of the sternum it was still heard double, but the diastolic sound was the loudest and the longest. The double sound was also heard at and under the left nipple, but very soft and faint. After a few weeks, he left the hospital much relieved. He ceased

working in the pits, and set up a horse and cart. He has been seen occasionally during the last three years; the palpitations are less severe and less frequent, but the physical signs remain unaltered.

CASE 32.—*Aortic Regurgitation.*

An iron-roller, æt. 25, at no time very strong, but accustomed to hard work, had enjoyed good health until five years ago, when he began to be troubled with occasional palpitations without cough or dyspnœa. Nine months ago he caught a cold, and has had a bad cough ever since, accompanied by expectoration, in general thin, clear, and frothy, at times thicker and more viscous, but still light-coloured. At first he spat a little clear red blood, not amounting altogether to a dessert-spoonful. Two months since a little œdema of the ankles appeared, and the palpitations increased in frequency and severity, and dyspnœa set in, which has been increasing ever since. He has had no pain all along. Five weeks ago he was obliged to desist from work, and although he tried to work again after a little rest, the severity of the palpitations and dyspnœa compelled him to desist. He has lost flesh greatly during the last two months.

On his admission into the Birmingham General Hospital his complexion was yellowish, transparent and waxy. He had no pain, but felt weak across the loins, and had a dry cough which troubled him much at night. The palpitations were less severe than they had been. There was a trace of œdema about the ankles. His appetite was good, his bowels were open, and his urine was healthy and plentiful. On placing the finger on any of the arteries, a sharp, quick blow was given to it, like a drop of water fallen in vacuo. They were seen to beat wherever they were superficial. Respiration 30. The pulsations of the heart were visible over the whole of the left side of the chest. A heaving pulsation and a little thrill were felt by the finger pressed down behind the sternum. The sound over the chest was nowhere very clear, but was very dull all around, below, and outside of the precordial region. A fine crepitation was heard behind the lower part of each side. The head was raised by the stroke of the heart against the stethoscope. Two inches below the left nipple, in an oblique outward direction, a systolic, shrill rasp-sound was heard, with a slight trace of diastolic sound. This sound decreased above the nipple, but was heard all up the sternum and to the right of it without any diastolic sound. Under the lower part of the sternum it was double. A loud single rasp-sound was heard behind both sides, particularly near the lower angle of the left scapula.

In a fortnight's time some œdema of the feet came on, but no pulsation of the veins of the neck could be discovered.

He left the hospital and was not seen until very lately, when he was considerably better than at the time he was in the hospital, but the physical signs remained much as they were.

The signs of aortic regurgitation were present in both these cases, and it is further probable that the dyspnœa in Case 32 was caused by mitral regurgitation in addition, and yet both these persons lived on in a tolerable state of health.

It therefore appears that a considerable amount of disease of the aortic orifice may exist for a number of years without seriously affecting the health, more particularly if hypertrophy of the left ventricle is joined with it, and that, consequently, this affection has little or no direct influence in retarding the venous circulation and producing engorgement of the vessels of the pulmonary or systemic circulation.

These cases furnish us with an answer to the question as to the mode in which the circulation is carried on when there is excessive narrowing of the aortic orifice. An unusually *prolonged* rasp-sound was heard, commencing with the systole of the heart, proving that the muscular contraction which gave rise to it was also *prolonged*, by which means a kind of compensation was effected; an amount of blood being thus *squeezed* through the contracted aortic orifice, which could not have been sent by the ordinary systolic action. This, of course, was effected by the ventricle having become hypertrophied.

Mitral Orifice.—In the 84 cases of pulmonic congestion there was disease of the mitral orifice in 67, 30 of them being obstructive only, and 37 of them regurgitative. In the 23 fatal cases there were 4 of obstructive, and 13 of regurgitative mitral disease; and in the 36 cases of pulmonary apoplexy there were 4 of obstructive, and 9 of regurgitative mitral disease. It follows, therefore, that mitral regurgitative disease was the most frequent and

direct cause of *pulmonary congestion*. A certain amount of congestion is, doubtless, produced by obstructive mitral disease, and yet it may exist to a great extent, and for a long time, without producing fatal or even very distressing symptoms, as occurred in Case 88.

Mitral regurgitation has been seen to arise from shortening, thickening, or adhesion of the valves, and from vegetative growths and calcification, with or without dilatation of the orifice, but rarely from this latter affection alone; for in 117 cases of dilatation of the left ventricle, there were only 17 in which the mitral orifice was notably dilated. Its mean circumference is estimated by Bizot at four inches. In general, the two valves can more than close this space, so that a certain amount of dilatation may exist without giving rise to regurgitation. It should be mentioned also that in a few cases, although the mitral orifice was dilated, the valves were large in proportion, so that they effectually closed it, and thus no regurgitation took place.

SYSTEMIC CONGESTION. — *Hypertrophy*. — Systemic congestion, more particularly as evidenced by anasarca or cerebral congestion, existed in the whole of the 116 cases constituting the *systemic group*. There was hypertrophy of the right ventricle alone in 2 of them; of the left alone in 13; of both in 34.

There were thus 45 cases of hypertrophy of the left ventricle, in 7 of which there was also mitral, in 13 aortic valvular disease, and in 43 tricuspid regurgitation; so that there was hardly a case of systemic congestion in which simple hypertrophy of the left ventricle existed unaccompanied by some other morbid change in the walls or valves, calculated either directly to produce venous congestion, or else to counteract any tendency of the ventricular action to produce arterial congestion. On the other hand, cases similar to that which follow show that excessive hypertrophy may exist for a long time without inducing congestion.

CASE 33.—*Slight Aortic Obstruction—Hypertrophy and Dilatation—Sudden Death.*

A young man, of delicate constitution and slender make, æt. 19, had never enjoyed such an amount of health as would allow him to follow any employment. He had experienced strong palpitations at the heart from his earliest recollection, which were much increased by exertion, and were attended with a pain and throbbing up his neck to his head. He had generally some cough, with slight and clear expectoration. On one occasion he spat up three or four table-spoonfuls of blood after walking quickly. The pulse was full, hard, and resilient. The action of the heart was heaving, and was seen all over the chest, and all the arteries near the surface were observed to pulsate violently. A strong pulsation was felt over each clavicle, and over the sternum on pressing down the finger. A thrill was also felt up the right carotid, over the right clavicle, and behind the sternum on deep pressure. There was extensive dullness around the region of the heart. The cardiac sounds were distant and confused when listened to over the precordial region. At the top of the aorta, and up the right carotid, was heard a double rasp-sound. The respiratory sound was natural.

He was seen occasionally both by Mr. Alfred Baker and myself until the time of his death, which took place suddenly rather more than three years after this. He had been sleeping in his chair, when he awoke and suddenly placed his hand on his heart. He dropped his head and died, his sister declaring she heard a noise at his heart.

Inspection.—The body was opened under a coroner's warrant, much against the wish of his friends, and to satisfy them a promise was given that the chest only should be examined.

There was some little serum in both pleural cavities, more in the left than the right. The bottom of the left lung was carnified. The pericardium contained between three and four ounces of serum. The heart had some large milky patches on it, and was of enormous size, the bases of its ventricles measuring fifteen inches and a half round, and the left ventricle nine inches from its apex to its base. The walls were firm and red. The tricuspid orifice was five inches in circumference, and its valves were very large and sufficient to close it. A mass of calcareous matter adhered to the base of one of the sigmoid valves, not quite so large as a small horse-bean.

The heart in this case had no appearance of disease, but seemed rather the healthy heart of a giant. There is every reason to believe that it was unnaturally large from birth.

Here were the conditions which have been supposed by Andral to be peculiarly favourable to the production of serous effusion, viz., great disproportion between the size of the cavities of the heart, and that of the aorta; and yet no other evidences existed of obstruction of the general circulation than were furnished by the presence of a little serum in the pericardium and thorax. Aneurism of the arch of the aorta was at one time suspected.

The connection between *hypertrophy of the left ventricle* and *cerebral congestion* demand especial attention, because some authors have maintained that the latter affection generally results from the pressure of blood thrown into the cerebral arteries by the violent contraction of the hypertrophied ventricle.

Death resulted from cerebral congestion or apoplexy in 17 out of the whole 155 cases; in seven of them preceded by anasarca. In 2 of them there was extensive mitral disease, and in these 2 there is every reason to believe that tricuspid incompleteness would have been found, had it been looked for. In 12 such regurgitation was seen, so that there remain only 3 uncomplicated cases of hypertrophy of the left ventricle. In the 2 following cases death resulted from cerebral congestion, and yet there was less hypertrophy of the left ventricle than in the case above quoted, where there was no systemic congestion:—

CASE 34.—*Tricuspid Regurgitation—Congestive Apoplexy.*

A railway sub-contractor, æt. 34, had been under my care with symptoms of hypertrophy of the heart, which followed rheumatic fever. There were no signs of any valvular disease. Having been one day engaged during some hours in superintending the laying of rails, and having very often stooped down to run his eye along the line, he was suddenly taken ill, and expired in five minutes.

Inspection.—The heart was red, large, and firm, being hypertrophied and dilated. The tricuspid orifice measured five inches in circumference.

The veins of the brain were gorged with dark blood, an immense quantity of which issued from the jugular veins.

CASE 35.—*Congestive Apoplexy—Diseased Arteries—Tricuspid Regurgitation.*

A coach-harness filer and dresser, of temperate habits, was admitted into the Birmingham General Hospital. His wife stated that five months before his admission he was seized, while at work, with what he described as a weakness in his right arm. This was speedily followed by a fit, in which he struggled violently for half an hour, after which he became delirious, and talked and raved incessantly for three or four days, during which time he could not answer any questions. He was much weakened by this attack, and in ten days' time he complained that his right side and limbs were weak and cold. He partially recovered from this state in the course of a month, but had constant headaches, which did not prevent him from resuming his employment.

A month since he had another struggling fit, succeeded by delirium, which lasted for four days. On recovering from this state, his articulation was slightly affected, his hearing impaired, and his sight rather dimmed, and he had less power in the right than in the left half of his body.

The notes of his state on admission, &c., are mislaid. He died from an attack of cerebral apoplexy.

Inspection.—All the vessels of the membranes and substance of the brain were very full of blood, and those at the base of it were ossified. The arachnoid membrane over the hemispheres was opaque, and the white substance of the brain was of a dusky colour. The upper layers of the floor of each ventricle in the middle were rather softened. An ounce or two of serum escaped from the base of the cranium and spinal cord.

The lungs were pale and bloodless.

The heart was large, in consequence of the dilatation of its right side, while the cavity of the left ventricle, the walls of which were red and firm, and of the natural thickness, could not contain more than an almond. The circumference of the tricuspid foramen measured five inches. The aortic valves were thick and cartilaginous at their bases, but acted well. The mitral orifice admitted two fingers. The aorta was much diseased, being thickened and roughened with atheroma and calcareous scales, as also were the arteries of the neck and arms. In one spot of the aorta the inner coat was totally, and the middle coat was partially, destroyed by ulceration.

Now, the difference in these cases was this: that in the two latter tricuspid regurgitation existed, a most powerful

and direct cause of systemic congestion, in Case 34 being strongly aided by hypertrophy of the *right* ventricle. Although the arterial coats were much diseased in Case 35, yet that was not the cause of death, as no vessel was ruptured.

In case 85 there was fatty degeneration and dilatation; death taking place from the rupture of a cerebral artery, preceded by enormous venous congestion, and tricuspid regurgitation, recognized during lifetime.

It may be inferred, therefore, that cerebral congestion and apoplexy were not directly induced in these cases by hypertrophy of the left ventricle. There can be no doubt, however, that in some cases these affections are indirectly promoted by hypertrophy of the *right* ventricle adding force to tricuspid regurgitation; and also that hypertrophy of the left ventricle might in some degree indirectly add to the congestion, by more strongly opposing the current of regurgitation.

Viewed in its proper light, this inference is quite consistent with what was observed by Andral and others. In fact these cases confirm in a remarkable manner the co-existence of hypertrophy of the left ventricle with cerebral apoplexy. Now, as general hypertrophy of the heart, involving both ventricles, has been found nearly twice as often as that of the left alone, it is highly probable that in the greater number of cases observed by these writers, there was hypertrophy of both ventricles, accompanied as it always is, by their *dilatation*. Again, it will be seen on referring to the tables, that in almost every case of hypertrophy and dilatation of the right ventricle, the tricuspid foramen was also dilated to such an extent as to allow of regurgitation. Had they looked, therefore, for this cause of systemic congestion, they would doubtless have found it; but at the time they wrote, it appears to have been almost entirely overlooked. This will clearly appear when we examine more closely into the state of the tricuspid orifice in its relation to the systemic circulation.

Dilatation.—In the 116 cases of systemic congestion, there was dilatation of the right ventricle alone in 22, of the left alone in 9, and of both in 83. So that either one or both were dilated in 114 cases. The right ventricle was therefore dilated in 105, the left in 92.

Of the 105 cases of dilatation of the *right* ventricle, 66 were without hypertrophy (attenuation), and 39 with it. Of the 92 cases of dilatation of the left ventricle, 40 were without hypertrophy (attenuation), 53 with it.

So that the co-existence of dilatation with systemic congestion in the great majority of cases cannot be questioned.

Of these, the 66 cases of *dilatation with attenuation* occurring on the right side of the heart, are the only ones which can be considered causes of systemic congestion, by favouring the accumulation of the venous blood in the heart, and thus impeding its return from the brain and the capillaries generally. There would then remain 39 cases of dilatation with hypertrophy, and in these cases the cause of systemic congestion would be, so far as we have seen, unaccounted for.

Valvular derangements. Aortic orifice.—In the 116 cases of systemic congestion, there were 22 of aortic disease, in 6 combined with mitral disease, so that their influence on its production was not marked.

Mitral orifice.—There were 43 cases of mitral, combined in 6 with aortic disease. In 34 of them there was also pulmonic obstruction, and in 30 dilatation of the right cavities of the heart. So that mitral disease would appear to act *indirectly* in producing systemic congestion, by acting through pulmonary congestion. The following cases, however, shew that it may proceed to a great extent without doing so:—

CASE 36.—*Mitral Contraction—Pulmonary Congestion.*

A gentleman, æt. 36, had been ill some time, suffering chiefly from dyspepsia and dyspnœa. Shortly before his death he was seen by

Mr. Saunders, when the dyspnœa was very distressing. Hemoptysis of dark coloured blood succeeded, and he died suffocated, having never exhibited any signs of anasarca.

Inspection.—The heart was large, being dilated and hypertrophied; and there was such an amount of calcareous deposit around the mitral orifice as to reduce it to a very narrow slit. The lungs were amazingly congested with dark blood, and some patches of pulmonary apoplexy were found in them.

CASE 37.—*Mitral Obstruction—Pulmonary and Cerebral Apoplexy.*

A female, æt. 39, was in good health when she ran violently with her breast against a post. From that time she felt a pain in her chest, and her breathing became short. Whilst under the care of Mr. Saunders, she had several attacks, both of cerebral apoplexy and of hemoptysis. In one of these attacks of apoplexy she died, having never had any trace of anasarca from first to last.

Inspection.—The lungs were studded with large patches of pulmonary apoplexy. The heart was large, and the mitral orifice was excessively contracted by fibro-cartilaginous and calcareous deposits, so that the little finger could not pass through it.

These cases prove that diseases of the valves of the left side of the heart may proceed to a great length without inducing the slightest trace of obstruction of the general venous circulation. It is probable, however, that congestion of the veins of the general circulation had occurred in Case 37, and that the fatal attack of apoplexy was the result of it. The state of the tricuspid orifice, however, was not noted.

CASE 38.—*Mitral, Aortic, and Tricuspid Regurgitation—Dropsy—Effusion on the Brain.*

A filer, æt. 38, was a patient of the Birmingham General Hospital.

All the notes are mislaid except those of the inspection of the body, and of a clinical lecture delivered on his case. From these it appears that his earliest and leading symptoms were dyspnœa and great pain at the precordial region. His pulse was resilient and small, and was visible at the wrists. A double rasp-sound was heard, of which the systolic portion was very rough, and very much prolonged, and was at

its maximum below the nipple, neither of the sounds extending up the course of the aorta. Subsequently, venous pulsations of the neck appeared, followed by œdema of the feet, which rapidly gained the thighs, scrotum, and abdomen. While on the night-stool he became suddenly comatose, and died soon after.

Inspection.—The vessels of the brain were much congested, and there were four ounces of serum in its ventricles. There was a little serum in the right pleural cavity, and much more in the left. The right lung was congested, the left very much so, and its posterior portion adhered to the chest.

The pericardium was distended with straw-coloured serum. The heart was greatly dilated, and the walls of the left ventricle much hypertrophied. The mitral orifice was nearly obliterated by calcareous deposits, and its valves were short and cartilaginous, not free to act. The aortic valves were short and corrugated. The tricuspid orifice measured nearly six inches round; the valves were thickened. The aorta was dilated, and roughened by atheroma. The liver was large and congested.

The mitral disease was of long standing, but, formidable as it was, no symptom of obstruction of the general circulation appeared, till venous pulsations in the neck announced the accession of tricuspid regurgitation, which arose from the inability of the valves to close the greatly dilated orifice.

The *pulmonary orifice* was incomplete in 4 cases, but in 3 of them there was an open foramen ovale, which would have the effect of dividing the force of the regurgitating blood, and would thus prevent any very great effect being produced on the systemic circulation.

The state of the *tricuspid orifice*, however, was so remarkable as to arrest attention at once. Its valvular apparatus worked so incompletely as to allow of regurgitation in no less than 106 cases out of 116 of systemic congestion, and in 111 out of the 155 including both groups. Of these 51 arose from disease of the valves, with or without dilatation of the orifice, and in 60 from the latter cause alone. As therefore there was tricuspid disease in 51, and mitral in 67, either singly or jointly, with the aortic valves, it follows that the tricuspid valves were nearly as often

diseased as the mitral. But the diseases were very different both in *nature* and *appearance*. Thus the effects of ordinary inflammation on the endocardium and valves probably existed in an equal degree in both cases, but at the mitral orifice atheromatous degeneration, calcification, and vegetative growths were frequently superadded, but only occurred in 3 cases at the tricuspid orifice, in which arterial blood was admitted to the right side of the heart by an open foramen ovale. It must, however, be borne in mind that these results are all taken from *fatal* heart diseases; but had they been taken indiscriminately from the bodies of persons who had died from various diseases, a very different state of things would, doubtless, have been revealed; for incompleteness of the tricuspid orifice is almost always, sooner or later, fatal, whilst disease of the aortic and mitral valves may exist for a great many years without destroying life. Consequently disease of the valves would generally be found much more frequently on the left than on the right side of the heart.

In 10 cases there was adhesion of one valve to the side of the ventricle, of which the following cases are instances:—

CASE 39.—*Tricuspid Regurgitation—Pulmonary Apoplexy—Dropsy.*

A married lady, æt. 29, had not enjoyed good health since an attack of rheumatic fever in early youth. She had for four months suffered from dyspnœa, during which time she constantly expectorated clear mucus, sometimes mixed with dark blood, and had occasional attacks of severe pain in both sides, with fever.

When visited in consultation with Mr. S. Partridge, she could not lie flat; her urine was not coagulable by heat. In about a month after this her legs began to swell, and a slight systolic bellows-sound was heard over the precordial region, when she was hurried or excited. The veins of the neck, which had been always rather full, now became very turgid, and pulsated strongly. The œdema of the legs increased, ascites appeared, and she died, the subject of extensive anasarca, three months after my first visit.

Inspection.—There was a considerable amount of serum in both sides of the chest, and in the abdomen. Some patches of circumscribed pulmonary apoplexy were seen in the lungs.

The heart was more than twice the size of the fist, being hypertrophied and dilated throughout. *Two flaps of the tricuspid valves acted imperfectly, one being partially, and the other entirely adherent to the right ventricle.* The tricuspid foramen was greatly dilated, and its circumference measured five inches and a half. An immense transparent coagulum, of the consistence of jelly, was lying in it, and extended into both the auricle and the ventricle. The other organs were healthy.

CASE 40.—*Tricuspid Regurgitation—Dropsy.*

A girl, æt. 14, pigeon-breasted, had rheumatic fever twelve months since, with very severe pain under the left breast, and distressing dyspnœa.

When visited with Mr. Jones, she complained of great dyspnœa, and occasional severe pain in the precordial region. The urine was not coagulable by heat. On the left side the chest sounded dull from the second rib downwards. A purring thrill was felt under the left nipple. At the same spot a bellows-sound was constantly heard, and for some distance around it. In two months' time the veins of the neck were observed to fluctuate obscurely. In another fortnight the ankles and legs became œdematous, ascites soon followed, and in three months from my first visit she died extensively anasarcaous.

Inspection.—A large quantity of clear lemon-coloured fluid was found in the abdomen, about a pint in each pleural sac, and three ounces in the pericardium. The lower part of each lung was slightly carnified, and they were otherwise healthy.

The heart was dilated to nearly double its natural size, the walls retaining their usual thickness. The valves were not thickened, but a *part of the tricuspid valve was glued down to the side of the right ventricle*, and thereby prevented the closure of the foramen, the circumference of which measured four inches and a quarter. The other organs were healthy.

In 20 cases the chordæ tendineæ were shortened, so that the valves were prevented from rising up into the place of closure, and effectually closing the tricuspid orifice.

CASE 41.—*Adherent Pericardium—Mitral Obstruction—Tricuspid Regurgitation—Dropsy.*

A widow, æt. 55, ever since the catamenia disappeared, five years since, had felt dyspnœa, palpitations on exertion, with cough, and expectoration of clear, grayish, lumpy mucus. Three weeks since she took cold, when her cough became aggravated, and she felt a pain at her heart; the dyspnœa and palpitations became worse. She was unable to lie down for a week, and her legs had swelled, and pitted on pressure for four days. Her urine was scanty.

When visited her pulse was small, irregular, and feeble. There were large undulations visible in the veins of the neck, which were turgid, particularly those on the right side. The dullness over the precordial region was extended. The pulmonary sound was accompanied by bass-viol and cooing sounds, and some mucous rattles. The action of the heart was very tumultuous, irregular, and feeble. The sounds were extended and weak, but natural.

She improved for a time under a generous diet, belladonna frictions over the heart, and tonic and diuretic medicines. But relapsing, she entered the Queen's Hospital. Finding herself getting worse, she again went home.

On being summoned to her I found her deeply jaundiced, labouring under extensive anasarca, and in a dying state.

Inspection.—The lungs were engorged, and there were several large masses of pulmonary apoplexy, with well defined edges.

The pericardium adhered closely to the heart. The heart was large, yellow-mottled, flabby, and soft. The auricles were greatly dilated. The tricuspid orifice measured four inches and a half in circumference, its valves were opaque and thickened, and had *very short shrivelled chordæ tendineæ*. The mitral ring was osseo-cartilaginous, and contracted so as only to admit one finger, the valves were cartilaginous, but would close the orifice.

The liver was rather large, its substance bordering on cirrhosis. It adhered to the abdominal walls.

The progress of disease was well marked in this case also. Obstruction at the mitral valve and shortening of the cords of the tricuspid valves had probably existed for some time; but the pericarditis which led to adhesion having set in about three weeks before the patient was seen, caused a rapid dilatation of the tricuspid orifice, and thus a formidable regurgitation would take place.

In 16 cases the valves were thickened and shrivelled, so as to work stiffly, and so diminished in size as to be incapable of performing their office.

CASE 42.—*Adherent Pericardium—Mitral and Tricuspid Regurgitation—Emphysema of the Lungs—Dropsy.*

A brassfounder, æt. 35, of temperate habits, had a cough, with clear frothy, expectoration, as long as he could remember, with dyspnoea, which was aggravated every winter. Nine months since he experienced pain under his heart, and strong palpitations, which were quickly followed by a diminution in the quantity of his urine, by œdema of the feet and ankles, and by urgent dyspnoea, which, as also the palpitations, were much increased by work, or any violent exercise. All these unpleasant symptoms were relieved by his club surgeon, Mr. Cartwright, and the œdema disappeared. Four months since they returned, and on applying to me he was again relieved by tonic and diuretic medicine, but I have no notes of his state at that time. For the last six months dyspnoea had greatly increased.

On admission into the Birmingham General Hospital he was seen to labour under general anasarca. His face was puffed and had a livid tint, particularly the lips; his eyes were rather injected. He complained of pain at the epigastrium, and of a very troublesome cough, which disturbed him at night, and was attended with copious expectoration of mucus. His pulse was 90, small, feeble, and rather jerking. The urine was scanty, turbid, and contained much lithate of ammonia, but no albumen. The veins of the neck were very turgid, particularly on the right side, and exhibited broad undulations synchronous with the systole of the ventricles of the heart.

Percussion elicited a very clear sound all over the chest, the usual precordial dullness being imperceptible. The pulmonary sound was very faint, mixed here and there with bass-viol sounds, and small, fine muco-crepitant rattle. The impulse of the heart was moderate. A weak systolic rasp-sound was perceived, and was at its maximum around the left nipple, but was not heard at the top of the sternum. He died in three days.

Inspection.—The veins of the neck and the lungs were gorged with blood, but there was no pulmonary apoplexy. There were patches of emphysema interspersed through the lungs, more particularly along the free margin, which overlapped the heart.

The pericardium adhered to every part of the heart by cellular tissue and clear gelatinous matter; it was, however, easily separated by the

finger. The heart itself was not much larger than usual. There were patches of atheroma in the aorta. The corpora Arantii of the aortic valves were thickened and enlarged, more particularly one of them, which projected considerably. The apices of the mitral valves were much thickened and corrugated, and one of them was only a quarter of an inch deep, and was divided into three or four portions, each with a separate cord, thus resembling a tricuspid valve. The tricuspid orifice nearly admitted all four fingers and the thumb, and measured six inches and a half in circumference. *The valves were thickened and corrugated at their free edges*, and attenuated at their bases. They were much shortened, the depth of one being less than an inch, of another half an inch, and that of the third still less.

The pericardium being so easily separated from the heart had probably not long become adherent. The state of the mitral valves could hardly have been produced otherwise than by original malformation, which would account for the habitual dyspnœa from birth. When dilatation came on, the mitral and tricuspid orifices would both admit of regurgitation, and hence the formidable train of symptoms. It is remarkable that the patient should, under these circumstances, have been twice so much relieved.

CASE 43.—*Bronchitis—Mitral and Tricuspid Regurgitation*
—*Dropsy.*

A gardener, æt. 74, with fine ruddy complexion, and of shortish stature, had enjoyed uninterrupted good health until twelve months ago, since which time he has taken cold upon every slight exposure to the weather. During the last three months he has suffered from dyspnœa, constriction of the chest, orthopnœa, and cough, at times suffocating, with scanty muco-purulent expectoration. Within ten days his legs began to swell, and on his admission into the Birmingham General Hospital they were much enlarged, and pitted deeply on pressure. His bowels were regular, and he passed a pint and a half of urine daily. His pulse was under 70. The radial artery was tortuous, and felt like a cord. The chest sounded clear on percussion, except that the precordial dullness extended rather lower than usual. The pulmonary sound was accompanied by a mixture of bass-viol and cooing sounds, and mucous rattles. The impulse of the heart was feeble and slow. Both its sounds were prolonged and muffled, and were not heard beyond the precordial region.

In spite of treatment his symptoms became aggravated. The dyspnœa and orthopnœa increased greatly. His nights were restless. Besides general anasarca, there was ascites, and effusion into the left pleural cavity was detected by dullness on percussion varying with position, tubal breathing, and tremulous bleating resonance of voice. A gangrenous slough appeared on the right leg, and he sank in less than three weeks after his admission.

Inspection.—The right lung adhered to the side; it was congested in its most dependent parts. The left lung was compressed to half its proper size by two quarts of serum in the left pleural cavity, and was congested. Portions of the lining membrane of the bronchial tubes were congested and softened. They contained much muco-purulent fluid.

There were about three or four ounces of amber-coloured serum in the pericardium. The heart was fatty, and nearly double its usual size from an increase of its left side. The fibre was firm and dense, but was the colour of brown paper. The cavity of the right ventricle was much diminished, and encroached upon by the left. The tricuspid orifice measured five inches and a half in circumference. *Its valves were short, and all connected together by reticulated tendinous fibres.* The walls, carneæ columnæ, and septum of the left ventricle were all much hypertrophied. The posterior mitral valve was ossified, and the chords of both were short and thick. The circumference of the mitral orifice was four inches and a half. The aortic valves had lost some of their elasticity, but were not opaque or thickened. The coronary arteries were ossified. The aorta, and, indeed, all the vessels of the arterial system, were lined with atheroma, and here and there by calcareous deposit.

In addition to these changes, vegetations were found on some of the valves; not large, horny, and vermiform, as seen on the mitral valves, but smaller and bead-like, closely attached to their free edges.

In other cases the valves were attenuated and perforated. In some again one of the chords had apparently been broken, and was curled up into a small nodule, like a pin's head. This last affection would probably not much affect the working of the valves, for it has been found in the bodies of persons who have never presented any traces of heart disease, or obstructed circulation. In some of these cases, more particularly in those where a valve adhered to

the ventricle, milky patches, and sometimes lymph were detected close by, indicating the inflammatory origin of this morbid change.

There was a marked difference in the appearance of these valvular affections on the different sides of the heart. Those at the mitral orifice were usually such as prominently to meet the eye; atheromatous-degeneration, calcification, and large vegetative growths being often engrafted on the previously inflamed valves; whereas the adhesion of a valve to the ventricle, or the shortening of one of the chordæ tendineæ, two of the most frequent and powerful causes of tricuspid regurgitation, must generally be looked for to be seen. In Case 39 the observer was on the point of closing what he supposed to be a most unsatisfactory examination, when he caught sight of the adhesion of a tricuspid valve to the right ventricle. His attention having been thus directed to this valve, he observed that the tricuspid orifice was so much dilated, that, had all its valves been free to work, they could not possibly have closed it. He then referred to his notes, and found that venous pulsation had existed during lifetime. In the belief that he had thus discovered the long-sought-for cause of cardiac dropsy in certain cases, he measured the tricuspid orifice and valves in every body which he subsequently examined.

When tricuspid regurgitation was found to result from simple dilatation of the foramen, without disease of the valves, it was in nine-tenths of the cases connected with dilatation of both right ventricle and auricle. The difference in this respect between the two sides of the heart is not difficult of explanation. In the first place the dilatation of the right cavities was carried to a much greater extent than that of the left, although it would be impossible to give the exact relative measure of the two, from the difficulty experienced in taking them accurately. In the second place the fibrous zone of the tricuspid orifice was less frequently affected with disease tending to thicken and harden it than the mitral zone; the inflammation which attacked

the former diminishing the cohesion of its parts, and disposing it to dilate, and not to make them rigid by calcification, which occurs so much on the left side of the heart.

Whatever were the reasons, however, the fact is that dilatation of the tricuspid orifice was found in 91 out of 105 cases of that of the right ventricle. It was not noted unless the circumference of the orifice exceeded five inches in the male, and four and a half in the female. Bouillaud makes its average circumference in the male four inches, and Bizot four and a half. Whilst the greatest reliance may be placed on Bizot's accuracy, the measurements in these and other cases, would lead to an estimation something under four and a half inches.

Now to cover the area of a circle whose circumference varies from four to four and a half inches, each segment should have a height of 8 or 9 lines. If they are of unequal sizes, the joint height of two, or the diameter of the circle, should be from 16 to 18 lines or thereabouts; and if the tricuspid foramen be dilated to such an extent that its circumference reaches six inches, each valvular segment should be about one inch in height. If they are less than this a space is left open in the middle, through which the blood can regurgitate during the contraction of the ventricles. Some allowance must be made for the diminution of the aperture, by contraction of the base of the ventricles, but this probably does not take place to any great extent.

If, therefore, the valves barely close the orifice in health, it is clear that when it is dilated regurgitation must take place during the systole of the heart, unless the valves are proportionately increased in size. This took place in 15 out of 18 cases of dilatation of the right ventricle and tricuspid foramen, which occurred in the *pulmonic group*, and consequently there was no regurgitation, and no systemic congestion.

The two following were amongst them :—

CASE 44.—*Bronchitis and Emphysema—Effusion in the Chest*
—*Dilated Tricuspid Orifice, with large Valves.*

A labourer, æt. 60, had been subject to cough and shortness of breath two winters, and had been in bad health for twelve months, during which time his feet and ankles had occasionally swelled. On his admission into the Birmingham General Hospital his lips were livid and his face bloated. He laboured under excessive dyspnœa, with orthopnœa. He had cough, attended with scanty muco-purulent expectoration. The skin was cool, the pulse not frequent, but firm and quick. The bowels were regular. The urine was scanty and loaded with lithates, but contained no albumen. He had occasional palpitations and considerable œdema of the feet and ankles. The external jugular veins were tumid, and slight undulations were seen in them. The precordial dullness was much more extended than usual; and the chest was bombed out over this spot. The pulmonary sound was masked by loud cooing sounds and mucous rattles of different kinds. The action of the heart was slow and heaving. Its sounds were limited to the precordial region, and were distant and muffled. Ten days after this his urine was increased, and the œdema of the legs was nearly gone, from his having kept a horizontal position, and having taken diuretic medicines. But dyspnœa had greatly increased. He lay on his left side, with his head raised, not being able to lie on his back or right side without a sensation of suffocation. His face was very much puffed and very livid; his cough frequent and dry. The left side sounded dull below the fifth rib, varying with position, and here only an obscure gurgle was heard on deep inspiration, but there was no other respiratory sound. There was bleating resonance of the voice along the margin of the fifth rib. Above this rib, on the right side, the pulmonary sound was coarse, and mixed with mucous and muco-crepitant rattles. The impulse of the heart was very feeble, and the sounds distant and prolonged. He died from dyspnœa in four days more.

Inspection.—The left pleural cavity contained a pint and a half of serum, and the right half a pint. The free edges of both lungs were studded with patches of emphysema, and their posterior parts were congested. The left lung was somewhat dense and compressed. The lining membrane of the bronchial tubes was livid, softened, and thickened irregularly. The pericardium contained ten ounces of serum. The heart was large, and weighed twenty-eight ounces. All its cavities were somewhat dilated. The walls of both ventricles and the carnæ columnæ were slightly hypertrophied. There was much

atheroma in the aorta. The aortic valves were a little thickened at their bases. The tricuspid foramen measured five inches and three-quarters in circumference, but the *valves were unusually large*, being on an average nearly one inch in height. There was no serum in the abdomen.

The first and principal diseases in this case were doubtless bronchitis and emphysema of the lungs, and the pulmonary congestion attending them was the cause of the dilatation of the right side of the heart. As the tricuspid valves were uncommonly large, there would be no regurgitation, but yet there were undulations of the jugular veins.

CASE 45.—*Mitral and Aortic Obstruction—Bronchitis.*

A widow, æt. 50, had always laboured under delicate health, and for some years had experienced dyspnœa on exertion. Latterly this had much increased, and was followed by pain in the head and sickness, and then by palpitations and occasional diarrhœa. When visited in consultation with Dr. Skerrett, she had a troublesome cough with muco-purulent expectoration. There was some œdema of the ankles. The tongue was clean and the bowels regular. The urine was turbid, and not coagulable by heat. There was no pulse perceptible at the right wrist, in the left it was frequent.

The precordial dullness was extensive. On the right side of the chest there was a mixture of cooing sounds, and slightly ringing mucous rattle. The sounds of the heart were distant and confused; the diastolic sound having none of its usual clearness. There were no venous pulsations in the neck. In a few days' time she died from increasing dyspnœa.

Inspection. Both lungs were universally adherent to the chest. They were much congested, and the lining membrane of the bronchial tubes on the right side was in many places swollen, livid, and softened; they contained much muco-purulent fluid.

The heart was dilated, particularly on the right side, but not hypertrophied. The *tricuspid valves, however, were large, and supported fluid, although the orifice was upwards of five inches in circumference.* The mitral valves were fibro-cartilaginous, and their bases adhered to each other, so that one finger only could be introduced between them. On the auricular side of one of them was a softish mass of semi-organized fibro-cartilage. On the lining membrane of the auricle were old patches of white lymph, and also recent bloody

patches. The bases of the aortic valves were ossified, and numerous patches of atheroma were found in the aorta. The *arteria innominata* was nearly obliterated by similar deposits in its middle coat, and between it and the lining membrane.

The kidneys were yellowish, and slightly lobulated and mottled.

The right side of the heart being considerably dilated in this case, and with it the tricuspid orifice, from the naturally small size of the tricuspid valves, it might have been expected that a portion of the dilated orifice would have been left uncovered by them; they were, however, unusually large in this case, and effectually closed the tricuspid foramen. Death was produced by bronchitis occurring in lungs previously congested by mitral obstruction.

Thus the valves were increased in size in 15 cases out of 121 in which the orifice was dilated; a very different result from what Hope supposed took place, as he conjectured they "generally increased in size with the dilatation of the orifice," and that consequently such dilatation did not induce systemic congestion by means of tricuspid regurgitation.

Although the accuracy of Mr. W. King's experiments may be allowed, the soundness of his deductions from them may fairly be questioned, viz., that regurgitation acts as a safety valve to relieve the lungs from arterial congestion produced by a too vigorous action of the right ventricle. For it has been seen that congestion, both pulmonic and systemic, arises not so much from the force and amount of the current of arterial blood thrown into the lungs or capillaries, as from obstruction to the blood leaving them; and that consequently any impediment to the *venous* circulation must induce it. Now, what can be a greater impediment to the blood returning from the general circulation, than regurgitation through the tricuspid orifice, by which the advancing stream is met by another coming from an opposite direction? in the same manner as a high tide meets the current of a river, and causes it

to overflow its banks. And what, again, can be a greater obstacle to the blood returning from the lungs than regurgitation through the mitral foramen?

We have seen that the tricuspid valves were diseased, or that the orifice was dilated to an unusual size in 126 cases. In 15 of these the valves had increased in size so that they perfectly closed the foramen during systole, and there was no regurgitation. In 3 more there was an open foramen ovale, which divided the force of the ventricular contraction with the venous current, and in these also there was no regurgitation. There remain, therefore, 105 cases of tricuspid regurgitation occurring in 116 cases of systemic congestion.

Now, if under these circumstances tricuspid regurgitation be not recognised as the most direct and constant cause of systemic congestion, it must be shown that it frequently takes place without producing such an effect. Dr. Walshe quotes one case of this kind, but does not state the size of the tricuspid valves. In the following case, dilatation of the tricuspid orifice was joined to disease of its valves, one of which was glued to the wall of the ventricle, and strong venous pulsations were recognized during lifetime; and yet only slight œdema of the ankles was visible, and after death no traces of anasarca could be found. The patient, however, was carried off by an attack of acute bronchitis, so that the heart disease had not run its full course.

CASE 46.—*Aortic Obstruction—Tricuspid Regurgitation—Slight Anasarca.*

A gun-stocker, æt. 60, a patient of the Birmingham Dispensary, felt palpitations of the heart when in bed a year ago, which occasionally reappeared for six or seven months, and then ceased; great dyspnoea then came on. He had lost flesh for three weeks, and for three days had pain below the left breast. The veins of the neck pulsated most violently, and felt as firm and hard as arteries. The pulse was converted into a tremulous vibration. There was cough, with expectoration of much clear mucus, sometimes streaked with blood. The chest sounded dull about three inches around the heart. One coarse,

hollow, and amazingly prolonged sound was heard synchronous with the ventricular systole, and its maximum was to the right of the lower part of the sternum. Cooing sounds were heard over the chest. There was slight œdema of the ankles and of the legs. Five weeks after this he was carried off by an attack of acute bronchitis.

Inspection.—The body was emaciated, and presented no traces of anasarca. The veins of the neck were very much distended with dark blood. The pericardium was opaque and thickened, and had on its cardiac portion a patch of soft yellow lymph as large as a crown-piece. The heart was very firm, and was nearly three times the size of the fist, from general hypertrophy and dilatation. The aortic valves were unfolded, grown together, and calcareous, so that the passage for the blood was contracted into a small triangular opening, which could barely admit a quill. The aorta was studded with calcareous scales. The arch was slightly dilated, and its middle coat easily torn. One of the tricuspid valves adhered to the right ventricle. The tricuspid foramen measured five inches in circumference. The other organ were healthy.

One or two other such cases have also been seen; but these exceptional cases, so very few in number, cannot invalidate the inference drawn from the large number here examined. The following cases illustrate the course of simple, uncomplicated tricuspid regurgitation.

CASE 47.—*Aortic Obstruction—Tricuspid Regurgitation—Dropsy.*

A wood-turner, æt. 62, had an attack of acute bronchitis eighteen months ago, since which time he has felt a tightness of the chest; for the last nine months dyspnœa, and lately palpitation. Six weeks ago his legs swelled for a few days; this went off, and returned three weeks since. Dyspnœa was most urgent. The jugular veins were seen to pulsate chiefly on the right side, but were not turgid. The urine was not coagulable by heat.

There was dullness, more extensive than usual, around the region of the heart. The pulmonary sound was coarse in places, with slight cooing. The action of the heart was tumultuous, heaving, and irregular. The pulse was irregular, but moderate. The sounds of the heart were feeble and obscure, but on the right side of the sternum the systolic sound was louder, and slightly coarse and prolonged. In two or three days' time he began to expectorate dark, clotted blood. This,

with increasing dyspnœa and anasarca, continued up to his death, which took place three weeks after my first visit.

Inspection.—The legs and thighs were anasarcaous; a little fluid was found in the abdomen. At the inner edge of the back, and lower portion of the upper lobe of the right lung (both of which were otherwise healthy), was a mass of pulmonary apoplexy, of the size of an orange, with abrupt edges. The heart was immense, and measured fifteen inches around the outside of the base of the ventricles. It was generally hypertrophied, and dilated, firm, and red. Within the folds of one of the aortic valves was a patch of semi-osseous matter, which did not prevent the free action of the valves. The circumference of the tricuspid foramen measured full six inches. One valve was large, the other two small. They were roughened by small hard substances, rather less than split mustard seeds, particularly on their free edges. The coronary vessels were large, but healthy, and the aorta was studded with numerous soft elevations produced by atheromatous deposit. The other organs of the body were healthy.

CASE 48.—*Endocarditis—Tricuspid Regurgitation—Dropsy.*

A porter, æt. 32, was attacked with acute rheumatism twelve years ago, and again eight years since, when he had very severe pains in the left breast, troublesome cough, and dyspnœa. Eighteen weeks previous to my visiting him his ankles began to swell, then his legs and abdomen. There was much dyspnœa. The urine was not coagulable by heat. The pulse was hard, sharp, and vibratory. The jugular veins were turgid, and pulsated.

There was dullness on percussion at the bottom of each side of the chest. The systolic sound of the heart was accompanied by marked bellows-sound. In a few days' time intense pain at the precordial region suddenly made its appearance, with most distressing dyspnœa. The pulse rose to 120, and was very sharp, hard, and full. Venesection having been freely employed, and having been joined with the administration of digitalis and mercurial frictions, the acute symptoms were removed; the anasarca and the pulsation of the jugular veins disappeared. The pulse remained very hard and vibratory.

In a few weeks venous pulsations of the neck, rapidly followed by anasarca, reappeared, and he gradually sank.

Inspection.—Much fluid was found in the abdomen and thorax, and four ounces in the pericardium. This membrane was thickened and opaque in patches.

The heart was generally hypertrophied, and dilated to more than

twice its natural size. The tricuspid valves were a little thicker and larger than usual, but could not nearly close their foramen, which would admit four fingers and the thumb nearly up to the knuckles, and which measured nearly seven inches in circumference. The mitral valves were thickened, but acted well. The lining membrane of the left auricle was thickened and opaque, and was studded with several horn-like patches. On one of the aortic valves was a patch of soft, reddish, recently-organized false membrane, of the size of a fourpenny-piece. The lining membrane of the aorta seemed swollen, and puckered unevenly. The other organs were healthy.

CASE 49.—*Tricuspid Regurgitation—Emphysema—Dropsy.*

A labourer, æt. 55, had, during several winters, cough, with wheezing, dyspnœa, and expectoration of pearly mucus; and last winter he had œdema of the legs. In the spring all these symptoms, except the expectoration, disappeared.

When visited at the commencement of another winter, he was found to have been ill for some weeks. There was great œdema of the legs, thighs, and scrotum. His urine was scanty and high coloured, and not coagulable by heat. The respiration was hurried, and he had frequent palpitations. There was dullness on percussion around the heart. Muco-crepitant rattle was heard all over the chest. The sounds of the heart were sharp and clear, and were heard over the whole thorax, but seemed distant when listened to over the precordial region. He died in fourteen days.

Inspection.—The lungs were gorged with serum. Large patches of emphysema existed at the summit of each lung; some cells were of the size of a horse-bean, and were bounded by tough, inelastic walls.

The heart was dilated to three times the size of the fist, its parietes being of the usual thickness. The right ventricle and auricle were particularly large, and the tricuspid foramen measured six inches in circumference. Its valves were small and thin, and could not close the aperture.

The other organs were in a healthy state, except that the lining membrane of the stomach was of an uniform bright scarlet colour, and rather mammillated at its great curvature.

There was no structural change in the valves of such a nature as to interfere with their action in any of these cases; but in every one of them there was dilatation of all the cavities of the heart and of the tricuspid orifice; and

in the first three there was also hypertrophy. In these three cases the dilatation probably arose from inflammation; in Case 49, as in Case 44, from pulmonary engorgement. Tricuspid regurgitation was, therefore, the only cause of the obstruction of the general circulation which was found in these cases.

An examination of 34 cases of cardiac dropsy recorded by Bouillaud, shews that tricuspid regurgitation took place in 22 of them; and that in 7 others the right auricle and ventricle were both dilated; whence, from what we have seen in other cases, we may infer that the intermediate orifice was also dilated, although no express mention is made of it, and that consequently regurgitation took place in these 7 cases also. In 2, the right ventricle was stated to have been much contracted, which state, had it existed during life, would have promoted obstruction. So that of 34 cases, there were only 3 in which there was not either tricuspid regurgitation or obstruction; a proportion very similar to that observed in the cases here recorded.

The announcement of this fact must have occasioned much surprise at the time it was made, and was doubtless received with incredulity by some, as it seemed impossible that it should have been so entirely overlooked by previous observers. In explanation of this, it must be remembered that it was long a stereotyped opinion that there was little or no disease of the valves on the right side of the heart, and that those which have been shewn to exist in these cases were of such an appearance as by no means prominently to meet the eye, unless of one who was looking for them; and that some persons, again, led away by the notion of the tricuspid orifice being a safety-valve in certain cases, would attach but little importance to its simple dilatation.

Be that as it may, it is now more than 20 years since the existence of this direct cause of obstruction of the systemic circulation was shown in a paper read before the Medico-Chirurgical Society; and it is 17 years since

the results of the cases contained in the above tables were made public. Since then there have been opportunities and ample time for disproving the correctness of the inference drawn from them, had it not been sound. It has, on the contrary, however, been fully confirmed. Other causes, such as the poverty of the blood in anæmia, chlorosis, &c., &c., doubtless may give rise to anasarca, more especially when co-existing with some obstruction at the heart, but the existence of one cause does not ignore that of others.

Here, then, we have the solution of the doubt which existed as to the cause of obstruction of the systemic circulation under certain circumstances.

Dilatation is confirmed as the main cause of such obstruction; not, however, as supposed by Hope, because the walls of the ventricle become attenuated, for, in fact, they were as often hypertrophied as attenuated; nor yet, as supposed by Andral, because "there is an excess of the capacity of the heart relative to that which has been preserved in the blood-vessels;" but because it is accompanied by *incompleteness of the tricuspid valves*, in consequence of which a powerful back current is forced against the blood returning from the veins of the general circulation.

It is unnecessary here to trace out the effects which may be produced on the different organs by this powerful cause of impediment to the venous circulation. It can easily be imagined that many diseases may be thus induced; indeed, when it is remembered that dilatation is so often joined with hypertrophy, and when thus united is more frequent on both sides of the heart than on the left alone, it is more reasonable, after what has been seen, to attribute the greater number of attacks of cerebral congestion and apoplexy which occur in connection with diseased heart, as much or more to tricuspid regurgitation than to hypertrophy of the left ventricle.

It is probable also that this impediment to the circulation exists in many other cases which do not terminate fatally. It has been customary to attribute the anasarca that ap-

pears after great depletion, fever, and other debilitating causes, to weakness of the heart alone. It may be, however, that in consequence of this weakness of the ventricles they become dilated, and with them the tricuspid orifice. Indeed, in many of these cases anasarca has been accompanied by venous pulsations in the neck. On the renewal of health and strength this dilatation may subside, and the causes of obstruction cease. This is illustrated in the following:—

CASE 50.—*Cardiac Dropsy — Tricuspid Regurgitation — Recovery.*

A clergyman, æt. 50, having been subject to much worry, fretted a great deal, and became very dyspeptic. Finding that the digestion of his food gave him much uneasiness, he began to eat very sparingly, and to live very low. On Sunday, whilst officiating, he felt the church very hot, and fainted. When he came to himself he vomited a quantity of dark fluid, which he supposed was bile. From that time he became weaker and weaker, and his complexion was very sallow, like that of an old Indian, as he himself described it, and he passed sleepless nights. Œdema of the ankles appeared, and was followed by palpitation of the heart, and anasarca gradually spread upwards. When seen in consultation with Mr. Gabb he was generally anasarcaous, and was troubled with great dyspnœa. The urine was scanty, but not albuminous. He was extremely weak, and could not lie flat. The veins of the neck were seen to pulsate very markedly, but not strongly. Precordial dullness was very slightly increased. The sound of the heart was feeble, and its action weak and irregular. On one occasion a faint systolic bellows-sound was detected. A combination of tonic and ordinary diuretics failing to act on the kidneys, the full dose of infusion of digitalis, with ammonia, &c., was administered, and at the same time he was kept well up with brandy and wine. In a short time the kidneys acted freely, and the anasarca was quickly removed. He was well sustained with generous diet and tonics, and eventually was completely restored to health, and has remained well during the last four years.

Weakness of the heart in this case probably arose from imperfect nutrition, depending on a deficiency of proper food, and greatly impaired powers of digestion. In the

following case it would seem to have arisen from sub-acute bronchitis supervening on the disease existing in a chronic form, and accompanied by a thickened state of the mucous membrane of the air tubes, which induced an amount of pulmonary congestion, which the right ventricle was unable to overcome without dilating:—

CASE 51.—*Feeble Heart—Venous Pulsations in the Neck—Dropsy—Recovery.*

A miner, æt. 66, had been troubled with a cough and shortness of breathing for a year or two, with occasional palpitations. Three weeks since, his legs, and subsequently his thighs and scrotum, swelled. On his admission into the Birmingham General Hospital his urine was scanty, 1024 sp. gr., and contained no albumen. His tongue was clean, and his appetite was tolerably good. The pulse was under 100, soft and feeble, but regular. The veins of the right side of the neck were large, tortuous, and undulated. The respiratory sound was rather feeble. In different parts of the chest, particularly at the posterior parts, bass-viol and cooing sounds were heard, with occasional large, dry crackling.

The heart was seen to beat two inches below the left nipple towards the outer side of the chest, and also at the xiphoid cartilage. At the former spot a soft and rather prolonged bellows-sound accompanied the systole of the heart, diminishing in intensity in every direction. Under the use of tonic and diuretic medicines, and a generous, full diet, the anasarca gradually disappeared, as did the engorgement and undulations of the veins in the neck, and in less than eight weeks he left the hospital, feeling better than he had done for years.

CASE 52.—*Venous Pulsations—Dropsy—Recovery.*

A gentleman, æt. 68, of nervous temperament, had enjoyed good health until within two or three years, and during that time he had applied for advice whilst suffering from influenza. An examination of the chest after his recovery from this attack showed that he laboured under chronic bronchitis with some emphysema. On this occasion dyspnœa was most urgent. He had œdema of the legs and feet. There were strong pulsations in the external jugular veins. His pulse was full and hard. There was slight fluctuation in the abdomen. The respiratory sound was feeble, and mixed with cooing and bass-viol sounds, with here and there some small mucous rattle. At the bottom

of each side it was absent, and there was dullness on percussion. The action of the heart was heaving. An obscure systolic bellows-sound was heard a little to the left of the lower third of the sternum, but not extending in any direction. Simple diuretic medicines produced no effect, but under the use of infusion of digitalis with ammonia he rapidly recovered; the anasarca, dyspnœa, and venous pulsations all disappearing.

Eighteen months after this he had a similar attack, but of a more obstinate nature, his legs being much swelled and very red; and he complained greatly of their itching. The bellows-sound again appeared. After the lapse of some time, and the employment of elaterium, digitalis, &c., the anasarca left him. He has continued in his usual state of health for the last two years.

CASE 53.—*Venous Pulsations—Dropsy—Recovery.*

A lady, æt. 43, was married at the age of 30, but has had no family. Previous to her marriage she was very pale, and was subject to palpitations, dyspnœa, and swelling of the ankles. Menstruation was, at that period, very scanty and irregular. Since her marriage a slight improvement has taken place in this respect, but at times she suffers in an exactly similar manner. For the last four months she has menstruated very slightly, and has suffered much from flatulence and other dyspeptic symptoms. In this state she applied for advice. The sounds of the heart were feeble, but clear, and were heard over every part of the chest. The veins of the neck were slightly swollen. She derived much benefit from tonic and alterative medicines, which in a great measure removed the dyspepsia. In a few months' time she returned, with œdema of the legs and thighs, and a trace of ascites. When she was seen in consultation with Mr. Baynham, the dyspnœa and palpitations were oppressive. The most active diuretic medicines had been employed without the least benefit. The veins of the neck were now seen to be very much more swollen, and a pulsation in them was manifest. The urine was not coagulable by heat.

A small quantity of blood was taken from the arm, and tonics were combined with diuretics. In less than three weeks the swelling and pulsation of the veins subsided, and every trace of dropsy disappeared.

The cause of dilatation in all these cases was most probably subacute bronchitis supervening on chronic bronchitis. Indeed, I had on several occasions attended the

subject of Case 52 for such attacks. It is probable that many cases of dropsy from bronchitis, as they are commonly called, are thus produced.

In the following case anasarca was relieved, although mitral disease remained :—

CASE 54.—*Mitral Disease—Venous Pulsations—Dropsy—Relief.*

A tradesman's daughter, æt. 15, had a severe attack of rheumatic fever, which left her troubled with dyspnœa and palpitations. Dropsy having come on, and not being speedily relieved, her parents brought her to me for advice.

The action of the heart was feeble, and the pulse very small. The veins of the neck were swollen, and pulsated slightly. Undulations were seen between the second and third left ribs, and below the nipple. The precordial dullness extended upwards. A strong and rather coarse bellows-sound was heard below the left nipple, and also near the lower angle of the left scapula, seeming in the latter situation to be close to the ear. Cooing sounds were heard in different parts of the chest. She was relieved, and the anasarca disappeared, the veins of the neck remaining rather full. Some months after this the anasarca returned. Seen in consultation with Mr. Pye Chavasse, the dyspnœa was so great as to threaten suffocation, and the legs and abdomen were amazingly distended. The strongest diuretic medicines failed to act on the kidneys, nor was any relief obtained from various remedies. It was proposed to employ acupuncture on the thighs, but objected to by the parents. In a few days' time large bullæ rose on the legs and burst, the sores putting on a black gangrenous aspect. Much serum ran from them, and in a few days more the anasarca had greatly diminished, and the sores on the legs assumed a healthy appearance. The dyspnœa was much relieved, and the kidneys resumed their functions. In a word, the patient gradually recovered her ordinary health, and is now alive; but the physical signs of valvular disease remain the same, and she cannot do any work.

This is indeed a remarkable case. After witnessing it, who would ever despair of a favourable termination in cardiac dropsy? The physical signs of dilatation of the left auricle and of mitral regurgitation could not be mistaken. Here, then, was a temporary recovery from dropsy,

although mitral regurgitation remained and still remains. The *direct* cause being recognised, it is therefore possible that this formidable sequel of heart disease may sometimes be temporarily arrested, although the *indirect* cause may be altogether beyond reach.

The following conclusions would seem to be fairly deducible from the examination of the above cases :—

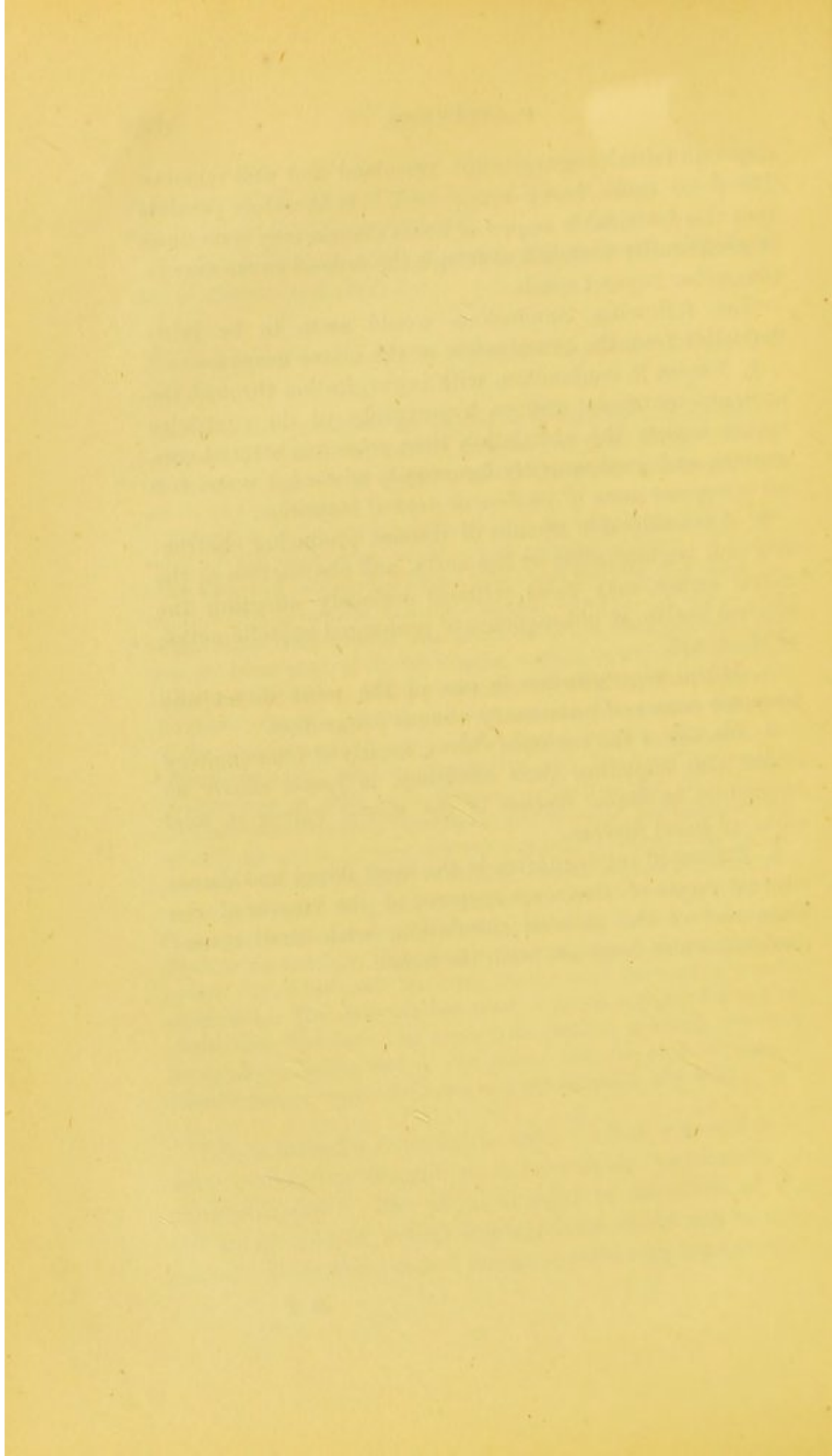
1. Except in conjunction with regurgitation through the auriculo-ventricular orifices, hypertrophy of the ventricles rather assists the circulation than promotes arterial congestion, and consequently *hypertrophy of the left ventricle is not a frequent cause of cerebral or general congestion.*

2. A considerable amount of disease producing obstruction and regurgitation at the aorta, and obstruction at the mitral orifice, may exist without seriously affecting the general health, in consequence of prolonged systolic action of the heart.

3. Mitral regurgitation is one of the most direct and frequent causes of pulmonary venous congestion.

4. Disease of the tricuspid valves, mostly of inflammatory origin, and impairing their efficiency, is found nearly as frequently as similar disease of the mitral valves in *fatal* cases of heart disease.

5. Tricuspid regurgitation is the most direct and almost *constant* cause of that engorgement of the vessels of the brain and of the general circulation, with their consequences, which originate with the heart.



PART II.

DIAGNOSIS.

DISEASES OF THE AORTA, PERICARDIUM, AND
HEART.

THE
BIBLIOTHECA

OF THE
MUSEUM OF THE
CITY OF BOSTON

DIAGNOSIS.

CHAPTER XI.

DIAGNOSIS OF DISEASES OF THE THORACIC AORTA.

AORTIC DISEASE. — ANEURISM. DIRECT SIGNS. — *Pulse — Shape of Chest — Pulsations — Thrill — Percussion — Auscultation — Murmurs — Character of Murmur — Seat of Murmur.* INDIRECT SIGNS *from contact with the Heart, Blood-vessels, Vena Cava, Nerves, Trachea, Bronchi, Pulmonary Tissue, Œsophagus — Aneurism within Pericardium — Aneurism below the Bifurcation of the Trachea — General Conclusions.*

AORTIC DISEASE.—There are no signs clearly indicative of disease of the coats of the thoracic aorta. There is, it is true, a peculiar sharpness and rapidity of stroke in the pulse, which is often associated with diminished elasticity of the arterial coats, which might lead to a suspicion of a diseased state of the thoracic aorta; and again this might be inferred if calcification were discovered in the coats of small arteries that reach the surface; but beyond this there are no diagnostic signs. It was well marked in Case 95. When, however, such disease has been followed by *aneurism* from *dilatation* or *perforation* of the coats of the vessel, or both, various signs, indicative of what has occurred, may be found.

ANEURISM.—The signs of *thoracic aneurism* are of two sorts, *direct* and *indirect*; the former furnished by the aneurism itself, and the latter by the disturbance that is produced in the functions of neighbouring organs.

The *direct* signs are derived from the *pulse, shape of the chest, pulsations, thrills, local dullness on percussion, sounds detected by auscultation.*

Pulse.—It may be easily imagined that when the whole or part of the blood which issues from the left ventricle has to pass through an aneurismal pouch, its motion may be modified in such a manner that a shock of a peculiar nature may be communicated to the finger laid on an artery. Some writers have described the pulse as having a resilient character in certain cases of aneurism, which they attribute to a second impulse given to the blood by the reaction of the aneurismal pouch after its distension. Undoubtedly such a pulse is sometimes felt, and the cause assigned for it is probably the true one; but it is no less certain that a very similar kind of pulse is often found in certain cases of disease of the heart uncomplicated with aneurism. Now, supposing that we were enabled by a very nice sense of touch, and very long practice, to distinguish between the two kinds of pulse, such a sign would be almost valueless from the difficulty of appreciating it. But it is one thing to distinguish between the extremes of the pulse in respect to strength, rapidity of stroke, fulness, and frequency, and another to detect the variations of the pulse which arise as well from aneurism of all sizes and shapes as from certain affections of the walls and valves of the left side of the heart; and the more expert the practitioner has become in detecting the difference of the one from the other, the more will his observation have convinced him that his skill in this respect is of little practical value either as regards diagnosis or treatment. We shall not be surprised at this when we consider the different forms under which aneurisms present themselves to our view; in some cases having thin and elastic, in others firm, inelastic walls; or adhering in

such a manner to the neighbouring parts, as to be incapable of expansion; associated sometimes with a powerless, softened heart, at other times with this organ vigorous, and even largely hypertrophied; some of them nearly filled with layers of solid coagulum, others containing only liquid blood. These opposite states must each affect the pulse in a manner different the one from the other, and in many cases produce effects on it, similar to those which result from certain forms of diseased heart, which it is unnecessary here to describe.*

A valuable sign, however, is sometimes furnished by the absence or comparative smallness of the pulse at the wrists or in the axilla; because this may be caused by the plugging of the origin of the vessel by a clot connected with an aneurismal pouch.

CASE 55.—Dilated Aneurism of the Arch of the Aorta, communicating with the Left Lung, and bursting into the Left Pleural Cavity.

A Thames waterman, æt. 32, accustomed to drink hard, four months since had an apoplectic fit, for which he had been bled, cupped, and blistered, with relief, but was left with impaired motory power of the left side of the body, and a constant dull aching pain on the left side of the head.

Admitted into St. Thomas's Hospital, under the care of Dr. Elliotson, he was found labouring under considerable dyspnoea, which was much increased on exertion. His appetite entirely failed him, and he could not sleep. The bowels were costive. No pulse could be felt in either arm. He was troubled with violent coughing on attempting to lie flat.

* It is probable that when the beautiful sphygmograph invented by M. Marey has been used by a great many observers, the results of numerous observations by it carefully recorded, and the inferences tested by experience, we shall be able to draw most important conclusions of great value, with regard to diagnosis. Papers on this subject will be found in Virchow's Archiv., vol. xxx., in the Physiologie Médicale, De la Circulation du Sang, Paris, 1863, and Czermak's Mittheilungen, Wien, 1864.

The chest sounded dull on percussion over the upper third of the sternum, and over a small space a little to the left of it. The respiratory sound was natural. The action of the heart was strong and heaving. Over the precordial region both sounds were heard, the systolic sound being rather distant and coarse. Between the cartilages of the second and third left ribs a single systolic, hollow, rasp-sound was heard; the intensity of this sound diminished in all directions as the ear receded from this point. On regarding it attentively, a slightly elevated spot, rather larger than a shilling, was perceived, which communicated a strong single pulsation, and a purring thrill to the hand placed on it. One night he expectorated some blood; and a week after his admission, being agitated and excited in conversation, he suddenly fell back, became deadly pale and insensible; and gasping with slow respiration, whilst his pupils were fixed and dilated, for twenty minutes, he expired.

Inspection.—On cutting through the cartilages of the left ribs, a quantity of red coloured serum escaped. The left pleural cavity was found to contain a gallon of the same kind of fluid; and the left lung was imbedded in an enormous clot. The heart was firm and red, and the parietes of the left ventricle were near the base one inch in thickness. The aortic valves were thickened; and patches of a fibro-cartilaginous consistence and atheroma extended up the aorta.

At the commencement of its arch the aorta was dilated into an aneurism, which might have contained a small fist, in which after three or four lines the coats of the vessel seemed destroyed, or at least were not to be distinguished from the indurated lung, which formed a nest for it. At the upper left corner was an opening, communicating with the left pleural cavity, in which was a recent clot. Slightly adhering to the internal surface of the sac, at its upper and anterior portion, was a soft, mottled, laminated, pyriform concretion as large as a date. The arteria innominata was partially, and the left subclavian artery entirely, plugged up with firm clots. Permission was not obtained to remove the diseased parts.

The aneurism was in this instance detected before the tumefaction, pulsation, and thrill were observed. The absence of the pulse in both arms led to a close examination of the chest. The stethoscope was applied over a thin flannel shirt, when to the left of the upper part of the sternum a loud hollow rasp-sound was heard, which at the precordial region was so faint as to be with difficulty heard at all; it could not, therefore, be generated in the heart.

Dullness being perceived on percussion at the same spot, the existence of aneurism, containing some coagulum, which extended into the origin of the vessels at the upper extremities, was rendered almost certain. This latter circumstance also proved that it originated in dilatation. When the tumefaction and pulsation were discovered, it became evident that in one spot the coats had given way.

A very great dissimilarity in the two pulses at the wrists was also observed in Case 21; but there were other signs that led to a careful examination of the chest, which revealed the existence of an aneurism, some of the coagulum from which was found on inspection plugging up the origin of the right subclavian artery.

In the following case, the calibre of the arteries of the arms was reduced by atheromatous degeneration, and not by aneurismal clots:—

CASE 56.—Hypertrophy and Dilatation of the Heart—Large Tricuspid Valves—Diseased Arteries—Sudden Death.

A married woman, æt. 50, had a severe illness eight years ago, with pain in her left side, since which she has occasionally felt pulsations extending from her heart up to her head, and down her arms, with giddiness. She has had latterly a constant cough, and when examined the pulse was small. Pulsations were felt above each clavicle. The action of the heart was heaving and tumultuous. The carotid arteries pulsated very strongly. Double saw-sound was heard, its maximum being under the bottom of the sternum. A systolic saw-sound was heard under each clavicle, loudest under the right, without any diastolic sound.

Two years after this, being excited, she suddenly placed her hand over her heart, dropped her head, and died in half an hour.

Inspection.—The heart was very much increased in size, being generally hypertrophied and dilated, more especially the left ventricle. The tricuspid foramen measured five inches in circumference; but the valves were hypertrophied in substance and extent, so that they effected the closure of the foramen perfectly. The aortic valves were slightly cartilaginous, but acted well. The lining membrane of all the arteries, particularly the aorta, was much diseased, being puckered and

having much atheroma under it, and being deficient in some places. The arteries of the arms were diminished in calibre by the deposition of atheroma in their middle coats. The other organs were healthy.

Here was considerable general hypertrophy and dilatation, without any symptoms of obstruction of the general circulation.

In this case, aneurism of the arteria innominata was at one time suspected. The double saw-sound was not accounted for by any post-mortem appearances.

The sharpness and rapidity of the stroke of the pulse alluded to above, as often associated with loss of elasticity in the arterial coats, although of no value in itself, would, as in the last instances, induce a practitioner who had discovered it carefully to explore the chest, with a view of discovering its cause, and whether there were any traces of incipient aneurism; and knowing that such a disease *might* be developed under these circumstances, he would place his patient on such a plan as would be most likely to prevent arterial dilatation or rupture.

Shape of the Chest.—Although it has been seen that aneurisms, when they reach the surface of the chest, frequently produce an alteration in its shape by forming prominences, the bases of which vary from the size of a shilling to that of a large saucer, yet it must be remembered that such appearances are sometimes produced by other causes.

CASE 57.—*Circumscribed Pleuritic Effusion.*

A young lady, æt. 16, whilst on a visit to some friends, was observed to be troubled with a short, dry cough, and to be short-breathed. At the upper part of the left side of the chest there was dullness on percussion, and an absence of respiratory sound, from which congestion of the lung was inferred by the gentleman who saw her. On her return home she was seen by her usual medical attendant, who found that immediately under the left clavicle there was considerable bulging out of the ribs. Some weeks after this she was seen in consultation. She had been improving in health for some time, and had almost lost her cough, but for the last few days she had been at a stand-

still, and was still very weak. There was now no bulging of the chest, and but little dullness on percussion over the upper fourth of the left side of the thorax, before and behind. In the left scapular region some fine to-and-fro creaking was heard, mixed in two or three spots with the crepitant rattle of pulmonary œdema. As she had been kept for some time in the house, owing to the prevalence of ungenial weather, and the timidity of friends, they were advised gradually to take her into the air, and she soon regained her health.

When the *bulging* of the chest was first observed it was clear that this could not arise from mere congestion of the lung, and a question arose as to the existence of aneurism. In that case, however, the dullness on percussion, and absence of respiratory sound, would hardly have extended into the scapular region, to say nothing of there being no pulsation or murmur. The subsequent progress of the case proved it to have been one of circumscribed pleuritic effusion; and that the lower two-thirds of the left lung had adhered closely to the side, after an attack of pleurisy in childhood, whereby the pleural cavity was limited to the upper part of the side, in which alone any effusion could take place.

CASE 58.—*Mitral Obstruction—Tricuspid Regurgitation—Dropsy.*

A lad, æt. 13, had a severe attack of rheumatic fever when five years old. Four weeks since he felt palpitation, and three days back he felt for the first time a pain under his left breast. When seen with Mr. Bracey he could not lie down, and dyspnœa was distressing. The pulse was small and vibratory; there were strong palpitations under the xiphoid cartilage. From the second down to the seventh rib of the left side, the chest was bombed out an inch beyond the level of the other side. This space gave out a dull sound on percussion, and over it existed a purring thrill. A loud coarse bellows-sound was heard all over the chest.

In a month's time the projection of the left side had much decreased under the employment of iodine and mercurial frictions. A pulsation and thrill were then felt between the fifth and sixth ribs, alternating in time with another between the second and third ribs. In another month the veins of the neck became very turgid, the feet and legs

became œdematous, and ascites followed. The urine was not coagulable by heat. Four months from my first visit he was extensively anasar-cous; the whole of the left side of the chest sounded dull, and the respiratory sound could only be heard on that side close to the spine. He then died.

Inspection.—The heart was enormous, extending up to the second rib, and more than two inches to the right of the sternum. The lining membrane of the left auricle was thickened and opaque, and was covered with patches of a horn-like substance, which extended into the mitral valves. These were much thickened, and would not allow the passage of two fingers; the bases of the aortic valves were also thickened; the right ventricle and auricle were immensely dilated and attenuated; the tricuspid foramen measured five inches and a half in circumference. One of the tricuspid valves had its chordæ tendinæ much thickened and shortened, so that it could not rise into the plane of closure; consequently the valve was incomplete. The other organs were healthy.

The prominence here observed was not such as is usually seen in aneurism, and extended lower down the chest; still, as it was accompanied by dullness on percussion, strong *thrill* and coarse bellows-sound, a suspicion of aneurism was necessarily entertained. The history of the case indicated diseased valves, and as this, with *effusion into the pericardium*, might have given rise to the signs observed, means were taken to promote the absorption of any liquid that might be effused. The subsidence of the prominence proved that such had been the case; and the heart being now allowed to reach the surface of the chest, was seen to be so greatly dilated, that pulsations propagated through the left auricle were plainly visible; and disease of the mitral orifice was recognised as the cause of the thrill, and the coarse bellows-sound.

Of all the sources of error, however, arising out of an observance of a bulging of the walls of the thorax, that caused by malignant tumour in the anterior mediastinum is the most difficult to detect.

CASE 59.—*Cancerous Tumour in Mediastinum and Liver,*
ſc., &c.

A gentleman, æt. 63, had for some months suffered from severe pains in the chest, shooting from under the sternum, supposed to be of a rheumatic character, during which time he had lost much flesh and strength.

On inspecting his chest, all the veins ramifying on it were seen to be unusually full and tortuous. Nothing further was discovered at that time, but in two days, as compression of the superior *vena cava* was suspected, the chest was again closely examined. No abnormal sound could be detected, but on regarding attentively the chest en profile, a slight prominence was seen to the right of the upper part of the sternum. In three or four more days the bulging in this spot was plainly visible, and over it slight dullness on percussion was perceived. The existence of a mediastinal tumour therefore, lying between the surface of the chest and the superior *vena cava*, was evident, and it was predicted that sooner or later there would be œdema of the head, neck, and arms; but whether this tumour was cancerous or aneurismal was left an open question. The tumour increased rapidly, and bulged out over the whole of the upper part of the right side of the thorax; œdema of the head, neck, and arms came on, persisted twelve or fourteen days, and then gradually subsided. Severe pain was then felt in the right hypochondriac region, and the edge of the liver was found to be indented with tuberos projections.

Had any doubt of the nature of the disease existed before, it now disappeared; the tumour was evidently cancerous. The disease of the liver increased to a great extent, and the patient eventually died worn out by the agonies he endured.

The inspection of the body took place in London, and the notes of it were transmitted, but have been mislaid. Large masses of cancerous growth were found in the spots indicated during lifetime, partly of schirrous and partly of encephaloid consistence; and some in other organs.

The *vena cava* superior was lodged in a deep groove situated on the posterior surface of the mediastinal mass.

A tumour of a similar nature existed in the mediastinum and compressed the superior *vena cava* in Case 69; but although it adhered to the upper edge of the sternum it had not caused any prominence.

We have hitherto only referred to projections from the surface of *the chest*, but swellings may appear above the sternum and clavicles, and may arise from aneurism, as in Case 23 above quoted.

The swelling in this case might have been occasioned by an abscess, and it is curious that one should actually have existed during childhood at this very spot, and have left its scar behind it. Such an abscess, if it were in contact with the arch of the aorta, or the origin of the large vessels of the neck, might have propagated some pulsations, but not so *strong* and *liquid* as were felt in this case. The nature of the affection having been recognised, the deposition of coagulum in the sac was most clearly traced by the increasing firmness of the tumour, and by the pulsations becoming more feeble and less liquid. It might have been expected that the sac would have been diminished by this process, but in this case the coagulated fibrin was not firmly attached to its walls, so that the current of the blood was enabled to reach and dilate them.

Neither a bulging of the walls of the chest, nor a liquid tumour above the sternum and clavicle, necessarily arises from the presence of aneurism; so that neither can by itself furnish a sign by which to recognise the existence of this affection; but they serve to direct attention to the spot, and it will probably soon be discovered by other signs what is the nature of the disease which gives rise to them.

Pulsations.—Dr. Hope was of opinion, that “pulsation underneath the sternum or ribs, at the superior part of the chest, was one of the least equivocal signs of aneurism, although not without ambiguity;” that when occurring *below* the clavicle it is produced by sacculated aneurism, and when felt *above* the sternum and clavicles it generally results from dilated aneurism. This opinion was doubtless founded on a consideration of the mode of formation, and the progress of this disease, for when the aorta is dilated the arch is generally involved, against which the current

of the blood impinges strongly; and hence may be inferred a tendency to the production of pulsation *above* the sternum and clavicles. A sacculated aneurism, on the other hand, usually stretches laterally or towards the surface of the chest *below* the clavicles, except in such Cases as 23 and 60; and consequently pulsations are to be looked for in these situations. Such probably would be the case when there are pulsations owing their existence to thoracic aneurism. But experience has shown, that pulsations, both above and below the clavicles, often exist without aneurism, and that aneurisms no less frequently occur without pulsation in these situations. The fact is that hypertrophy of the left ventricle is alone sufficient for their production; and in Cases 33, 56, and others, they were thus produced. On the other hand aneurisms of considerable size sometimes appear close to the surface of the chest without giving rise to any pulsations. In Cases 72 and 26, no pulsation existed, and none were felt above the clavicles in Cases 72, 25, and 24; in all of which an aneurism adhered to the trachea, which might have been expected to have propagated its pulsations upwards. Nor were any perceived, either above or below the clavicles, in Cases 21 and 55, although a pulsation was felt on the spot where the tumour touched the surface of the chest. Out of 32 cases of thoracic aneurism arising without the pericardium, there were only 10 in which pulsations, immediately above or below the clavicles, were perceived, and in 9 of these the aneurisms had reached the surface of the chest at the time of the examination. It is probable, therefore, that some authors have described what they conceived ought to exist, rather than what they have themselves actually observed.

It has been seen that the force of the heart's action is by itself capable of producing pulsations; when, therefore, hypertrophy is joined with aneurism, it necessarily favours their production; and, on the contrary, a feeble and attenuated state of that organ prevents their formation.

This is strikingly illustrated in some of the cases here recorded. Thus in one of two cases in which an equal amount of uniform dilatation of the aorta was found after death, pulsation was distinctly felt on pressing the finger deeply down behind the top of the sternum, and in the other none could be detected. In the former case the heart was large, firm, and red; and in the latter it was in a state of brown, fatty degeneration.

In Case 25, the force of the pulsation was two or three times notably diminished after copious hemoptysis; and in the following case, when the strength was nearly exhausted, and death was approaching, pulsation, once evident to the patient herself, could not be detected; and a rasp-sound which had been distinctly heard by her medical attendants no longer existed.

CASE 60.—*Mixed Aneurism of the Arch of the Aorta compressing the superior Vena Cava—Bronchitis.*

A married female, æt. 55, had long laboured under troublesome cough, with copious yellowish and grey-coloured expectoration, and wheezing respiration, with some dyspnœa. In this state she applied for advice, when the chest sounded clear on percussion, but the respiratory sound was coarse, and was accompanied by cooing and bass-viol sounds, and in some places by uneven, moist rattles. The sounds of the heart were clear, but distant.

She was lost sight of for three months, when she was visited in bed. She stated that she had not been well since she was last seen; that dyspnœa had gradually increased; and that a "beating substance" had risen above the sternum, which had receded during the last week. Her medical attendants, Mr. Green and Mr. Carter, both stated, that during their attendance a softish rasp-sound was heard at the top of the sternum. She was labouring under most distressing dyspnœa, and was not able to assume the recumbent position for a moment. Her breathing was accompanied by a loud whistling, as if the trachea were compressed, and by a large, moist, tracheal rattle. The top of the sternum, for a distance of two inches down, was pushed out and bent forwards. Above this a fulness could be perceived, but no pulsation could be felt. There was expectoration of thick yellow mucus. There was œdema of the head, neck, and arms, but of no other portion

of the body. The sounds of the heart, particularly the systolic sound, were feeble but distinct, and were heard rather louder over the top of the sternum, and over the upper third of the front of the left side of the chest, than in the precordial region, when the breath was held. The upper third both of the sternum and of the front of the left side of the chest sounded very dull on percussion. The respiratory sound was lost in the tracheal rattle. Six hours afterwards, on attempting to lie down, she suddenly expired.

Inspection.—It was found, on removing the sternum, that at its upper part, for two inches in length, it was closely adherent to a subjacent tumour. This portion was therefore separated from the remainder of the bone, and left adhering to the tumour. This was found extending a little to the right of the sternum, but principally backwards, where it lay on and compressed the trachea; and to the left, where it greatly encroached on the upper third of the left lung, and was double the size of the fist. This portion of the left lung was carnified, and of a dark slate colour. On being cut into, numerous drops of thick primrose-coloured fluid exuded from the bronchial tubes. It adhered most closely to the ribs. The lining membrane of all the bronchial tubes was pale and thickened. The heart was of a natural size, rather pale and flaccid. The valves on its left side were slightly thickened, but acted well. The left vena innominata and the superior vena cava were found closely adherent to, and compressed by the tumour, the latter being lodged in a superficial groove on its surface. The tumour was found to be a dilatation of the aorta, which commenced a little above its origin, and was continued to the origin of the left subclavian artery. The coats of the aorta much thickened and corrugated, with deposits of atheroma and fissures, could be traced for some distance, but were deficient at the upper and anterior portion. Here there was an adhesion to the upper portion of the sternum, which was denuded of periosteum. To the right of this and above it, traces of the inner coats of the vessel were also lost, and over the whole of this space there was a large mass of firm coagulum. This coagulum filled half the sac, the remainder of which might have contained six ounces of fluid. A clot of fibrin hung like a flap over the origin of the innominata, and when pressed against it closed it.

The preparation is in the Museum of King's College, London.

There was here clearly a tumour in the mediastinum, compressing both the trachea and the superior vena cava. If the accuracy of the statement of the patient could be relied on, this tumour was an aneurism, very similar to that in Case 24, which had decreased in size, owing to the deposition of fibrinous clot within it. So again, if sound

had at one time been engendered in the pouch, it must have ceased from the decrease of the muscular power of the heart, as she approached her end; for the deposition of coagulum in it would have tended rather to increase than to diminish the murmur. Had the patient been examined at an earlier period, it is probable that a pulsating tumour would have been felt behind the top of the sternum, and a rasp-sound would have been heard in the same situation. As it was, however, the nature of the affection was apparant.

An abscess or mediastinal tumour connecting the aorta or heart with the surface of the chest is said sometimes to give rise to pulsations similar to those occurring in aneurism. Although several such cases are recorded in these pages, the following is the only one in which well-marked pulsations were discovered:—

CASE 61.—*Carcinomatous Mediastinal Tumour.*

A gentleman, æt. 70, had been suffering from dyspnœa for some weeks, occurring latterly in very severe paroxysms, which were supposed to arise from spasmodic asthma. When the paroxysm had passed off he was still short of breath, and the respiration was accompanied by a well-marked *whistling* sound. The trachea appeared to be compressed backwards. Examined with Mr. Penhall, slight dulness was perceived towards the left of the top of the sternum. In this spot a *pulsation* was felt, not powerful or heaving, but rather liquid. The existence of a mediastinal tumour was, therefore, clearly indicated, but it was doubtful whether it was of an aneurismal or carcinomatous character. The probability was in favour of the latter, as there was no murmur or thrill of any kind. If it were so, the tumour must have pushed the trachea backward, and the aorta forwards, in such a manner as to cause it to displace the lung to a certain extent, and to remove it from contact with the surface of the chest. In a few days' time he died from exhaustion, after a violent fit of coughing.

Inspection.—A malignant tumour was found in the mediastinum. It was of an oval shape, about the size of a turkey's egg. It lay between the trachea and the arch of the aorta, adhering to the former and compressing it backwards, and pushing the aorta forwards, thus causing it to displace the lung, and reach the surface of the chest.

Pulsations may, however, be produced by the heart being dilated largely, and the left auricle thus brought up

against the ribs, or by its being connected with the walls of the chest by adhesion with the pericardium.

Thus in Cases 58 and 89 the left auricle touched the surface of the chest between the second and third ribs, and gave rise to *pulsations*. In Case 28 undulations were seen between the seventh and eighth left ribs, caused by a threefold connection between the heart, the pericardium, to which it closely adhered, and between the pericardium and the lung, which latter organ again adhered to the ribs. It is hardly necessary to remark that the seat of pulsation produced by the action of the heart varies with the position of that organ, which has been seen to be temporarily or permanently displaced by pleuritic effusion or other causes. When, however, a pulsation is limited to a small spot at the upper part of the chest, it demands close attention; and when in that spot there is an unusual prominence, it has invariably denoted the existence of a subjacent thoracic aneurism.

The study of the nature or quality of a pulsation thus discovered is of the utmost importance, varying as it does from a dull heaving to a sharp, quick stroke, which gives the idea of fluid almost striking the finger, and which has been here denominated as a *liquid* pulsation. In the former case either the tumour has not fully reached the surface of the chest, or else it is more or less filled with coagulum, which is interposed between the finger and the blood. In the latter case the walls of the pouch at this spot are thin, and the blood impinges directly upon them. Thus by studying the nature of the pulsation we are enabled in some degree to trace the onward progress of the disease, as in the following case:—

CASE 62.—*Sacculated Aneurism springing from the ascending portion of the Arch of the Aorta, and bursting externally.*

A working jeweller consulted Mr. Baynham, and stated that eight months previously he had felt pains in the loins, and that about five months ago he was "much put out," when he felt a sudden snapping

under his breast-bone, accompanied by pain, which had continued ever since, striking from under the right nipple to the back between the shoulders. He also stated that he could not lie on his left side without greatly increasing the pain in his chest.

When visited in consultation with Mr. Baynham, there was a violent throbbing of all the arteries, the pulse being sharp, quick, and rather full; and a strong heaving impulse at the precordial region. A purring thrill was felt to the right of the upper third of the sternum. Over this spot there was a dull sound on percussion. A bellows-sound was heard following as close as possible upon the systolic sound, all over the chest. Ten days after this, he stated that he had latterly felt violent pains in the back, between the spine and the angle of the right scapula. Pulsation was felt where the dullness and thrill existed.

The notes of the case are incomplete, but, nine months after he was first seen, the upper half of the front of the right side of the chest projected forwards, over which part very *liquid pulsations* existed. Over the whole of the swelling a double rasp-sound was heard. He could not support cold applications.

In three months more the projection of the upper half of the front of the right side of the chest had almost entirely receded; but a conical tumour pointed close to the middle of the sternum, a little to the right of the mesian line. Over this spot there was violent pain. He was still unable to bear cold applications, owing to the pain they occasioned. The legs became oedematous. The tumour rapidly increased in size, and its apex became red and shining; blood began to ooze from it, and at length it burst externally, and death ensued.

Inspection.—The upper two-thirds of the right half of the sternum, as well as the ribs and cartilages on that side, from the first to the fourth inclusive, were found adherent to a large tumour, which occupied the upper half of the right side of the chest, and had compressed the upper lobe of the lung into a very small space. The heart was larger than natural, from hypertrophy of its walls, chiefly those of the left ventricle. It was red and firm. The aortic valves were thickened, but acted freely. The internal surface of the aorta was thickened, and studded with many patches of atheroma and horny plates. At the convexity of the ascending portion of the arch, from within an inch of the semi-lunar valves to the origin of the arteria innominata, was a circular opening as large as a crown-piece, which communicated with an enormous pouch, which constituted the tumour above mentioned, and which was the size of a melon, and could contain more than a pint of fluid. Its lining membrane seemed intimately blended with that of the aorta, but the middle coat of the latter terminated

about two inches from the aorta, all round the aperture. In this sac there was no coagulum. A portion of the walls of this pouch consisted of the right half of the upper two-thirds of the sternum and of the costal cartilages, and a part of the ribs which were inserted in it. The greater part of this portion of the sternum was devoid of periosteum, and was much thinned by erosion. The cartilage of the first rib was nearly, and those of the second and third ribs were entirely, separated from the sternum, and from their intercostal muscles. Between the second and fourth cartilages was an aperture of the size of a half-crown piece, which led into another sac external to the sternum, eight inches in circumference and an inch and a half in height. In it were some old, reddish coagula, and some recently clotted blood, and it was partially lined within by a smooth membrane. The integuments were very thin, and had given way at the spot where the dark purple appearance had been observed during lifetime.

The history of this case, coupled with the post-mortem appearances, lead to the belief that the disease commenced by dilatation of the ascending aorta, and was followed by rupture in a fit of passion. When he came under my notice there were evidences of hypertrophy of the heart, and there was a strong presumption in favour of the existence of a sacculated or mixed aneurism approaching the surface of the chest to the right of the upper third of the sternum. This presumption was based on the existence of a purring thrill and dulness on percussion over this spot, on the extent to which the rasp-murmur was heard, and on the fact of its not being exactly synchronous with the impulse of the heart. This was soon converted into certainty by the appearance of a *liquid* pulsation. The sheath and the pleura seem at length to have given way, and the blood passing forward between the ribs, was bounded for a time by the integuments of the chest alone. The murmur was probably formed in the roughened aorta.

Again by the same means we are enabled to trace the formation of coagula *within* the sac, and the consequent retrogression of the disease, as in the following :—

CASE 63.—*Sacculated Aneurism springing from the Ascending Aorta, and bursting into a Tubercular Cavity in the Right Lung.*

A married female, æt. 27, two years ago had a severe illness, which was considered a liver complaint, and for which she was salivated, since which time she had never felt well, and had a constant ringing in her ears, with deafness and a severe cough. At the time of that illness she had great pain under the xiphoid cartilage, which shot up the right arm and shoulder; and also a beating at the heart, which seemed to extend up her throat, and at times interrupted her swallowing. For twelve months she had perceived a swelling to the right of the upper third of the sternum, which had greatly increased in size during the last two months.

Being admitted into the Birmingham General Hospital, dyspnoea on movement was most urgent, and threatened suffocation; she was very deaf, and complained of noises in the head, and of great pain about the precordial region, striking up to the right shoulder and down the right arm; of a troublesome cough, accompanied by white frothy expectoration, and of inability to lie on the left side without dyspnoea and great increase of the cough. Her pulse was small and feeble, but regular and very frequent. Both a pulsation and a purring thrill were felt above each clavicle and above the sternum. To the right of the upper third of the sternum a swelling of the shape and size of half an orange, resting on an outer slightly elevated ridge about two inches wide, was perceived. Over the middle of this tumour a single and very liquid pulsation was felt, and a purring thrill, seeming as if the fluid within the tumour struck the finger at each pulsation through a very thin wall. Nearly the whole of the upper half of the right side of the chest sounded duller on percussion than that of the left side, more especially that portion of it which was occupied by the tumour. The respiratory sound was nearly absent over the upper third of the front of the right side of the chest, and rather blowing and yet more feeble at the upper third of the back part of the right side than over the corresponding portion of the left side. No rattles were detected. The action of the heart was natural, and also its sounds. A well-marked, but not intense, rough, double saw-sound was heard over the tumour, but was much more distinct during the systole of the heart than during its diastole.

She was ordered to be kept perfectly quiet, to have a light, nutritious, but unstimulating diet, including meat daily, to take some camphor and hyoscyamus, and to have a bladder of refrigerating

lotion kept over the tumour as constantly as she could bear it. Under this treatment, with two or three applications of four leeches to the epigastrium and to the right axilla, and with the occasional employment of belladonna frictions over the chest, the dyspnœa and anxiety in a great measure disappeared; the pulse became less frequent, the tumour sensibly decreased, and in four months it was reduced to the size of half a walnut, inclusive of its edges, and in another six weeks the chest was nearly level. What could be then felt of the tumour was firm and hard; the strength of the pulsation was much diminished; it no longer seemed liquid, but felt distant and less sharp; the purring thrill was gone. The saw-sound was become very feeble, and only diastolic. One day she vomited, as she said, a mixture of matter and dirty-coloured lumpy blood, amounting to about an ounce.

She remained much in the same state for nine months, suffering occasionally from severe headaches and paroxysms of great pain in the epigastric region, accompanied by pyrosis and loss of appetite, and acceleration of the pulse, a state which was generally relieved by moderate doses of the diluted hydrocyanic acid, and some leeches. The tumour occasionally increased in size after her having taken cold and coughed violently. Having been for some time at home, she was again received into the hospital after an attack of influenza, when the tumour was found to have slightly increased in size, and she could not lie down flat without great dyspnœa. Her cough was troublesome, and she expectorated much frothy mucus. She had intense pain, extending from the sternum up the right side of the neck. The pulsation in the tumour was strong, but not liquid; only a faint sound was heard over the tumour, alternating with the heart's impulse. The back part of the upper half of the right side of the chest was duller than before, and the respiratory sound was much more feeble over the whole of the right side than over the left, and there was much mucous rattle over both sides of the chest. This bronchitis was soon relieved, and then she varied little until she left Birmingham for Ambleside, where she was attended by Mr. Fell, and was seen occasionally by Dr. John Davy, and where she died suddenly, suffocated with blood, about two years after her first admission into the Birmingham Hospital.

Inspection, by Dr. John Davy and Mr. Fell.

Both lungs adhered to the chest. The left lung was small, but in a healthy state; there was a little blood here and there in its bronchial tubes. The left lung was dense, and there was much dark coagulated blood in its tubes. There were large patches of grey induration in its middle lobe. In the upper lobe was much of the same matter, con-

taining tubercles in it, and also several tubercular excavations, one of which was of the size of a chesnut, and was quite at the apex of the lung. A large tumour, about the size of a child's head, lay under the upper part of the sternum, extending a little to the left of it, but principally to the right, where it adhered to the diseased lung, and to the sternum and costal cartilages. This was found to be an aneurismal pouch, springing from the right side of the ascending aorta just above the valves. It ran up the outer edge of the arteria innominata, which was not involved in it, so that the origin of this vessel had a valvular appearance. This sac was nearly filled with fibrin, the outer layers of which were very firm and discoloured. In one spot there was an ulcerated opening which would have admitted a crow-quill, and which communicated with a small tubercular cavity. A portion of the sternum in contact with the sac was eroded; and on the right of it, over a space of the size of a crown-piece, the sac was bounded by the common integuments, and produced a slight external protuberance. The heart and its valves were healthy.

The nature of the affection could not here be mistaken. A pulsating tumour, a purring thrill, and a double murmur distinct from the sounds of the heart, at the upper part of the right side of the chest, rendered the diagnosis certain. The aneurismal sac being in part bounded by the integuments, the finger was enabled to trace what was going on *within* it. In proportion as its size diminished, so did the liquidity of the pulsation, and the increase of solid matter between the finger and the blood could distinctly be perceived.

The post-mortem appearances seem satisfactorily to explain why the murmur was at one time only diastolic. On the cessation of the systole of the heart, the sac would compress the ascending aorta and arteria innominata, and thus offer an obstacle to the blood flowing onwards, which would not exist when these vessels were fully distended by the heart's action; and also the edges of the orifice of the pouch giving way to the force of the blood, would favour its entrance into the pouch, but would obstruct its onward flow through the aorta during diastole, by protruding into the vessel, as in Case 101. And the same

may be observed in Case 103, which will hereafter be quoted.

Thus in certain cases pulsation may afford us no assistance, whilst in others it may prove a sign of the highest possible value.

Thrill.—Purring thrill denotes the proximity to the walls of the chest of a cavity through which blood is passing, that has been thrown into agitation by some unusual obstacle to its course. Hence it is observed when the heart is dilated, and at the same time there is obstructive disease of its valves; also when the coats of the aorta are dilated and diseased; and, although in a less degree, when a sacculated aneurism approaches the surface of the chest. In the latter case, the larger the sac the less marked will be the thrill from the increased force required to put the whole of its contents into motion. Hence Dr. Hope found it less marked in sacculated than in dilated aneurism. *Cæteris paribus*, its strength, like that of pulsations, must vary with the force of the heart's action.

In Case 62, it existed before any elevation was perceived, and thus proved to be a diagnostic sign of much value. In the following case it was perceived also without any elevation, and in connection with a very loud rasp sound. It was so remarkably strong, and appeared to be produced so immediately under the finger, that its seat was almost necessarily referred to the pulmonary artery, and by these two signs alone the nature of the affection was discovered.

CASE 64.—*Aneurism of the Pulmonary Artery—Constriction of the Aorta, &c., &c.*

A thin pale girl, æt. 19, a screw-wormer, was a patient of the Birmingham General Dispensary, under the care of Dr. Fletcher, and by his kindness was submitted to my examination. She complained of violent pain in the head, and of some pain in the left side of the chest, great dyspnoea, and troublesome cough.

Immediately under the left of the sternum, between the second and third ribs, there was dullness on percussion over a space measuring nearly four square inches. Over this spot a very liquid pulsation was

felt, accompanied by a purring thrill so rough and so close to the surface, that it seemed almost to grate on the fingers. At the same place a loud, hollow rasp-sound was heard with the systole of the heart, and also for a moment at the commencement of the diastole, as if the blood was receding.

This case has been detailed at length by Dr. Fletcher, in the 25th vol. of the Medico-Chirurgical Transactions, from which it appears that the patient lived a year after this examination, and, after repeated attacks of bronchitis and pneumonia, died rather suddenly.

Inspection.—The pulmonary artery was found dilated into a pouch, the interior circumference of which measured nearly six inches, and which pointed principally in the anterior direction, where the internal and middle coats were wanting, and where a fibrinous clot was found. One of the pulmonary valves was contracted. The aorta was first a little dilated and then contracted, particularly where it was joined by the ductus arteriosus. There was also a permanent communication between the two ventricles.

Dr. Fletcher's description is accompanied by a drawing, and is well worthy of perusal; and he has been kind enough to show me a beautiful preparation of the morbid parts.

The extreme proximity of the thrill and rasp-sound to that part of the surface of the chest which covers the pulmonary artery, led to the belief not only that this sound was engendered in that vessel, but that it must have been brought up close to the surface by its dilatation. Doubtless a part of the sound was engendered at the constricted portion of the aorta, as in Cases 65 and 67.

In Case 55, the purring thrill existed over the elevation; in Case 63, over the tumour and above each clavicle; in Case 68, both over the prominence and above the sternum. In the following case the thrill was occasioned by a constriction of the aorta just below the arch, but was probably made more perceptible by the arch of the aorta lying in a higher position than usual.

CASE 65.—*Constriction of the Aorta below the Arch—Malformation of the Aortic Valves—Acute Phthisis.*

A female servant, æt. 20, had enjoyed good health until three months ago, when, after hard work, she felt pain in the chest, short-

ness of breath, and headache, which gradually increased, and she became very feverish and thirsty, and was troubled with a dry cough.

On her admission into the Birmingham General Hospital she complained of dyspnoea and inability to lie down, with troublesome dry cough, occasional giddiness, throbbing pains in the head, and slight shooting pains under the left breast. The tongue was dry and brown in the middle, white and moist at the edges. Her appetite was impaired, and she was very thirsty. The skin was dry and hot, the urine plentiful and high-coloured. The bowels were open, but the evacuations were scanty.

Between the second and third ribs on each side of the sternum, and also above the top of it, a well-marked purring thrill was felt. Under the cartilage of the second right rib a loud systolic bellows-sound was heard, gradually diminishing as the ear receded from this spot. It reappeared, but was very faint, between the fifth and sixth left ribs. During the course of her illness the intensity of the bellows-sound varied, but its maximum was always under the cartilage of the second right rib, and near the right sterno-clavicular articulation, where it sometimes amounted to a rasp-sound. The purring thrill also was constant. She died in three weeks from the time of her admission, her chief symptoms having been rapidity of pulse, slight lividity of countenance, great dyspnoea, and hurried respiration, dry cough, with occasional expectoration of clear mucus, now and then tinged with blood, coarse respiratory sound, with cooing sounds, and sometimes muco-crepitant rattles; a combination of symptoms which indicated the existence of unsoftened tubercles or gray granulations throughout a large extent of lung.

Inspection.—The arch of the aorta was found to reach as high as the first rib, and was slightly dilated. Just beyond the origin of the left subclavian artery the coats of the aorta suddenly became attenuated round the circumference of the vessel for half an inch, so that in this spot their thickness scarcely exceeded that of an ordinary adult's radial artery; and not only were the walls thinner, but the diameter of the vessel was considerably contracted at this spot. At the origin of the aorta were only two semilunar valves, one large and one small, partially adherent to each other, and to the sides of the vessel nearest the mitral valve; so that they formed a funnel-shaped projection into the aorta. They maintained a column of water in the vessel, but offered a slit for the passage of the blood, which only admitted the passage of the little finger. The lungs were densely studded with minute semi-transparent gray granulations, which became rather larger and more opaque towards the apex of each lung.

In this case a loud bellows-sound, being at its maximum where the thrill existed under the cartilage of the second right rib, led to the suspicion that there was a dilated aneurism at this spot, which was not confirmed on inspection, although the aorta was slightly dilated.

Purring thrill is sometimes the result of a diseased state of the aorta about the origin of the carotid arteries when it is carried up their course; and the same thing may occur when obstructive disease at the aortic valve is joined with great hypertrophy of the left ventricle, as in Case 33, and a suspicion of aneurism has sometimes arisen in these cases. The absence or presence of other signs will generally enable us to decide this point.

Auscultatory Signs.—Percussion.—When an aneurismal pouch reaches the surface and displaces more or less of the spongy pulmonary tissue, it of course renders the sound on percussion dull over that spot, but the dulness is not of a different character to that produced when any other solid or liquid thus displaces the lung. Circumstances, however, may occur which render the sounds yielded on percussion of great diagnostic value. Thus in Case 25, above quoted, the extent of dulness on the left side of the thorax notably diminished after each copious expectoration of blood. Now, as there was abundant evidence of the existence of a tumour compressing the trachea, œsophagus, and recurrent nerve, and lying in contact with the aorta, with the left lung and the spine, it would seem only reasonable to infer that this tumour was a sacculated aneurism embedded in the left lung.

Aneurisms of considerable size, however, may exist without giving rise to dulness on percussion. In Case 26 none existed, and in 71 it was very slight.

Sounds produced by the current of the Blood.—Bearing in mind the manner in which* sounds are engendered or modified in passing through elastic tubes by the diminution or enlargement of their calibre, or by an alteration in their

* Observations on Diseases of the Chest, by Dr. Blakiston, p. 50.

lining membranes, it will be found that valuable diagnostic signs are furnished by such modifications in thoracic aneurism.

But little can be learned from the *intensity* or *quality* of abnormal sounds, for these properties depend in a great measure on the force of the heart's action. When there is positive evidence of the existence of aneurism, with little or no murmur, and at the same time the action of the heart is strong, we may conclude that the aneurism is large and sacculated, and that it communicates with the aorta either by a very large or a very small aperture; or else that it is from some cause very inelastic; a conclusion, however, which would be drawn more from a consideration of the manner in which such sounds are produced than from actual observation. For in all the cases here recorded, when the impulse of the heart was great, some abnormal murmur was heard, except in Case 104, in which no opportunity was afforded for a post-mortem examination. In Case 64, the intensity of the murmur, actually mounting up into the ear, was such that, coupled with the existence of an amazingly strong thrill close under the finger, it was taken to indicate the dilatation of a main artery to such an extent as to cause it to lie immediately under the integuments and the sternum. From the situation, this could only be the pulmonary artery. In Case 62, the intensity of the murmur decreased with the *extension* of the aneurismal sac, but in Cases 103 and 63, it decreased with the *diminution* of the sac, caused by the deposit of coagulum and consequent loss of elasticity.

There was one quality of sound that was never heard but in a *dilated* aneurism, and that was a peculiar *hollowness* of tone. It was well marked in Cases 26, 64, 74, 55, and in the early stage of 21, but subsequently the murmur lost this character when the aneurism became sacculated.

The *seat* of the murmur is of great importance. When it is heard at the precordial region it is of much less value than when it is *limited* to some spot near the upper part of

the sternum; because it might in the former instance be confounded with a murmur engendered at the aortic orifice, even were it heard louder near the upper part of the sternum than over the seat of the aortic valves. It may also, under such circumstances, be accompanied with circumscribed dulness on percussion, and still not denote the existence of an aneurism, because the dulness may be caused by the consolidation of a small portion of the lung, which being in contact with the aorta may transmit the valvular murmur with great intensity to the surface of the chest. This is illustrated in the following:—

CASE 66.—*Phthisis Pulmonalis—Aortic Regurgitation.*

A merchant, æt. 55, accustomed to free living, began to complain of shortness of breath, which was followed by cough, accompanied by clear expectoration, frequently streaked with blood. His strength failing, and losing flesh, he placed himself under Mr. Wickenden's care.

At that time there was *dulness on percussion an inch and a half below the right clavicle*, and an absence of respiratory sound, and buzzing bronchophony. *A double rasp-sound was heard at the precordial region, increasing in intensity until the ear reached the right of the top of the sternum.*

Soon after this I saw him and examined him at his request, but not being in attendance on him I took no notes of his case.

On talking the matter over with Mr. Wickenden, I found that latterly he had had several attacks of hæmoptysis. We considered his case to be one of phthisis; but others considered it might be one of thoracic aneurism.

Soon after this the signs of tubercular softening at the apex of the right lung became very manifest, and he died.

Inspection.—The aortic valves were so thickened and shortened that they must have allowed the blood to regurgitate. There was a small dilated aneurism of the abdominal aorta. The lungs were extensively tuberculous.

The murmur was heard both at the precordial region and over the aneurismal tumour in 21, 62, 74, 55, and 103. It was, however, joined with other signs, which clearly indicated the existence of aneurism; in Case 21 there being pulsation and absence of the pulse in one wrist; in Case 74

pulsation, but the nature of the affection was masked by pleuritic effusion. In Cases 62 and 103 there was a *pulsating tumour*, and in Case 55 a pulsating tumour, purring thrill, and absence of pulse in one wrist.

But even should a murmur be heard *confined* to a spot near the upper part of the sternum, it would not *necessarily* indicate the existence of aneurism, as may be seen on reference to Case 65, and also in the following:—

CASE 67.—*Tubercular Infiltration of the Lung—Constriction of the Aorta.*

A gentleman, æt. 48, of studious habits, who had scrofulous ulceration of the neck in his youth, complained of a difficulty of breathing which had been coming on for some time. This was followed by a very troublesome cough, with slight clear expectoration. He then consulted Mr. Wickenden, who found the whole of the left side of the chest dull on percussion. On this side there was no trace of respiratory sound. On the right side there was coarse respiratory sound, here and there mixed with large and small moist rattles. *A loud, single, prolonged rasp-sound was heard over the left side, its maximum being between the second and third costal cartilages.* At the precordial region it was heard more faintly, and also in the back. He died, apparently, from increasing dyspnoea.

Inspection.—In the right lung there were some grey granulations and unsoftened yellow tubercles, singly and in small groups. The whole of the left lung was converted into a yellowish-white hard mass, of almost fibro-cartilaginous consistence, which was mottled in spots with a pink colour. At the point where the ductus arteriosus joins the aorta the coats of this latter vessel were thickened, *and its calibre was reduced to less than half its proper size for nearly the distance of an inch.* There was atheroma all along the aorta. The heart was generally hypertrophied and dilated, but not to any great extent. The valves acted well.

In these cases, therefore, the murmur was caused by the constriction of the aorta, in the same manner as it has been seen to arise from narrowing of the orifice of the arteria innominata.

A reference to the manner in which aneurismal murmurs are produced will show that their value as diagnostic

signs must, in some measure, depend upon their connexion with the systole or diastole of the ventricles of the heart. A diastolic valvular murmur can only be formed by aortic regurgitation, or by a moderate amount of obstruction at the auriculo-ventricular orifices. In neither of these cases is murmur carried to such a distance from the spot where it is engendered, as when it arises from aortic obstruction. Hence a diastolic murmur heard *very much* louder on either side of the upper part of the sternum than at the precordial region, is generally, if not always, characteristic of aneurism. If it is limited to such a spot, it is absolutely characteristic of this disease, because we can suppose no other state of the vessel which could give rise to it.

CASE 68.—*Aneurism of the Thoracic Aorta.*

A female servant, æt. 25, received a violent shock from a fright eleven months ago, since which time she has been very nervous and hysterical. She has, in the course of this period, had several attacks, which were chiefly characterized by severe pain in the chest, dyspnœa, palpitation, and on two occasions she lost her voice for some days. After one attack her right hand and arm swelled, and became of a purple colour, which lasted ten days or a fortnight.

Having been admitted into the Birmingham General Hospital, her face was seen to be turgid, and she complained of occasional giddiness, tinnitus aurium, and throbbing of the vessels of the neck, of pain at the precordial region, shooting towards the left shoulder and down the arm, and of constant severe pain between the shoulder-blades. She was unable to lie down without experiencing the sensation of suffocation, and violent palpitations appeared on the slightest exertion or mental emotion.

The impulse of the heart was strong and jerking. There was violent pulsation in the carotid arteries; a strong purring thrill was felt on pushing the finger down behind the summit of the sternum, and also between the third and fourth right costal cartilages. This spot sounded dull on percussion. The precordial dullness was lower than usual. There was a little coarseness in the respiratory sound generally. There was a soft systolic bellows-sound heard mostly at the situation of the apex of the heart, and also behind the left side, accompanied by the diastolic sound, which was rather less clear than usual. At the

point to the right of the sternum, where the dullness and purring thrill were perceived, there was a double saw-sound, which became single on approaching the right clavicle, and was slightly prolonged into the carotid artery.

She remained in the hospital some time, during which she had an attack of acute articular rheumatism, after which the pulsations, thrill, and murmurs were much more feeble. She was relieved, and has not been since heard of.

Although there was probably in this case some disease of the valves of the left side of the heart, and much hysteria, yet the evidences of aneurism were also clear and unequivocal. At the spot where there were dullness and a thrill, there was a diastolic sound, which was not heard at all at the heart. The aorta was dilated at its arch, as could be distinctly felt with the finger.

Not only does a diastolic murmur in these situations indicate aneurism, but it denotes that it is sacculated. It existed in Cases 25, 68, 62, 64, 74, 103, and 63, in all of which there was sacculated aneurism, except in Case 25, in which it was mixed. There was, it is true, no post-mortem examination in Cases 68, 102, and 103, but in 101 and 103 there was a pulsating elevation on the surface of the chest, and the blood was at one time felt close under the finger. In Case 25, however, the diastolic sound was only heard on one occasion a little before death, and might have arisen from some temporary obstruction by a portion of clot. In Case 64 the sound was possibly caused by regurgitation through the incomplete valves of the pulmonary artery. The murmur was double in Cases 68, 62, 64, 74, and 102, and single and diastolic in 101, 103, and 63. In Cases 101 and 63 it was found that the mouth of the sac was constructed in such a manner that it would yield to the current of blood, so that it was allowed freely to enter the sac; but on its exit the calibre of the aorta was diminished, and thereby the current was impeded in its onward course. In Case 62 the sound was at first single and systolic, and afterwards double on the aneurism becoming sacculated.

When, therefore, aneurismal murmurs occur, they are often of much value in enabling us to form a just estimate of the nature and size of the tumour; but it must be remembered that they depend a great deal on the force of the heart's action, and that some aneurisms attain to a large size without giving rise to any murmurs at all.

The *indirect* signs of thoracic aneurism arise from their coming into contact with the *heart, blood-vessels, nerves, trachea, bronchi and pulmonary tissue, and the œsophagus.*

Heart.—A small aneurism in close contact with the heart, such for instance as originates within the pericardium, may irritate this organ, a large one may embarrass its movements by pressure, and a dilated aorta may oppose a considerable obstruction to the onward flow of the blood. In this manner palpitations, syncope, irregular action, fluttering, and a sense of constriction may be induced.

All these symptoms, however, may result from various organic diseases of the heart, as well as from its sympathetic action in certain cases of dyspepsia, anæmia, and hysteria, or other nervous excitement; and therefore they are of no value as diagnostic signs of aneurism, but merely serve to direct the attention to the heart and its appendages.

Blood-Vessels.—The *arteries* of the neck and arms are sometimes compressed in a peculiar manner. Thus in Case 63 the sac crept along the *arteria innominata*, and when distended must have compressed this vessel. Hence there would be inequality in the circulation, which possibly gave rise to the headache, with which the patient was tormented. So also in Case 25, the left carotid artery was compressed from behind, and this patient also suffered from intense headaches, particularly when lying on his right side. As headaches are common, and arise from a variety of causes, they would not serve to point out the nature of the affection under consideration.

Pressure on the vessels of the lungs is interesting in a pathological point of view, but as it would give rise to a

state of the lungs which might arise from other causes, it furnishes no diagnostic signs.

When the descending *vena cava*, however, is compressed, a curious and valuable sign is developed; its existence being revealed by anasarca confined to the head, neck, arms, and chest. For pressure on the descending *vena cava*, which is the only trunk that unites the veins from these parts, could alone give rise to such a state. The existence of a tumour, therefore, in the immediate vicinity of this vessel is thus clearly indicated. Such tumour, however, is not necessarily aneurismal, as is proved by the following:—

CASE 69.—*Carcinomatous Tumour compressing the Descending Vena Cava, and producing Œdema of the Head and Arms*
—*Similar Tumour around the Stomach—Cyst in the Brain.*

A gilder, æt. 43, having felt poorly for some months, which he attributed to over-exertion in his business, consulted Mr. Wickenden, complaining of severe pain in his head, and uncomfortable sensations at his stomach. His breath was fetid, his gums spongy, and his bowels constipated. His pulse was slow, but natural in other respects. The sounds of the heart were heard over the whole of the front part of the chest. He thought he should be well if he could live without eating, since food caused him great uneasiness. Soon after this a tremulous motion of the hands was observed, and he complained of great pain in his head, shoulders, and arms. Having joined Mr. Wickenden in consultation, I continued to attend with him until the death of the patient. His chief complaint was of intense pain in the head. The pupils were small and contracted, and the iris inactive. The pulse 40. The pain in the head was slightly relieved, and the pulse rose to 70 under the use of the cold douche. He went to the seaside for three weeks, during which time it appeared that he had three epileptic fits. On his return his symptoms seemed aggravated. His memory of words was affected, and his sight became slightly impaired. He was very irritable, and wandered in his mind. In a few days the pain between his shoulders became excessive, and he was rather violent. Strong narcotics relieved this pain, but that in his head remained, although blisters were repeatedly applied down the spine.

Six months after his first seizure he had no pain, except in the head;

but dyspnœa came on, and œdema of the lower right eyelid. There was slight dullness to the right of the upper part of the sternum. The respiratory sound was natural; the sounds of the heart were extended but normal. The pupils were now dilated, the pulse small and quick, 120. *Edema of the head, neck, arms, and upper part of the thorax came on.* The sounds of the heart were heard louder, the respiratory sound more feeble, and the sound given out on percussioa a little duller than elsewhere, at the right of the top of the sternum. There was whistling respiration as if the trachea was compressed. He could not lie down, but spent his time leaning forwards. He complained of pain in the head, and wandered at times. Slight œdema of the legs and scrotum next appeared, and when seen again, he was in a profuse perspiration, leaning forwards, and with the greatest difficulty drawing in his breath; and he complained of a pain in the head.

He died in this state four months after he was first seen, and about seven months after his first seizure.

Inspection.—The face, neck, arms, and breast were very œdematous, the legs and scrotum slightly so.

In the inferior and posterior portion of the left hemisphere of the brain was a cyst of the size of a hen's egg, which rested on the tentorium cerebelli. It was thick, and was surrounded by small tumours, varying in size from that of a pea downwards, hard, white and lardaceous. It contained about an ounce of clear fluid, and around it the brain was softened.

The right pleural cavity contained three, the left two pints, and the pericardium two ounces of clear fluid. The heart was dilated, large, soft, and flabby. A white, hard, lardaceous tumour, with a spot or two of softening in it, stretched from the bronchial glands to the right edge of the upper part of the sternum and sternal end of the right clavicle. The *vena cava superior* was imbedded in it and flattened. The lesser curvature, and indeed the greatest part of the stomach, was embraced by a solid mass of a similar kind to that found in the chest, which also invaded the mesenteric glands, and the liver contained a mass, rather larger than a walnut, of the same deposit.

The signs of a mediastinal tumour were unequivocal, but it was not supposed to be an aneurism; because, although it evidently was in contact with the surface of the chest to the right of the sternum, and conveyed the sounds of the heart with unusual clearness to the ear, yet there was no trace of pulsation over that spot. The same sign was observed in Cases 26 and 60, but there were other

signs in these cases which proved the tumour to be aneurismal.

The absence, however, of anasarca of the upper part of the body does not prove the non-existence of a tumour near the descending vena cava, because there was a tumour in this situation in Cases 72, 62, 105, and 63, but the vessel was not compressed by it.

It is said that the thoracic duct is sometimes thus compressed, and that inanition follows from the flow of the chyle into the blood being interrupted. Possibly such was the case to a certain extent in Case 21, but this point was overlooked in the inspection of the body.

In Case 61 a similar tumour existed, and was discovered from observation of the tortuous swollen state of the veins on the chest emptying themselves into the superior vena cava. It was in this case predicted that œdema of the head, neck, and arms would soon appear, as in fact it did. As there was no murmur, thrill, or pulsation, no opinion was at first given as to the exact nature of the tumour. The same signs were observed in Cases 26 and 60, but there were others which proved the tumours to be aneurismal. In one a hollow rasp sound was heard, limited to a small spot to the right of the upper part of the sternum; and in the other a "beating substance" had at one time been seen over the top of the sternum, and a rasp sound had been heard under the upper part of the sternum, where it projected outwards, which sound ceased when the force of the heart's action was diminished in articulo mortis.

Nerves.—The pains so often felt in the chest and down the arms in thoracic aneurism, are doubtless caused by the nerves being in some degree implicated; and they have this value, that they induce patients to apply for advice, and thus lead to the discovery of the affection, if present, by means of other more certain characteristic signs. When the recurrent nerve is involved, the muscles of one side of the larynx are sometimes paralysed, so that the vocal plate

on that side cannot rise up into the proper plane for the formation of the voice, which therefore becomes hoarse and jerking, or is reduced to a whisper. Dr. Todd has related a case of this kind.* It also occurred in Case 25. The position of the tumour was the same in Case 24, so that it probably took place there also, but this point was overlooked in examining the body. The voice was similarly affected in Case 73, but whether this arose from stretching of the nerve, or ulceration of the larynx, was doubtful.

Trachea.—When an aneurismal tumour comes in contact with the trachea, particularly if adhesion takes place between them, violent cough and suffocating dyspnœa are produced, more or less accompanied by whistling sounds. In some cases the group of symptoms exactly resembles those produced by laryngitis; and it has occasionally happened that tracheotomy has been performed in the hope of giving relief from impending suffocation, supposed to be caused by laryngitis, when in fact the distress has arisen from the pressure of an aneurism. Such a circumstance nearly occurred in the following case:—

CASE 70.—*Aneurism of the Arch of the Aorta communicating with the Trachea.*

A tradesman, æt. 38, had suffered some months from hoarseness and partial loss of voice. He was constantly troubled with a cough, and had several attacks of hæmoptysis, in which black coagulated blood was generally brought up, mixed with a little of a bright red colour. He constantly felt a constriction of his throat, and his respiration was accompanied by a slight whistling. The trachea seemed to recede backwards from the sternum, but was not turned on either side. No trace of abnormal sound could be detected over any part of the chest.

In this state he was one day suddenly seized with symptoms of acute laryngitis. Mercury was rapidly administered, and preparations were made by Mr. Lawrence, under my advice, to perform tracheotomy, had not relief been obtained. He was, however, quickly salivated, and recovered, but the cause of these attacks still remained concealed from view.

* Medico-Chirurgical Transactions, vol. xxvii.

Some months after this, he was seized with symptoms of severe bronchitis; on this occasion he was seen by Mr. S. F. Palmer, who was kind enough to send me the particulars of the attack, and of the post-mortem appearances, and to show me the diseased parts. From his account, it appears that previous to death the patient was expectorating a thin sanious fluid, that whilst conversing he was suddenly seized with symptoms of suffocation, gasped eagerly for breath, attempted to thrust his fingers down his throat, as if to remove some obstacle to respiration, sank back upon his bed, and died.

Inspection.—There was much atheromatous and some calcareous degeneration of the aorta. The arch was dilated uniformly into a pouch, at the back of which was an oval opening, an inch long and half an inch wide, with smooth rounded edges, leading into a sac which would have held a chesnut. This sac was bounded at the sides by the arterial coats, and at the back by the trachea, to which it closely adhered, and with which it communicated by a small aperture with smooth and rounded edges, capable of admitting a crow-quill. It was filled with concentric layers of fibrin, the most external of which were tough and light coloured. This sac pressed on the trachea, and very much diminished its calibre.

“The larynx presented evident signs of chronic or repeated inflammation, the submucous tissue being evidently thickened. There were signs of inflammatory action in the lining membrane of the trachea.”

There was not, from first to last, a single sign that could be taken to indicate the existence of aneurism. Neither the dilatation of the arch of the aorta, nor the pouch, were sufficiently great to interfere with any other part than the trachea. The whistling respiration might have been produced by chronic laryngitis; and both this and the hemoptysis might have been referred to tuberculization of the lungs; but the absence of all physical and general signs of that disease excluded this cause. To me the symptoms were inexplicable; but it is possible that during the attacks of hemoptysis some fragments of fibrinous clots might have been expectorated, which, had they been seen, would have raised a suspicion of the nature of the affection, as in Cases 24 and 84.

When the tumour is small, as it was in this case, the

difficulty of distinguishing between the effects of aneurism and laryngitis is insurmountable; the more so as inflammation of the trachea and the larynx are sometimes induced by the aneurism in contact with them. It is possible that the new method of exploring the larynx may in some cases assist in clearing up the obscurity in which the differential diagnosis is thus involved.

But even should the larynx be found to be secondarily affected, the exciting cause may be a tumour, *not* of an aneurismal character, as occurred in Case 73.

When the trachea is thus compressed it is sometimes pushed backwards from the sternum, as in Case 70, or turned aside from the median line, as in Case 25, and a valuable sign of the existence of a tumour in this region is thus afforded. In other cases a marked difference is produced in the symptoms by the alteration of position; a great mitigation of the cough and dyspnœa occurring when the patient leans forward. By this we are assured, not only that there is a tumour, but that it is so moveable that it falls on the trachea when above it, and falls from it when below its level. Such an effect may be produced by a largely dilated heart, as in a case recorded by Morgagni. A careful exploration of the precordial region would lead to the discovery of the true cause of the symptoms observed. From this a very valuable sign is eliminated, because no other substance except the heart or an aneurismal tumour in this position could have the necessary amount of mobility. Strumous or cancerous disease would have a different character altogether, being fixed, and comparatively immovable.

Bronchi.—Sometimes the pressure is chiefly exerted on one of the bronchi.

CASE 71.—*Aneurismal Pouch springing from the Arch of the Aorta, and compressing the Right Bronchus.*

A married woman, æt. 50, had been subject to asthma for some years, and to occasional paroxysms of urgent dyspnœa. In one of

these she was admitted into the Birmingham General Hospital, and died before she could be carefully examined.

Inspection.—The right lung was much smaller than the left, and contained but little air. A few air tubercles were scattered through its apex, but were not found in any other part of the body.

An aneurism, of the size of an orange, was seen springing from the right side of the ascending aorta, and *compressing the right bronchus and the vessels* of the corresponding lung. It communicated with the aorta by a circular aperture of an inch and a half diameter. The middle coat of the aorta was traced for a short distance into the aneurismal pouch, which was lined with fibrinous layers.

Had it been possible carefully to examine the chest in this case, it is probable that the compression of the right bronchus would have been discovered by the absence of respiratory sound on the right side of the chest, in which case, had an aneurismal murmur been heard in that spot alone, the nature of the affection would have been made out, as in Case 26. But no physical signs of less value than this would have sufficed, as the bronchus might have been compressed by a solid tumour.

In the following case, although more or less fixed to the trachea, it obstructed a large bronchial tube :—

CASE 72.—*Aneurismal Pouch springing from the upper and right portion of the Arch of the Aorta, and compressing the Trachea and the Upper Branch of the Right Bronchus.*

The adjutant of a dragoon regiment, æt. 45, of athletic frame, had been accustomed to take a great deal of violent exercise. When a young man he had drunk hard, but of late years had been very temperate. For three years he had suffered more or less from dyspnoea and cough. About a year since these symptoms became aggravated, and a severe pain came on between his shoulders, with a sense of tightness across his chest; and his cough invariably became urgent on lying down. Having been much fatigued and chilled by a long march on a very cold wet day, and being also much excited, his eyes were observed almost to start from his head, and his face became very livid. A pain in the right side succeeded, and he was attended by Mr. Hodgson, in consultation with Dr. Pilkington, the surgeon of the regiment, when symptoms of slight pleuro-pneumonia at the lower part of

the right side were observed. Dyspnœa, cough, and constriction of the chest remained, however, after the acute attack had subsided. Seen in consultation with Dr. Pilkington, he stated that he had not been able to lie down for 15 weeks. He had a harassing cough with a slight amount of frothy expectoration, and complained of great tightness of the chest, and severe pain shooting from under the sternum backwards and down each arm. He breathed freely when considerably inclined forwards, and in that position he could run about the room without distress; but the moment he assumed either the erect or recumbent position, he was threatened with suffocation. He was cheerful, and stated that his appetite was good and his bowels regular. His pulse was feeble, but natural, his tongue slightly coated. No pulsation or thrill could be felt in any part of the chest. The impulse of the heart was feeble.

The chest sounded rather dull over a small spot to the right of the upper angle of the sternum, and also near the bottom of the posterior portion of the right side. The pulmonary sound was rather coarse, and with it were traces of small crackling and bass-viol sounds. When he raised himself upright the respiratory sound totally ceased over the upper third of the right side of the thorax, and a whistling sound was heard at a distance from the patient. The sounds of the heart were natural, but were heard rather louder to the right of the upper corner of the sternum than in the precordial region. He was much relieved by belladonna frictions over the chest; but in a few weeks' time he was seized suddenly with a violent paroxysm of coughing, having a few days previously taken cold, as he thought. This continued for three hours incessantly, when he died.

Inspection.—A tumour of the size of a large orange was found lying under the upper part of the sternum and to the right of it, but not adhering to it; and seemed to have been nearly covered by the left lung when inflated. It adhered closely to the trachea immediately above its bifurcation, and for three inches upwards, and considerably compressed and even flattened it. It also overlapped the right bronchus, more especially a large upper branch, which was given off almost immediately below the bifurcation of the trachea. This tumour was found to be an aneurismal pouch, partly formed by the dilatation of the upper right angle of the arch of the aorta and the orifice of the arteria innominata, and partly by distension of the cellular coat. It was about half filled with layers of old-formed, discoloured, fibrinous coagula. The heart was of moderate size, and of healthy appearance. Both lungs, but more especially the right, were much engorged. At the bottom of the right lung, towards its posterior part, was a mass of gray tough induration, about the size of an orange.

There were not in this case any of the usual signs which are supposed to indicate aneurism. There was no pulsation or thrill, nor any abnormal sound whatever, except the whistling respiratory sound which denoted compression of the trachea. The tumour, in fact, had hardly reached the surface of the chest, so that no thrill would be felt, nor any pulsation, except it were propagated up the trachea. Had the left ventricle been hypertrophied it might have produced a strong current through the pouch, and thus have given rise to murmur, which in its natural state it could not be expected to do. But in the absence of all ordinary signs, there was one which clearly indicated the nature of the affection. For it was not only evident that the trachea was permanently compressed by some tumour, but that this tumour extended considerably to the right, and was so moveable that in a certain position it fell upon the upper division of the right bronchus, and on that alone. Had the tumour consisted of diseased glands, it would not have been thus moveable, but have been one solid mass, as in Case 60. An aneurism springing from the right of the arch of the aorta alone remained as the possible cause of this peculiar sign. This, therefore, was the diagnosis given.

Lungs.—When the substance of the lungs is encroached upon, an additional sign, namely hemoptysis, may show itself. This may occur in different ways; by ulceration between the sac and a bronchial tube, or even a tubercular cavity, as in Case 63. In Cases 70 and 24, it arose from the sac ulcerating into the trachea. In most cases this symptom would induce a suspicion of phthisis rather than of aneurism. Yet in Case 24 the nature of the material expectorated, blood and fragments of discoloured coagula, materially assisted the diagnosis; and in Case 25 hemoptysis produced a remarkable alteration in the physical signs, which, properly interpreted, might have determined the nature of this affection.

Œsophagus.—Difficulty and pain in swallowing may result

from interference with the œsophagus, but we have never seen it occur in a permanent and marked degree. This symptom may arise from the pressure of any tumour, and in the following case was produced by cancerous deposit:—

CASE 73.

A fisherman's wife, about 60 years of age, had been suffering some time with pain in the throat, and a hacking cough. She occasionally experienced much difficulty and pain in swallowing solid food, and sometimes brought up by a kind of regurgitation a little dark venous blood. The voice was peculiarly hoarse and yet very feeble, sometimes amounting only to a whisper. Seen, in consultation with Mr. Gabb, there was a fulness at and below the larynx, but no decided tumour could be felt, nor was there any pulsation or thrill. There was some whistling respiratory sound, but no abnormal sound was detected by auscultation.

Inspection.—A cancerous tumour, about the size of a walnut, was found between the lower part of the larynx and the œsophagus, adhering to both. The œsophagus was ulcerated through to the tumour, the larynx nearly through.

Below the bifurcation of the trachea the œsophagus has been seen to be pushed aside by a large aneurism, without giving rise to any symptoms of such an occurrence during lifetime.

It may happen, however, that the existence of a thoracic aneurism which under ordinary circumstances would give rise to signs both direct and indirect, is hidden from our view by the presence of another disease which completely masks it. This happened in

CASE 74.—*Aneurismal Pouch springing from the Right Side of the Ascending Aorta—Right Pleuritic Effusion.*

A widow, æt. 44, had experienced palpitations at the heart for three years, and had been subject to occasional attacks of rheumatism. Six months since, she was suddenly seized with acute pain in the right side; this was succeeded by great dyspnoea and flying pains in her shoulders, and fixed pains in two of her fingers, which were red and swollen.

Seen, in consultation with Mr. Meek, she was found rather emaciated, with some œdema of the extremities. Her pulse was feeble, and her bowels constipated. She had a slight cough, with clear, frothy expectoration. She suffered from severe palpitations, and occasional paroxysms of most urgent dyspnœa, accompanied by a great sense of terror; so that she would hardly allow any one to approach her at such times.

Below the nipple, the right side of the chest measured two inches more than the left side, and on a line with the axilla one inch more. To the right of the upper part of the sternum a pulsation was felt much stronger than in the precordial region, and also, though faintly, all over the right side. Excepting immediately under the clavicle, the whole of the right side of the chest sounded as dull as possible. On the left side the precordial dulness was rather more extended than usual. For two inches below the right cavicle, and nearly half way down the back of the right side, a strongly marked blowing tracheal sound was heard, and which appeared very distant, as if it were propagated from the bronchus of the opposite side: below this point no pulmonary sound could be detected. On the left side the respiratory sound was rather coarse, with some traces of muco-crepitant rattle. Over the precordial region a double bellows-sound was heard, which became louder and more hollow and coarse towards the right of the upper part of the sternum, where it amounted to a rasp-sound. She lived only a few days after this examination, and died in one of her paroxysms of dyspnœa.

Inspection.—The right side of the chest contained more than a gallon of serum. The right lung was compressed against the spine, and devoid of air. Several membranous bands passed from this lung to the sides of the thorax, and to the diaphragm. The left lung was very voluminous, and adhered closely to the chest. The heart was very large. Under the upper half of the sternum was seen a round tumour of the size of a large orange, which projected two inches to the right of the sternum, and adhered to that bone over a space as large as a shilling. It lay over the right bronchus and descending vena cava, but was not adherent to either. It was found to be an aneurismal pouch springing from the right of the ascending aorta.

The true nature of the affection was in this case disguised by the pleuritic effusion in the right side of the chest. When I first saw the patient the chest had not been measured, and I certainly mistook the impulse of the

aneurism for that of the heart, which I thought had passed over to the right side, as in cases 97, 98, and 99. It was impossible to examine the case thoroughly, owing to the excited state of the poor woman. The murmur might have been produced at the heart; the dulness on percussion extended over the whole side, and the pulsation modified by the fluid in the pleura, more resembled the impulse of the heart, than an aneurismal pulsation. From whatever cause, the nature of the affection was not detected.

From what has been seen of the progress and termination of thoracic aneurism, it follows, that there must be two kinds which are not likely to give rise to any of the signs above enumerated, and these are aneurisms *within the pericardium, and those of the descending aorta below the bifurcation of the trachea*. In the former case they invariably burst, and prove fatal before they attain a sufficient size to reach the surface of the chest, and give rise to *direct* signs; or to produce *indirect* signs by interfering with any important organ, except the heart; and if any signs were thus produced by pressure on the heart, they could not be distinguished from those resulting from disease of the organ itself. If, for instance, a murmur were caused, it would be so near the valves that it would be confounded with valvular murmur. Aneurisms occurring below the bifurcation of the trachea, may attain a large size, without either reaching the surface, or materially interfering with the œsophagus, apex of the heart, &c. It will, therefore, excite no surprise, that in the following cases the existence of the disease was not discovered during lifetime.

CASE 75.—*Aneurism springing from the Root of the Aorta, and bursting into the Pericardium.*

A married woman, æt. 30, of very intemperate habits, had completely lost her appetite, and complained of severe gastrodynia and frequent vomiting. On being visited as a patient of the Birmingham Dispensary, she was found with an anxious expression of countenance.

The pulse was rather hurried. She had a cough with slight expectoration, and tenderness in the epigastric region. She had occasionally palpitations of the heart.

The chest sounded clear on percussion. There were some slight cooing and bass-viol sounds heard in different parts. *The sounds of the heart were perfectly natural.* She improved considerably for some days, when, being excited, she suddenly dropped down, and was found dead by Mr. Bromhall, who was passing her residence at the time.

Inspection.—There were signs of chronic inflammation in the coats of the stomach, and on the lining membrane of the bronchial tubes. The section of the liver had the appearance of nutmeg.

The pericardium was distended with blood, on removing which, a tumour was seen nearly as large as a walnut, slightly shrivelled up. This was an aneurism which communicated with the aorta about one-third of an inch above the valves at the back part of it, by an orifice as large as a sixpence, with rounded edges. The middle coat could be traced some distance into the sac; but the convexity of the sac was formed by the pericardium alone, and was lacerated in such a manner that the blood had escaped into the cavity of the pericardium. It was half filled with fibrinous layers, some of which were rather tough, and a small piece projected through the lacerated opening above-mentioned.

It is seldom that an opportunity occurs of examining cases of this kind before death, the aneurism being generally so small at the time of the perforation that the inconvenience resulting is not sufficient to induce the patient to seek advice. In this instance she applied for the relief of gastrodynia. On careful exploration no unusual sounds were detected in the precordial region. Even had a sound been engendered by the aneurismal pouch, it would undoubtedly have been mistaken for valvular murmur, on account of the proximity of the aneurism to the valves of the heart. It is, therefore, next to impossible to detect, during lifetime, the existence of aneurisms in this situation, where they seldom reach the size of a walnut. It is supposed by some that they are invariably formed by dilatation of all the coats; but in this case, the tumour was formed at first by dilatation, and subsequently by the rupture of the inner and middle coats. This may

also be inferred from the fibrinous layers found within the sac. A very similar case is related by Morgagni, and in Dr. Smith's case, before alluded to, the greater part of the sac was formed by the pericardium alone.

CASE 76.—*Aneurism springing from the Root of the Aorta, and bursting into the Pericardium.*

A coach-harness forger, æt. 26, was taken suddenly ill at half-past seven in the morning. Mr. Meek was called in and found him in a state of collapse. His intellects were clear, and he complained of pain and a sense of tightness at the epigastrium. The pulse was almost imperceptible. His lips and the surface of his body were blanched and very cold, more especially the extremities.

The dullness of the precordial region was greatly extended and very marked. The respiratory sound was clearly heard over both sides of the chest. The sounds of the heart were very distant, and the diastolic sound indistinct; but there were no cardiac murmurs. He sank in ten hours.

Inspection.—There were traces of atheroma in the aorta. In one of the sinuses of Valsalva there was a dilatation of the size of a cherry, which had burst in the middle; and through this aperture, a large quantity of blood had escaped into the pericardium, much of which was coagulated.

The absence of murmur in this case proves nothing; as, unless it had been of a very marked character, it would, probably have ceased when the action of the heart became oppressed by effusion of blood into the pericardium.

I have seen other cases very similar to this; but, as the patients had not complained, and consequently were not examined during lifetime, a detailed account of the post-mortem appearances would be useless.

CASE 77.—*Aneurism springing from the Concavity of the Arch of the Aorta, and bursting into the Pericardium.*

A wheelwright, æt. 40, of intemperate habits and addicted to spirit-drinking, had an attack of acute rheumatism three years since; and

from that time had suffered occasionally from flying pains of a rheumatic character; and for some weeks he had felt some pain across his chest. He was attended by Mr. Meek, who, on a careful examination of the chest, *failed to detect any abnormal sound.*

The evening before his death, he boasted of being quite well and free from pain. He had bread and cheese and a pint of beer for supper, and retired to bed at half-past ten. A little before midnight he got out of bed, but did not complain. About two o'clock in the morning his wife awoke and spoke to him. Finding he did not answer, she raised him up in bed, when he smiled at his child, gave a gasp, and died.

Inspection.—The pericardium was filled with liquid and coagulated blood. The heart was very fatty. The aorta was studded with atheroma, which had extensively invaded its middle coat. In the concavity of the arch of the aorta was a hole with rounded edges of the size of a fourpenny piece, that opened into a pouch of the size of a hen's egg, which passed downwards, and the back of which closely adhered to the pericardium. The middle coat of the aorta only passed for a short distance into it. It crossed the bifurcation of the pulmonary artery. Having been opened immediately on its removal from the body, the aperture through which the blood passed into the pericardium could not be discovered, having probably been very small, and having been cut directly through with the scalpel.

There was not a single sign of aneurism present in this case; indeed, it is difficult to see how there could be. For although the sac sprang from the concavity of the arch of the aorta outside of the pericardium, yet its adhesion to this membrane caused it to burst into its cavity without attaining any large size. As far as relates therefore to diagnosis, it may be classed with aneurism arising from the root of the aorta within the pericardium.

An analysis of the signs observed in from 40 to 50 cases of thoracic aneurism, leads to the following results:

1. Aneurism within the pericardium was not indicated during lifetime by any characteristic signs.
2. Aneurism of both kinds outside the pericardium existed without the slightest trace of *pulsation* or *murmur*.
3. On the other hand, pulsations both above and below the clavicles were frequently seen without aneurism.

4. A *pulsating, circumscribed tumour* indicated a sacculated or mixed aneurism.

5. The growth of an aneurismal tumour, and the coagulation of fibrin within it, were in some cases traceable by corresponding alterations in the *quality* of the pulsations.

6. The presence of *purring thrill* over a *circumscribed spot assisted* in determining the nature of the disease, in *conjunction with other signs*.

7. The *intensity* of aneurismal murmur was in a great measure proportioned to the force of the heart's action.

8. A *systolic murmur* heard at a distance from the precordial region, even if distinct from the sounds of the heart, only afforded evidence of aneurism when it was combined with other signs denoting the existence of a circumscribed tumour.

9. A *double* or *diastolic* murmur distinct from the sounds of the heart, indicated the existence of a sacculated or mixed aneurism.

10. When such murmur, double or single, had a peculiarly *hollow* character, a *dilated aneurism* was present.

11. Although there were several cases in which not one of these signs was present, yet there was hardly one in which a careful examination of the chest did not sooner or later reveal the nature of the disease.

Thus in one case, whilst there was an absence of all such signs of aneurism, its existence was discovered by the fact that a moveable tumour fell on or from a branch of the right bronchus, according as it was placed by the position of the patient *above* or *below* its level. In another, the expectoration of fragments of mottled laminated coagula shewed that the tumour, which was known to compress the left bronchus, was aneurismal. In a third, the notable diminution of dullness on percussion of the left side of the chest after copious hæmoptysis, rendered it highly probable that the tumour known to exist in that situation, and to encroach on the pulmonary tissue, was of an aneurismal character.

Hence the importance of the young practitioner carefully studying the records of well observed cases of this kind; constantly referring to them, and comparing with them any doubtful cases occurring in his own practice; rather than trusting to a code of diagnostic rules, too often based on hasty and unsound general deductions.

CHAPTER XII.

DIAGNOSIS OF DISEASES OF THE PERICARDIUM.

PERICARDITIS, INCIPIENT STAGE.—*General Signs—Countenance—Position—Disturbance of Brain—Local Signs—Pain—Dyspnœa—Palpitation—Syncope—Inspection—Percussion—Auscultation—Friction Sounds.* EFFUSION.—*General Signs—Countenance—Position—Pulse—Cerebral Disturbance—Local Signs—Pain—Respiration—Circulation—Inspection—Percussion—Auscultation—Effect on Friction Sound.* TERMINATIONS.—*Absorption—Adhesion.*

PERICARDITIS.—Observation of the progress and termination of pericarditis, and of the pathological changes which take place in its course, lead to the inference that each stage must give rise to signs more or less peculiar to itself. We shall therefore consider separately those observable in the *incipient or dry stage*, in that of *effusion*, and in the *terminations* of the latter state.

INCIPIENT STAGE.—The *general signs* of this stage may be gathered from the *expression of countenance, appearance, position, and movements* of the patient; and from *certain derangements in the functions of other organs.*

The *expression* of the patient's *countenance* at the outset of the disease varies in a direct ratio with the amount of pain. When this is violent the expression becomes

more anxious, evincing great distress and dread; and this is more especially observable when the pain occurs in paroxysms. There are cases, however, of a mild character, where the pain is but trifling, in which a pinched and anxious expression of countenance may be observed; but in such cases, and indeed in many of those where pain is more marked, there is usually some pleuritic affection also.

CASE 78.—*Pericarditis.—Pleurisy.*

A youth, æt. 16, was recovering from an attack of influenza, and was on the point of returning to a public school, when his mother remarked that he seemed in low spirits. On my attention being thus directed to him, I was struck with the peculiar *pinched and anxious expression of his countenance*. On questioning him, he said he felt pretty well, with the exception of a slight stitch under his left breast. His pulse was 110, small, and slightly jerking and irritable. The pain in his side was found to be increased by pressure, not only over the precordial region, but also a little further towards the outer part of the left side of the thorax. No unusual dullness was found on percussion, but on auscultation a distinct, fine, to and fro creaking, feeble but superficial, was heard over the precordial region. So that there was evidently a mild attack of pericarditis and pleurisy. He remained in a delicate state for some time.

Neither the *appearance, position, nor movements* of the patient are much affected in this stage. It is true that in certain cases symptoms of high fever are manifested, with restlessness, throwing about of the arms, and inability to lie in particular positions, whilst a copious moisture bathes the skin; but this only occurs when acute rheumatism or pleurisy co-exists with pericarditis. As, therefore, they are not observed during the invasion of the disease under other circumstances, it is but reasonable to attribute these symptoms more to the acute rheumatism and pleurisy than to the pericarditis. In such cases the pulse is often full, strong, and hard, and has a peculiar, irritable, jerking character. In other cases it is, on the contrary, small, unequal, and irregular.

Cases have been described in which the *first* indications of disease were furnished by *disturbances in the functions of the brain*; delirium, unconsciousness, paralysis of the limbs, &c., where pericarditis was the only lesion discoverable after death; but such cases are rare, cerebral disturbance taking place mostly at a later stage.

Local signs may be derived from the sensations of the patient, *pain, dyspnæa, palpitation*, and a *tendency to syncope*; and from an examination by *inspection, touch, percussion*, and *auscultation*.

Very severe *pain* is sometimes felt at the onset of the attack; it has a sharp, stabbing character; lies chiefly at the precordial region, darting up to the left shoulder, and even down the arm; is sometimes increased, and at other times uninfluenced by pressure over the precordial region, and at the epigastrium. In the latter case the existence of some diaphragmatic pleurisy is to be suspected; indeed, when the disease is complicated with pleurisy, it is difficult to say how much of the pain perceived is due to inflammation of the pericardium. Occasionally pain is produced by pressure which did not exist previously. In some cases no pain whatever is experienced, and between these extremes there is every shade of gradation.

Dyspnæa is not always experienced in this stage of the disease, nor is the act of respiration usually seen to be hurried, except when it produces or increases pain, which is seldom the case unless pleurisy is also present.

The action of the heart, commonly unfelt, may now be perceived, and may amount to strong *palpitations*, often seeming irregular and tumultuous; only, however, in violent attacks. It seldom or ever fails to such an extent as to cause a *sensation of faintness* so early in the attack.

On *inspection* of the thorax the impulse of the heart is sometimes *seen* to be greatly increased, and the same may be perceived by the *touch* when the hand is laid on the precordial region at the commencement of the attack. As soon as

lymph is deposited on opposite surfaces of the pericardium, we should expect that the friction of two roughened surfaces might give rise to a *thrill* appreciable to the *touch*. Such is the case, but it has not the same character as the purring thrill felt when an aneurismal pouch approaches the surface, but seems further removed from the hand. Its intensity, however, much depends on the state of the walls of the heart. If they are thin or degenerated, little or no thrill will be engendered, while it may become considerable when they are hypertrophied, or when the heart is excited to violent action. The only instance I ever knew in which a purring thrill could have been confounded with it was in Case 64, when it arose from diseased pulmonary valves joined to dilated aneurism of the pulmonary artery, a most rare occurrence.

Percussion shows nothing abnormal; but it should be carefully practiced in this stage, in order that the extent of dullness caused by displacement of the lung may be accurately determined, when the patient is in the upright as well as the recumbent position. By this means the future progress of the disease may be more certainly ascertained.

Auscultation.—All the signs hitherto considered sink into insignificance in comparison with those which may be revealed to us by auscultation. At first it helps to discover nothing, but it should be very carefully practised, because we shall by this means learn much of the actual state of the heart before the attack; whether its walls were thickened or had degenerated, and whether any of its valves were affected with chronic disease. Not unfrequently, however, at the very onset a soft bellows-sound is heard either systolic or diastolic; generally this sound is engendered at the mitral valves, where inflammation has been seen so often to accompany pericarditis, and it may therefore be of recent origin. It may be distinguished generally from an exocardial murmur by being clearly heard at the back. It has been

said that exocardial murmur can also be heard in this situation. I have never known a single instance in which this was proved to be the case. Dr. Walshe supposes that in such a case the sounds are engendered in the pleura, and are excited by the heart's movement. It is more probable, however, that such sounds were really generated in the mitral valves, and co-existed with exocardial sounds heard in their usual situation in front.

As the disease, however, progresses, lymph is thrown out on different parts of the pericardium, which being thus roughened can no longer glide noiselessly on its opposite surface, but must necessarily give rise to sounds, which from the mode of their generation are styled friction sounds. They vary extremely in quality, and may be described as *rubbing*, *creaking* (like new leather), *blowing* or *whistling*; sometimes having a lengthened rumbling character, and at others consisting of separate clicks. Generally they are to and fro, or double; sometimes systolic, and very rarely diastolic. These varieties in the quality of the sounds are supposed to depend upon the nature of the exudation, and the absence or presence of a moderate amount of fluid. This is an extremely difficult point to determine, and one of no clinical importance. They vary in intensity from the loud, coarse rasp-sound to a fine distant and almost imperceptible blowing. This depends in a great measure on the action of the heart, the maximum of sound occurring when it is hypertrophied, and in some degree on the spot where it is engendered; becoming more feeble as this is further removed from the walls of the chest. In this latter case it is often impossible to distinguish these sounds from endocardial murmurs. In the majority of cases there is no difficulty in distinguishing them from the latter.

There are, however, two other sounds, both exocardial, and both often superficial. The one is that which occurs in an aneurismal pouch which comes up to the surface, and the other is that which is occasionally

heard in incipient phthisis, and which most probably arises, as has been conjectured by Dr. Kirkes, from slight compression of the aorta or the pulmonary artery, more especially the latter, by an unresisting portion of indurated lung. They are more likely, however, to be confounded with each other than with pericardial murmurs, because they are neither of them heard in the locality of the latter, both being higher up.

Sometimes the pericardial murmurs are made louder by the patient leaning forward, and also by firm pressure of the stethoscope; but the same may occur in regard to endocardial murmurs, although not in so marked a manner. They may in some degree, however, be distinguished from aortic murmurs by not being propagated up the course of the aorta, and from those engendered at the mitral orifice from not being heard behind. Still cases will occasionally occur in which the most skilful auscultator will not be able to pronounce whether the sounds he hears are endocardial or pericardial.

Now, if all, or the greater part of, the general signs were present, and were moderately developed, we should be enabled unhesitatingly to diagnose the existence of the disease. We have seen, however, that very often almost all the signs, general and local, are either absent, or else are so feebly developed as to excite little attention. Andral divides the cases into three groups; those in which the greater part of the signs here described are present, especially pain; those in which dyspnœa is the only sign; and those in which there are no such signs. Practically these run into one another by insensible degrees.

One general sign alone is constant, and that is *frequency of pulse*. This is by no means characteristic, and, therefore, valueless itself as a diagnostic sign; but in another point of view it is of great value, as whenever it exists a careful practitioner will explore the precordial region, and then, should pericarditis or endocarditis be present, he will in all

probability discover their existence by detecting some form of friction-sound or valvular murmur. I have sometimes been led by this sign to suspect the development of endocarditis during an attack of continued fever, and subsequently have found the correctness of my surmise established.

So that although the approaches of pericarditis are often most insidious, and apparently latent, yet if they are looked for, a careful examination will in almost every case enable us to detect them. Is it possible, however, to account for the extraordinary difference which exists between these opposite extremes; those in which the signs are most strongly developed, and those in which none appear? My experience leads me to infer that these signs, more particularly pain, are more strongly marked in proportion to the strength and the activity of the vital powers of the patient at the time of the attack; such as is seen in the early stage of rheumatic fever, and when coming on in conjunction with pleuritis, in a person previously in vigorous health; and that as the strength becomes lowered by illness, such as uræmia, great losses of blood, depressing fevers, and more especially serious accidents and operations, the signs become less and less marked, until they are lost.

CASE 79.—*Endocarditis and Pericarditis after Amputation of the Leg.*

A boy, æt. 5, was received into the Birmingham General Hospital with extensive laceration of the right leg, which had been caused by a waggon-wheel passing over it. The tibia was denuded, and the superficial and deep-seated muscles of the calf were torn from each other. He was restless in the night, and early the next morning he was seized with convulsions, alternating with rigid extension of the trunk and limbs. The pupils were dilated and insensible to the action of light. He was unconscious, but occasionally cried out. His pulse was very feeble, and so frequent that it was difficult to count it. At 10 a.m. the limb was removed by Mr. Hodgson, apparently without the boy being sensible of the operation. The convulsions and spasms ceased in about four hours, and never returned.

He improved rapidly for three weeks, when he was found one day feverish, with flushed face, hot skin, and white tongue. The pulse was quick in its stroke, from 90 to 100. He was fretful and had a slight cough, and the respiration was accelerated. On examining the chest, a little cooing sound was heard here and there, and the action of the heart was quick and jerking. The next day he was still flushed and feverish, and had an anxious expression of countenance. The respiration was more embarrassed. His cough was frequent and dry. The pulse was 100, full, hard, and jerking. The precordial region was dull on percussion up to the third rib. A coarse bellows-sound was heard below the left nipple and up the course of the aorta, but was not heard at the back. Four leeches were immediately applied over the region of the heart; some calomel and Dover's powder were administered, and strong mercurial ointment was rubbed into the precordial region. The next day all his symptoms improved. There was still much cooing sound. He gradually but completely recovered.

It would be out of place to comment on the extremely interesting points of this case in reference to its surgical treatment, any more than to remark that the state of the nervous system seemed to demand rather than to counter-indicate the operation; for by this means the main source of irritation would be removed, and the reflex action controlled. The discovery of pericarditis was unexpected, and shows the necessity for carefully examining the chest for a long time after accidents and operations, since pericarditis is not the only affection which thus insidiously creeps on and destroys the patient.

CASE 80.—*Pericarditis after Amputation of the Leg.*

A fine healthy lad. æt. 15, whilst employed on the Birmingham and Derby railway, was thrown down, and a heavily loaded waggon passed over his right leg and thigh. On being brought to the Birmingham General Hospital it was found that he had received a severe compound fracture of the leg, with great laceration of the soft parts, and injury to the knee-joint. In about half an hour the limb was removed by Mr. Hodgson above the knee.

On the third day after the operation he felt a pain at his heart, with dyspnœa and "catching" of his breath. The pulse was 130, hard and small, the skin was dry and hot, the urine high-coloured and

scanty. The whole of the abdomen was rather tender, and the precordial region very painful on pressure. The respiration was hurried. The action of the heart was quick, but not strong. There was no precordial dullness; but a creaking sound was distinctly heard two inches below the left nipple towards the sternum, but not above it, which persisted during the suspension of respiration. A soft, systolic bellows-sound was heard chiefly to the left of the sternum, between the second and third ribs. Eighteen leeches were applied, with strong mercurial frictions over the precordial region, and calomel and opium were given. The next day the creaking sound was heard higher up the chest, but still near the sternum. The bellows-sound was also heard all up the course of the aorta; but the precordial pain had left him. In three days more the abnormal sounds at the heart disappeared, with the exception of a slight prolongation of the systolic sound. He recovered completely.

The general signs of pericarditis were less obscure in this case, but still were by no means strongly marked.

These two cases were examined at the request of my friend and colleague Mr. Hodgson, and the notes are published with his approbation.

In illustration of this view, it may be mentioned that in applying morphia endermically in a large number of cases of sciatica, it was found that distressing symptoms, as faintness, numbness of the extremities, nausea and vomiting, were felt in a direct ratio with the strength and vigour of the patient. The curative effect was the same in all cases, but in delicate and weakly persons little or no discomfort was induced by the remedy.

EFFUSION STAGE.—*General signs.*—Although the expression of the countenance is affected more by pain than by any other cause, yet it often becomes more anxious when fluid is effused into the pericardium, and the action of the heart becomes thereby more impeded. Then, also, if the circulation becomes affected either by the fluid compressing the flabby walls of the heart, or from their contractile power being diminished by inflammatory action, the appearance of the countenance may become bloated and livid.

The *position* of the patient becomes for the most part

prone, and not unfrequently the slightest movement of the trunk induces a sensation of faintness.

The *pulse* is affected in a remarkable manner. If, before effusion took place, it was strong, full, and bounding, or even in its normal state, it is now more or less feeble and small, and in most cases irregular as to rhythm and power, but still jerking; from the first frequent, the least movement or mental emotion will rapidly raise it by twenty beats in the minute. This alteration in the pulse is very characteristic.

Why *disturbance of the great nervous centres* should frequently appear at this stage, as indicated by symptoms varying from slight twitchings and mental wanderings, to violent tetanic convulsions and wild raving delirium, and on what causes it depends, it is difficult to say. As sudden mental emotion, however, brings on palpitation, or on the contrary, syncope, so affections of the heart may in turn act on the brain. This cerebral disturbance has been seen to such an extent, as to have masked the original disease, and yet it was of a purely functional nature, nothing to account for it having been found in the brain after death. But it must not be forgotten that cases have occurred where the symptoms observed in lifetime seemed to point out the exact locality of mischief in the brain without such being discoverable. A woman was admitted into the Birmingham General Hospital, with hemiplegia and amaurotic blindness; both of which symptoms had come on about ten days before her admission. She rapidly sank, and after six hours' careful examination of the body, no trace of disease could be discovered. It would be useless, therefore, to speculate on the cause of such a complication occurring in any particular case of pericarditis.

Local signs.—*Pain* does not make its *first* appearance in this stage, being neither promoted nor increased by pericardial effusion. A sensation of weight, however, is sometimes experienced when the sac is much distended with fluid.

Respiration is less affected by effusion than might be

imagined; a large quantity sometimes existing without any notable disturbance of the respiratory process; which often depends more on the amount of pain in the pericardium itself, or in the neighbouring pleura, preventing the full expansion of the chest; and on pneumonia, or the compression of a weak flabby heart by the effused fluid, inducing pulmonary congestion, rather than on any encroachment on the lungs by the distended pericardial sac.

The *circulation* is often much affected. The action of the heart previously normal or much increased, may now become more feeble, and more tumultuous and unequal; and strong palpitations may be converted into tremulous fluttering, and this, in proportion as pressure is exerted on the walls of the heart by the effused fluid. Much must therefore depend on the condition of the heart previous to the attack; for, if the walls are hypertrophied, the pressure would have little effect upon them in comparison to what it would have if they were fatty and attenuated, or even in their natural state. In the former case a tendency to syncope may be induced by slight circumstances.

Inspection may reveal a bulging out of the precordial region, more or less marked, as the patient is young and the cartilages of the ribs yielding and elastic; at the same time the intercostal spaces may be obliterated or even bulge outwards; and in some cases, after a time, the integuments over the precordial region may become œdematous, and the heart may be seen beating higher than its normal position.

On the *hand* being *laid* on the chest, the impulse of the heart will be found to be very much less than it was before the effusion, although if the heart is hypertrophied it may be still far from weak. In the majority of cases where the effusion is at all considerable, it will not be felt at all. If there was a thrill perceptible before, it will soon rapidly disappear, as the opposed rough surfaces become separated from each other by the effused liquid.

The signs derived from *percussion* are of great import-

ance. In proportion as the pericardium becomes distended with fluid, more or less of the interposing lung is pushed aside; so that the resonance it affords on percussion is changed into a heavy dull sound, and the finger laid on the spot becomes aware of a dead weight as it were underneath. The extent of the precordial dullness is thus notably increased, and sometimes extends up to the first rib, and even to the clavicle. It has more or less of a triangular shape, the apex pointing upwards; but its shape may be somewhat varied by gravitation of the fluid on the patient changing his position to either side.

Before, however, we interpret extended dullness over the precordial region, as a sign of pericarditis, all sources of error must be excluded; because such dullness may arise from other causes. An increase in the size of the heart itself, arising either from hypertrophy or dilatation, or both combined, may have this effect. In that case the dullness will have existed from the beginning. The shape of such dull space will be as wide at its upper part, and even wider, than at its base, the reverse of what occurs in pericardial effusion. When the auricles are dilated, as is usual when the extent of dullness is much increased, a soft undulating pulsation is often both seen and felt; and when much hypertrophy exists the impulse of the heart is strong and heaving, instead of being weak and fluttering as in simple effusion.

A mediastinal tumour or aneurism of the aorta approaching the surface may also occasion dullness on percussion; but neither the shape nor locality will be identical with that arising from effusion, to say nothing of other signs belonging to these forms of disease, which will be discussed when they are treated of.

Condensation of the lung, pleuritic effusion, and enlargement of the left lobe of the liver may all, more or less, increase the extent of dullness; but it is quite unnecessary to point out the obvious distinguishing signs of these affections.

When a careful observer finds the precordial dullness increasing *day by day*, he may safely conclude that it arises from effusion within the pericardium ; and thus in this stage of the disease, percussion is almost an unerring guide to sure diagnosis.

If *auscultation* has been practiced from the onset, and a friction sound has been detected, a time will come when such sound will become fainter and fainter, and, eventually, cease, having been last heard near the base of the heart. If at the same time percussion shows the extent of precordial dullness to be gradually increasing, we may be certain that effusion has taken place, has separated the folds of the pericardium, and prevented their rubbing against one another ; such separation not being complete at the base of the heart, where the membranes are reflected from the outer sac on the walls of the heart, until a considerable amount of fluid has been poured out. At the same time the natural sounds of the heart will become more and more feeble, and in some cases will be hardly perceptible when the patient lies on his back ; and they will be found irregular as regards intensity and rhythm, often intermitting.

TERMINATIONS.—The termination of an attack of pericarditis by *absorption* is attended by a gradual diminution of the extent of the precordial dullness, while the attrition sounds may, more or less, reappear for a time as the roughened surfaces of the pericardium are once more allowed to come in contact with each other, and before the lymph is reduced to the state of a milky patch.

When absorption does not take place, and the heart remains surrounded by fluid and solid exudation until the patient sinks, the precordial dullness of course remains ; and sometimes a friction sound of a peculiarly moist, gurgling character, will be heard near the base of the heart.

When, on the other hand, *adhesion* takes place, partial or general, whilst the process is going on, after the absorption of the greater part of the effused fluid has taken place, loud friction sounds are often heard, varying in locality and

intensity, sometimes resembling a succession of *clicks*. When *complete adhesion* has taken place, all dullness on percussion and friction sounds necessarily cease. Various signs have been brought forward by different writers as indicative of *adhesion*. Great stress has been laid on a peculiar *jogging action of the heart*. This was only noted by me in one of 12 cases or more. In fact the action of the heart varied, as it may be well supposed it would, according as that organ was hypertrophied, or weak and flabby, dilated, &c. In one case it was "strong and quick, like the stroke of a hammer," in two or three, very feeble and irregular, and in others moderate in force.

Some writers describe the heart as having been forced higher up than its normal position by the effused fluid, and retained there by adhesion of the pericardium, evidenced by its beat; but the position as well as the action of the heart must depend more on its *size* than anything else. Even, however, were it so, unless the precordial region had been carefully examined before, or at the very onset of the attack of pericarditis, the difference in the position of the heart would hardly be discoverable in practice.

Again, the comparative *immobility of the heart*, and the *diminished effect on precordial dullness* produced by inspiration and expiration, may be assumed *theoretically*, but *practically* have not been found by me to be sufficiently marked to be considered signs diagnostic of adhesion.

Undulations or retraction of the integuments over the precordial region, or at the epigastrium, synchronous, or nearly so, with the ventricular systole, may certainly sometimes be observed in these cases, as was pointed out by Dr. Saunders; but not unless adhesion has also taken place between the outer surface of the pericardium and the neighbouring costal pleura and diaphragm. Such an appearance has been observed between the seventh and eighth left ribs, particularly in Case 28, when it was caused by the adherent pericardium being connected with the costal pleura in that spot by long bands of false membrane.

On the whole it cannot be affirmed that there are any constant and infallible signs of adhesion of the pericardium; although I believe it may frequently be pronounced to have taken place when the precordial region has been carefully explored from the commencement of the attack.

It was suspected in one or two cases, of which the following is one:—

CASE 81.—*Adherent Pericardium—Mitral Obstruction—Dropsy.*

A girl, æt. 8, was for nine months afflicted with articular rheumatism in the elbows, wrists, and finger-joints, the latter being quite distorted with swellings, some of which were as large as nuts. These gradually disappeared, and after some months' time she was in tolerably good health. Four months after this she took cold, from sitting in a cold damp place for some hours. She became an out-patient of Addenbroke's Hospital, Cambridge, but after a week was too ill to attend, and I visited her at her own home. She was suffering from intense dyspnœa and orthopnœa, and complained of great pain and tightness across the region of the heart. There was slight œdema of the ankles. The action of the heart was violent, and its pulsation strongly marked under the left nipple. About this point a slight systolic bellows-sound was heard. The chest sounded well on percussion, and the respiratory sound was natural. Leeches were applied to the precordial region, and this was followed by strong mercurial frictions. Effusion, however, gained the abdomen, and the patient quickly sank.

Inspection.—The pericardium was universally adherent to the heart. The left auricle was greatly dilated, and filled with a black clot. The mitral valves were fringed with osseo-cartilaginous vegetations, with sharp-pointed spiculæ, one of which was a quarter of an inch long.

This case occurred in 1834, when shortening of the chordæ tendineæ would certainly have been overlooked by me. If, however, the valves were perfect, they must have closed the tricuspid orifice, and then this would be a case in which dropsy occurred without any apparent obstruction to the circulation on the right side of the heart. I have met with two other such cases in the practice of others.

CHAPTER XIII.

DIAGNOSIS OF DISEASES OF THE HEART.

ACUTE DISEASE—*Endocarditis*. CHRONIC DISEASES.

Not Organic—Hysteria—Dyspepsia—Anæmia—Murmur—Angina Pectoris—Organic—Walls and Cavities—Increase of Contractile Power—Decrease of Contractile Power—Fatty Degeneration—Pulmonic Congestion—Polypoid Coagula—Globular Coagula—Endocardium and Valves—Aortic Orifice—Constriction—Murmur—Seat—Direction—Regurgitation—Visible Pulsation at Wrists—Murmur, Diastolic or Double.—Mitral Orifice.—Constriction—Diastolic Murmur—Regurgitation—Pulsation between Second and Third Ribs—Systolic Murmur—Seat—Direction. Pulmonary Orifice—Tricuspid Orifice—Regurgitation—Anasarca—Cerebral Congestion—Venous Pulsations in Neck—Absence of Murmurs. General Conclusions.

ACUTE DISEASE.—*Walls*.—The existence of general *carditis* being problematical, or at any rate of extreme rarity, and partial *myo-carditis* being always mixed up with pericarditis or endocarditis, I know of no signs by which they can be discovered.

Cavities.—*Acute endo-carditis* does not reveal itself in its earliest stage by any diagnostic sign. There is usually

little or no pain, and the palpitations and irregular action of the heart simply indicate irritability of that organ, which may arise from various causes. It is not until a notable effect has been produced on the endocardium, and more especially the valves, and a *murmur* has been engendered, that we are enabled to recognize the existence of endocarditis. Even then, caution is required in interpreting the meaning of the murmur; for if, as is often the case, pericarditis is also present, it is sometimes very difficult to distinguish the exo-cardial from the endo-cardial sounds; and when it occurs alone, or in the course of acute rheumatism, it is impossible at first to determine whether the murmur is the result of a previous attack, or has been engendered recently, unless the sound has been developed under our ears, as it were. Some authors, indeed, conceive that they can distinguish between a recently engendered and an old standing murmur. When, however, a soft systolic bellows murmur is heard in rheumatic fever, near the mitral or aortic orifices, accompanied by signs of cardiac irritability, we may generally infer the co-existence of endo-carditis.

There are certain cases which often puzzle the most experienced practitioner, in which it is not impossible that a sub-acute form of endocarditis may be present. The patient complains of some slight ailment, it may be in the head, chest, or abdomen, and is treated accordingly; but after such symptoms have been relieved, the pulse remains frequent and rather small. The sounds of the heart are seldom affected, except that sometimes the diastolic sound is unusually sharp. Phthisis is often, and with just cause, suspected; but there is little or no dyspnoea, and no abnormal sounds can be detected in the lungs. After a time the pulse falls, and the patient recovers. In four of such cases, which were kept steadily in view, after an interval of from two to five years, *valvular disease supervened*.

We now proceed to the consideration of the signs afforded

by the more *chronic* diseases of the *walls* and *cavities* of the *heart*, the result of previous inflammatory action, or of morbid nutrition.

CHRONIC DISEASES.—*Non-organic*.—The various organic diseases, the formation, progress, and termination of which we have seen, give rise to *general* and *local* signs during life-time, more or less indicative of their existence. But amongst these signs are some which occur *without any organic disease* such as *pain*, *palpitation*, *irregular action of the heart*, *dyspnœa*, *syncope*, and even *murmur*. We need not feel surprised at this, when we consider that these symptoms may be produced under certain circumstances in persons otherwise perfectly healthy. Thus, palpitations, dyspnœa, irregular action, and even pain, are induced by violent running or other exercise. In this case the heart is stimulated by an unusual amount of blood being forced into it by compression of the veins. Sudden and unexpected news, again, will bring on some of these symptoms; and also possibly take away the appetite for the time; the impression made on the brain being through the pneumogastric nerve, communicated to the heart and the stomach. In others, a sudden shock will arrest the heart's action, and induce syncope.

The same result takes place in the course of certain diseases, more especially *dyspepsia*, *hysteria*, and other forms of *nervous irritability*, *anæmia*, &c.

It might be imagined that the inconstancy of these symptoms in such diseases, contrasted with their persistence in organic affections of the heart, would assist materially in distinguishing between one and the other. But this is not the fact in practice; for even in serious and permanent diseases of the heart, the general signs are often intermittent, and remain for a time quiescent. In the great majority of cases, we can certainly distinguish the one from the other, but it requires time, care, and skill to do so. On the one hand, we must, by careful exploration of the precordial region, find whether there are other traces of organic disease, and if none,

then we come to the affections resulting from functional derangement *par la voie d'exclusion*. On the other hand, we must search for characteristic signs of the different non-cardiac affections, and shall not have much difficulty in distinguishing between, *dyspepsia, hysteria, anæmia, &c.*

In the two following cases it was at first very difficult to ascertain whether or not there was any organic disease of the heart or aorta :—

CASE 82.—*Jaundice—Pulmonic Congestion and Apoplexy.*

A gentleman, æt. 56, was attacked with congestion of the liver and the lower part of the right lung. He soon became slightly jaundiced. He was proceeding favourably, when, walking upstairs contrary to orders, he was suddenly seized with the most urgent dyspnœa, accompanied by distressing palpitations, the pulse rising from 80 to upwards of 140. He remained several days in such a state, that the least exertion, even the movement of his hands, induced suffocating dyspnœa, the pulse remaining at the same high rate. During this time he was expectorating some very dark thick blood. The dullness of the right side gradually diminished, and the breathing became tranquil. One day the pulse suddenly fell to 76. An eminent physician from London, since dead, saw him and considered he was labouring under aneurism of the thoracic aorta, but this was not the opinion of those who watched him constantly; the symptoms leading them to attribute the dark bloody expectoration to pulmonary apoplexy. He slowly but steadily recovered, the peculiar heart symptoms remaining unexplained. About eighteen months afterwards he was attacked whilst in London with symptoms indicative of the lodgment of a large amount of fecal matter in the caput coli. Whilst recovering, he was suddenly attacked after his first drive, and whilst walking upstairs, in an exactly similar manner to that above described as occurring in his former illness. This attack passed off in the same manner after some days, the pulse one day suddenly falling to 72.

The subsequent history of this case leads to the conclusion that there was no organic affection whatever of the heart, but that it was excited in this peculiar manner by sympathy with another organ; a different one in each attack.

CASE 83.—*Hysteria.*

A young lady, æt. 27, was attacked one day whilst ascending the stairs with sudden dyspnœa and giddiness, followed by tumultuous palpitations. Seen the next day, it was found that the most trifling movement of the body or arms, or even an attempt to speak, brought on the most intense panting dyspnœa. The breath sounds were entirely masked by a panting with a metallic ring to it, exactly like the noise of a railway train at a great distance.

It seemed almost as if one of the lungs must have given way and collapsed. The next day, by taking great precautions, an examination of the chest was effected without the panting being excited; and it was then found that no abnormal pulmonic or cardiac sound of any kind could be discovered. It was, therefore, concluded that the heart's action was induced by sympathy with some other organ. Nothing could be detected near the root of the lungs, nor was there any spinal tenderness. It was found, however, that there had been some irregularity of the catamenia, and that three or four years ago she was suffering from what was called spinal irritation. The uterus, therefore, seemed the only organ which could be fixed on as the exciting cause, and the disease was pronounced to be hysteria. The patient slowly and steadily recovered, without any trace of organic disease of the heart.

The murmur which so often accompanies anæmia has been seen to resemble most closely the bellows-sound heard in certain permanent affections of the valves; very frequently, however, it will be found that in the former case it disappears, or is very much weakened in repose, particularly during sleep; and sometimes, too, its intensity can be increased by pressing the stethoscope on the precordial region, and the *bruit de diable* can be made to appear by gentle pressure on the veins; for although its formation may, in some degree, depend on the peculiar state of the blood in spanæmia, yet its intensity bears a certain ratio to the force of the heart's action. A systolic murmur occurring in other cases where there is no organic disease, arising from the violent jerking action of the heart, which often takes place in hysteria and other deranged states of the nervous system, is only heard during the time these palpitations occur, and therefore would not

be confounded with permanent valvular murmur of confirmed cardiac disease.

Angina Pectoris.—There is one form of pain which demands particular attention, and has obtained the name of *Angina Pectoris*. It generally comes on suddenly in the precordial region, shooting backwards and down the left arm. It is of a stabbing character, and at once seems to knock down the patient, and stamps the countenance with an expression of intense anxiety and dread. It has been seen to occur in almost every form of organic disease of the heart, but most frequently when there is ossification of the coronary arteries, and fatty degeneration of the walls of the heart. It is, therefore, of no value as a diagnostic sign, revealing any particular affection, but merely denotes some organic lesion, and calls for immediate measures for relief, if, indeed, any such can be devised.

Organic Diseases of the Walls of the heart have been seen to give rise to an *increase* or a *decrease* of their *contractile power*.

Increase of contractile power, if constant, must arise from *hypertrophy* of the left ventricle, or both. If it be great, the *impulse* communicated to the hand laid on the chest will be considerable, and will have a *heaving* character; and violent pulsation will be perceived, not only in the arteries of the neck, but above and below the clavicles, as seen in Case 56. Here the state of the pulse did not correspond with that of the carotid, as the calibre of the arteries of the arm was diminished at their origin by atheromatous degeneration. The extent of *precordial dullness* is increased, not extending so much upwards as when it arises from effusion in the pericardium, but more downwards and laterally, because, as has been seen, more or less *dilatation* accompanies the hypertrophy. The intensity of the systolic sound is diminished, and it assumes somewhat of a muffled character, because a thicker medium is interposed between the valves and the ear.

In hypertrophy by *extent*, however, when the walls

are not thicker than in their normal state, the *quality* of the systolic sound may be unaffected, whilst its *intensity* is increased, and it is heard at a greater distance than usual from the point where it is engendered. In simple hypertrophy neither dyspnoea nor dropsy are to be expected, because it has been shown that hypertrophy of the ventricle does not of itself produce any notable congestion of the vessels, either of the pulmonic or the systemic circulation, although it may augment it when induced by other direct causes. But in a very large number of cases dropsy will be found, because the dilatation, which accompanies the hypertrophy so often, renders the tricuspid valvular apparatus incomplete; and thus the most direct cause of congestion of the systemic circulation is superadded.

In these cases the extent of heart uncovered by lung, and consequently in immediate contact with the walls of the chest is often increased, and thus a metallic resonance is communicated to the sounds of the heart. The following simple experiment will show how this arises:—Let the hand be formed into a hollow shape, as if for the purpose of receiving and retaining water; and then let it be placed over the ear, so that no part shall touch the external ear. If the back of this hand be struck with the finger of the other hand, nothing particular will be observed. Then let the hand be opened, and laid flat on the external ear; on its being struck a metallic ringing sound will be most strongly engendered. The air which was contained in the hollow of the hand, and which separated it from the ear, represents the spongy air-filled lung, which is usually interposed between the heart and the ear, except over the small space uncovered by lung. When a larger portion of lung is displaced by a dilated right ventricle, a much larger portion of the heart is brought into immediate contact with the ear, and hence the metallic ringing sound engendered.

This is well illustrated in the following:—

CASE 84.—*Adherent Pericardium—Mitral and Pulmonary Regurgitation*

A gas-burner maker, æt. 30, a confirmed drunkard, after taking cold, was ill three months, and ever since has had a pain at the heart; and one night, whilst smoking, felt as if something had burst there. Since then he has had more or less of palpitation, but has continued to work. Three months since he got wet through, and began to cough and expectorate white phlegm; the pain at the heart became severe, and he had also pain in the epigastric region. Palpitation increased, he lost flesh and strength.

He was bled and blistered without much relief. On his admission into the Birmingham General Hospital he had strong palpitation, and complained of violent pain at the heart on the least exertion. He had slight cough, with clear expectoration, and great dyspnœa on moving. He was much troubled with dreams. His bowels were confined, and his urine was scanty and high-coloured, and deposited a sediment like bran, but contained no albumen. The pulse was 116, feeble.

The chest sounded nowhere clear on percussion, but was very dull from the sternum to the outer edge of the left side below the left nipple. The respiratory sound was weak over the front of the left side of the chest. The action of the heart was very strong and quick, like the stroke of a hammer. The sounds were sharp, and were heard all over the chest, the diastolic sound being a short bellows-sound, which was afterwards occasionally absent. *A metallic resonance accompanied the heart's impulse.* He soon died without any trace of dropsy, and apparently from dyspnœa.

Inspection.—The lungs were congested but not friable. The pericardium adhered to the whole of the heart by means of a gelatinous lemon-coloured substance. The heart was placed almost transversely across the chest, its apex reaching to the extreme left side of it, *and the whole of this space was uncovered by lung.* It was greatly dilated, more particularly on the left side. The parietes of both auricles were hypertrophied, especially the left, which in one place were four lines in thickness. The lining membrane of the left auricle was opaque and thickened, having horny patches on it, but inseparable from it. The mitral orifice was a little contracted, so as with difficulty to admit two fingers. The free edges of its valves were covered with fibro-cartilaginous vegetations, of the consistence and appearance of soft horn; two of them being three-quarters of an inch in length, five lines wide, and two lines thick. These bodies were fringed with vermiform vegetations, about the size of crow-quills. The valves were thickened and

large, but seemed adapted to each other, the vegetations hanging into the ventricle when they were closed. The orifice of the pulmonary artery was so much dilated that it admitted four fingers.

The stomach was pushed downwards by an immense liver and spleen. The liver was hard, and had a mottled nutmeg appearance. The spleen measured 11 inches by 6. Its upper half was reduced to a dirty red pulp, and adhered to the diaphragm. The edge of the softened part was coated with yellow cheesy-looking matter, of which considerable patches existed in the remaining half of the spleen. The cortical substance of the kidneys was pale.

This patient would appear to have had repeated attacks, both of pericarditis and endocarditis; the gelatinous substance which connected the pericardium being the product of the last recent attack. The enormous vegetations on the mitral valves must have materially interfered with their action, and probably sometimes got between them, and thus allowed regurgitation to take place. Had the diastolic bellows-sound been occasioned by regurgitation through the pulmonary orifice, it would have been constantly heard. The space allowed for regurgitation was probably too large to give rise to sound. It is more likely that this sound arose from obstacles to the flowing of the blood through the mitral orifice, varying with the position of the large horny excrescences.

Mitral regurgitation can have little effect in obstructing the systemic circulation, otherwise more decided symptoms of such an obstruction would have shown themselves in this case. Case 87 also illustrates this.

Death probably resulted from the joint effects of pericarditis and mitral obstruction.

Decrease of contractile power may be the result of *attenuation*, or *degeneration* of the ventricular walls, and possibly of *adhesion of the pericardium*.

When simple *attenuation* exists, or when the muscle becomes notably softened by *fatty degeneration*, the impulse of the heart is often greatly diminished, and sometimes even cannot be felt at all. When, as is generally the case, the attenuation is the result of simple dilatation,

the extent of the precordial dullness is increased. In that case the sounds of the heart, although often very feeble, are clear and well defined, and are heard to a much greater distance than usual from the spot to which they are ordinarily confined. When the ventricles are softened by degeneration, especially if they are at the same time hypertrophied, the sounds are extremely feeble, sometimes indeed inaudible, and almost always muffled and confused. The following case illustrates this :—

CASE 85.—*Fatty Degeneration — Tricuspid Regurgitation — Dropsy—Death from Cerebral Apoplexy.*

A female, æt. 60, some years ago, had an attack of acute rheumatism, ever since which time she had a cough, with copious clear expectoration. During the last seven months she had suffered much from dyspnœa and palpitations. The pulse was feeble and irregular. The veins of the neck were much swollen and knotty, pulsating feebly. The ankles were œdematous.

The precordial dullness was greatly extended both downwards and laterally. The action of the heart was fluttering and irregular, and its impulse almost imperceptible. A soft blowing sound, thought to be systolic, was heard under the middle of the sternum, very near the ear, but *no other cardiac sound could be heard*. The respiratory sounds were natural. There was no trace of albumen in the urine. In a short time serous infiltration had gained the thighs, when she was suddenly seized with cerebral apoplexy, and died in twelve hours.

Inspection.—A great quantity of black coagulated blood was spread over the surface of each hemisphere of the brain, between the dura mater and the arachnoid membrane. There was a clot, with laceration of the interior of each hemisphere.

There were milky patches on the pericardium, one thicker than the others, of the size of a shilling, on the upper surface of the right ventricle. The heart was dilated to an enormous size; its walls, of the usual thickness, were soft and flabby, of a yellowish mottled colour, and crepitated like a piece of lung between the fingers. The valves were healthy. The circumference of the tricuspid foramen measured nearly six inches, and its valves could not effect its closure. The other organs were healthy. There was atheromatous degeneration of the arteries, both of the chest and the head.

The state of the veins of the neck from the first

indicated incompleteness of the tricuspid valvular apparatus. The causes of the bellows-sound, and of the precordial dullness, and the extreme feebleness of the heart's action were, however, considered doubtful. Effusion in the pericardium might have, in some degree, caused the two latter phenomena, whilst the bellows-sound might have been valvular. On the other hand, the extreme proximity of this sound to the ear led to the suspicion that it was exocardial, in which case the dullness on percussion could hardly have arisen from effusion into the pericardium, for this would, by separation of the surfaces of thin membrane from each other, have prevented the formation of any friction-sound. The dullness would then arise from a very dilated heart, and its feeble action from softening, and degeneration of the walls of the ventricles. Inspection alone cleared up the doubt.

In this class of cases dyspnœa, dropsy, or cerebral congestion may be naturally looked for, because as congestion of both circulations has been found to be chiefly *venous*, it would be greatly favoured by any causes which tended to enfeeble the heart, and diminish its contractile power, whereby the circulation of the blood being retarded, it would have a tendency to collect and stagnate in the venous capillaries.

Morbid Products within the Cavities.—When *polypoid concretions* are formed towards the close of life by the coagulation of the blood within the cavities of the heart, it is natural to suppose that symptoms of obstruction to the circulation thus occasioned should somewhat *suddenly* come on. Accordingly, some of those we are about to enumerate are thus occasionally observed, and are doubtless due to such a cause. Thus the pulse rapidly increases in frequency, and becomes at the same time small, weak, and irregular; faintness, coldness of the surface, and an anxious expression of countenance are seen, and are sooner or later followed by lividity, dyspnœa and other symptoms of pulmonary congestion. But when such concretions are formed

more gradually, and more particularly when they assume the form of warty excrescences and globular vegetations, I know of no means by which their presence can be ascertained. The following case, however, is too remarkable to be passed over :—

CASE 86.—*Globular Vegetations in the Right Ventricle of the Heart.*

A muffin-seller was admitted into Adenbroke's Hospital, December, 1834. He had long suffered from dyspnœa and frequent attacks of bronchitis. Six months since he took a violent cold, and had constant dyspnœa ever since. He felt slight pain over the whole of the chest, rather severe at the precordial region, and had frequent and violent fits of palpitation. The expectoration consisted of thick, nummular, semi-transparent, gelatinous sputa, interspersed with much black pulmonary matter, but presenting no trace of pus. The action of the heart was quick and irregular, and communicated a metallic vibration to the ear laid on the chest. The sound was heard at some distance from the chest. Dullness on percussion and vocal resonance was perceived here and there over different parts of the chest, so loud behind as almost to sting the ear; and in spots coarse bronchial respiratory sound was mixed with vesicular and puerile respiration. A mixture of sub-crepitant and sonorous rattles was heard all over the back, and a part of the left side of the chest. In a few days he began to sink, the pulse became very weak and rapid, and his mind wandered. At every fifth or sixth beat of the heart a sound was heard like that of a bubble bursting in a hollow metal tube or bottle, loudest at the apex of the heart, and diminishing in intensity as the ear receded from this point, so that it could not be engendered in the stomach. As a similar sound had never before been heard, the attention of Dr. Haviland, and Mr. Johnson, the house-surgeon, was drawn to it, and it was verified by them. The next day he died.

Inspection.—Under the idea that the peculiar sound heard in this case might have been occasioned by air in the pericardial sac, it and the heart were opened under water with the greatest care; but no air whatever escaped.

The greatest portion of both lungs were œdematous, very pale, and studded with much black pulmonary matter. Several masses of yellowish grey induration were scattered here and there, which gave out no pus on pressure (*chronic pneumonia*). The pericardium contained three ounces of lemon-coloured serum. The walls of the left

ventricle were firm and red, nine lines thick near their bases; those of the right were flabby, six lines thick, of which four and a half consisted of yellow fat. There was atheromatous degeneration at the base of the mitral valves, and the aortic valvular zone was calcified. In the cavity of the right ventricle were a great number of yellowish-white globe-shaped bodies, varying in size from that of a mustard seed to a hazel nut. The smaller ones were solid, with a red speck in the centre. The larger ones were hollow, and contained a dirty, purulent-looking fluid. Their walls were about as thick as the coats of the femoral artery, and a section of them showed that they were composed of concentric rings. There was much atheromatous degeneration throughout the arteries, including the coronaries.

Never having heard a sound similar to that which was perceived a little before death in this case, the works of those authors who had written on the subject of chest diseases were consulted; and, strange to relate, an exactly similar case was found in Laennec's *Treatise on Auscultation*.* It was that of a female who died of pulmonary phthisis. The sound was described as "resembling that which a bubble produces in disengaging itself from a fluid, or the "cliquetis" of water agitated in a glass decanter with thin walls;" and a number of globular vegetations exactly similar to those in this case were discovered in the right ventricle. Laennec observes in a note, "one might attribute this phenomenon to the existence of the globular vegetations in the heart, but I do not attach much importance to this sign. I have heard it in other cases, particularly in one of hydro-pericardium, with pneumo-pericardium." Nearly thirty years have now passed, but I have never heard a similar sound; and although it is remarkable that it should have been heard in both these cases of globular vegetations, I can offer no explanation of the mode of its production.

If *derangement of the valvular apparatus* is suspected, we have to determine its *seat* and *nature*. Here attention is at once directed to certain orifices of the heart by the general signs present. From what has been seen of the progress

* *Traité de l'Auscultation*. Paris, 1831. Vol. 3, p. 239.

and termination of these diseases, if there be neither urgent dyspnœa, nor any signs of obstructed systemic circulation, we shall suspect the aortic orifice; if great dyspnœa be present, we shall look to the mitral foramen; and if signs of obstruction of the systemic circulation, we shall expect to find tricuspid regurgitation, with or without disease of the valves on the left side of the heart, according as evidence of urgent pulmonary congestion exists or not. We will now then proceed to examine in succession the signs of derangement of the valvular apparatus at each of these orifices.

Aortic orifice.—The signs indicative of disease of the aortic valves, vary in some degree with the form such disease assumes, and the manner in which the flow of the blood is thereby affected. In one form there is simple *constriction* of the orifice, whereby *obstruction* is produced; in another there is *insufficiency* of the valves to close the orifice to which they are attached, in consequence of which *regurgitation* takes place during the ventricular systole.

Obstruction.—When this exists alone, or in a moderate degree, it gives rise to no marked *general* signs; and indeed it may be very extensive and of long standing, without inducing any notable disturbance of the health. The pulse is but slightly if at all affected, and neither dyspnœa, dropsy, nor any other signs of impeded circulation make their appearance. Or else some signs may show themselves, and afterwards disappear as the case progresses. Thus in Case 30, violent and protracted fits of palpitation occurred at intervals for some years, and then gradually subsided, as the heart accommodated itself to the obstruction.

The chief *physical* sign is afforded by the manner in which the first sound of the heart is modified or replaced by a murmur; a slight thickening of a deposit on the valves giving it a dull muffled character; and vegetations, or calcareous concretions, engendering a murmur which varies from a gentle soft blowing sound, to that resembling

rough sawing or rasping. Such murmur is of course *systolic*, and its *intensity* is in an exact ratio to the force with which the left ventricle contracts, whether increased temporarily by nervous excitement, or permanently by hypertrophy; or decreased, on the other hand, by attenuation and softening. When this murmur has a soft and blowing character, it is sometimes impossible to distinguish it from exocardial or inorganic murmurs, even with the aid of such distinguishing marks as have been pointed out when treating of their diagnosis.

There is no peculiarity in the *nature* of such sound when engendered at the aortic orifice to distinguish it from similar sounds formed at other orifices of the heart.

It has been asserted, however, that such murmurs are heard loudest at the exact spot on the surface of the chest which lies immediately over the orifice in which they are respectively engendered. Thus, disease of the aortic valves would give rise to a murmur, the greatest intensity of which would be under the left edge of the sternum below the third rib, when the heart was not displaced by excessive hypertrophy or any other cause. Not unfrequently, however, the maximum intensity of such sound is heard considerably to the right of this point. When this has occurred, however, there has generally been more or less hypertrophy of the left ventricle, as in Case 46.

It has therefore often been found impossible to determine the orifice at which the murmur was engendered, from the spot at which it was most distinctly heard in lifetime.

A more valuable sign, however, is furnished by the *direction* in which valvular murmurs are propagated, as was noticed by Dr. Williams. Thus, when one is formed at the aortic orifice, it can often be clearly traced for some distance up the course of the aorta. When it is heard louder over the arch than elsewhere, it has been held that it is engendered in the aorta itself; but this is not necessarily so, as has been shewn in the remarks on aneurismal murmurs, and as was illustrated by Case 66.

In fact, the transmission of such sounds depends more on the state of the lungs, the conducting medium, than on any other cause; a small consolidated portion between the aorta and the walls of the chest, serving to carry aortic valvular murmur to the ear at some distance from the point of generation with remarkable distinctness, as in the case above quoted.

Regurgitation.—When the valves are insufficient to close the orifice, and regurgitation takes place, there are still no marked signs of impeded circulation. The pulse, however, is peculiarly modified, becoming more or less resilient, and even double; thus resembling that which is sometimes found in dilatation of the aorta.

But the chief sign is afforded by *visible pulsations* of the arteries at the wrist and other places where they approach the surface of the body, as first noted by Dr. Corrigan. Rarely, if ever, does aortic regurgitation take place without visible arterial pulsations. It must be remembered, however, that visible pulsations have been seen in cases of marked hypertrophy, without any defect at the aortic orifice; and therefore they can only be considered as a sure sign in the absence of hypertrophy.

The murmur in this case is either *diastolic* or *double*, according as there is simple inefficiency, such, for instance, as would result from shortening of the valves; or as there is a permanent obstruction also, such as might take place when the valves were glued together, so as to make a fixed open ring.

While the systolic sound is more or less propagated up the course of the aorta, the diastolic sound has a tendency to spread towards the lower part of the sternum.

CASE 87.—*Aortic and Mitral Regurgitation.*

A gun-barrel filer, æt. 30, the notes of whose previous history are mislaid, at the time of his admission into the Birmingham General Hospital was emaciated, and laboured under great dyspnœa. All the arteries which approached the surface were seen to pulsate violently.

There was a strong heaving impulse two inches below the left nipple. The pulse was resilient. Above the nipple and up the course of the aorta there was a double rasp-sound, the diastolic being stronger, coarser, and more prolonged than the systolic sound. Two inches below the left nipple the systolic sound alone was heard. Fourteen days after this a purring thrill was felt up the course of the right carotid, but not up the left. During the act of coughing a lump as large as a nut was seen to rise up above the right clavicle, over which a purring thrill was felt. In that spot a single systolic rasp-sound was heard. To the left of the sternum the diastolic sound was faintly heard, but *under the middle and bottom of the sternum it was rough and prolonged.* No more notes of this case during lifetime can be found. He died five weeks after his admission into the hospital.

Inspection.—The pericardium adhered to the side of the thorax on a level with the sixth rib. It contained five ounces of clear lemon-coloured serum. There were milky spots on the heart, which was firm, red, and rather small. The wall of the left ventricle was eight lines thick across the middle. The aortic valves were thick, shrivelled, and small, with cartilaginous bases; they could unfold, but were insufficient to close the aortic orifice. The circumference of the mitral orifice measured four inches. The ventricular side of the mitral valve nearest the aorta was covered with patches of warty vegetations, and was eight lines in height. The other valve was thick, shrivelled, and much shorter, and all the chordæ tendineæ were shortened and thickened. Two of the tricuspid valves were one inch deep, and the other half an inch. The circumference of the tricuspid orifice measured three inches and a half. The arteria innominata was contracted at its origin and dilated beyond that point. There were some patches of old adhesion between the bottom of the left pulmonary pleura and the side. The top of the left lung was surrounded by a cartilaginous cap, and the lung itself at this spot was puckered and contracted. Both lungs were much engorged, and there were patches of brown-coloured, dryish hepatization, in which there was no crepitation, nor was there any offensive odour. There was no trace of dropsy.

The notes of a clinical lecture on this case, which have been preserved, show that the leading symptoms throughout were dyspnœa and palpitation, and that death ensued from what may be styled chronic suffocation, without a trace of anasarca having been manifested. A comparison between the physical signs during lifetime and the appearances after death is remarkably instructive.

Mitral Orifice.—Obstruction.—The general signs indicative of disease of the mitral orifice are more marked than in valvular aortic disease. Thus the impulse of the heart is often irregular in force and rhythm, and the pulse small, tremulous, and irregular; but these are far from being characteristic. Dyspnoea is the most constant, and in certain cases, the most significant sign. It is usually accompanied by cough and watery or bloody expectoration. When these symptoms are urgent, and a careful exploration of the chest affords no evidence of primary pulmonary or pleuritic disease, we at once turn to the mitral orifice for its cause.

The chief physical sign of mitral obstruction is a diastolic murmur. It is rarely, however, that such a murmur is engendered, because the size of the auriculo-ventricular opening is so large that a considerable amount of disease may exist without sufficiently narrowing it to give rise to sound. On the other hand, the amount of disease may be such that the aperture is contracted to a very narrow slit, through which the small stream passes noiselessly.

CASE 88.—*Aortic and Mitral Obstruction—Sudden Death from Syncope.*

A tradesman's wife, of small stature and delicate constitution, a native of Poland, had rheumatic fever at six years of age. Latterly she had suffered from palpitations and dyspnoea on over-exertion or excitement. The action of the heart was heaving, irregular, and tumultuous, but *its sounds were normal*. During the last week of her life she had felt better than usual, and was, with their husband, spending the evening with some friends in a small room with a large fire in it, where she appeared very happy, and not excited. Having taken a little bread and cheese and half a glass of ale for supper, she complained of the heat, and removed further from the fire. Presently she suddenly threw out her right hand as if to grasp something, turned deadly pale, and dropped her head on her husband's shoulder. I saw her in less than ten minutes, supported in an upright position, and quite dead.

Inspection.—The vessels of the brain were almost empty. The

upper third of each lung adhered to the thorax, and both were engorged with blood, except at their very summits. The heart was double its natural size, from hypertrophy and dilatation of its left side. *The bases of the mitral valves adhered to each other in such a manner as to allow the passage of only one finger.* The lining membrane of the left auricle was opaque, and partially thickened. The aortic valves were much ossified, and one rigidly projected from the side and could not lie flat.

Death was directly produced in this instance by syncope; but the patient had a short time before her death been under my treatment for palpitation and dyspnoea on exertion; and no abnormal sounds of the heart were then detected.

It is instructive to observe how much disease may exist on the left side of the heart without giving rise to urgent symptoms. Here were both aortic and mitral obstruction.

The absence of murmur in these cases, however, mainly arises from the comparatively slight force of auricular contraction.

Regurgitation.—The general signs are much the same as in obstruction, perhaps slightly more marked, particularly in those accompanied by pulmonary congestion.

When, however, the disease in question is joined with considerable dilatation of the left auricle, a sign has been observed in some of these cases which it is believed has not been noticed elsewhere. A faint pulsation or undulation was seen between the second and third left ribs, and it was often difficult to discover whether or not it was synchronous with the systole of the left ventricle. On an inspection of the bodies the left auricle was found greatly dilated, its appendix was uncovered by lung, and was in direct contact with the walls of the chest.

CASE 89.—*Mitral Obstruction—Tricuspid Regurgitation—Dropsy.*

A girl, æt. 14, had articular rheumatism, with some pain under the left breast, two years and a half ago. Six months after that she began

to perceive palpitations, which have occasionally reappeared ever since, accompanied by depression, nausea, and dyspnœa on exertion.

When visited, with Mr. Jones, she complained of constant nausea, long and violent attacks of palpitation, occasional headaches, and a dry cough. The pulse was feeble and variable in size. The left side of the chest sounded dull from the second rib downwards, and over this space the respiratory sound was inaudible. A faint pulsation was felt between the fifth and sixth ribs, alternating with a similar one between *the second and third ribs*. A grating sound was heard with the systolic sound of the heart. Six months after this, turgescence and strong pulsations of the jugular veins were seen. The feet, legs, and abdomen became gradually and successively infiltrated. The urine became scanty, but was not coagulable by heat. A double grating sound was heard in the precordial region, and continued till her death, which took place nine months after she was first seen.

Inspection.—There was much fluid in the thorax, pericardium, and abdomen. On either layer of the pericardium there were several rough patches, which felt like a calf's tongue. The heart was hypertrophied, and dilated to more than double the size of the fist. The chordæ tendiniæ of one of the tricuspid valves were shortened and thickened. The tricuspid foramen measured nearly five and a half inches in circumference. The valves could not close it, a large space being left uncovered in the middle, not only from the inability of one of the valves to rise up to the plane of closure, but from the disproportion which existed between the size of the valves and that of the foramen. These valves were thickened, and adhered to each other at their bases, so that two fingers could not pass between them. The lining membrane of the left auricle was thickened and opaque, and near the mitral valves was granulated.

The other organs were healthy.

The undulations here seen were doubtless caused by the blood being driven by ventricular contraction through the patulous mitral foramen into the left auricle, dilated so as to be in contact with the walls of the chest. The grating sound was caused by pericardial friction. The same undulations, arising from similar causes, were also observed in Case 58, above quoted.

The chief local signs are furnished by a systolic murmur. Dr. Hope took it for granted that such murmur was always present when mitral regurgitation took place. This,

however, is not the fact. In the following case, and others in which regurgitation through the mitral orifice must have taken place, no trace of murmur was detected.

CASE 90.—*Mitral Regurgitation—Diseased Bronchial Glands—Pulmonary Emphysema.*

A lad, æt. 19, of diminutive stature, not looking more than twelve years of age, stated that he had never been put to any kind of work, having been, from a child, unable to bear exertion; because his body was covered with an eruption, which, about five years ago, was cured, soon after which his breathing became very short.

On his admission into the Birmingham General Hospital there was slight spinal curvature to the left perceived. The chest was bombed and rounded by the filling up of many of the intercostal spaces. The respiration was laborious and chiefly abdominal. His face was livid and bloated. He complained greatly of dyspnœa, amounting to a sense of suffocation on endeavouring to walk quickly, and of a troublesome cough and wheezing.

Percussion everywhere elicited a clear sound, but more especially under the sternum and behind at the lower third of the left side, where it was ringing. The pulmonary sound, everywhere feeble, was absent in certain parts, particularly under the sternum. In front it was accompanied by varieties of moist rattles; behind the lower two thirds of each lung there was a tolerably even and fine muco-crepitant rattle. The voice resounded more clearly than usual under the sternum and behind the left side. *The sounds of the heart were natural.*

This lad improved considerably after having been leeches between the shoulders, and after having taken the mist. ammoniaci with mild laxative medicines. Nine days subsequent to his admission he was seized with great dyspnœa after exerting himself in walking, and died in two hours.

Inspection.—On opening the chest the lungs did not collapse. Nearly the whole of the free edge of each lung was studded with patches of emphysema, the cells of which were not larger than mustard seeds; similar patches existed at different parts of the surface of the lungs, especially behind the left lung. The bronchial glands were much enlarged, and rose up into the anterior mediastinum, varying in size from that of a pea to a pigeon's egg. They resembled the spleen in colour and consistence. In one near the root of the lungs was a yellow mass as large as a horse-bean, resembling atheroma, but of the

consistence of chalk. Much blood and serum ran out on cutting into the lungs, the lower lobes of which were greatly engorged. The mucous membrane of some of the air-tubes was thick, soft, and violet-coloured. The heart was equal to that of a full-sized person, but the circumference of the aorta did not exceed that of the forefinger. The heart was of a yellowish-brown colour and flabby. The edge of one of the mitral valves was divided into three half-lozenge-shaped points, having a great many very short chordæ tendinæ, with little or no carneæ columnæ. *The chords were so short, and the valve had so little depth, that it could not rise up into the plane of closure. The mitral orifice was four inches and a half in circumference.*

The origin of deranged circulation may here be traced to congenital malformation of one of the mitral valves. On the enlargement of the cavities of the heart by natural growth, whilst this shallow valve remained tied down, a certain amount of regurgitation must have taken place through the mitral orifice. This would have a tendency to increase from a still further dilatation of the orifice itself, produced by the pressure of the blood from the lungs, and the back current during the systole of the ventricles. A diminished supply of blood being thus poured into the aorta, it would cease to grow by a well-known law, accommodating its calibre to the amount of duty required of it. The body, also, would not grow with its usual vigour in consequence of receiving a deficient supply of arterial blood, and hence this lad's stunted appearance. That he should have reached 19 years of age is only to be accounted for by his never having been put to any laborious employment.

CASE 91.—*Mitral and Tricuspid Regurgitation—Emphysema—Dropsy.*

A wire-drawer, æt. 56, had been a collier twenty years since. Nine years ago, after having taken repeated colds, he had an attack of rheumatism, and was laid up during nine months with swelling of the legs, thighs, and body, and has been asthmatic ever since. When received into the Birmingham General Hospital he had been ailing two months.

There was slight angular curvature of the spine, with anasarca of the lower extremities and abdomen. His face was purple and bloated, and he laboured under great dyspnœa. He had cough, with expectoration of thick muco-purulent fluid. The pulse was 84, firm and steady. There were strong venous pulsations on the left side of the neck. The precordial dullness to the left of the sternum was more extended than usual, but the sternum itself sounded very clear. The pulmonary sound was everywhere feeble, and was mixed with cooing sounds, and behind the left side with large mucous crackling. *The sounds of the heart were clear and well defined.* He was not relieved by any treatment; the dyspnœa and anasarca increased, and he died in thirteen days.

Inspection.—The right lung adhered slightly, and the left lung extensively to the thorax. There was rather more than half a pint of clear, light-coloured serum in each pleural cavity, and a considerable quantity in the abdomen. The upper half of each lung was studded with emphysema; most of the dilated cells being of the size of a mustard seed, and one or two reaching the size of a horse-bean. The heart was firm and red, and covered with milky spots in the shape of strings of small beads. The auricles were both dilated, but the ventricles were of a natural size. *One of the mitral valves was shrivelled up to a mere thick, ragged-edged fringe, not more than two lines deep, with a great many chordæ tendinæ much thickened, and three stiff, hardened, and much hypertrophied carneæ columnæ.* The mitral orifice measured four and a quarter inches in circumference. Two of the tricuspid valves were in the same state as that of the mitral valve above described.

In all these cases the progress of the disease seems to have been from the left to the right side of the heart, the symptoms of venous obstruction coming on with the incompleteness of the tricuspid valves.

Here were both mitral and tricuspid regurgitation, without a trace of murmur. On the other hand, in two or three cases a systolic murmur was heard chiefly where the beat of the heart was perceived, and on inspection the mitral orifice and its valves were found complete. In the following case the ventricular sides of the valves were covered with rough vegetations, so that probably the murmur was caused by the blood passing over this

roughened surface. It is difficult to conceive how such a sound as this so produced could be distinguished from that caused by mitral regurgitation.

CASE 92.—*Mitral and Tricuspid Regurgitation—Anasarca.*

A lawyer's clerk, æt. 21, had from a child felt a fluttering at his heart, and dyspnœa, after being hurried in any way, and particularly after running. He had rheumatic fever at 14 years of age, ever since which time he has had severe palpitations. He had another similar attack of a slight nature at the age of 18, and since then he has gradually become more and more pigeon-breasted.

When first seen he was labouring under great dyspnœa. The precordial space was dull over a larger extent than usual, and it was bombed out. There was no tumefaction or pulsation of the veins of the neck. The pulse was small. The systolic sound of the heart was accompanied by a very shrill musical saw-sound, the maximum of which was under the sternum, a little above the level of the nipple. It was continued, as it were, into the diastolic sound, so that the systolic sound terminated with a kind of jerking increase.

He was occasionally seen and relieved from time to time for several months; chiefly by belladonna frictions, ether, expectorants, &c. At length he complained of great constriction across the epigastric region, and of his feet being constantly cold.

When seen in consultation with Dr. Skerrett, the pulse was a little quick and sharp, and the precordial dullness extended. A musical saw-sound was heard during the systole of the heart, and was at its maximum under the left nipple, its intensity diminishing upwards from this point. It was heard plainly at the lower angle of the left scapula, but with less shrillness than in front of the chest. No diastolic sound could be heard. A strong purring thrill was felt around the left nipple, and towards the outer side of the chest. Soon after this the veins of the neck became turgid, and pulsated; œdema of the feet and ankles appeared, and was quickly followed by general anasarca and death.

Inspection.—The cellular tissue was loaded with serum. The lungs were engorged. The heart was dilated, particularly both the auricles. The mitral valves were thickened and shrivelled, and their free edges fringed with long horny vegetations, one or two nearly an inch long, so that they could not close the mitral orifice, which was four inches and three quarters in circumference. On one of the tricuspid valves was a bundle of soft, red vegetations, as large as a bean. The orifice was five inches in circumference, and the valves rather small.

CASE 93.—*Mitral Obstruction and Tricuspid Regurgitation—Dropsy.*

A bargeman, æt. 22, of athletic frame, who had been much exposed to wet and cold, and had been subject to rheumatism, was admitted into the Birmingham General Hospital labouring under great dyspnœa and orthopnœa, with harassing cough and mucous expectoration streaked with blood, pain at the heart, and palpitations. His face and lips were turgid and rather purple. He had an anxious expression of countenance and a hurried manner.

The precordial dullness was extensive. Shrill, cooing sound was heard all over the chest. The impulse of the heart was heaving, and violent palpitations were frequent. *The systolic sound was prolonged, shrill, and raspish, and was most distinct below the nipple. The diastolic sound was short and clear, and was heard distinctly near the angle of the left scapula.* Both sounds were heard in some degree over the whole of the chest.

Under the use of small doses of digitalis and nitre his health was much improved for a time, and the blood disappeared from his sputa; but in a short time all the bad symptoms returned, and, in addition, the veins of the neck became turgid and pulsated, and œdema of the legs appeared. The systolic murmur became stronger, the hemoptysis severe, and dyspnœa very distressing with orthopnœa; the urine more scanty, the pulsations most distressing, and he died five weeks after his admission.

Inspection.—On opening the thorax, the lungs, which were very large, did not collapse; they were congested and very emphysematous, particularly at their free margins. In the upper and middle lobes of the right lung were three or four yellow, soft tubercles, rather larger than peas. The base of the left lung was of a yellowish red colour, uncrepitating, heavy, and friable. In one part of it was a patch of a greyish-colour, from which bloody pus exuded on pressure. In another part of it was a patch of pulmonary apoplexy of the size of a pigeon's egg, with well-defined edges. The whole of this hepatized portion of lung was drier than in ordinary pneumonia.

The pericardium was thickened and opaque, but contained no serum. On the front of the heart were several thickish, milky patches; the organ was large, and both ventricles were dilated; the walls of the left ventricle being firm, red, and hypertrophied. The tricuspid orifice was six inches in circumference, and the valves of the ordinary size. The zone of the mitral foramen was thickened, and only admitted one

finger to pass freely. *The valves were thickly fringed with warty vegetations, which extended into the auricle.*

Contrary to what was observed in aortic valvular murmur, the direction in which mitral murmurs are propagated does not assist in tracing them to their origin, whilst their seat is indicated by the spot on the surface of the chest where they are heard loudest. This is where the beat of the heart is perceived in the front of the chest, and near the angle of left scapula behind it. A systolic murmur, most intense in these spots, always indicated the presence of mitral regurgitation.

It appears, therefore, that mitral regurgitation is very frequently unaccompanied by any murmur; and that when one is present it is not always possible to fix with certainty on the mitral orifice, as the spot in which it is engendered.

Pulmonary Orifice.—Affections of the pulmonary orifice are so rare, that I have only seen five of them, of which three were associated with open foramen ovale. Of the other two, one (Case 64) was joined with dilated aneurism of the pulmonary artery; and in the other the orifice was uncommonly dilated, but mitral regurgitation also existed. So that nothing can be said respecting the diagnosis of obstruction or regurgitation existing in this situation.

Tricuspid Orifice.—Disease inducing simple *constriction* and *consequent obstruction*, so rarely occurs at the tricuspid orifice, that it is unnecessary to inquire into the means of discovering its existence.

Regurgitation.—When this takes place from inefficiency of the valves to close the orifice, signs indicative of congestion of the systemic circulation have been seen sooner or later to arise. The principal of these is anasarca, commencing at the feet and lower extremities. When this is found to be the case, and there are no traces of albuminuria, contracted liver, or other sufficient cause for its production, we may be almost sure that it arises from regurgitation through the tricuspid orifice. The symptoms

of cerebral venous congestion are not so plainly marked, and not unfrequently an attack of apoplexy affords the first sign of its existence.

A most valuable local sign of this affection is furnished by venous pulsations or undulations in the neck. They are generally much more marked on the right than on the left side of the neck. Dr. Hope was of opinion that they often arose from simple hypertrophy; but they were only seen in three of these cases, unaccompanied by regurgitation. If they often arose from hypertrophy alone, there would surely have been some traces of them in Case 33, and others of a similar kind.

On the other hand, the absence of venous pulsations by no means proves the non-existence of regurgitation, for in several cases of decided regurgitation, in which the walls of the heart were either attenuated or softened, there were no pulsations. In fact venous pulsations, although they raise a strong presumption in favour of the existence of regurgitation when they are present, are by no means a measure of the amount of obstruction which is opposed to the circulation by such regurgitation. Their strength is rather a measure of the contractile force of the heart. Thus if both ventricles are hypertrophied, a strong venous current is met by a strong regurgitating current, the shock is great, and the pulsations up the veins of the neck are strongly marked. If both ventricles are softened or attenuated, a feeble venous current is met by an equally feeble current of regurgitation, the thrill and pulsation is slight, but the flow of venous blood is just as much impeded in one case as in the other.

I know of no auscultatory sign of tricuspid regurgitation. It was at one time generally assumed that a systolic murmur was the result. So far, however, is this from being the case, that a murmur was hardly ever heard in any of the cases here recorded, which could be fairly traced to this cause. The following cases show that a strong regurgitation may take place without giving rise to any murmur:—

CASE 93*.—*Tricuspid Regurgitation—Dropsy.*

A single woman, æt. 57, a cook, had enjoyed good health up to the age of 34, when menstruation ceased, and she then suffered from dyspnœa and profuse leucorrhœa. Eighteen months since she first felt a dull pain in the right hypochondrium, which was attended with considerable derangement of the stomach, and whitish-coloured stools. She was at that time consuming a great quantity of animal food, ale, and spirits. Her symptoms were relieved by treatment. A few weeks since her feet and ankles, and subsequently her abdomen swelled, and she was received into the Birmingham General Hospital.

There was little or no dyspnœa, and no cough. The chest sounded clear on percussion, except that the dullness in the precordial region was more extended than usual. The respiratory sound was natural. The heart's action was weak and irregular, and its sounds rather obscure. There was great turgescence, and some pulsation in the veins of the neck. The urine was not albuminous.

With rest, and under the use of tonic and diuretic medicines, and a tolerably generous diet, the anasarca disappeared, and she left the hospital and took a cook's place.

Six months after this she was again admitted. Her legs and abdomen were greatly distended with fluid. The stools were pale and offensive. The urine yellowish and turbid, abounded with phosphates, but contained no albumen. The veins of the neck were greatly distended and pulsated. She could only sleep in a semi-reclining posture, and laboured under great dyspnœa. The dullness over the precordial region was very extensive, and there was also dullness at the bottom of each side of the chest varying with position. There was no cough or expectoration. The pulmonary sound was clear, but rather coarse, except at the bottom of each lung, where it was very feeble, and occasionally mixed with a fine muco-crepitant rattle. The sounds of the heart were both distant and muffled, running into each other from the great irregularity of the heart's action, *but were unaccompanied by murmur*. This time medicines made no impression on her; her symptoms continued to become more and more aggravated, and she died in less than a month.

Inspection.—The abdomen and the integuments of the body generally were greatly distended with fluid, and a considerable quantity existed in both pleural cavities. The lungs were healthy.

There were four ounces of fluid in the pericardium. The heart was large, the right auricle being more particularly dilated. There was a great deal of fat in the walls. The sigmoid valves acted well, although

the corpora Arantii of two of them were a little thickened. The tricuspid orifice measured 4 inches in circumference. The back valve, as well as a thick fleshy column fixed to its apex without any cord, were closely glued to the ventricle. The edges of the other two were thickened and shrivelled; the height of one was half an inch, and the other a quarter of an inch. There were milky patches on the lining membrane of the right ventricle.

The liver was rather hard. The kidneys were lobulated, and their appearance was slightly granular on being laid open.

CASE 94.—*Tricuspid Regurgitation—Dropsy.*

A nurse, æt. 48, married, had been asthmatical for eight or nine years; but for the last four years she had suffered greatly in winter, and in foggy weather she felt "bloated up for breath." About six months since her legs began to swell, and she had pain at the heart and urgent dyspnœa. On being admitted into the Birmingham General Hospital, her symptoms chiefly consisted of broad undulations in the veins of the right side of the neck, in addition to the pain mentioned, and general anasarca. She was relieved by diuretic and tonic medicines, and by belladonna frictions over the heart, and left the hospital, every trace of venous pulsation and anasarca having disappeared.

Some time after this she applied again as an out-patient. She had been exerting herself, had been exposed to alternate heats and chills, and anasarca had reappeared; after any unusual exertion, her hands felt benumbed until they had been rubbed. Upon stooping and rising again, she felt a pain under the xiphoid cartilage. Her appetite was bad. Her breathing was but very little oppressed. The pulse was small but regular. She had a troublesome cough, with some wheezing and expectoration of thick blackish gray mucus. The action of the heart was natural. Broad venous pulsations were observed in the neck, stronger on the right than on the left side. The pulmonary sound was coarse, accompanied in most parts of the chest by bass-viol and cooing sounds. On the left side there was some moist crackling, the pulmonary sound commencing with crackling, and terminating in cooing and bass-viol sounds. The diastolic sound of the heart was stronger and coarser than usual, and was heard all over the chest; but there was no other abnormal cardiac sound up to the time of her death, which took place in six weeks' time, the anasarca gradually increasing.

Inspection.—There was much serum in each pleural cavity, and a

very little in the pericardium. The lining membrane of the air-tubes was thickened in places, as in chronic bronchitis.

The right auricle of the heart was dilated. The tricuspid orifice measured 5 inches in circumference, and all the valves had very short thick chordæ tendinæ, so that the valves could not rise up into the plane of closure. The aortic valves were thickened, but acted well.

The adhesion of one of the tricuspid valves to the ventricles which occurred in these cases, as well as in Case 29, must have allowed regurgitation, which was doubtless much increased by the dilatation of the right side; in Case 39 arising from congestion and pulmonary apoplexy of the lung; in Case 40 from inflammation accompanying rheumatic fever; and in Case 93*, from that subacute form of inflammation to which spirit-drinkers are subject, and the traces of which were here found in several organs. The inflammatory origin of the adhesion was clearly marked in this case by the thickened milky patches on the lining membrane of the ventricle near the adherent valve.

The dyspnoea, which in all these cases preceded the dropsy, arose, in Cases 39 and 94, from the state of the lungs, and in the other two cases possibly from the effect of inflammation on the muscular walls of the heart.

The influence of rest, diet, and treatment in removing the dropsy for a time, in Case 93, while its cause remained untouched, is remarkable.

CASE 95. — *Diseased Arteries — Tricuspid Regurgitation — Dropsy.*

A bootmaker, æt. 46, an old soldier, who had lost the right leg above the knee, had been accustomed to drink spirits to excess. Nine months since he felt a pain under the sternum, followed by dyspnoea, both of which have continued ever since. When visited with Mr. Clark, the dyspnoea was most distressing, he was unable to lie down, and had a strong sensation of constriction across his chest. There was œdema of the legs and thighs, and some ascites. The jugular veins pulsated strongly. There was dullness on percussion for some inches around the precordial region. The respiratory sound was natural. The action of the heart was heaving, and its sounds distant. The pulse was very

full and hard, and its stroke remarkably quick and sharp, which character it retained till death took place, three weeks after my first visit, having been preceded by the expectoration of dark clotted blood for two days.

Inspection.—There was serous effusion in the chest and abdomen. A patch of pulmonary apoplexy, as large as an orange, existed about the middle of the concave portion of the right lung, near the pericardium, and was continued gradually into the healthy portion of the lung.

The heart was enormously hypertrophied and dilated. On its surface was a patch of thick, yellow lymph. The dilatation of the right cavities was very great; the circumference of the tricuspid foramen was five inches and three-quarters; one of the valves had short, thickened chordæ tendinæ, they could not close the foramen. The aortic valves were thickened, but acted well. There were many patches of atheroma under the lining membrane of the left auricle. The aorta was a mass of disease, being thickened and puckered, and in many places denuded of its lining membrane, and all the large arteries were similarly diseased.

The liver was of a nutmeg colour. The kidneys and other organs were healthy.

The following is a summary of the most usual signs of valvular derangement observed in these cases:—

Of the Aortic Orifice.—*Constriction.*—Systolic murmur, traceable up the course of the aorta; in certain cases very prolonged.

Regurgitation.—*Visible* arterial pulsations.

Diastolic murmur, slightly traceable to the right of the aorta.

Mitral Orifice.—*Obstruction.*—Signs of pulmonary congestion; *very rarely* a diastolic sound heard near the apex of the heart, and at the lower angle of the left scapula.

Regurgitation.—Signs of pulmonic congestion. Systolic murmur heard at the apex of the heart, and the lower angle of the left scapula, without visible arterial pulsations. Increased intensity of diastolic sound.

Tricuspid Orifice.—Signs of congestion of the systemic circulation. Venous pulsations in the neck.

It need hardly be remarked, that in certain cases the

position in which these various normal or abnormal sounds of the heart are heard will be changed by the displacement of the whole organ. This occurs temporarily when a large amount of effusion takes place in the left pleural cavity, by which the heart is completely tilted over to the right side of the chest. A permanent displacement, however, is sometimes observed, and has been attributed by Dr. Stokes to the absorption of fluid previously effused into the *right* pleural cavity. The three cases that follow, in some degree confirm his opinion.

CASE 96.—*Permanent Displacement of the Heart to the Right Side.*

A girl, when she was 12 years old, had been run over by a cart, the wheel of which passed over her back and upwards across the right shoulder. She was laid up a long time with very severe pain in her right side. Ever since that time she has suffered from cough, with slight frothy expectoration, and considerable dyspnœa on exertion; and she has felt her heart beat on the right side, which it did not before the accident. On admission into the Birmingham General Hospital, whistling respiratory sound was heard at a considerable distance from her. The left side of the chest measured an inch and a half more round than the right side under the nipple. There was but little action of the right ribs. The lower two-thirds of the right side of the chest sounded quite dull on percussion, and the upper third was slightly duller than the corresponding part of the left side. The pulmonary sound was pure but intense over the whole of the left side. It was coarse and feeble under the right clavicle, and absent below this in front. Behind it was distant, blowing, and jerking. Nasal resonance of voice was heard near the root of the right lung. There was no impulse in the precordial region in any position of the patient. The heart was felt to beat between the fourth and fifth ribs external to the right nipple. In that place the healthy sounds of the heart were heard at their maximum. They were also heard clearly behind the right side, but nowhere on the left side.

She has gradually improved up to this time, which is eight years since the accident, and five years since she was first seen. The dyspnœa is now less, but is urgent on exertion. The cough troubles her but little. The heart still beats on the right side, but not so far from the sternum as when first seen. The whistling respiratory sound is as

loud as ever. The dullness on the right side is less marked. Both sides of the chest now measure the same. Some feeble and coarsish pulmonary sound is heard all over the front of the right side, very faint towards the bottom, but is totally wanting at the lower half of the posterior part.

CASE 97.—*Permanent Displacement of the Heart to the Right Side of the Chest.*

A miner, æt. 53, had been troubled with a dry cough for five years, and dyspnœa on exertion. Twelve months since he took cold, when his breathing became more laborious, and his cough more troublesome, and was accompanied by expectoration of froth, and by a dull aching pain under the left nipple. For the last two months he has had spasmodic cough with scanty expectoration, and great dyspnœa. On his admission into the Birmingham General Hospital, there was slight lateral curvature of the spine towards the left. Below the nipple the right side measured three-quarters of an inch less than the left side. The heart was seen and felt to beat to the right of the sternum in a line with the nipple. The left side of the chest sounded clear on percussion, and the lower part particularly so. Over the right clavicle, and three inches below its sternal end, the sound was very dull, and a slight dullness was observed at the spot where the heart was seen to beat. The upper third of the right side behind sounded also dull. Whistling respiration was heard at a distance. Very little respiratory sound was perceived on the right side, but under the right clavicle it was tracheal, and in other parts was masked by dry rattles. Behind the left side there was large, uneven, mucous crackling. He left the hospital in a few weeks relieved, his heart continuing to beat to the right of the sternum.

CASE 98.—*Permanent Displacement of the Heart to the Right Side of the Chest.*

A collier, æt. 39, had been subject to dyspnœa and cough, with muco-purulent expectoration, for ten years, which was attended with increased dyspnœa and constriction of the chest, followed by the expectoration of dark liquid blood on taking fresh cold. He had on these occasions night sweats, and had gradually year by year been losing flesh. Four years ago he caught cold, and suffered much from a severe cutting pain under his right nipple, increased by deep inspiration, and shortly afterwards he noticed his heart beating on the right side of the sternum, where it has remained ever since. On his admis

sion into the Birmingham General Hospital, the left side measured half an inch more than the right side on a level with the nipple. His breathing was rather stridulous. The heart was both seen and felt to beat to the right of the sternum a little below the nipple. The left side of the thorax seemed bulged out and the right side flattened; there was slight spinal curvature to the left. The breathing was chiefly abdominal. The whole of the right side sounded duller than the left on percussion, more especially behind and under the clavicle. The pulmonary sound was coarse, and was prolonged during expiration under both clavicles; it was accompanied by some crackling over the left side, and was quite masked by mucous rattles on the right side. Incomplete pectoriloquy was heard under the right clavicle, and strong buzzing bronchophony below the angle of the right scapula. The sounds of the heart were heard clear and distinct on the right side, but very remotely on the left side. He left the hospital relieved.

The evidences of pleurisy on the right side in Cases 96 and 98 are strong and conclusive, and the time when the heart passed over to the right side is accurately marked. The general signs in Case 97 are more obscure; but there can be little doubt that effusion had once existed in the right pleural cavity. Indeed the result of such effusion was seen in this as in the other cases, in a diminution of the size of the right side of the thorax.

Dr. Stokes seems to think that the transposition of the heart chiefly arises from the rapidity with which the fluid is absorbed, not allowing time for the ribs to fall in. But in all these cases the side was actually contracted, and the cavity of the right side was diminished. It is probable, therefore, that in the earlier stage of the disease, the lung had been more or less bound down to the spine by adhesive bands, and was prevented from expanding into the space left by the absorption of the fluid, which has become filled up by the heart passing over from the left side. When the same thing occurs on the left side, the right lung expands, and often passes over considerably to the left of the median line.

It is curious to observe that stridulous breathing was heard in all these cases, as if the trachea had been com-

pressed. This must have occurred from the aorta having been dragged across it. This effect was not observed, however, in those cases where the heart was pushed over by fluid in the left side. But in the latter instance the heart meets with a fully inflated lung, which buoys it off from the trachea; whilst in the former it lies on a shrivelled lung, the lower part of which is more or less bound down to the spine.

CASE 99.—*Aortic and Tricuspid Regurgitation—Dropsy.*

A labourer, æt. 33, of athletic frame and intemperate habits, accustomed to violent exercise, but who had never had rheumatism, was admitted into the Birmingham General Hospital. His legs were anasarcaous, as also were the walls of the abdomen, but to a less extent. His face was tumid and bloated, his lips purple, his expression of countenance anxious. The respiration was very laboured, the shoulders being raised, and he could not lie down. He had pains at the region of the heart, and constant palpitations. His urine was scanty and loaded with lithates; the pulse soft and feeble. There was cough with aqueous expectoration. The external jugular veins were seen to undulate above the clavicles.

The lower half of the right side sounded dull on percussion, the level of the dullness varying with the position of the patient. The left side sounded dull from the fourth to the eighth rib, and from the sternum to the outside of the nipple. The pulmonary sound over the upper two-thirds of the right lung was coarse, and attended with cooing sound; over the lower third it was feeble; over the left lung it was accompanied by some muco-crepitant rattles. The impulse of the heart was felt over the dull space around the precordial region, and was quick, jerking, and tumultuous. The sounds of the heart were heard all over the chest; the systolic sound at a distance from the precordial region being dull, and the diastolic sound longer and less clear than usual. Below the left nipple the systolic sound was prolonged, and of an acute shrill rasp quality, extending up the course of the aorta. At the middle of the sternum near the left fourth cartilage, both sounds were raspy and prolonged.

His symptoms became aggravated, the dyspnoea greatly increased, and he died in seven days.

Inspection.—The left lung was universally adherent to the chest, and had apparently been some time in this state. There were four

pints of serum in the right pleural cavity. The lungs were congested. The lining membrane of the bronchial tubes was of a purple tinge. The pericardium was opaque and thickened, and contained five ounces of yellow serum. The heart was very large, and weighed twenty-six ounces; it was soft and flaccid, and had many milky patches on its surface. Its cavities were all greatly dilated, and contained much black blood. The walls were not much hypertrophied. Two of the aortic valves were thickened and shrivelled, but could fall back during the systole of the ventricle, but the third valve was transformed into cartilage, and rigidly stood out from the side of the vessel. The mitral valves were slightly thickened. The tricuspid orifice was full six inches in circumference, and its valves were of the ordinary size. The lining of the aorta up to its arch was thickened, and studded with atheroma and calcareous scales.

CASE 100.—*Diseased Aorta, Hypertrophy, and Dilatation of Heart—Tricuspid Regurgitation—Pericarditis—Pleuritis.*

A labourer, æt. 40, an habitual drunkard, having drank cold water when he was heated ten weeks since, began to feel palpitations at the heart, followed by pain across the epigastric region, dyspnœa, and cough with expectoration of thick yellow phlegm. For the last four weeks he has been unable to lie down, and has had œdema of the ankles seven or eight days, and scantiness of urine during three days.

Admitted into the Birmingham General Hospital, he said he had less palpitation, but he had pain across the epigastrium, a bad cough with hardly any expectoration, distressing dyspnœa and orthopnœa; a little pain in the head and up the left side of the face. The legs were anasarcaous, and the scrotum and abdomen slightly so. The urine was scanty and high coloured, not albuminous; the pulse was rather full and hard, under 100. There were violent pulsations of the arteries visible at the wrists. The respiration was 30, and its sounds were natural. There was extended dullness around the region of the heart, with heaving action of that organ; pulsation over each clavicle, and a thrill was felt on deep pressure over the sternum. The diastolic sound of the heart was very faint, and the systolic sound like that of the fourth string of a violoncello, its maximum being up the course of the aorta under the sternum. Digitalis was given and a blister applied. He got much worse, however, and in a few days the scrotum being greatly distended, was punctured by Mr. Freer. He was ordered to take half a grain of elaterium twice a day, with an ammonia mixture, which produced strong action of the bowels; and though he was dis-

tressed by vomiting, he was relieved, and the anasarca was greatly diminished. Eighteen days after this, venous undulations were plainly seen in the neck, chiefly on the right side. Percussion was good, except, as before, over the precordial region, and below the fifth ribs behind. The respiratory sound was loud in front, coarse behind; the heart's action feeble and slow; a fine rasp-sound was heard at the extreme left of the side between the seventh and eighth ribs; near the nipple to the left of the sternum a soft systolic bellows-sound was heard, which became raspish as the right of the sternum was approached, and musical up the sternum, its maximum being about the middle of that bone.

The dyspnoea increased, and his legs became red, and were attacked with gangrenous sloughs. He died six weeks after his admission into the hospital.

Inspection.—The cellular tissue was loaded with serum, a few pints of which were found in the cavity of the abdomen. The lower third of the right side of the thorax was also filled with serum, in which flakes of yellow lymph floated. This space was bounded by pleura, covered thickly with reticulated yellow lymph; in one or two spots on the lungs it was an inch thick, and resembled concrete pus. This lung was carnified at its lower half, and engorged all over; there were a few patches of pulmonary apoplexy in it, and also in the lowest portion of the left lung, which was otherwise healthy and very voluminous.

The heart was very large, firm and red. About three ounces of serum were in the pericardium, and on each fold of this membrane were rough patches of yellow lymph, not adherent to each other; one patch, rougher and thicker than the others, was on the left side near the apex, and one on the corresponding part of the sac. It was not of very recent formation. The mitral valves were slightly thickened, but acted well, and were fully equal to close the mitral orifice, which was four inches in circumference. The aortic valves were prevented from folding back on the vessel from their free edges being in several places transformed into cartilage, but they acted so as to prevent regurgitation. The tricuspid foramen measured five inches round, and had only two valves, one of which was a deep and long one, and the other was less than a quarter of an inch in height. The aorta was slightly dilated, and was lined with atheroma and steatoma, in which were some calcareous concretions.

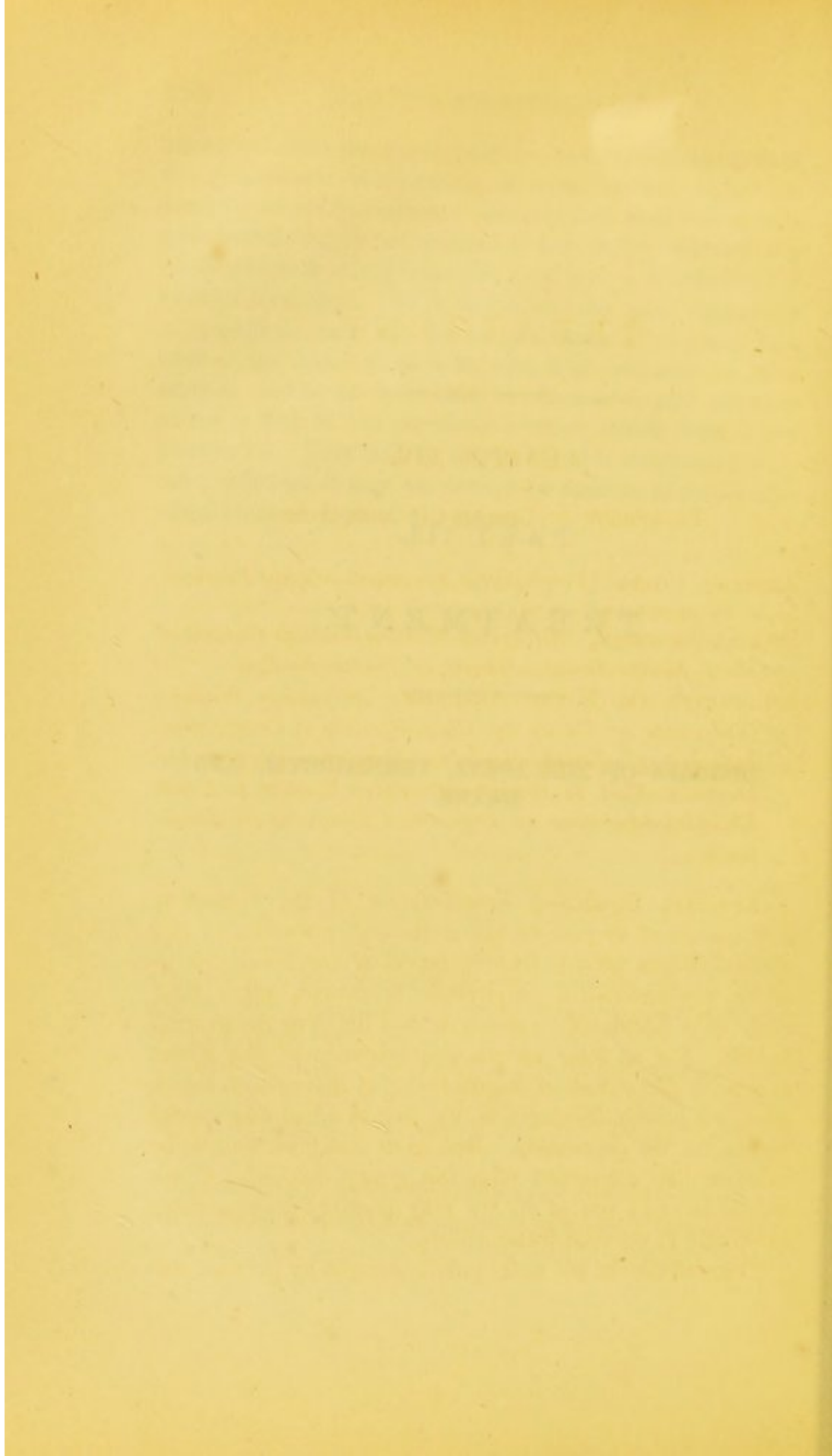
A careful study of the cases on which the above conclusions are founded, shows that it must frequently be difficult, if not impossible, to determine the exact seat or

amount of valvular derangement ; and consequently that the refinements in diagnosis of some writers cannot be carried out at the bedside. Happily this is of less consequence than might be imagined at first sight. Correctness of diagnosis is chiefly valuable, as it lays a foundation for rational treatment. With this object the main endeavour of practical men will be to determine if organic disease of the heart be present, and, if so, whether its contractile power be increased or decreased, and whether either or both of the circulations which result from it are obstructed. This will be accomplished in most cases with ease, although it may not always be possible to predict the exact state of each of the valves.

PART III.

TREATMENT.

DISEASES OF THE AORTA, PERICARDIUM, AND
HEART.



TREATMENT.

CHAPTER XIV.

TREATMENT OF DISEASES OF THE AORTA.

ARTERIAL COATS. *Prophylactic Treatment—Gouty Diathesis—Dyspepsia.*

DILATED ANEURISM. *Strengthen Walls—Diminish Current of Blood—Diet—Drugs—Danger of Depletion—Rest.*

SACCULATED AND MIXED ANEURISM. *Strengthen Walls—Obliteration of Cavity by Clots—Quality of Coagulum—Influence of Depletion—Iron—Sedatives—Purgatives—Digitalis—Cold to Surface—Results—Objection to Great Depletion—Influence of Differential Diagnosis on Treatment.*

ARTERIAL COATS.—A consideration of the formation and causes of disease of the coats and aneurism of the thoracic aorta, affords little prospect of our being able to adopt any successful prophylactic treatment, other than such as is calculated to preserve and improve the general health. For as long as we are ignorant of the direct causes of atheromatous degeneration of arteries, on which aneurism principally depends, we cannot adopt any special means for its prevention. But, as it has been seen to be more or less connected with the gouty diathesis, if not indeed to arise out of it, we may possibly do something indirectly to prevent its approach.

Thus advice is not unfrequently sought by persons suf-

fering from that form of dyspepsia in which lithates abound, and which often terminates in gout and arterial degeneration. In such cases the stomach can be assisted in its performance of the first process of digestion by the administration of pig's pepsine and nitrohydrochloric acid *before* meals; any excess of lactic or similar acids, being neutralised by bicarbonate of potash and nitre *after* meals. The action of the liver, if sluggish, may be stimulated by rhubarb, and acetic extract of colchicum, and, if necessary, by the application of nitro-hydrochloric acid over the right hypochondrium.

Perspiration may be promoted by moderate and constant out-door exercise, daily ablution, and mild friction; whilst great precautions can be taken against nervous depression from whatever cause arising.

The diet, too, may be so regulated as materially to aid in thus promoting good digestion and assimilation, by the interdiction of malt liquor, and wines rich in grape sugar, such as port, brown sherry, &c.; and by substituting for them claret, hock, manzanilla, and other *dry* wines, brandy, whisky, &c., as well as by enforcing a plain but nutritious mixed diet, such as may be found most suitable to each individual case.

When there is reason to believe that atheromatous degeneration of the arteries has actually taken place, it is of the greatest importance to guard against over exertion and excitement of every kind. For there is now the danger before us, either of dilatation of the aorta, or rupture of one of the arteries, giving rise to sacculated aneurism, or cerebral apoplexy.

ANEURISM.—When aneurism of the thoracic aorta is once formed, the nature of the treatment which tends to afford relief, and to arrest the progress of the disease is clearly indicated.

The objects to be borne in mind are two-fold.

1. The prevention of an increase or rupture of a dilated aneurism.

2. The obliteration of a sacculated aneurism by the deposition of fibrinous clots within it.

In some respects these objects are similar, and in others dissimilar the one from the other. Consequently writers who, up to a comparatively recent period, have proposed plans for the treatment of aneurism generally, irrespective of these differences in the objects to be attained, have failed in adapting their remedies to meet both cases.

It is proposed therefore to consider separately the treatment of *dilated*, and of *sacculated* or *mixed aneurism*.

DILATED ANEURISM.—The prevention of further dilatations or of rupture might be facilitated by *restoring the elasticity*, and *strengthening the walls* of the pouch, or by *diminishing the force of the current* of blood through it.

There are no means, however, of *restoring the elasticity* of the coats of an artery that have been invaded by atheromatous degeneration and calcification.

The only method by which we can hope to *strengthen the walls of the pouch* is by inducing an inflammatory action in them, whereby lymph might be thrown out between the coats. This effect might be produced by mechanical irritation, and possibly by the use of a highly stimulating quality of food. Such a proceeding, however, would be very hazardous; because, in the first place, it is more than probable that the benefit derived from thus strengthening the walls of the sac, would be more than counterbalanced by other effects of this inflammatory action. Thus the quantity of the general nutrient fluid might be increased, and the exudation into the arterial coats rendered moister by which means ulcerative softening of the atheromatous mass might be promoted, and with it the tendency to rupture and dilatation. So also, adhesive inflammation, once excited, might be propagated to the contiguous parts, such as the trachea and vena cava, and cause them to adhere to the pouch, and death might thus be produced as in Cases 26, 72, 24, 71 and 70.

On these grounds therefore, supposing it were possible to induce adhesive inflammation in the walls of the pouch by stimulating food or other means, it would be hazardous to adopt this treatment. Besides which the heart itself would also be stimulated, and the force of the current of blood increased, although this might to a certain extent be counteracted by measures presently to be detailed.

The balance then on the whole being against adhesive inflammation in the walls of a dilated aneurism, we are called upon rather to prevent than promote it.

The endeavour thus to strengthen the walls of the aneurism being abandoned, the only alternative that remains is the *diminution of the force of the current of blood*. This may be accomplished by greatly lessening the amount of blood in the circulation, reducing the action of the heart by low diet, sedative and purgative drugs, and mental and bodily repose. In practising venesection, there are two things to be guarded against. One is the danger pointed out by Drs. Watson, Hope, and others, which arises from a state of irritability which is so commonly associated with weakness, under the influence of which the heart's action would occasionally be more violent than in the ordinary state of health, as was seen in Dr. Hope's experiments on animals. The other is the evil arising from abstracting large quantities of blood at one bleeding, in the recumbent position, whereby syncope might be induced, from which the heart might be unable to recover, in consequence of the obstacle to the circulation offered by a dilated aneurism of the arch of the aorta full of blood. The same objections lie against an extremely poor diet, and an immoderate use of purgatives; in addition to which such a low state of the system might be induced as would favour the softening of the atheromatous coats, although in a different manner from that in which such a result is produced in a plethoric state of the body.

In the selection of *sedative drugs*, the fear of syncope must still be borne in view. Extract of belladonna, with

laudanum, rubbed into the precordial region, has appeared to act more beneficially in tranquillising the heart than any other drugs in whatever manner administered.

In thus endeavouring to diminish the force with which the blood is impelled into the aorta, the danger above pointed out may be avoided, but the heart's action may be reduced to such a low point that it may be insufficient to force the blood properly through the capillaries, so that venous engorgement and its consequent evils may result. The greatest caution, therefore, is required in carrying out this plan.

To the enforcement of mental and bodily repose there cannot be any objection urged, unless the latter were carried to such an extent as seriously to derange the organs of digestion. Indeed, as nothing tends more powerfully to induce heart diseases than mental excitement, so is there nothing which in a more effectual manner promotes their alleviation or cure than mental quietude and repose. The ill effects of a want of it are seen in Cases 21, 62, 55, and 101.

The number of cases to which this line of treatment is applicable must necessarily be limited; because dilated aneurism is of itself rare, and is seldom detected before the coats have given way, and it has assumed the character of *mixed aneurism*. Of those here recorded it was adapted to Cases 72, 26, and the early stage of Case 21. In Case 72, however, no great hopes of relief were entertained, from the circumstance that the tumour was already large, and was firmly compressing the trachea. It was not therefore pushed to such a degree as to inconvenience the patient. In Case 21, the necessary amount of rest and quietude could not be attained.

SACCULATED AND MIXED ANEURISM.—An attempt to strengthen the walls of the sac by the deposition of lymph is not open to the same objections as in the case of dilated aneurism; for there is no longer any middle coat to be destroyed by atheromatous softening at that part of the sac

which is most prone to burst; nor is the sac any longer bound down to the contiguous parts in such a manner that inflammation could cause it to adhere to them, and so greatly to compress them; because it can expand in other directions. Hence there would be no reason why we should not induce a moderate amount of inflammation in the walls of the sac, if we were able to do so; and *à fortiori* we should not be justified in having recourse to depletion, and lowering with the view of preventing rupture or adhesion to neighbouring parts.

The obliteration of the cavity of the sac by the formation of fibrinous clots within it, as demonstrated by Hodgson, is doubtless our chief object. The coagulation of the blood would be favoured by an inflammatory condition of the walls of the sac, as has been seen in regard to what takes place within the heart.

It would also be favoured by a retardation of the circulation. This would be promoted by the means suggested for the treatment of dilatation of the aorta. Coagulation within the sac might therefore be assisted, and possibly induced by such means, and thus its increase under the expansive force of the current of blood might be temporarily prevented.

But a cure might still be very far off; for the sac may become filled with coagulated blood of a very soft consistence, so that, not adhering to its walls, the current of blood might pass round it and burst forth, as in Case 23. In order, therefore, that the process of obliteration may go on satisfactorily, it is necessary that the clot should be rich and firm, and become agglutinated to the walls of the sac. Now the copious abstraction of blood, and the restriction of the patient to a low diet, has a strong tendency to impoverish the blood that remains, and to cause it to coagulate into a soft, gelatinous mass, with little tendency to cohesion.* Becquerel and Rodier state that "bleeding

* Simon's Chemistry. Syd. Ed. p. 278.

exerts a remarkable influence on the composition of the blood, the greater and oftener it is repeated;" and that in proportion to the number of venesections, "the blood becomes impoverished and watery." Simon* states that "in anæmia arising from excessive loss of blood, the composition of the blood is changed, becoming poor in corpuscles and fibrin; that the solid constituents are diminished; that the clot, if it forms at all, is small, soft, and diffuent, and the fibrin, after it has been separated by whipping, not tough or firm." He† also states that Geddings, in the examination of the heart and large vessels of anæmic inhabitants of the morasses of the Carolinas, found either "scarcely any coagulated blood, or else a clear red, or greenish dirty-looking fluid, almost entirely devoid of solid or colouring constituents, containing but few blood corpuscles, and which could not be coagulated, either by heat or nitric acid." Others have not found the fibrin diminished.

In order, therefore, to promote the formation of *tough* and *firm* coagula, which may adhere to and strengthen the walls of the sac, and on which they may contract, and thus form a solid fibrinous tumour, we should rather enrich the blood by animal food, than impoverish it by low diet and extensive depletion. It behoves us then to seek for other means to promote coagulation within the sac which are not incompatible with this last-mentioned object. These may be found in the administration of sedative drugs, purgatives, and chalybeates; mental and bodily repose; and the application of cold to the external tumour.

An attempt has also been recently made to effect this object by the introduction of iron wine into the aneurismal sac.

Were the treatment confined to the administration of sedative medicines, and the enforcement of complete rest, whilst a full nutritious diet was allowed, there might be a

* Op. cit. p. 308

† Op. cit. p. 309.

danger of inducing a plethoric state of the system, and a troublesome derangement of the chylo-poietic viscera. These evils, however, might be counteracted by the employment of saline purgatives, which would relieve the system, and remove some of the watery portions of the blood, at the same time that they would not greatly diminish those parts of it which enter most largely into the formation of the solid clot. Here again small doses of the tincture of digitalis may doubtless be of great value; by diminishing the frequency of the heart's stroke, and thus favouring coagulation within the sac, without weakening the patient, or in any way injuring the heart. It has only been employed, however, in two or three of these cases, in consequence of an alteration of views in regard to its mode of action.

We are not entirely excluded from the use of the lancet in these cases, because it may happen that an extraordinary tension of the vascular system might require relief by the abstraction of a *very small* quantity of blood.

The administration of iron has been found by Simon, and by Andral and Gavarret, to have an extraordinary influence on the state of the blood, in some instances doubling the amount of its solid constituents. It has generally been given in these cases in the shape of the tincture of the sesquichloride of iron combined with hyoscyamus, and it has seemed to have been of the greatest value.

The application of cold cloths and cataplasms, or bladders of ice to the surface of aneurismal tumours, was practised by Guerin, and subsequently by Pelletin; he joined it, however, to excessive depletion, and very low diet. Rejecting the latter part of the treatment, it has been employed for the purpose of producing coagulation within the sac, but it has been combined with the plan of treatment above detailed. In some cases the pain induced by the application of cold was so intense that it could not be borne. The following is a case in point, and so was Case 62:—

CASE 101.—*Sacculated Aneurism springing from the Ascending Aorta, and bursting into the Left Pleural Cavity.*

A jobbing smith, æt. 54, stated that when a young man he had been much addicted to drink; that last Christmas he began to feel a pain in his breast, shooting through to his back.

When visited in consultation with Mr. Berry, he complained of some pain in his breast; of palpitations and cough. His expectoration was said to be generally white, occasionally of a yellowish colour. He could lie flat on his back, but not on either side. He had a natural pulse.

A slight elevation was perceived between the cartilages of the second and third left ribs, which was of the size of a half-crown piece. Over this spot a single pulsation was felt. Here also a dull sound was given out on percussion. The respiratory sound was natural. The sounds of the heart were normal above the left nipple, but over the elevation a faint diastolic bellows-sound was heard. It was louder when the patient sat up than when he lay down. The extent and projection of the elevation gradually increased, and the pulsation over it became more liquid. He did not bear well the application of cold. He rather suddenly expired some months after being first seen.

Inspection.—The left pleural cavity was full of serum and recent black coagulum. The cartilages of the second and third left ribs were detached from the sternum by erosion of this bone, which, as well as a portion of the left lung, was found adherent to a tumour of the size of a small melon, which was seen lying under and to the left of the sternum. The ascending portion of the arch of the aorta was slightly dilated and studded thickly with atheroma and calcareous scales. At the upper, left, and anterior portion of it was a circular aperture, as large as a shilling, with well-defined edges, leading into a pouch which formed the tumour above described. The inner membrane of the aorta was insensibly blended into that which lined this pouch, but the middle coat terminated abruptly around the aperture. The cellular coat might be seen doubled back upon itself for nearly half an inch, where the pouch rested as it were on the aorta, and then, leaving the vessel, formed the covering of the sac. Where the sac was thus in contact with the aorta, all the coats seemed separated from each other by thickened cellular tissue and atheroma. The anterior and right side of this sac was lined with laminated, tough, discoloured coagula. At the spot where the cartilages of the second and third left ribs had been detached from the sternum, the blood had passed in front of the ribs, and another small pouch was formed, and was filled with some

old and some recent coagulum. This formed the elevation which was observed during lifetime. At the upper part of the back and left side of the large sac, the lung was denuded over a space as large as a shilling, and for some distance around this spot the lung seemed infiltrated with blood, and resembled pulmonary apoplexy in colour, but was much softer in consistence. From this place a channel could be traced for six inches between the pleura and lung, to an opening in the pleura, about six inches from the apex of the lung, and about midway across its posterior portion. Through this opening the blood, having previously formed the channel to it, by dissecting the pulmonary pleura from its subjacent lung, had burst forth into the pleural cavity. The appearance of the heart was natural.

The existence of an aneurism was here clearly pointed out by the pulsating tumour, which sounded dull on percussion, over which a murmur was heard which was not audible in the precordial region. The murmur being diastolic, led to the belief that the aneurism was sacculated; because it is difficult to conceive in what way a diastolic murmur can be produced in a dilated aneurism. During the systole of the ventricles, the edges of the aperture of the sac must have yielded to the force of the blood, and allowed it a free passage into the sac. On yielding in a similar manner to the current passing out of the pouch during diastole, the edges of the aperture would project into the aorta, and thus diminish its calibre, and offer an obstacle to the blood flowing backward under the influence of the recoil power of that vessel.

The inability to bear external cold applications is sometimes overcome by using a cold conium poultice.

The rational method, therefore, of treating sacculated and mixed aneurisms, founded on observations of their formation, progress, and termination, would appear to consist in the use of sedative drugs, with iron and digitalis, and occasional doses of saline purgatives; a moderate amount of food of a nutritious, but not over-stimulating quality, absolute rest of mind, moderate bodily exercise, and the application of cold to the surface of the chest near the sac.

Its effect may be seen in the following:—

CASE 102.—*Aneurism of the Arch of the Aorta.*

A widow, æt. 42, took cold from exposure four years ago, since which time she has had a dry cough, and her breath has gradually become shorter. Two years since she fretted very much in consequence of the loss of an only child. During the last nine months she has constantly experienced a little pain, which seemed to shoot from the left of the upper part of the sternum backwards through her breast, and has been much increased by coughing, and which has been rendered excruciating by pumping or any other violent exertion. During the last six months she has had frequent palpitations, which have been always aggravated by exertion or mental emotion.

The pulse was small, particularly when compared with the impulse of the heart, which was very strong three inches below the left nipple. No pulsation or thrill could be felt over the sternum or clavicles.

Immediately to the left of the upper third of the sternum a prominence was observed, rising gradually to the height of about half an inch, and covering a space about twice the size of a crown piece. Over this spot there was a faint pulsation felt, which was not at all liquid. A coarse double murmur was also heard in the same place. At the precordial region both the natural sounds of the heart were heard, unaccompanied by any murmur. The respiratory sound was natural. Leeches and belladonna frictions were ordered to the region of the heart, a mild anodyne mixture was prescribed, and a bladder, containing a refrigerating mixture, was directed to be kept constantly on the tumour. In a few weeks' time the tumour had sensibly diminished, and the pain and palpitations were much less severe. She has not been since heard of.

The aneurismal pouch, in all probability, already contained fibrinous clots when the patient came under examination. The existence of a double murmur confined to the spot where a pulsating tumour was seen, afforded conclusive evidence of the nature of the tumour, and clearly proved it to be a sacculated aneurism of the aorta.

This treatment was also attempted to be carried out in Cases 90, 62, 63, and 23. In 60 there was no time for its trial, and 55 occurred in the practice of another physician.

In 62 and 100 the applications of cold could not be borne, from the excruciating pain they caused. In 93 the inability of the patient to obtain rest from bodily labour prevented his deriving benefit from any treatment. In 60, coagulation in the sac was effected; but the patient was so much depressed by long suffering and straightened circumstances, that the clot when formed was soft and gelatinous, and thus the blood burst forth before the cure could be completed. Two of the following cases were supposed to be alive when they were published, and the third died of another complaint, and thereby the success of the treatment was clearly revealed.

CASE 103.—*Aneurism of the Arch of the Aorta.*

A pensioner, æt. 46, formerly 20 years in the horse artillery, and latterly a nurseryman, had invariably enjoyed good health until within two years, when he was one day suddenly seized with a kind of snatching or shooting pain at the back of his head, which was increased by the recumbent posture. It persisted for eight or nine weeks, and was replaced by a sensation of lightness in the head, and once or twice when coming home from work, he partially lost his senses and fell down, and he has experienced this sensation occasionally ever since. Four months since he had been engaged for some days in hard trenching among tough roots, when he one day received a strong shock from striking his spade against a stone. He immediately felt a severe pain under the sternum, and pulsation a little to the right of its upper part, so strong that a person whom he requested to feel it thought the heart was beating in that situation. He continued to feel the pain and pulsation for some weeks, when being unable to continue his work, he came to Birmingham by recommendation of his medical attendant, and was admitted into the General Hospital.

When examined, he complained of some severe pain and beating to the right of the sternum when quiet, which were much increased by exertion. He stated that he had lost flesh of late, and that the night before he felt a pricking on the right side of the front of the chest, extending down the right arm. A pulsation was felt over the right clavicle, and a trace of it over the left; none over the top of the sternum. The pulse was natural, but fuller and larger in the right than in the left wrist. Close to the right of the sternum, on a level with the second rib, a slight elevation was seen, the base of which was

of the size of a crown-piece, and over this spot was felt a strong single systolic pulsation. The impulse communicated by the stroke of the heart was not great above the left nipple. Dullness was perceived on percussion over the elevated spot. The respiratory sound was natural. The sounds of the heart above and to the left of the nipple were rather confused, and the diastolic sound was accompanied by a long, soft, rasp-sound. Over the lower third and middle of the sternum this sound became a saw, rasp-sound, coarse, hollow, and louder than over the nipple. Over the elevation it was more hollow, but less intense, still diastolic, but the systolic sound of the heart was also heard in all these places. Five weeks after this he complained of much pain and constriction of the chest, and down the right arm. The elevation was, however, less marked, and the pulsation in it weaker. Six leeches were applied near the right axilla. In another fortnight he was bled to 3vj, and a bladder of cold water was ordered to the elevated spot. In a few days he complained of much pain in the chest, and was bled to 3xvj, with relief. The elevation was much diminished in extent, and the ribs were easily traceable through it. The pulsation was much less liquid, the elevation feeling firmer and more solid. The rasp-sound became much more feeble. A few weeks after this the tumour had nearly disappeared, and his pain and uneasiness were very much diminished. What remained of the tumour was firm and hard, and the pulsations over it had not the least trace of liquidity. He was then discharged.

It is probable that in this, as in Case 62, the aorta was first dilated, and then ruptured on his receiving a shock. It is doubtful whether the diastolic murmur was generated at the mouth of the aneurismal pouch, or by regurgitation through the orifice of the aorta. The liquid pulsation of the tumour was enough, however, to indicate the nature of the disease; the blood seeming almost to strike the finger when he was first examined.

CASE 104.—*Sacculated Aneurism of the Arch of the Aorta.*

A labourer, æt. 53, enjoyed tolerable health until three years since, when he was attacked with severe cough, dyspnœa, and pain in the chest, and in a few days a pulsating swelling of the size of a walnut appeared to the right of the upper part of the sternum. Leeches were applied, and the swelling, pain, and cough disappeared, but considerable dyspnœa remained, particularly on exertion. Eleven months ago he

had a similar attack, when he was admitted into the Birmingham General Hospital; and for some time the tumour increased, but eventually receded considerably after he had been leeches, placed under the influence of digitalis, and had cold applications to the tumour. He was discharged. Last week he experienced another attack, and on one or two occasions he spat up a little bright red blood.

On his readmission into the hospital he was seen to be emaciated, was pale and sallow; he had an anxious haggard expression of countenance. He complained of total loss of appetite, great dyspnoea, increased by exertion, severe pain under the sternum, darting up to the right clavicle and to the right hypochondrium; he lay easiest on his right side. Between the first and second right costal cartilages a tumour projected which was seen to pulsate, and on which pulsation by no means liquid was felt with the fingers. It covered a space about the size of a crown-piece. The upper part of the sternum was slightly projected forwards. The tumour sounded dull on percussion. The respiratory sound was rather feeble under both clavicles, and was in places accompanied by cooing sounds. The action of the heart was heaving, and both its sounds rather muffled and prolonged. No sound was heard over the tumour.

The patient was, after a time, relieved by cold applications to the tumour, &c., and has not since been heard of.

Although the pulsation in the tumour was not very liquid, it was sufficiently so to prove that it was not propagated from the aorta by any solid or liquid lying before it. The tumour was clearly a sacculated aneurism containing some fibrinous clots. Its pulsations were propagated over the whole of the upper part of the chest.

CASE 105.—*Sacculated Aneurism springing from the Ascending portion of the Arch of the Aorta.*

A shopkeeper, æt. 38, had, during the year 1837, experienced more or less of pain in the front of his chest. In November of that year he had an attack of illness, which was said to present the character of bronchitis. Soon after this he felt pains in the thorax, running down the right arm, which he supposed to be rheumatismal, his right arm often feeling as if it were asleep. In a short time after this his medical attendant perceived a swelling to the right of the upper third of the sternum, over which he felt distinct fluctuation and a strong pulsation.

In November, 1838, he took cold, and the pain in the chest became aggravated, being most urgent when he was sitting quiet.

The tumour had now attained the size of half a large melon. He was placed on a rigidly spare diet, was bled repeatedly, and ordered digitalis. On visiting him in February, 1839, a very large swelling was seen over the upper half of the right side of the thorax, and a very liquid pulsation was felt over the whole of it with heaving impulse. All the sounds of the chest were natural, but the double sounds of the heart, which were clear, were heard distinctly over the tumour. A bladder of cold lotion was ordered to be kept almost constantly over the tumour; great quietude was enforced, and mild sedatives and nutritious diet were ordered.

In a few weeks the tumour was seen to decrease, and his diet was gradually improved. In August the swelling was reduced to the size of half an orange, and felt firm and hard, the pulsation being liquid only in one small spot. The cold application was discontinued, and animal food and gentle exercise allowed every day. Towards the middle of the month he was seized with a violent attack of acute bronchitis, which carried him off in eight days.

Inspection.—A smooth spheroidal tumour, with a dense, shining, aponeurotic covering, was seen lying in the upper part of the right side of the thorax and under the upper third of the sternum; it was as large as a small melon, and had pushed aside and compressed the upper lobe of the right lung. The substance of the lungs was healthy, and the compressed portion was easily inflated. The lining membrane of the bronchial tubes was livid, puffed, and in some places softened; they contained much ropy mucus. The tumour adhered to the sternum and to the cartilages of the second and third right ribs, and partly to the second rib, which had become detached from its cartilage by erosion. The tumour was found to be an aneurismal sac, springing from the right and anterior portion of the ascending part of the arch of the aorta, with which it communicated by an oval orifice. With the exception of a small portion immediately behind the sternum, every part of the sac was lined with dense, firm coagulum, from half an inch to two inches in thickness. There remained room in the sac for about eight ounces of fluid. The great size of the tumour, and its extension on the right side of the chest, had caused it to push the large vessels further back from the sternum than usual. The left vena innominata and the superior vena cava lay in deep grooves on the tumour, and each contained coagulum. A prominence in the interior of the sac was found to correspond with these grooves. The descending aorta, innominata, carotid, and subclavian arteries were all healthy.

One glance revealed the nature of the affection. It was very interesting to watch the gradual diminution in the size of the tumour, and its increased solidity from the formation of coagulum within it. Before the fatal attack of bronchitis the patient was enabled to attend to his shop.

In Case 63 death resulted from a communication having been established between the sac and a tubercular cavity in the lung. Mr. Fell, of Ambleside, in describing the result of the inspection of the body, remarked, "There can, I think, be no doubt as to the effect of your treatment in arresting the tendency to rupture externally, and prolonging her life; and had not the disease been coupled with pulmonary ulceration, affecting the sac (which seemed sound all around the opening, where the fatal hemorrhage took place), it is impossible to say how long she might have lived, or whether she would ultimately have died of aneurism." The morbid specimens both from 63 and 105, are in the museum of King's College, London.

In carrying out this line of treatment, great assistance was derived from closely watching the nature of the pulsations; gradually diminishing the employment of means adopted to promote coagulation in the sac, as the decreasing liquidity and clearness of the pulsation indicated the progressive formation of clots. Deducting Case 21, in which the circumstances of the patient prevented the treatment being carried out, there remain eight cases, in five of which great relief was obtained, and in three of which the treatment failed.

Thus, as far as the limited number of cases allows of any inference being drawn from them, practical observation confirms the soundness of these views of treatment, which have been suggested by the nature and progress of the affection.

Success, however, has been claimed for a line of treatment, in some respects the very opposite of that here suggested and worked out, viz., the depletory plan as

practised by Valsalva. Dr. Hope has pointed out the serious objections which exist against this treatment, and has proposed a plan more approaching to that which has been here advocated. Drs. Beatty, Stokes, Watson, Copland, O'Brien and others, have also advanced sound reasons against our employing Valsalva's method. The extreme severity of this treatment would prevent its being rigorously carried out, in most cases, even were there not so many weighty reasons against it; so that, in fact, it is highly probable that in many instances the cures claimed for it took place under the use of a diet more generous than was suspected by the medical attendant.

Now, in selecting the treatment to be adopted, as we cannot always at once determine whether an aneurism is dilated or sacculated, it is a safe rule to adopt the treatment suitable for dilated aneurism, in those cases in which the tumour has not reached the surface of the chest, and the treatment for sacculated aneurism in those in which an external prominence is perceived. Rarely a dilated aneurism might be included in the latter class, as Case 25, but never one in which the coats were entirely preserved. Sometimes, also, a sacculated aneurism might be included in the former class (Class 62, in its earlier stage); but even if this were so, the danger of adhesion to an important contiguous part, as the trachea might be diminished by mild antiphlogistic treatment, and on the sac reaching the surface, the treatment would be immediately changed.

CHAPTER XV.

TREATMENT OF DISEASES OF PERICARDIUM.

RHEUMATIC FEVER—*Prophylactic Treatment—Alkalies—Sudorifics and Diuretics—Opium—Sthenic form—Venesection—Objection—Strength only apparent—Calomel and Aperients—Asthenic Form—Stimulants and Tonics—Spirit Drinkers—Mercurial and Opiate Frictions, &c.*

PERICARDITIS—*Alkalies—Early Administration of Stimulants—Opium—Leeches—Strong Mercurial and Opiate Frictions—Rest—Recumbent Position—Stimulants for Delirium—Blister for Effusion—Iodine Friction in Chronic form.*

As a knowledge of the causes of any disease forms the only sound basis on which to construct a plan for preventing its approach, so the study of its progress and termination furnishes us with the best means of devising a line of treatment calculated to stay its course, and arrest its tendency towards a fatal termination. The two main causes of pericarditis have been seen to be—the morbid state of the blood which occurs in rheumatism; and a low debilitated state of the system.

These points, therefore, must be kept steadily in view in our endeavour to ascertain a satisfactory prophylactic treatment. Although it has been seen that in some few cases pericarditis has preceded an attack of acute rheumatism, yet, as a general rule, it appears in its course.

Therefore, the treatment of rheumatic fever must engage our attention, in some degree.

RHEUMATIC FEVER.—Whatever effect exposure to wet and cold may have in bringing on an attack when a person is predisposed for it, this cannot be considered as its cause, nor will it in general be thus induced in a person in perfectly good health. The blood is in an abnormal state, lactic or some other allied acid existing in it, and showing itself in the perspiration and the urine. How far this may depend upon hereditary taint, or how much on the habits and mode of living of the individual, it may be difficult to determine. That it arises from mal-assimilation cannot be questioned, and although we cannot hope to alter a constitutional tendency to disease, we may adopt measures for the improvement of the digestion and assimilation. And this it is often in our power to do, because persons in whom the rheumatic diathesis exists, have usually premonitory attacks of some sort or other previous to any violent outbreak of acute rheumatism. In these cases, the line of treatment previously laid down* for the gouty diathesis may advantageously be put in practice.

When, however, the attack is fully developed, we have to decide upon the line of treatment to be adopted. It would be useless to recapitulate all the different modes and medicines that have been employed during the last forty years with the arguments for or against each of them. There can be no doubt that rheumatic fever has occasionally run a very favourable course under every variety of treatment. I shall confine myself to describing that mode which experience has shown to be on the whole most satisfactory, and most in accordance with the discoveries that have been made in pathology.

The subjects of rheumatic fever present themselves to us under two very different aspects—the plethoric person, in apparently otherwise robust health, and the sickly, delicate-looking person, evidently in a low asthenic state. Yet we

* Page 270.

are well assured that these two persons, so different in appearance, have this one point in common, a state of the blood in which there is present a large quantity of lactic or similar acid, and an excess of fibrine held in very weak solution in consequence of the presence of this acid. The administration of alkalies is indicated for the purpose of neutralizing the acids; the salts formed being more easily eliminated from the blood; and to prevent the fibrin from being separated and forming injurious morbid products. At the same time elimination is to be promoted by exciting increased action of the skin and kidneys. This may be done by the administration of citrate or acetate of ammonia to increase perspiration; and nitrate or acetate of potash to stimulate an additional secretion of urine. It is important that there should be an excess of alkali. If the skin be acting very freely, as is often the case, bicarbonate of potash and nitre can be given. In many cases, from the time when the urine becomes very alkaline from the administration of the above salts, the patient progresses favourably.

But these two classes of cases may have other symptoms in common. Thus there may be much pain and great irritability, and both may, therefore, require to be treated by opium, sometimes in very large doses. In some cases, indeed, in which there is excessive pain and swelling of the joints, and in which there is profuse perspiration, large doses of opium have been given alone with the best results, as evidenced by the following:—

CASE 106.—*Acute Rheumatism treated by Opium.*

A tradesman's wife in Birmingham, 35 years of age, was seized with articular rheumatism about twenty-five years since. The joints, particularly the wrists and ankles, were much swollen, and exceedingly painful. There was great irritability, and her screams were loud and incessant. She was constantly bathed in perspiration. The urine was high coloured, but by no means scanty, and the bowels were in a good state. There were no abnormal sounds in the precordial region. Opium in grain doses was at first given every two or three hours, and

was rapidly increased to three grains, so that she took about half a drachm in twenty-four hours for nearly a week; during which time some strong beef-tea was given, and a large quantity of weak brandy and water. An ointment of opium and mercury was lightly spread on lint and laid on some of the joints which were most swelled and painful. At the end of this time, the symptoms mitigated, and the opium was gradually discontinued. She then for the first time recognised the physician in attendance. Her convalescence was rapid, and for many years she never had any return, nor had she any thickening of the joints or any affection of the heart.

This occurred before the value of the alkaline treatment was fully recognized, as occurring in the practice of Dr. Fuller and others. Since then the affected joints have usually been moistened with an alkaline and opiate lotion, generally with great relief.

Again, in both cases there is the probability of pericarditis supervening. Therefore, with a view to its prevention, the precordial region may be rubbed with an ointment containing both mercury and opium. Some practitioners strongly recommend the administration of mercury by the mouth and general inunction, in such quantities as to produce salivation. It has been found by others, however, to have a much better effect when rubbed in *locally*. In the cases in which the late Dr. Taylor observed pericarditis to come on whilst the patient was in a state of salivation, mercury was introduced by the *mouth* or by *general* inunction.

In the course of practice on which these observations are grounded, whilst in inflammation of the brain and its membranes great benefit was often derived from purging by large doses of calomel, in that of the peritoneum, pleura, and pericardium, the local inunction of mercury in combination with opium has given more successful results.

We have now arrived at a point where the lines of treatment for these two subjects of rheumatic fever diverge. Having in the case of the previously strong and vigorous, endeavoured to correct the poison in the blood, to allay pain and irritability, and to prevent peri-

carditis, what shall be done to calm the violence of the fever, and controul the force of the circulation? Here is *apparent* strength capable of bearing any amount of depletion. Bouillaud, Macleod, and others of their school would have taken half the blood from the body of such a subject by repeated bleedings. As this practice has been entirely abandoned, in this country at least, it would be useless to furnish facts and arguments against it. Such will be found in Dr. Fuller's* work.

Practical men must often have observed how persons apparently strong and robust are prostrated by venesection in rheumatic as well as in other fevers; but even when it is not so, the *result* might well deter us from this practice; for it has been seen that a low and debilitated state of the system predisposes more than anything else to pericarditis and endocarditis. Dr. Fuller† well remarks, "Even if a predisposition to cardiac inflammation be not engendered, as I believe it to be, by copious and repeated blood-letting, still convalescence is retarded, the patient weakened and rendered liable to frequent relapses."

Some persons assert that one small bleeding at the outset is often very beneficial in subjects such as we are supposing to be under treatment. It will be seen, however, that no abstraction of blood is generally required.

It has been found that the object we have in view is better accomplished by the administration of two or three doses of calomel of five grains each, followed by a brisk saline purgative containing some colchicum wine; and even sometimes by mild doses of castor oil. But rheumatism is no exception to other inflammatory acute affections.‡ The strength and vigour of its subjects are often more apparent than real; at any rate they are not unfrequently succeeded by a state of great prostration. The cautious practitioner will therefore always be on the

* On Rheumatism, p. 78

† Op. cit., p. 79.

‡ Introduction, p. 17.

look out for its earliest indications, prepared to anticipate it, if possible, by the administration of bark and stimulants; feeling his way by small doses, and increasing them in proportion as the powers of the patient diminish. Often a rapid amendment in all the symptoms follows the first employment of these remedies; at any rate a quick convalescence is often promoted, and an attack of cardiac inflammation is prevented. This is amply proved by comparing the statistics of the modern treatment of the disease with those furnished by that pursued by the advocates of bleeding, purging, and salivation.

The following Case illustrates this mode of treatment, and is given, not on account of the rapidity of the cure, for in fact the disease ran rather a protracted course; but as an instance of the disease occurring in an aggravated form, with a strong tendency to cardiac inflammation, eventually brought to a successful issue without leaving behind it any traces of its occurrence.

CASE 107.—*Rheumatic Fever—Recovery.*

A carver and gilder, æt. 38, a strong healthy-looking man, says he never had a day's illness in his life, but has occasionally had cramp in his limbs, and some palpitation at the heart. Five days since, without being in any way exposed to cold, he experienced stiffness and aching in the fore and middle fingers of the right hand, and in the soles of his feet. During the following night his feet and hands began to swell, and the next day all the joints of the body became affected; he had much headache, and lost his appetite and rest at night.

When seen he was very restless, but afraid to move on account of the agonizing pain, which prevented his bearing the weight of the bed-clothes. He had a most anxious expression of countenance; his skin was bathed in perspiration, having a strong acid odour; tongue moist and furred; pulse 112, full and bounding; respiration 24; urine high coloured, very acid, specific gravity 1020, containing no excess of urea. The next day it was found that the pain had become, if possible, more severe in the night, and shifting from one joint to another; that he complained of a "catching pain at his heart," and violent palpitation, and of being unable to lie on his left side. He had slight difficulty of breathing, and a little cough. The sounds of the heart

were natural. Coarse bronchial respiratory sound, mixed with sonorous rattle, was heard more or less all over the chest. Bowels confined.

Ordered. Potassæ bicarb. gr. xv. }
 „ nitrat, gr. v. } 4 tis. horis.
 Træ. opii. m. x.

Liq. morphinæ hydrochlor. m. xx. o. nocte.

Next day. Ol. ricini, ʒss.

Potassæ bicarb., increased to ʒss.

Turpentine stupe alternately to back and front of chest, followed by linseed poultice over the heart.

Three days after he was first seen, the pulse having risen to 120, and there being great debility, 4 oz. of brandy were ordered daily, and soon raised to 8 oz. in consequence of symptoms of delirium appearing.

This treatment was continued for 10 days, when liq. cinchonæ m. x was added to each dose of alkaline mixture. He gradually improved, and after a time the bi-carbonate of potash was reduced by half, and five grains of iodide of potassium added. The brandy, too, was gradually reduced. He was quite well nine weeks from his first seizure.

There was at the onset of this case great irritability of the heart, and pericarditis seemed imminent. It was probably warded off by the calming effects of the laudanum, the counter-irritation, and the sustenance of the strength by the early administration of alcohol. What might have been the result of the depletory treatment, for which this seemed so favourable a case, it may be difficult to say; but it is not impossible that the irritability of the heart might have been increased by it, and an attack of pericarditis induced. At any rate this case is valuable as illustrating the successful result of an opposite line of treatment.

Let us now return to our point of divergence, and inquire what is to be done for the asthenic subject of rheumatic fever. Having, as in the former case, employed remedies to correct the poison of the blood, and eliminate it from the system, to allay pain and irritability, and, if possible, prevent the inflammation of the heart, what shall we do to sustain the strength of our patient, and

thus prevent his sinking rapidly, or being attacked with pericarditis, which so often appears in a debilitated state of the body? Bark, ammonia, and alcohol are now our sheet-anchors, and we must commence with one or all of them at once; at first by gentle doses, but raising them fearlessly and rapidly, should the forces appear to be failing. Many of these patients, particularly in hospital practice, will on inquiry be found to have been habitual spirit-drinkers, and will therefore require a larger amount of stimulant to keep them up than if they had been temperate. As in these cases cardiac inflammation is most imminent, local inunction with mercury and opium should never be omitted.

This slight sketch of the treatment of acute rheumatism can be filled up in accordance with the requirements of each case, and will have to be varied as one or other of the symptoms predominate. Complications not unfrequently occur, too, such as pneumonia, pleuritis, nephritis, &c., which require attention, and would be fully entered into on this occasion, were we not concerned with rheumatism simply as a forerunner of pericarditis, and arising from a common cause, for which reason no attempt will be made to indicate the treatment of its chronic form.

PERICARDITIS.—Whatever may be the differences between the nature of pericarditis and endocarditis, their mode of termination and the signs to which they respectively give rise, in a practical point of view, no distinctions can be carried out in their treatment.

There is this peculiarity in cardiac inflammation, that it almost always only occurs in combination with that of neighbouring organs, or in course of other diseases. Therefore it is only in the former case that it can present itself to our view in a sthenic form; because even where it is of a rheumatic character, it does not usually appear until the original disease has in some degree lowered the strength; and this takes place to a still greater extent when it arises in the course of chronic pyæmia, or after serious acci-

dents or operations. Hence irritability and debility are the two leading characteristics of the early stages of pericarditis. Nor is it much otherwise when it appears in the sthenic form in combination, for instance, with pleuritis, for it then runs such a fearfully rapid and destructive course, that the strength is at once knocked down, and the same irritability and debility make their appearance.

Therefore these are the indications for our treatment of the first attack of the disease. To neutralize and eliminate any poison there may be in the blood, and to support the powers and allay irritability. Secondly, to promote the absorption of the products of the inflammatory action.

The best method for purifying the blood in rheumatism has already been pointed out; but we have no direct means of doing the same in uræmia or pyæmia, nor after operations and accidents, otherwise than by keeping up the subjects of them from the first by judicious management and diet.

At the outset of a combined attack of inflammation of the pericardium and pleura, &c., the treatment adopted will be in accordance with the views of each practitioner. Whilst hardly any would, in this country and in these days, employ large and oft-repeated bleedings, many would do so moderately, or apply leeches, and would at the same time give calomel to salivation, and purge freely. Others would have little faith in the abstraction of blood or mercurial salivation, but would employ diuretics, diaphoretics, and opium, combined with mercurial inunction and counter-irritation.

Our chief business, however, is with the treatment of pericarditis in its usual form occurring in the course of other complaints, and we have to conduct it in accordance with the indications above mentioned, to support the forces and allay irritability. This will be accomplished by the

administration of tonic and sedative remedies ; the former in the shape of bark, ammonia, and alcohol, or ether, in some shape ; the latter by opium.

A convenient form for exhibiting tonics, consists of effervescing draughts containing liq. cinchonæ, nitre, and ammonia in excess, which will tend to keep up the action of the skin and kidneys ; at the same time liq. opii. sed., or laudanum, can be added ; but it will probably be found more useful to give this drug in the shape of half grain doses of the gum more or less frequently as the symptoms may indicate.

It will generally be found necessary to give alcohol in addition to other medicines. Spirits have this advantage over wines, particularly those which contain much sugar, as port and brown sherry, that they are less calculated to increase the acidity of the blood. Brandy or whisky have usually been found suitable. It is impossible to lay down any laws or limits for the exhibition of stimulants and sedatives. The former must be given in sufficient quantity to sustain the forces, if they are at all sustainable, in accordance with the suggestions given for their increase or decrease in the introductory chapter. The extent to which opium may be given with advantage has been seen in Case 106.

But the pain may be very severe and of a stabbing character at the onset, in which case if dry cupping fails to relieve it, it is sometimes removed by the application of a few leeches, care being taken not to allow them to bleed long when the patient is faint. All this time the precordial region is rubbed every four hours or so with mercurial or opium ointment,* and omitted on the slightest appearance of the gums being affected ; a large linseed-meal poultice being applied after each inunction.

- * Ung. Hydrarg. fort ʒss.
 Camphoræ gr. xv.
 Pulv. Opii (well rubbed down) ʒi.

Whilst pursuing this treatment, it behoves us to be constantly on the look out to prevent the ill effects of disturbing causes. Thus the most perfect rest both of mind and body is requisite, because sudden motion or alteration of position have alike been known to bring about a fatal syncope. Above all, the patient should on no account be raised, or allowed to raise himself in bed.

Again, both delirium and choreic convulsions are by no means of unfrequent occurrence. Formerly these would have been attributed to a kind of metastasis of inflammation to the membranes of the brain; but it is now well known that they arise from irritation of the nervous centres, propagated through their branches from the seat of mischief, and they indicate exhaustion rather than active congestion. Hence if stimulants and sedatives have not yet been given, they must at once be administered; or if they have been given, they must rapidly be increased with no unsparing hand. It need hardly be remarked that from the first, strong meat-tea, milk, and other nutritive but easily digested aliments should be given in small quantities at frequent intervals.

In mild cases resolution may now take place under this treatment, or the products of inflammation may be absorbed almost as quickly as they are secreted. On the other hand, however, lymph and serum may be copiously secreted into the pericardial sac, and the endocardium and its valves may have become more or less swollen, thickened, and coated with lymph. Mercury has been rubbed in; it only remains for us to apply turpentine stupes, or what is generally more efficacious, blisters over the precordial region, covering them over when the vesication has been produced, by large cataplasms.

In spite of all treatment, however, some cases will run into a chronic state, in which sero-plastic effusion will neither be absorbed nor effect adhesion between the two

folds of pericardium. In that case iodine friction may be employed, for the purpose of promoting absorption; and ammonia may still be freely given, with a view of preventing fibrinous deposition on the endocardium and its valves, which it is said to do by keeping the fibrin of the blood in more complete solution.

CHAPTER XVI.

TREATMENT OF DISEASES OF THE HEART.

CASES WITHOUT NOTABLE CONGESTION—*Debility and Irritability—Increase of Power—Objection to Laennec's Treatment of Hypertrophy—Sedatives—Rest and Quietude—Decrease of Power—Danger of Dilatation—Of Syncope—Avoidance of Heated Rooms—Recumbent Position—Sedative and Tonic Drugs—Local Inunction—Tincture of Sesquichloride of Iron—Digitalis a Tonic—Diet—Climate.* PULMONIC CONGESTION.—*Danger of Tricuspid Dilatation—Mitral Obstruction—Mitral Regurgitation—Expectorants—Tonics—Leeches—Open Blisters.* SYSTEMIC CONGESTION.—*Treatment varies with strength or weakness of Heart's Action—Purgatives—Sudorifics—Diuretics—Squill—Acetate of Potash, &c.—Infusion of Digitalis—Drastic Purges Ineffectual—Acupuncturation.*

Acute myo-carditis and endocarditis have been seen to be so intimately connected with *pericarditis*, and to be so similar to it in their nature, origin, and progress, that the same line of prophylactory and curative treatment must be suitable for the one and the other; and as this has been discussed in regard to *pericarditis*, it would be superfluous to repeat it here.

Chronic diseases, however, of the walls and valves of

the heart, whether occurring as sequels of acute inflammations, or arising from other causes, demand special treatment. As the two principal of such causes have been shown to arise out of the arthritic and the rheumatic diathesis; prophylactory treatment must, of course, be directed towards a modification of these states of the system. This, too, has already been entered on; in the one case under the head of aortic disease, and in the other under that of pericarditis. There remain for consideration the means of alleviating these chronic diseases, and preventing their running a rapid and fatal course when they have already commenced.

Systematic writers, following the classification they have adopted, have laid down the treatment which they have deemed respectively suitable for hypertrophy, dilatation, various valvular affections, and other forms of chronic disease of the heart. But as these different states have been shown to be often complicated with each other, one in some cases tending to increase the injurious effects of the others, or, on the contrary, to neutralise or diminish them, it may be practically useful to consider their treatment in reference to the different stages which have been seen to occur in the progress of these diseases in general, and through which they reach a fatal termination.

These stages have been shown to be characterised—1st, by the *absence* of any notable obstruction of the circulation; 2ndly, by *obstruction of the pulmonic circulation*; 3rdly, by that of the *systemic circulation* or of *both*.

HEART DISEASE WITHOUT NOTABLE OBSTRUCTION OF THE CIRCULATION.—Various symptoms, indicative of temporary disturbance of the heart's action have been seen to arise, and to have been caused by dyspepsia, chlorosis, hysteria, &c., &c., perfectly independent of any organic lesion. These different states require their appropriate treatment, which it would be out of place to enter into largely here. It must be borne in mind, however, that in a great majority of such cases, the *irritability* of the heart which is thus

displayed, is connected with *debility*; and that although the muscular walls may be in no way diseased, yet their want of tone may sooner or later lead to dilatation of the cavities they contain, more particularly those on the right side of the heart, whereby incompleteness of the auriculo-ventricular orifices and consequent regurgitation may be produced, with all its train of fatal consequences. With a view, therefore, to prevent such an occurrence, and guided by the signs of irritability of the heart, we shall do everything in our power to sustain the forces; invigorating the constitution by generous diet, and suitable tonic medicines, and allaying excitement by anodyne frictions; enjoining quietude of mind and body, and favouring the circulation by position and other suitable means.

When, however, the symptoms of derangement of the heart's action are more persistent, and are dependent upon organic changes, our first inquiry must be whether they are such as lead to an *increase* or *decrease* of its contractile power.

Increase.—When the power of the heart is *increased* by hypertrophy of the ventricles, the choice of an appropriate treatment is often attended with great difficulties. The immediate effect of this change being to throw a powerful stream of blood into the arteries, and thus fill the capillaries with blood, active depletion, on the plan of Valsalva, has been strongly recommended by Laennec, not merely for the purpose of removing the congestion, but with the view of reducing the hypertrophy itself. Such a line of treatment, however, is based on the supposed correctness of two propositions, both of which have been shown to be unsupported by facts. One is, that the effect of hypertrophy on the circulation is always injurious, whereas it has been seen to have, in certain cases, a most beneficial influence on it, by compensating for obstruction caused by valvular defects. The other proposition is, that a very large number of cases of cerebral and pulmonary apoplexy are immediately produced by it; whereas it has

been seen that when such accidents have occurred in consequence of a congested state of the vessels, such congestion has, in the majority of cases, been of a venous, rather than of an arterial character, produced by obstruction to the return of blood through the veins to the heart; the commonest cause of cerebral apoplexy being rupture of an artery in consequence of atheromatous degeneration of its coats. It cannot be denied that arterial congestion and undue tension may be attended with certain injurious effects, and may call for a slight amount of venesection in particular cases, but not the wholesale abstraction of blood as practised by Valsalva.

Dr. Hope and others have pointed out the injurious tendency of such extensive depletion, from its inducing a state of anæmia, with its concomitant evils. But another powerful reason against this treatment is furnished by the observation of the progress and termination of heart diseases; for in proportion as the power of the muscular walls is diminished by this violent treatment, so is the tendency to dilatation and auriculo-ventricular regurgitation increased, whereby congestion of the pulmonary or general venous circulation is produced. With these dangers before us, it is impossible to carry the system of depletion to any great extent. We must be content slightly to relieve the tension of the vessels when it seems considerable. This plan will often succeed more effectually in quieting the action of the heart than copious blood-letting, which is often followed by strong reaction. Sedatives and rest materially assist; in fact, it is rarely necessary to abstract blood at all, if the patient be kept free from mental disturbance and strong bodily exertion. Of sedative drugs, belladonna and opium are those which most powerfully controul the heart's action; but hyocyamus and conium will often be found sufficient; and sometimes much benefit has been derived from the milder sedatives, lettuce or hop. Local anodyne frictions, particularly with a belladonna liniment, or an opium ointment, have seemed to

afford more relief than any medicine administered internally.

Great assistance is derived from promoting a due action of the skin by the aid of warm baths, the flesh-brush, and *moderate* exercise on level ground. At the same time, with a view of preventing the approach of diseases which are apt to ensue in a plethoric state of the system, the diet should not be too stimulating, although sufficiently nutritious well to sustain the strength; by which means the left cavities of the heart will not be irritated by the contact of blood formed from materials of too rich a nature. By adopting this line of treatment, we relinquish all attempts materially to diminish the size of the heart; and notwithstanding the extraordinary cases in which Laennec informs us the heart was reduced to a wrinkled state, it is probable that the majority of experienced practitioners will agree that nothing is lost by thus abandoning the hope of a cure so chimerical, which is obtained, if at all, by means so hazardous in their nature. Persons treated on the plan above suggested, often live on year after year, in comparative ease and comfort, greatly relieved, although still retaining a large heart.

Decrease.—If the action of the heart be *enfeebled*, whether from simple attenuation and dilatation, or from softening and fatty degeneration of the walls, in either case a tonic and sedative treatment is clearly indicated. Here again, however, we have to look to the future; for matters seldom remain very long without symptoms of further derangement showing themselves. Thus although in general there are no evidences of permanent pulmonary obstruction, yet they are apt to shew themselves on the occurrence of excitement or unusual muscular exertion, even on mounting stairs. Hence the danger of pulmonary complications, and of further dilatation of the right side of the heart, and consequent tricuspid regurgitation, as pointed out before. In fact there is more cause for

apprehension, and greater caution required in these cases, than in many others hereafter to be described, where there is a certain amount of valvular disease, which gives rise to marked signs of its existence. We have also to guard against a more imminent danger arising from *syncope*, more particularly when it is injudiciously treated. With a view to its prevention, in addition to the general rules of treatment which will presently be alluded to, we must strenuously interdict the frequenting of heated or crowded rooms, and substitute a recumbent or semi-recumbent position for an upright one, allowing at the same time a moderate amount of walking exercise; we must direct small doses of some stimulant to be given on the *first* sensation of faintness or exhaustion, such as ammonia, ether, spirits, or wine and water, &c., with immediate recourse to the recumbent position.

The patient may be so situated that this cannot be accomplished in the ordinary manner; as for instance when in a railway carriage. In that case the friends should be directed to bring the head down to a level with the knees, and to hold it there for a few seconds. Of course the same directions are applicable when syncope has actually come on. For want of this knowledge many cases of this kind terminate fatally, as was seen in Case 88.

In our endeavours to allay the irritability of the heart, we must trust more to tonic than to sedative drugs; great caution indeed is requisite in the employment of the latter. In general nothing stronger than hyocyamus and camphor, lettuce, or hop, should be given by the mouth, and only *very* mild opiate or belladonna friction locally employed. The most valuable permanent tonic will probably be found in the tincture of the sesquichloride of iron, combined with small doses of tincture of hyoscyamus. Many persons may think it extraordinary that digitalis should here be mentioned as a *tonic*, calculated to give increased force to the heart's action, whilst up to a recent

period it has been supposed to act in a diametrically opposite manner, and was so described in "Practical Observations on Diseases of the Chest." If, however, an opinion is found to be erroneous, the sooner and the more publicly it is renounced the better. In employing digitalis largely as a powerful diuretic in cardiac dropsy, it could not fail to be observed that it never seemed to depress the vital powers; and although this might be accounted for to a certain extent by the fact that stimulants were freely used in combination with it, yet even in cases where the stimulant had been considerably diminished or omitted, a depressing effect was never witnessed. The question also has sometimes presented itself as to the similarity between the action of digitalis and alcohol; why when both diminish the frequency of the pulse, the one should increase, and the other diminish the force of the heart's action? The researches of Drs. H. Jones, Dickenson, and Fuller, enable us, in answer, to assert that they do *not* act differently in this respect, but that both tend to increase the force of contraction; and that in fact digitalis in large doses produces violent muscular contraction and spasms.

In regard to diet, it is advisable not to fill the stomach at one meal, but to order small meals of nutritious food more frequently repeated; and very small doses of stimulants in some shape or other whenever languor and lassitude *begin* to show themselves, rather than any considerable quantity at meal times. With the tendency to congestion of the capillaries, the greatest attention must be bestowed on the state of the skin, more particularly during the winter months; its action being duly promoted by warm bathing, clothing, &c. In no class of cases is a residence in a warm, sheltered, and yet dry climate more valuable; such as is found on some parts of our southern coast; for whilst the saline particles in the atmosphere favour the circulation of the blood, the surface of the body is much protected from cold and damp, and

the out-door exercise necessary for the general health is duly kept up.

There are also cases, by no means of unfrequent occurrence, in which there is more or less of permanent valvular derangement without any notable or persistent symptoms of obstruction of either circulation. The aortic orifice is the chief seat of such disease, and it has been already shown how it is that it often exists to a great extent without materially disturbing the circulation. But little treatment is required, and its nature may be gathered from the remarks on that demanded by an increase or decrease in the force of the heart's action. The mitral orifice is also not unfrequently the seat of disease uncomplicated by disturbance of either circulation. But in that case the systolic murmur heard so frequently in the mitral region, near the left nipple, and under the lower angle of the left scapula, when unaccompanied by dyspnoea, will, it is suggested, be sometimes found after death to have been caused by roughening or vegetations on the ventricular side of the mitral valves or near them, rather than by any obstructive or regurgitative disease affecting the orifice itself; and this might of course persist an indefinite time without materially affecting the course of the blood, or the action of the heart.

Dilatation of the right side of the heart may also extend to the tricuspid orifice, and give rise to regurgitation through it, as evidenced by venous pulsation in the neck; and may even exist for a considerable time without giving rise to any, or at least only to faint traces of dropsy. But such cases must be very narrowly watched, for this is the state of the system in which cerebral apoplexy from venous congestion is to be dreaded. In such instances an attack has been brought on by a stooping position, as in Case 34. Great advantage is derived from dry cupping from keeping the head cool, and sometimes from the application of two or three leeches, or a small open blister behind the ears.

PULMONIC CONGESTION.—We are not now concerned with derangements caused by diseases of the lungs or pleura, but simply with those which depend on the intimate connexion that exists between the organs of circulation and respiration; whereby disorders of the former give rise to a disturbance of the functions of the latter.

Pulmonic congestion has been seen to arise from a decrease in the force of the heart's action, and from incompleteness of the mitral foramen; the state of the other orifices being little concerned in its production. In the former case the treatment suggested where there is a decrease of contractile power is equally applicable here, and as far as it is effectual in removing the cause of congestion, it will relieve the affection under consideration. Other remedies, however, may be added which will presently be detailed.

When mitral insufficiency is the main cause, the treatment may in some degree depend upon the nature of the valvular affection; whether it is such as to give rise to simple *obstruction* or to *regurgitation*. Because in the former case an increase in the contractile power of the left ventricle might assist the circulation, especially if, as is frequently the case, some aortic valvular disease were also present, or the elasticity of the coats of the aorta were notably diminished. Whereas when there is mitral *regurgitation*, forcible action of the left ventricle greatly augments the evil, by giving additional force to the current of blood which is driven back upon the lungs. Hence it is most desirable in this case to diminish the energy of the heart's action arising from hypertrophy of its left ventricle. But this can only be attempted by the mildest of the means proposed for this purpose; rest of mind and body, sedative friction, &c. For here again dilatation of the right ventricle and tricuspid foramen are very imminent, owing to the pressure thrown back upon the heart by the congestion of the lungs. Here the judicious employment of digitalis may be of the greatest use; for by this means

the circulation may be tranquilized, and rendered less frequent, whilst the walls of the heart are not weakened and rendered more liable to dilatation.

Other remedies are suggested by observing the manner in which nature often acts; giving great relief by promoting a copious secretion from the mucous membrane of the bronchial tubes. Indeed, it is extraordinary to what an extent and for what a long time this sometimes goes on, with the greatest possible relief to the patient. Hence the employment of expectorant drugs, amongst which squill in combination with ether, camphor, &c., is one of the most efficacious.

When dyspnœa comes on in paroxysms, it is often relieved by ethereal tincture of lobelia, or prussic acid, and more particularly by ammonia and chloric ether.

When pulmonic congestion is very urgent, the application of three or four leeches or the cupping-glasses at the back of the chest is often found very useful; but, above all, blisters, small and frequently repeated, or kept open, frequently afford great relief.

SYSTEMIC CONGESTION.—When congestion of both circulations occur simultaneously the case is formidable and is generally drawing to a close. Still, cases are occasionally met with in which much temporary relief is obtained, as in Case 54, 50, and others. In 116 cases of systemic congestion there were 53 of pulmonic congestion also; the latter affection in the great majority of cases resulting from an imperfect state of the valves on the left side of the heart, more especially those attached to the mitral orifice.

But when there are no signs of disease on the left side of the heart, although it may be impossible to pronounce whether there is structural derangement of the tricuspid valves, or whether the orifice is simply dilated, yet we can discover important guides to treatment, according as chronic disease of the lungs is present or not; or as the force of the heart's action is increased or decreased. In either case the treatment up to a certain

point will be conducted on the principles above laid down. Now, however, there is additional work to do; the engorgement of the capillaries must be relieved, and the serum that has been poured out must, if possible, be removed. This may be attempted by increasing the secretions from the kidneys, bowels, and the skin. The first of these plans is generally the most successful. In some cases simple diuretics will succeed; such as a combination of squill, nitric ether, acetate or nitrate of potash, spirit of juniper, &c., with drinks made of cream of tartar, broomtop, &c.

When these fail to act on the kidneys, digitalis will often succeed marvellously. The formula* in the note has been found very efficacious. Its effects may be seen in Cases 93, 94, 52, and 50.

Drastic purgatives, although valuable in certain forms of dropsy, were not found to be attended with good results, when the cause has lain at the heart. Elaterium often reduces the patient so suddenly that he rallies with difficulty. When the skin is hard and dry James's Powder has been found useful.

In Case 54, nature worked a great cure by creating an immense discharge of serum from large bullæ, which rose on the œdematous legs and burst. On the same principle acupuncture sometimes affords great relief; but it should not be performed in the scrotum, as gangrene has occasionally followed the operation in this situation. Blisters on the thigh will also sometimes act beneficially, but are not generally so efficacious as acupuncture.

* Infus. digital. (vet. Pharm.), ad f. ʒviii.

Spir. æther nit. f. ʒss.

Tinct. scillæ f. ʒii.

Tinct. cardamomi f. ʒss.

Potassæ acetatis ʒiii.

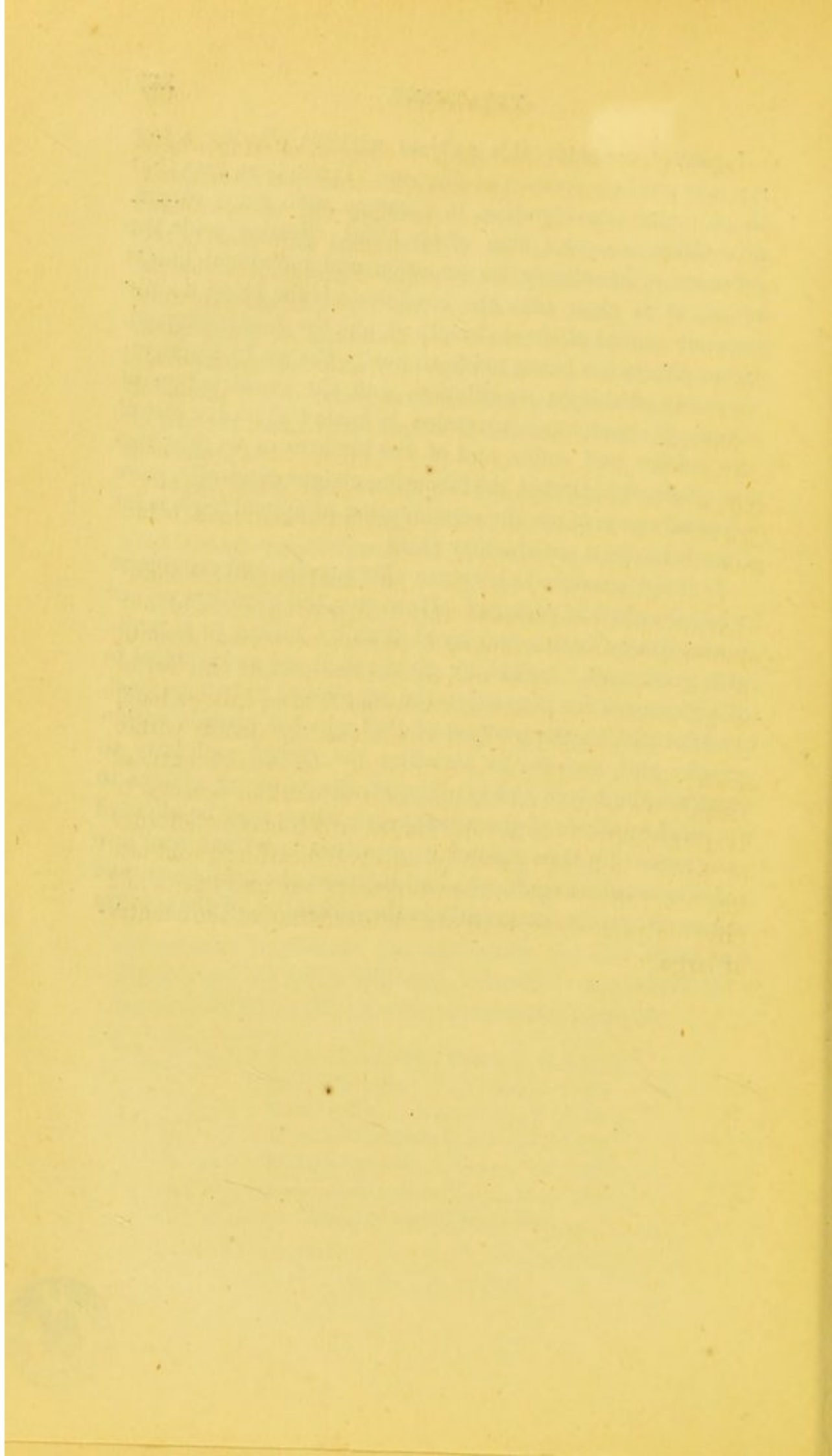
Ammoniæ sesqui-carb. ʒss.

Sumat ʒi bis vel ter indies.

I cannot conclude this subject without offering a few remarks on the *prevention* of diseases. It has been seen that those under consideration, in common with many others, arise from a *morbid state of the blood*. Setting aside the influence of hereditary transmission and individual habits of life, it is clear that the condition of the blood mainly depends on that of the *air* breathed, and the *food* consumed. Great efforts are being made to purify the *air* by improvements in drainage, ventilation, and the construction of dwellings; and much attention is bestowed on the *diet* of the soldier and sailor, and of the inmates of workhouses and gaols; but that of the labouring classes generally, more especially as regards the consumption of animal food, is far from being in a satisfactory state.

Is it not possible to increase this supply, and economize what we already possess? How can the members of our profession be better employed than by joining to promote this good work? *First*, by giving their aid and counsel to devise means for increasing the supplies of nutritious food, and for making the best use of that which is already within reach; and, *secondly*, by assisting the clergy and laity to instruct the wives and families of the working classes to avail themselves of the advantages which may be afforded to them. By thus throwing ourselves heart and soul into this benevolent work, we shall increase our usefulness, and raise our profession greatly in the estimation of the country at large.





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