Problems in cardiac pathology : dissertation for the degree of M.D. in the University of Oxford / by Fred. J. Smith.

University of Oxford.

Dissertation for the Degree of Doctor of Medicine.

The accompanying work entitled

Problems in Cardiac Pathology

by Frederick John Smith MRCS

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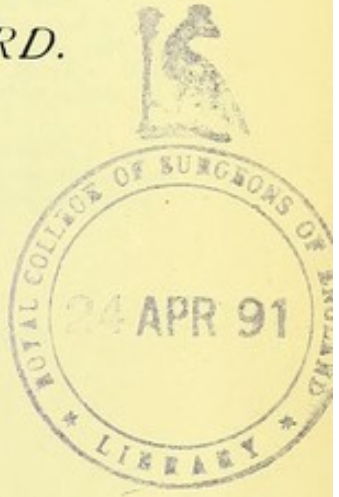
Oxford, March 1891 *Regius Professor of Medicine.*

DISSERTATION

FOR THE DEGREE OF M.D.

IN THE UNIVERSITY OF OXFORD.



PROBLEMS IN CARDIAC PATHOLOGY,

BY

FRED. J. SMITH, M.B., M.R.C.P., F.R.C.S.

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PREFACE.

It gives me great pleasure to record my thanks to the Members of the Staff of the London Hospital for their permission to make such use of their cases as I thought fit for the composition of this Dissertation; I hope that the treatment of the problems of Cardiac Pathology may be, in some degree, worthy of their approbation.

The historical sketch is introduced in conformity with the Statute. My matter is by no means exhausted, and I hope shortly to be able to publish portions of it which bear on the clinical aspect of heart disease.

FRED. J. SMITH.

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PART I.

HISTORICAL.

FROM the writings of Hippocrates—*De Morbis*—it would appear that he had some idea that a physical examination of the Chest would yield important results with regard to the condition of the thoracic organs. He believed, for example, that by immediate auscultation he had heard sounds calculated to distinguish hydrothorax from empyema. It was not, however, till many centuries after his death that the functions of the heart and the circulation of the blood were even guessed at, much less proved, and therefore it was impossible for him to have had any conception of the questions with which we are now dealing.

It is equally obvious that we shall find little or no reference to our subject in any of the writings which were produced between the time of Hippocrates and that of Harvey.

Harvey's period of activity occupied the earlier years of the seventeenth century. It certainly does seem a little remarkable that for nearly two hundred years his epoch-marking investigations into the action of the heart and circulation of the blood should have remained practically without result so far as the physical examination of the chest is concerned. It cannot be that heart disease was not common during those two centuries, for rheumatic fever is no new disease, and it is equally certain that there must have occurred many cases in which a noise of cardiac origin was recognisable at some little distance from the chest wall of the patient, circumstances which it is permissible to say we might have expected to lead to some auscultatory discoveries.

Section II. In 1763 Avenbrugger, a German physician, commenced in earnest the practice of simple percussion of the chest walls; after seven years continuous study of its phenomena

he published his observations ; so little attention however did they arouse that his labours went practically unrewarded. Corvisart—Laennec's master—seems to have taken the subject up next, and he carried this method—percussion—to a very considerable height of perfection and precision; but even now it yields comparatively little information about the condition of the heart, it does little or nothing to discriminate hypertrophy from dilatation, and is absolutely useless for estimating the condition of the valves.

Section III. The great discovery of Auscultation was left to a physician of the nineteenth century—to Laennec—a name equally worthy with Hippocrates', Harvey's, Hunter's, Charcot's, Lister's, Jackson's, &c., to be considered as that of one of the most enthusiastic founders of Medical Science. Firmly convinced of the great truths which Harvey taught, keenly and bitterly alive to the sad lessons which experience taught him on the hopelessness of advanced cardiac cases, and driven mad by the seeming impossibility of discovering such cases in an early, and therefore presumably curable stage, and withal convinced of the inadequacy of percussion as taught him by his masters, he adopted immediate auscultation of the heart, in the hope that it would yield him the information which he sought; nor was he altogether unsuccessful. Accident it was, however—how many discoveries in every subject are there which have not been made by trained intelligence taking advantage of accident,—which led him to the great advance of substituting mediate for immediate auscultation. To make the sketch complete we must turn to Laennec's own writings.

† He appears to have been as enthusiastic in the pursuit of the allied sciences as of Medicine itself, studying Heat, Light, Sound, &c., as far as the appliances and theories of the day would allow him. In 1815 he communicated to the Société de l'Ecole his first results obtained by the application of Acoustics, to the investigation of affections of the chest. This Thesis had reference solely to results obtained by contact of the ear directly with the chest. The accident, referred to above, took place in 1816, when—

"I was consulted by a young female who presented the general symptoms of disease of the heart, and in whose case the application of the hand and

† Translation edited by Dr. Herbert, published 1846.

percussion yielded scarcely any result, on account of her embonpoint. The age and sex of the patient forbidding that kind of examination of which I have just spoken—immediate application of the ear,—I happened to recollect a well known acoustic phenomenon, viz. that when the ear is applied to one end of a piece of timber, we can hear very distinctly the scratch of a pin made on the other end. It struck me that, in the present instance, advantage might perhaps be taken of this property of bodies. I procured a quire of paper, and formed it into a roll as compact as possible, one extremity of this I applied to the precordial region, and placing my ear on the other, was alike surprised and gratified to hear the pulsations of the heart much more clearly and distinctly than I had ever heard them by the immediate application of the ear."

In this extract is contained the discovery of the instrument which, perhaps more than all others, has contributed to the progress of Medicine as an exact science, though the object of the second part of this Thesis will be to show that we have yet a good deal to learn of the sounds heard by the stethoscope, and must not put a too absolute reliance on what it tells us.

Having discovered that mediate auscultation was capable of yielding excellent results, Laennec's next step was to try and perfect the details of his instrument. Again we quote from the same translation :—

"The first instrument I employed was a cylinder or roll of paper, sixteen lines in diameter, and a foot long, formed of three quires of pressed paper compactly rolled, kept together by pasted paper, and filed even at both ends. However compact such a roll may be, there always remains in the centre a passage of 3 or 4 lines in diameter, owing to the impossibility of rolling the quires that compose it, perfectly tight on each other. This fortuitous circumstance afforded me occasion to make an important observation, viz. that this canal is essential for the exploration of the voice. An entirely solid body is the best instrument than can be used for the auscultation of the heart."

As regards material, he goes on—

"Substances of medium density, such as paper, light woods, or Indian cane, have uniformly appeared to me preferable to all others—a result I have never known to vary. I am, therefore, at present in the habit of using a cylinder of wood, sixteen lines in diameter and a foot long, with a bore in the centre three lines in diameter ; and jointed in the middle by means of a tenon wrapped with thread."

Such was Laennec's original stethoscope, and with it I have little doubt that it was possible with practice to hear everything now to be heard in the thorax ; in fact the editor of the translation

from which I have quoted the description, remarks in 1846 that it was still the best.

Section IV. Since its introduction, the monaural stethoscope has undergone numberless variations in shape and material, according to the ideas and fancies, or whims, of various eminent physicians. In a catalogue of Instruments published in 1875, I find no less than twenty-seven different personal names given to various modifications, some eight of which are figured; the main point in which the majority differ from the original consists in having the two ends made broader while the connecting cylinder is brought down to very attenuated dimensions, and the length is reduced considerably below Laennec's standard: wood, vulcanite and cellulose are the principal materials. How far all these modifications have been real improvements, and not such in name only, I am unable to judge, but as none of them have kept, and strengthened by time a position, and claim to be the only reliable monaural stethoscope, we are forced to the conclusion that it is a case of *quot homines, tot sententiæ*, and that the stethoscope of the future has still to be found.

The first distinct advance was made about 25 or 30 years ago by Dr. (now Sir Andrew Clark), who introduced the binaural instruments. They possess many advantages over the single stethoscopes:—by blocklng both ears they render the observer less sensitive to extraneous sounds; by having flexible tubes connecting the ear and chest pieces, they are capable of being adjusted with a maximum of ease and convenience to all regions of a patient's chest with a minimum of movement on his part, this constitutes in itself an overwhelming advantage in pulmonary cases, and for cardiac sounds is an immense boon. Their only disadvantage is one shared in common with all instruments of precision, and consists in the difficulty in learning to disregard their intrinsic weaknesses; here, sounds originated by the instrument itself are heard, comparable to those audible on placing a shell to the ear, and these we have to learn to neglect; this is however by no means difficult, and with a little practice all thoracic sounds may be heard and identified most clearly and distinctly.

The shape of the removable chest piece, and the mechanism by which the earpieces are held in position, are the main

modifications which this instrument has undergone: now with well fitting ear pieces and a chest piece small enough in terminal calibre to get a complete contact in the narrow intercostal spaces of an attenuated subject, it remains unquestionably the best for ordinary use.

Some time prior to 1875, Dr. Scot Alison introduced an innovation, which, though now unaccountably neglected, is destined, I believe, to make the binaural the only stethoscope of the future. He termed his instrument the differential binaural, its principle consists in having two separate chest pieces, each connected by a flexible tube to an ear piece, by this means each separate ear receives the thoracic sounds which are generated beneath the point of contact of the corresponding chest piece. It thus becomes possible to differentiate with the nicest accuracy the exact time and character of sounds otherwise confused. By listening simultaneously to the two sides of the chest, we are well able to appreciate slight differences in respiratory sounds which might otherwise escape detection, and in cardiac cases where several bruits are present, I have found the use of this instrument most valuable, separate points of maximum intensity and varying directions of conduction being found and identified with the readiest ease.

It has only the slight drawback that it requires considerable practice to educate the two ears to listen simultaneously each to its own sound, and presumably a corresponding education of the psychical auditory centres must take place, for the ears like the eyes are accustomed to act together.

The history of the stethoscope has now been sketched from infancy to ripe maturity, and I think it little likely that any further improvements will take place, till science comes to our aid, and places a microphone in the market of such simplicity, that it may be easily adaptable to differential-binaural instruments, and of such moderation in cost as to be within the reach of all medical men. It now remains to sketch in a similar manner the gradual recognition of the cardiac sounds as heard by mediate auscultation.

Section V. It is not my intention to write a critical review of all the opinions of writers on auscultation; I wish merely to give

a very brief sketch of the rise of our knowledge of the physical signs of the heart which can be ascertained by the stethoscope. With this object I shall make full quotations from two or three pioneers in the work, noting those views which are still held to be correct, and leaving the omissions and errors to be accounted for by later extracts.

As the problems which I have, in the second part of this Thesis, endeavoured to solve, are chiefly concerned with the murmurs of the heart, these are the physical signs, to the history of the identification of which I have devoted most attention, omitting all mention of many most interesting phenomena, such as reduplication of the sounds, irregularity of rhythm, &c.

I have already quoted Laennec's remarks on the first case in which he listened to the cardiac sounds through a stethoscope. After considerable study and practice he remarks—

"Some of the signs obtained by mediate auscultation are very easily acquired, and it will be sufficient to hear them once in order to recognise them ever after; such are those indicative of ulcers of the lungs, hypertrophy of the heart in a high degree, fistulous communications between the pleura and bronchi, &c. But there are others which require more study and practice."

As a general statement this remains as true to-day as when it was written, though the examples of easily recognisable sounds are not perhaps so happily chosen as might have been the case.

Turning attention now to the cardiac sounds, Laennec says—

"In a healthy man who is moderately stout, and whose heart is well proportioned, its pulsations are audible in the precordial regions only; that is in the space comprised between the cartilages of the fourth and seventh left sternal ribs, and under the inferior extremity of the sternum. The movements of the left cavities are chiefly perceptible in the first point, and those of the right in the second; so that in cases of disease of only one side of the heart, the analysis of the pulsations of this viscus furnishes entirely different results in the two points."

"When the extent of the heart's pulsations becomes more considerable they are heard in succession in the following places: 1, the left side of the chest, from the arm-pit to the region of the Stomach; 2, the right side over the same extent; 3, the left posterior part of the chest; 4, lastly, but rarely, the right posterior part. The intensity of the sound decreases progressively in the order just detailed, and this I have found an invariable rule."

With regard to the cardiac impulse, Laennec remarks—

"The intensity of the shock communicated to the ear by the stethoscope,

is generally in an inverse ratio to the extent of the heart's pulsations, and in a direct ratio to the thickness of the walls of the ventricles. This impulse is but slightly marked in a person whose heart is in the proportions most favourable to the free exercise of the circulation; it is often, indeed, imperceptible, especially if there be any considerable degree of stoutness."

"The heart's impulse is felt only during the contraction of the ventricles; or, if in some rare cases that of the auricles produces a similar effect, it is easily distinguishable from the former. In fact, whenever a preceptible movement accompanies the systole of the auricles, it consists merely in a kind of vibration felt to be deeply seated in the mediastinum, and it seems even as if the heart were withdrawing from the ear."

This latter quotation can only refer to a presystolic mitral bruit, and is a wonderfully correct description, considering that the writer assumed that the second sound of the heart was the sound of the auricular contraction.

The succeeding sentence runs thus—

"A strong impulse must consequently be looked upon as the principal sign of hypertrophy of the heart; on the contrary, the absence of all impulse, joined to other signs, general and particular, is characteristic of its dilation."

Have we got any further since this was written? I think not in a direct line, though our knowledge in collateral directions has increased very materially.

On the two sounds of the heart, Laennec writes—

"In the natural state this—the sound of the heart's action—is double, and each stroke of the pulse corresponds to two successive sounds: the one clear, abrupt, resembling the click of the valve of a pair of bellows, corresponds to the systole of the auricles; the other, duller and more prolonged, coincides with the beat of the pulse, as well as with the sensation of the shock described in the preceding article, and which indicates contraction of the ventricles."

"The sound heard in the lower region of the sternum belongs to the right cavities; that of the left is heard between the cartilages of the ribs."

"In the natural state, the sound of the heart's contractions is similar and equal on both sides; on the contrary, under certain pathological conditions, each side emits a totally different sound."

"The sound produced by the heart's pulsation is stronger in proportion, as the walls of the ventricles are thinner, and the impulse weaker, and consequently cannot be attributed to percussion of the thoracic walls."

"When hypertrophy is extreme, the contraction of the ventricles is accompanied by a shock without a sound; and the sound of the auricle is so dull as to be scarcely heard."

"When, on the contrary, the walls of the ventricles are thin, the sound produced by their contraction is clear and rather sonorous, approximating to the nature of that of the auricles; and if there be marked dilatation it becomes nearly similar and almost as strong. Finally, in cases of considerable dilatation, the two sounds are not distinguishable from each other, either by their nature or intensity, but solely by their relative isochronism or anachronism with the arterial pulse."

"In the natural state, the sound of the alternate contractions of the heart is nowhere heard so strongly as in the precordial region, and it becomes weaker in the different points of the chest according to the progression already indicated."

But for his mistaken idea of the cause of the second sound, Laennec could hardly have been more accurate in describing the phenomena of hypertrophy and dilatation of the heart; his facts were marvellously accurately described, and only his theories require revision.

Of the abnormal bruits or sounds associated with or replacing the first and second cardiac sounds, Laennec recognised the following—

I. "The Bellows' Sound," on which he writes as follows:—

"The heart and arteries yield, under certain circumstances, instead of the sound which naturally accompanies their diastole, that to which I give the generic name of bellows' sound, from the exact resemblance it bears in the majority of instances to that produced by a pair of bellows when in use, and it is often to the full as intense. This comparison is perfectly appropriate. There are, however, many varieties of this sound, and some, indeed, are of such a description, that it is difficult to regard them as constituting in reality but one and the same phenomenon. But the rapidity of succession, and the insensible manner in which they pass into one another, remove all doubt as to the fact. They may be reduced to three, which I shall distinguish by the following names:—1, the Bellows sound, properly so-called; 2, the Saw or Rasp sound; 3, the Musical or Sibilant Bellows sound."

"Bellows Sound, properly so called.—This sound may accompany the diastole of the heart and that of the arteries, and is connected with them in such a manner as to replace and entirely do away with the sound which is natural to them, so that at each diastole the ventricle, the auricle, or the artery in which the phenomona take place, emits a distinct bellows sound which ceases during the systole. Most frequently the bellows sound is exactly circumscribed by the calibre of an artery or by the cavity of a ventricle. At other times, on the contrary, it is diffused, and appears to take place in a much larger region than that occupied by either the artery or the heart; so much so that neither the impulse nor form of the latter are recognisable."

"Saw or Rasp Sound.—The saw sound is precisely similar to that made by a saw when heard at some distance."

"Musical or Sibilant Bellows' Sound—This variety is presented by the arteries only—at least I have never met with it in the heart."

II. Purring Tremor.

"This is a denomination which I gave in the first edition of this work, to a particular sensation, which, in some cases, is perceptible to the hand, when this is applied to the region of the heart, and which, with Corvisart, who was the first to notice this symptom, I have pointed out as a sign of the ossification of the valves, and particularly of the mitral valve. Indeed this phenomenon is observed in almost every case in which there is some contraction of the orifices of the heart; but I have since frequently met with it without the existence of any organic lesion of this viscus. This purring is almost always limited to the left precordial region, to which the hand must be applied with some strength in order to feel it."

III. Pulsations of the heart heard at some distance from the chest. After stating that a tradition existed that such sounds were to be heard, and that Corvisart had once heard them, Laennec goes on to say:—

"The first opportunity I had of observing this phenomenon was in the case of a young girl in the year 1823. From this period I have been diligently looking out for it, and have convinced myself that if it is very rare in so high a degree of intensity as in the cases of which I have just spoken, it is very common in a less degree, and such that the heart may be heard at a distance of from two to ten inches from the chest. I have several times verified by the perfect isochronism of these beats, to those of the pulse, that the sound heard is that of contraction of the ventricles. I do not recollect meeting any cases in which it was afforded by the auricles.

On the causes of these phenomena, Laennec says:—

"I have seen a considerable number of patients die of very different acute or chronic diseases, who had presented the bellows sound during the latter period of their life, and sometimes for several months, very manifestly in the heart and different arteries; yet on autopsy I have discovered no organic lesion which constantly coincided with these phenomena, and which is not frequently met with in subjects who have not presented them. The bellows sound exists almost uniformly in the hearts of those labouring under contraction of the orifices of this organ; it is frequently met with in cases of hypertrophy and dilation, but still more frequently, both in the heart and in the arteries, of those who have no lesion of these organs and who are suffering from a very different affection."

After detailing some experiments, he concludes that the sound "is owing to a true spasmodic contraction either of the heart or arteries, and proves no organic lesion of either."

On the causation of the purring tremor, we find it written—

"I confess that, whatever pains I have taken, I have been unable to assign any satisfactory reason for it; all I can affirm is that it is unconnected with any constant organic lesion."

A similar paragraph explains Laennec's views on the sounds heard externally to the chest—

"I know not with any certainty to what state of the organs of the circulation they can be ascribed; but several reasons incline me to think that it is owing to a more or less abundant gaseous exhalation in the pericardium."

That these abnormal sounds did not help much, in the hands of their discoverer, to render early conditions of valvulitis easily recognisable is obvious when we read a little further on—

"Unless the vegetations—on the valves--are extremely numerous, I am of opinion that they can throw but little impediment in the way of the action of the valves, and consequently they can furnish no sign of their presence."

At the same time he came very near the truth when he adds—

"Yet, in one of the preceding cases, we have seen that three vegetations, a line long only, have been suspected."

Such is an outline of the history of the earliest recognition of cardiac bruits, and it must be confessed that the knowledge gained by Laennec was crude to the last degree: none of his abnormal sounds were associated with any constant lesion, and he was driven to the most curious and worthless explanations of them, while he expressly denies the existence of any auscultatory signs of Pericarditis.

In short, his chief claim to distinction—and that a very great one—lies in the fact that he invented the instrument which others perfected, and that he stimulated others to analyse the confusion of sounds with which his invention presented him.

Section VI. Scepticism of the value of the new method of thoracic investigation was very rife in the early days of its discovery, but nevertheless its position was too strong to be overthrown, and the advantages it offered were too tremendous to be rashly neglected. As a consequence, we find that the progress made was steady, if not phenomenally rapid, and in 1839, Dr. Hope wrote somewhat fully on the varieties of cardiac sounds to be heard. In this year he published the third edition of his work on the diseases of the heart, and it is from this edition that I shall quote to show the advances made since Laennec's death, and to give the position of the pathology of cardiac bruits at that time.

I will first give a few paragraphs from the introduction to the first edition to shew Dr. Hope's views as to the condition in which he found the subject—

"Notwithstanding the brilliant sunshine emanating from the discovery of auscultation by Laennec,—a discovery which, according to M. Bertin, 'has in a few years, more completely illumined the diagnosis of the diseases in question, than all the other modes of exploration had done for two centuries;' the great body of the profession still deny that the piercing ray has reached its destination, still doubt the utility of auscultation in reference to the primary organ of the circulation, still find the ordinary symptons beset with their accustomed difficulties, still complain, in short, that the obscurity which involves the diseases of which we speak is scarcely less profound than ever."

"Errors remain to be corrected, deficiencies to be supplied, inconsistencies to be reconciled; the subject—a confused and incongruous mass, requires to be moulded and compacted into a symmetrical and harmonious whole, the parts of which, while perfect in themselves, shall, by their justness of proportion and unity of design, afford relief and support to each other."

"The most prominent error which reigns throughout the doctrines of Laennec, and which has prevailed in the schools since the first publication of his work, is, that he mistook the nature of the action of the heart."

"Laennec and his predecessors have assigned to diseases of the heart, a certain series of symptoms (and I presume it would be fair to add a certain collection of physical signs) which they conceived to be common to the whole; but they had not analysed those symptoms, and ascertained which were peculiar to, and pathognomonic of, the several affections taken separately."

"He was not aware of a fact first noticed by the writer (Dr. Hope) in June 1825, namely, that murmurs are produced by regurgitation through the valves. This oversight alone naturally shook the confidence of many, and eventually of himself in his theory of valvular murmurs. For, the lesion being found in one valve, when according to that theory, it was expected in another, the inevitable conclusion was, that the theory was incorrect. At the same time, the cause of the murmur remained doubtful."

"Anœmic murmurs Laennec attributed to a wrong cause; hence he was unable to analyse and foresee the circumstances under which nervous and anæmic murmurs should occur, and, consequently to distinguish them from those occasioned by valvular disease."

"Several minor phenomena likewise, as the purring tremor, and the arterial thrill and bellows-murmur, he was, in consequence of the confusion created by the error in question, equally unable to explain."

We may now turn to the body of the work in order, by the aid of liberal quotations, to understand the position in which Dr. Hope left the subject which he so thoroughly investigated.

After devoting a chapter to describe the position of the heart in the thorax, he next enters with great minuteness of detail, into a description of the experiments which he undertook, to determine the nature of the action and sounds of the heart. I do not propose to quote any of these details, but shall merely state the conclusions at which he arrived. They run as follows—

"Of the motions of the Heart."—

"1. The auricles contract so immediately before the ventricles, that the one motion is propagated into the other, almost as if by continuity of action; yet the motion is not so quick that it cannot readily be traced with the eye."

"2. The extent of the auricular contraction is very inconsiderable, probably not amounting to one-third of its volume. Hence the quantity of blood, expelled by it into the ventricle, is much less than its capacity would indicate."

"3. The ventricular contraction is the cause of the impulse against the side."

"4. It is the apex of the heart which strikes the ribs."

"5. The ventricular contraction commences suddenly, but it is prolonged until an instant before the second sound."

"6. The ventricles do not appear ever to empty themselves completely."

"7. The systole is followed by a diastole, which is an instantaneous motion, accompanied with an influx of blood from the auricles, by which the ventricles re-expand, but the apex collapses and retires from the side."

"8. After the diastole, the ventricles remain quiescent, and in a state of apparently natural fulness without distension, until again stimulated by the succeeding auricular contraction."

"Of the Sounds."—

"9. The first sound is caused by the systole of the ventricles."

"10. The second sound is occasioned by the diastole of the ventricles."

"My conclusions respecting the immediate causes of these sounds may be summed up."

"The first sound is compound, consisting 1st of valvular sound; 2nd of the sound of extension—a loud smart sound, produced by the abstract act of sudden, jerking extension of the braced muscular walls; 3rd a prolongation, and possibly an augmentation by bruit musculaire."

"The second sound is produced by the sudden expansion of the semilunar valves, resulting from the recoil upon them of the columns of blood in the aorta and pulmonary artery."

With regard to the conclusions 2 and 6, we now know, or are taught, that they are erroneous, and that both the auricles and ventricles do completely empty themselves in systole. Dr. Hope's explanation of the second sound is still universally

acknowledged to be correct, and if his opinions on the immediate causation of the first sound are somewhat obscure, they, at any rate, are in practical accord with those now urged by Dr. M. Foster, differing in scientific, not in clinical details.

It is fair then to assume that, so far as the normal movements and sounds of the heart are concerned, we have already traced them to a position which is identical in its clinical features, at least, with that which they occupy at the present day.

It now remains to trace the further development of the clear recognition of abnormal sounds. With this object we will quote extracts from the fourth chapter of Dr. Hope's work.

"Section I."—

"Modifications of the motions and sounds by Hypertrophy and Dilatation."

"The first sound is duller and more prolonged than natural, in proportion as the hypertrophy is more considerable; and, when this exists in an extreme degree, the sound becomes nearly extinct, but never, according to my observation, wholly so, as stated by Laennec. The second sound is weaker than natural."

"By Simple Dilatation the first sound becomes loud, brief and clear like the second, while the second is more or less increased."

"The modifications occasioned by Hypertrophy with Dilatation are compounds of those of hypertrophy with those of dilatation."

This quotation if amplified a little, and interspersed with a few more descriptive adjectives, would very accurately express the modern views of the sounds to be heard in hearts hypertrophied and dilated without organic valvular disease, this part of the subject will consequently receive no further mention here.

"Section II."—

"Murmurs produced by Valvular Disease."

"To the murmur of Laennec, I added in the first edition of this work in December 1831, the murmurs from regurgitation, thus assigning to each valve a double murmur—one from the blood flowing in the natural direction; the other from its flowing retrograde when the valve was permanently patescent."

"The circumstances under which I have found murmurs produced in the several valves respectively, are as follows."

"Aortic Valves. 1. Systolic murmur. I have found a murmur attend the ventricular systole in every degree of fibrous, and other disease of the aortic valves sufficient to contract the aperture. The same remark applies to vegetations on the valves or in the orifice. I have even found a considerable murmur produced by mere asperity of the valves without appreciable

contraction. I have have also found murmur created when without contraction or roughness of the valve or orifice, the aorta immediately above the valves was dilated either in its whole circumference, or partially so as to form a pouch."

"2. Diastolic murmur of the aortic valves, I have known to be occasioned by any disease contracting or otherwise deforming one or more of the valves, so as to prevent complete occlusion of the orifice. Finally, I have seen regurgitation, with murmur from mere enlargement of the aortic orifice, whence the valves otherwise sound were incapable of closing it. This murmur, before the discovery of regurgitations, was necessarily and invariably mistaken for a murmur with the second sound from contraction of the mitral orifice."

"Pulmonic Valves. I have never once met with, and ascertained after death such disease of the pulmonic valves as created a systolic murmur during life."

He had heard the bruit due to various causes, but concludes--

"It would appear that the majority of cases of systolic murmur in the pulmonic orifice, are connected with lesions, not of the valves themselves, but, of contiguous parts."

"I believe a diastolic murmur of the pulmonic valves to be exceedingly rare, as I have never met with a case or been able to find one recorded. I created this murmur artificially by perforating one valve."

"Mitral Valve. 1. Systolic murmur; I have met with this murmur from every variety and degree of disease of the mitral valve and chordæ tendineæ, capable of holding the valve permanently open; also from vegetations; also from dilatation of the orifice rendering the valve, otherwise healthy, incapable of closing it."

"2. Diastolic murmur of the mitral valve. Any lesion of the valve capable of sufficiently contracting its aperture, may, with limitations, give rise to this murmur. It was one of the only two murmurs discovered by Laennec."

"Tricuspid Valve. 1. Systolic murmur. I have seen this occasioned by contraction of the valve; and also by dilatation of the ventricle rendering the orifice so large that the otherwise healthy valves were unable to close it."

"2. Diastolic murmur of the tricuspid valve, this is so rare that I am not satisfied that I have ever met with it."

On the actual characters of the bruits, as regards pitch, quality, &c., the following are Dr. Hope's conclusions.

"1. The ventricular systolic currents through contracted orifices, from being stronger than the diastolic, produce louder murmurs."

"2. Considerable contractions of a rough, salient configuration, whether osseous or not, produce the rough murmurs of sawing, filing, or rasping, provided the current be that of the ventricular systole, its diastolic currents being too feeble."

"3. The pitch or key of murmurs is higher in proportion as they are generated nearer the surface, and the currents producing them are stronger;

and *vice versa*. Also the key is lowered by distance, independent of depth, from reverberation through the chest."

"4. Musical murmurs indicate nothing more than ordinary murmurs."

"5. Rough murmurs, and even loud and permanent bellows murmurs indicate organic disease."

"6. Murmurs from regurgitation necessarily indicate organic lesions."

"7. Continous murmurs in the heart will probably be found to indicate—sometimes organic disease attended with regurgitation out of the aorta into the right ventricle or pulmonary aorta; sometimes churning of a little serum between layers of rough lymph on the pericardium; and sometimes, probably, dilatation of the pulmonary artery and compression of the vena innominata."

"Situation in which Murmurs of the respective valves are most audible."

"Authors had not pointed out these situations with any degree of accuracy, previous to the first edition of this work in 1831: nor was it possible for them to do so; for, as they were unacquainted with the regurgitations, they could not know whether a murmur with either sound was seated in an arterial or in an auricular orifice. Dr. Elliotson, indeed, attempted in his Lum. Lect. in 1830 to define the situations in question, as the following quotation shews."

"'If the impediment is in the left ventricle, at the mouth of the aorta, the murmur is loudest at the cartilages of the ribs to the left of the sternum; if in the right ventricle, at the mouth of the pulmonary artery, it is loudest at the sternum and to the right. The sound is often so loud, that it prevents the natural sound of the auricles from being distinctly perceptible, till the ear or stethoscope is removed from the region of the ventricles, higher, to the region of the auricles.'"

"'When the impediment is at either of the auriculo-ventricular openings, the morbid sound is heard at the moment of the auricular contraction, and is generally loudest at the superior part of the cardiac region. It is loudest at the cartilages of the left ribs when the left auriculo-ventricular opening is narrowed; loudest at the sternum and to the right when the narrowing is at the right auriculo-ventricular opening.'"

"Nothing can be more erroneous, contradictory and confused than this account, and there is imperative necessity for more precise and accurate rules on the subject."

"I am now enabled to offer a code of rules of an exceedingly simple and obvious nature."

"Murmurs seated in the semi-lunar valves are best heard immediately over those valves (that is, on the sternum, opposite to the inferior margin of the third rib when the patient is horizontal, and a little lower when he is erect), and thence for about two inches upward, along the diverging courses of the aorta and pulmonary artery respectively. A distinct murmur high up the aorta proceeds from the aortic valves as a pulmonic murmur is only feebly and

indistinctly transmitted in that direction. A distinct murmur high up the pulmonary artery, proceeds from the pulmonic valves as an aortic murmur is only feebly and indistinctly transmitted in that direction. These rules will even apply to semi-lunar regurgitations, notwithstanding, that their murmurs are weaker and not so well transmitted up the vessels in consequence of the current setting out of them into the ventricles."

"Murmurs seated in the auriculo-ventricular valves are best heard at that part of the precordial region, where, from the heart being in contact with the walls of the chest, there is dulness on percussion—in short about the apex. The upper and left side of the dull portion, being nearest to the mitral valve, is the best point for exploring its murmurs, the auscultator has to place his stethoscope about an inch above the spot where the apex impinges."

"The upper and right side of the dull portion being nearest to the tricuspid valve is the best point for exploring the murmurs of this valve, and the point will generally be found on or near the sternum, at the same level as on the opposite side."

"The only source of fallacy is in the case of regurgitation through the semi-lunar valves on either side of the heart; for here the murmur descends down the ventricle with the refluent stream."

"When both the semi-lunar and the auricular valves are diseased, it is perfectly easy to ascertain this by observing, according to the above rules, together with those for the pitch of murmurs, that there are two distinct sources of murmur."

It seems necessary that a few lines should now be given to the history of pericardial bruits, since they are liable sometimes to be confounded with endocardial sounds. Dr. Hope writes on them as follows—

"The honour of giving the first clue to this class of murmurs belongs to Collin, who in 1824 described the sound as the creaking of new leather, but the merit of satisfactorily unravelling the whole subject is, in my opinion, to be awarded to Dr. Stokes, who twice heard attrition murmurs in 1834, and accurately described them."

He then proceeds—

"The murmur is almost always double, accompanying the two sounds of the heart in correspondence with the movements of the organ backwards and forwards within the pericardium. It presents very diversified characters, it has generally more or less of a rough character, sometimes like the rasping of wood, or the grating of a nutmeg; or sometimes like the rustling of silk or even the crackling of parchment."

Space is then devoted to describing ordinary endocardial bruits which afford "presumptive evidence" of pericarditis, a presumption we are now hardly warranted in making. The

following rules are then laid down for differentiating peri- from endo-cardial bruits.

"Listen to murmurs of the sigmoid valves two inches or more up the pulmonary artery, where attrition murmurs are mostly inaudible; listen to murmurs of the auricular valves a little above the apex where they are sure to be loudest, whereas attrition murmurs will be loudest at the point where they are generated. Further attrition murmurs present the following distinctive peculiarities."

"1. They are usually of a much rougher quality of sound than the valvular, so that, when the two co-exist, the one may be heard through the other."

"2. When a murmur with the second sound is rough, as rasping, creaking, croaking, &c., it is almost certainly from attrition."

"3. Attrition murmurs are almost always attended with vibratory tremor, whereas valvular murmurs rarely present this phenomenon."

"4. Attrition murmurs are apt to undergo frequent and sudden changes of character, and of situation, valvular murmurs change little in character, and not at all in situation."

If to these rules we add—

5. Attrition murmurs are usually more superficial than valvular ones.

6. Attrition murmurs can often be altered in character by varying pressures of the stethoscope: valvular bruits remain unalterable by stethoscopic pressure.

The separation is as complete as it can at present be made.

These quotations give ample proof that Dr. Hope was an exceedingly skilful and accurate auscultator, and they incontestably shew that he had brought most of the cardiac physical signs up to their present level.

By experiment and observation he had recognised both aortic bruits, separated them, and pointed out the areas in which they were to be heard; both pulmonary bruits he had similarly elucidated chiefly by experiments; systolic murmurs at both auriculo-ventricular orifices he knew well in time and locality; pericardial rubs he had accurately separated from endocardial bruits; to complete this sketch there remains then only to discuss the direct obstructive bruits at the auriculo ventricular valves, and to mention the elaboration which the two simple bruits at any orifice, have undergone with regard to the exact period of their occurrence.

Section VII. Dr. Hayden, writing in 1875, says—

"At the meeting of the British Medical Association at Oxford in 1868, I read a paper in which I set forth my views on this subject, viz."

The exact period of occurrence of cardiac bruits. He classified them thus.

Presystolic, heard in the long pause.
Systolic, heard with the first sound.
Post-systolic, heard in the short pause.
Prediastolic.
Diastolic, heard with the second sound.
Post-diastolic, heard in the long pause.

"This classification, he says, may seem to involve unnecessary refinement, but if the distinctions be founded in nature, then it is sufficiently warranted, and needs no further justification, quite irrespectively of its imputed value as an aid to differential diagnosis."

He then enters into a somewhat lengthy discussion on the question, what bruit is indicative of mitral constriction? He quotes, in order, Gendrin in 1841, Hope in 1839, Skoda in 1853, Stokes in 1854, Bellingham in 1857, Ormerod in 1864, Blackiston in 1865, Gull in 1866, Tanner in 1869, Niemeyer in 1869, Aitken and Trousseau in 1870, shewing that all of them either did not recognise the bruit at all, or misplaced it in time, and missed its diagnostic significance. Fauvel, he says, in 1843, first really identified the bruit in question, and fully appreciated its significance. Austin Flint recognised it in 1859, and Markham, too, in 1854, but Gairdner it was, in 1861, who gave a full and clear exposition of the subject—the first in this country. Since this time the bruit, with its peculiar character and rhythm, and its indication of mitral narrowing, has become public property, but about the exact point in the cardiac cycle at which it occurs, discussion of the most assertive type still fitfully rages.

A post-systolic bruit indicates a slight reflux current through the mitral valve, at the acme of the systole, when heard at the apex, but when heard at the base it is invariably caused by an aortic-valve lesion of an obstructive character.

Of pre-diastolic bruits, Dr. Hayden had only heard one, and in that case there was aortic incompetency of a considerable degree.

A post-diastolic bruit, he concludes, is pre-eminently basic in location, and affords evidence of slight reflux through the aortic orifice at the acme of arterial reaction.

The last three of Dr. Hayden's refinements, though possibly existing in nature, are now but little, if at all, referred to in general works on medicine, and I shall not further allude to them here. His next point is to fix the locality of bruits arising at the four cardiac orifices respectively, and in this he so closely follows what I have already quoted from Dr. Hope, that I propose to make no further extracts, but to close this portion of my Thesis by stating succinctly the sources from which I have obtained my diagnosis of the bruits utilised in Part II.

1. For all bruits of the right side of the heart, I have accepted the visiting physician's diagnosis only.

2. For all presystolic mitral bruits, I have relied on either the visiting or house-physician's diagnosis.

3. For both aortic and for mitral systolic bruits, I have relied also on the above, or have utilised the notes (and that only in very few cases) in the following way.

a. If a bruit was stated to be systolic in time heard best at the apex and conducted round to the left axilla, I have called it mitral systolic.

b. If a bruit was systolic in time, and heard best over the midline of the sternum, about the level of the third cartilage (or space), or over the second right cartilage, and conducted upwards into the vessels of the neck, I have called it aortic systolic.

c. If a bruit was diastolic in time, heard best as in *b*, but conducted down the sternum, and heard in the epigastric space or at the apex, I have called it aortic diastolic.

d. *b* and *c* combined I have called a double aortic.

PART II.

CONTROVERSIAL.

In the British Medical Journal for February 1887, Sir Andrew Clark published a paper, giving brief details of 684 cases in which cardiac bruits were present without any cardiac symptoms. Relying on these figures, he stated that we ought, to some extent, to modify our views as to the serious nature of valvular lesions, when these were established by abnormal sounds alone.

It was this paper which led me to investigate in some detail all accessible cases of cardiac disease: and with this object in view, I tabulated all the cases I could find in the hospital records, of deaths due either (*a*) directly to cardiac valvular disease, or (*b*) to other diseases with cardiac complications (the latter not necessarily recognised until the autopsy).

The period I selected, as most accessible to investigation, was the eleven years from 1877 to 1887 inclusive: the Registers were first carefully searched for all fatal cases diagnosed as Morbus Cordis, and subsequently the Post-Mortem records of the period were carefully gone through, and every case was included in which valvular lesions were noted. The original notes taken at the bedside were then laid under contribution for the following points:—

1. Age
2. Number of days in Hospital.
3. Presence of Syphilis, Scarlet Fever and Chorea.
4. Presence of Rheumatism.
5. Dates and number of Rheumatic attacks.
6. Bruits present.
7. Family History or alleged cause of fatal illness.
8. Previous History.
9. History of fatal illness.
10. Date of onset of fatal illness.
11. Pulse in last week.
12. Temp. „ „

13. Albuminuria.
14. General appearance.
15. Special immediate cause of death.
16. Results of Autopsy

The cases were then sub-divided into five primary groups.

I. Those without any secondary or independent lesion; or with no P. M., and insufficient evidence of such lesion.

II. Those with evident secondary cardiac lesions; but with no independent lesion.

III. Those with independent lesions.

IV. Those with cirrhotic kidney only as a secondary or independent lesion.

V. A small group of congenital cases and others with some special point of interest.

Each group being further sub-divided into (1) those in whom Rheumatism was known to have occurred: (2) those in whom it had been of doubtful occurrence: (3) those in whom it was known not to have occurred.

The total number of cases was 956, distributed in years as follows:—

Table I.

	M.	F.	Total.
1877	38	33	71
1878	41	32	73
1879	34	32	66
1880	40	30	70
1881	49	24	73
1882	61	36	97
1883	56	49	105
1884	65	49	114
1885	50	35	85
1886	51	45	96
1887	59	47	106
Gross	544	412	956

On the data thus ascertained, I proceeded to consider some "Problems in Cardiac Pathology" which seemed worth elucidation.

Problem I.

In what proportion of deaths are cardiac lesions present, either diagnosed during life or found for the first time at the Post-Mortem.

Whether this proportion would hold good in a general community I am unable to say, but I see no reason for believing that the practice of a large general hospital differs materially in this particular point from that of private individuals.

The total number of admissions on the medical side and the total number of deaths for the period 1877–1887, are shewn in the first two main columns of Table II.: the percentages which my cardiac cases represent are shewn in the last two columns of the same table.

Table II.

	Admissions.			Deaths.			Percentage of Cardiac Deaths to Admission.			Percentage of Cardiac Deaths to deaths.		
	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.
1877	1506	1284	2790	285	155	440	2·52	2·57	2·54	13·33	21·29	16·12
1878	1380	1323	2703	272	161	433	2·97	2·42	2·70	15·07	19·87	16·86
1879	1344	1272	2616	268	157	425	2·53	2·51	2·53	12·69	20·38	15·53
1880	1454	1296	2750	265	156	421	2·75	2·31	2·54	15·09	19·23	16·63
1881	1553	1404	2957	292	164	456	3.15	1·71	2·47	16·78	14·63	16·01
1882	1730	1582	3312	314	211	525	3·53	2·27	2·93	19·43	17·06	18·48
1883	1620	1615	3235	301	231	532	3·46	3·03	3·24	18·60	21·21	19·74
1884	1931	1798	3729	328	215	543	3·36	2·72	3·05	19·81	22·79	20·99
1885	1806	1777	3583	332	213	545	2·77	1.97	2·37	15·06	16·43	15.59
1886	1965	1819	3734	351	242	593	2·59	2·47	2·54	14·53	18·59	16·19
1887	2000	1840	3840	339	265	604	2·95	2·55	2·76	17·40	17·73	17·55
Gross	18289	17010	35299	3347	2170	5517	2·97	2·42	2·71	16·25	18·99	17·33

From the gross totals it will be seen that the percentage of deaths to admissions is M. 18·30. F. 12·76. Gross 15·63.

The figures of the last main column may be taken as representing the relative vulnerability of the Heart, and are certainly somewhat startling. They indicate that out of every 100 people who die, no less than 17 have the valves of the heart defective. The figures for each individual year do not call for any detailed comment, as the cases in hospital are liable to such irregular fluctuations, that it becomes necessary to take a very large total to eliminate accidental variations; I would only call attention to the extreme figures 12·69 for the males in 1879, and 22·79 for the females in 1884.

Taking only the last line, where gross totals are considered, we see that out of every 100 people who die, no less than 17

have the valves of the heart defective. I may draw attention to one rather curious point indicating thus early in our investigations a marked difference between men and women with regard to heart disease. It will be noticed that the percentage of deaths to admissions is no less than 5½ less for women than men, but the percentage of what I may, for our present purpose, call cardiac deaths to total deaths is 2½ higher in women than men. I hope in a later paper to discuss this point more fully, merely remarking here that obviously, for some reason or other, women are more liable, proportionately, than men to valvular disease.

The figures of Table II. and the conclusions to be drawn from them are so profoundly modified by the solution of the next Problem, that it is useless to discuss them further.

Problem II.

In a given number of cases of valvular disease, how many will die directly from that cause?

For the solution of the Problem, I have drawn up in tabular form the numbers previously mentioned. The various headings explain themselves, and their indications can be seen at a glance.

Table III.

GROUP.	Table of Group. M.	Table of Group. F.	Gross.	Total of Subsection. M.	Total of Subsection. F.	Gross.
I. No Rheum, no other lesion	34	23	57			
II. With Rheum ,, ,, ...	55	46	101	113	92	205
III. ? Rheum ,, ,, ...	24	23	47			
IV. No Rheum $\bar{c}$ secondary cardiac lesions	39	27	66			
V. With Rheum ,, ,, ,,	64	60	124	127	103	230
VI. ? Rheum ,, ,, ,,	24	16	40			
VII. No Rheum $\bar{c}$ indep lesion	91	72	163			
VIII. With Rheum ,, ,, ...	85	59	144	290	209	499
IX. ? Rheum ,, ,, ...	90	62	152			
X. Cardiac cases $\bar{c}$ cir. kid.	24	16	40			
XI. Various cases	14	8	22			
Gross Total	544	412	956	544	412	956

In the first six groups, death was most decidedly due to heart disease and to nothing else: the next four groups contain cases in which the valvular disease was at the most only contributory: Group XI. I have here neglected.

A comparison of Table III. with II. will at once shew why I refrained from any detailed comment on Table II. for in III. we find that nearly half the cases have to be omitted to get a genuine cardiac death roll; this very strongly corroborates Sir Andrew Clark's statement which I mentioned at the beginning of my Thesis. When two investigators, working quite independently of one another, arrive by absolutely different paths at the same result, the probability of the truth of this result is very much more than doubled—is is rendered almost a certainty. Now

from the tables which I have constructed out of dead-house facts, might have been deduced as a not unreasonable probability, the very conclusion which Sir Andrew Clark had arrived at from the study of living patients, viz. that cardiac bruits may often be neglected in making a prognosis.

What are the grounds on which I rely? Taken as a whole they are—out of a total of 934 cases, where valvular disease was found, in no less than 499 it was comparatively innocuous, the patient dying of some intercurrent affection. Some little detailed criticism of these numbers will, however, modify them to a slight extent.

The intercurrent affections naturally divide themselves into three classes.

1. Those in which the cardiac disease was a very distinct predisposing factor—*e. g.* acute nephritis, cerebral softening, &c.

2. Those in which the heart trouble rendered recovery much more unlikely—*e. g.* extensive pleuro-pneumonia, cirrhotic liver, &c.

3. Those in which the condition of the heart can hardly be supposed to have had much effect—*e. g.* carcinoma, cerebral tumours, &c. Now the numbers in each of these three classes were as follows—Class I. 21, II. 35, III. 443: consequently we must deduct 56 from our original total: this leaves us 443 out of 934, or a percentage of 47·43—in other words a little less than half of those in whom cardiac bruits exist, will die of something totally unconnected with their heart disease.

I have not given the exact figures of the two sexes separately, but I may again note that there is a difference, distinctly marked, between them, which shews that when once women acquire heart disease, it is more likely to be a factor in the cause of death than in the case of men; if we consider for a moment the usual occupations of the two sexes, this statement will appear the more remarkable, and one requiring some further explanation.

Problem III.

What is the relative frequency of individual cardiac bruits, and of their combinations.

As a matter of mathematical curiosity, the actual number of possible variations in endo-cardial bruits is 255, not one quarter of these are ever heard, and a large number of that quarter are mere clinical curiosities, heard with extreme rarity.

Amongst my cases there were 768 in which bruits were heard, shewing altogether a total of 31 variations in combination; of these 31, seven occurred once only, and twelve more occurred less than ten times—leaving only twelve combinations likely to be met with.

The following Table IV. gives the results in tabular form, arranged in order of frequency.

As experience would, I think, suggest to any auscultator, we find that a single bruit suggestive of mitral incompetency is by far the most frequent abnormality, representing no less than 34.11 per cent. of the total.

The next, rather less than half as frequent, is a combination suggestive of aortic stenosis and incompetency associated with mitral incompetency.

The third in order, about half as frequent as the second, is a combination suggestive of mitral stenosis and incompetency, the other valves working satisfactorily; the relative numbers of the two sexes, in groups two and three, are very curiously complementary, but the contrast is intensified if we take aortic lesions on the one hand and mitral stenosis on the other, for males have as great a preponderance in the one class as females have in the other; the causes of this difference I hope to try to explain in a later paper.

These three groups, together, form nearly 60 per cent. of the total, the remaining groups scarcely call for any detailed comment, except to illustrate the rarity of the combinations.

Dr. Hayden is the only authority with which I am acquainted who has mentioned the matter of frequency of combinations of bruits. I do not know upon what facts his deductions were based, but his results differ very considerably from mine. He

Table IV.

Aortic.	Mitral.	Tric.	Rutm.	M.	F.	Total.	Per Cent.	Dr. Hayden.
...	S.	...	...	131	131	262	34·11	...
D. & S.	S.	...	...	90	37	127	16·53	...
...	P. & S.	...	...	14	53	67	8·72	4
S.	S.	...	...	26	21	47	6·12	3
D.	S.	...	...	32	8	40	5·21	7
D. & S.	...	...	...	29	1	30	3·90	1 & 2
D. & S.	P. & S.	...	...	20	9	29	3·77	...
...	S.	S.	...	11	16	27	3·51	5
...	P.	...	...	8	18	26	3·38	...
D.	...	...	...	18	7	25	3·25	...
D. & S.	S.	S.	...	9	4	13	1·69	...
...	P. & S.	S.	...	4	8	12	1·56	...
S.	P. & S.	...	...	4	5	9	1·17	...
S.	...	...	...	8	1	9	1·17	...
D.	P. & S.	...	...	4	3	7	0·91	...
S.	S.	S.	...	3	2	5	0.65	...
D. & S.	S.	...	S.	4	1	5	0·65	...
D. & S.	P.	...	...	2	2	4	0·52	...
D.	P.	...	...	3	1	4	0·52	...
D.	S.	S.	...	4	...	4	0·52	...
...	P.	S.	...	1	2	3	0·39	...
S.	P. & S.	S.	...	1	1	2	0·26	...
S.	P.	...	...	1	1	2	0·26	...
D. & S.	P.	S.	...	1	...	1	0·13	...
D.	P. & S.	S.	...	1	...	1	0·13	...
D. & S.	P. & S.	S.	...	...	1	1	0·13	...
D. & S.	P. & S.	P. & S.	...	...	...	1	0·13	...
S.	...	...	S.	...	...	1	0·13	8
S.	P. & S.	P. & S.	...	...	...	1	0·13	...
...	P.	P.	...	...	...	1	0·13	6
...	...	S.	...	...	...	1	0·13	...

omits single bruits from his calculations, and then arranges the combinations in the following order.

1. & 2. Aortic stenosis and regurgitation.
3. Aortic stenosis and mitral regurgitation.
4. Mitral stenosis and regurgitation.
5. Mitral regurgitation and tricuspid regurgitation.
6. Mitral and tricuspid stenosis.
7. Aortic and mitral incompetence.
8. Aortic and pulmonary stenosis.

Dr. Hayden's first group comes fifth on my list, his third is also my third, his fourth comes second with me, a result I strongly suspect to have been due to his imperfect acquaintance with the mitral presystolic bruit: in his fifth group we are nearly in accordance, but with regard to the sixth, I cannot but think

that he must have made a serious error, for neither from books nor from my own experience, can I find that tricuspid and mitral stenosis, occurring together and without any other lesion, is any but the rarest of lesions; when I discuss in the next problem the lesions present, it will be seen that from post-mortem evidence, I have only been able to collect five cases, and here in speaking of bruits alone I have only one example to offer. If his observations are accurate, I cannot offer any plausible explanation in the difference of our results. With regard to his seventh group, where the bruits would indicate aortic and mitral incompetency, there is again a serious discrepancy between us, and one which on reference to the next problem assumes even greater proportions; for I find there, that the double lesion in question is absolutely the commonest in cardiac morbid anatomy. I am consequently forced to a conclusion, that Dr. Hayden's auscultatory observations must have been inaccurate. His rarest group I find also exceedingly rare, so rare indeed, that I have not a single example to show from P. M. records, and only one case in which the murmurs would indicate the lesions. As in ordinary practice with the patient before us, the bruits are the chief means that we possess for estimating the exact condition of the valves, I have thought it fair to quote the results of my enquiry in the present point; but, as will appear from the next problem, the figures here given have very little real importance.

It is worthy of notice that while simple mitral bruits are very common, single aortics are comparatively rare. I have been able to collect only 19 cases of a single diastolic, and 7 of a single systolic aortic murmur.

Problem IV.

How far do the bruits heard during life correspond with the appearances found after death.

My data for solution of this problem are 545 cases in which autopsies had been recorded and in which murmers had been heard during life. The precise authority on which I have relied in the designation of the various bruits I have already recorded in the historical sketch which precedes these problems, but I may repeat them here as they are all important for any weight which my deductions may bear :—

(1.) For all bruits of the right side the heart I have relied on the diagnosis of the visiting physicians, and theirs only.

(2.) For all presystolic mitral bruits I have accepted only the same authority as for (1).

(3.) For the more frequent bruits I have relied on either (*a*) the physicians as above, or (*b*) the house-physicians, or (*c*)—and this is not more than 5 per cent of the cases,—the notes, translating the point of maximum intensity, and the direction of conduction in the ordinary methods laid down in text books of medicine.

(4.) Under no circumstances have I relied on the diagnosis of an unqualified clinical clerk alone.

Quite independently of one another I constructed the two following Tables V and VI ; in the first one (V) I took the bruits as the known quantity, and tabulated the post mortem appearances ; in the second, I took the P. M. appearances as known, and tabulated the bruits present ; the two tables were drawn up with some considerable interval of time between them, so that no impressions or prejudices interferred with a rigid regard to written facts. It is evident that the two should exactly agree *mutatis mutandis*, that they do not do so absolutely is strong evidence of *bona fides* in their construction, the discrepancies are so small as not materially to affect my conclusions.

A few words must still be added explanatory of the method employed in comparing the morbid anatomy with the abnormal sounds. I have ommited vegetations as a cause of stenosis, but have of course allowed them as possible causes of aortic and pulmonary systolic bruits, and have noted them as conceivable

Table V.

Bruits Present.				Total of Cases.	A. Inc.	A. Sten.	M. Inc.	M. Sten.	Tr. Inc.	A. Inc. M. Sten	A. Inc. M. Inc.	A. Sten. M. Sten.	A. Sten. M. Inc.	M. Inc. Tr. Inc.	M. Sten. Tr. Inc.	M. Sten. Tr. Sten.	A. Inc. M. Inc. Tr. Inc.	A. Inc M. Sten. Tr. Inc.	A. Inc. M. Sten. Tr. Sten.	A. Sten. M. Sten. Tr. Sten.	A. Inc. M. Inc. Tr. Inc. P. Inc.	A. Inc. M. Sten. Tr. Inc. P. Inc.	M. Sten. A. Inc. P. Inc.	Tr. Sten. P. Inc.	Tr. Sten. P. Inc. M. Sten.	A. Inc. M. Sten. Tr. Sten. P. Inc.	All Valves Comp.
Aortic.	Mitral.	Tric.	Pul-monary.																									
...	S.	...	...	175	11	1	65	20	...	12	40	2	1	6	4	...	5	2	1	2	1	...	1	1	...	...	...
D. & S.	S.	...	...	78	21	1	3	...	...	3	38	2	5	...	...	...	4	...	...	...	...	...	...	...	...	1	...
...	P. & S.	...	...	41	...	...	7	14	...	5	4	...	...	2	3	...	2	2	1	...	...	1	...	...	...	...	...
S.	S.	...	...	34	3	1	4	3	...	1	10	2	5	...	...	...	1	...	...	2	1	...	...	...	..	...	1
D.	S.	...	...	26	8	1	1	...	...	1	10	1	...	...	...	...	2	1	1	...	...	...	...	...	...	...	...
D. & S.	...	...	...	23	15	1	1	...	...	1	3	...	...	...	...	...	2	...	...	...	...	...	...	...	...	...	...
D. & S.	P. & S.	...	...	20	5	1	1	...	...	3	5	2	...	...	...	1	1	...	1	...	...	...	...	...	...	...	...
D.	...	...	...	19	8	...	...	1	...	1	7	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	1
...	P.	...	...	18	...	...	1	13	...	...	...	...	...	...	2	1	...	1	...	...	...	...	...	...	...	...	...
...	S.	S.	...	16	...	...	6	1	1	...	2	1	...	2	...	...	1	2	...	...	...	...	...	...	...	...	...
...	P. & S.	S.	...	9	...	...	...	1	...	1	1	...	...	...	1	1	...	1	...	1	...	...	1	...	1	...	...
D. & S.	S.	S.	...	8	...	2	...	...	...	1	2	...	...	...	...	...	1	...	1	...	...	...	...	...	...	...	1
S.	...	...	...	7	4	1	...	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
D.	P. & S.	...	...	6	...	...	1	...	...	3	1	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...
S.	P. & S.	...	...	4	...	...	...	1	...	2	1	...	...	...	...	...	...	...	...	..	...	...	...	...	...	...	...
S.	S.	S.	...	4	1	...	1	...	...	...	1	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...
D. & S.	S.	...	S.	4	1	...	...	...	...	...	3	...	...	...	...	...	...	...	...	..	...	...	...	...	...	...	...
D. & S.	P.	...	...	3	1	...	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
D.	P.	...	...	3	2	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
D.	S.	S.	...	2	...	...	...	...	...	...	2	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
...	P.	S.	...	2	...	...	...	...	...	...	...	...	...	1	...	1	...	...	...	...	...	...	...	...	...	...	...
S.	P. & S.	S.	...	2	...	...	...	...	...	...	...	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...
S.	P.	...	...	1	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
D.	P. & S.	S.	...	1	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
D. & S.	P. & S.	P. & S.	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...
S.	...	...	S.	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
S.	S. & P.	S. & P.	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
...	P.	P.	...	1	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...
...	...	S.	...	1	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
No bruits heard at all				34	6	...	9	6	3	2	3	...	1	1	...	...	3	...	...	...	...	...	...	...	...	...	...
					88	9	101	61	4	37	136	12	12	13	11	5	23	9	6	7	2	1	2	1	1	1	3

causes of mitral and tricuspid presystolic bruits, but in writing simply the words stenosis and incompetency I have only allowed old constricting lesions in the former and recent vegetations in the latter : of course old stiffened constricted orifices have been allowed to be natural causes of regurgitation, but at the same time these cases must go into a different class from those where the bruits gave the exact condition of the valve.

Taking the bruits in order of numerical frequency, Table V, gives us the following facts—

1. Mitral systolic bruit. Of 175 cases, 65 had mitral incompetency as the sole lesion ; 40 more had the aortic valves also incompetent : in 20, mitral stenosis was the sole lesion : in 11 cases the aortic valve alone was diseased : in 12, the mitral was stenosed, and the aortic valve incompetent : the remainder shewed very various lesions. In considering each group we may conveniently divide the cases into classes, as follows—

Class I, those in which the bruits accurately represented the whole of the lesions present : Class II, those in which the bruits represented a part only of the lesions : Class III, those in which the bruits indicated more than was found post-mortem in the theoretically affected valve, but did not indicate some further lesion : Class IV, those in which the bruits gave a totally wrong impression, the lesions, represented by them being entirely absent. Classified thus, we get the following division of the 175 cases :—

Class I. 65 or 37.1 p.c.
Class II. 97 or 55.4 p.c.
Class III. nil.
Class IV. 13 or 7.4 p.c.

In this group, Class II is allowed to admit all cases of stenosis of the mitral valve, an assumption which is hardly warranted in its entirety : the difference would only swell the numbers in Class IV. A similar allowance has been made in the succeeding groups, so that Class II will always be a little larger than strict facts would allow, and Class IV a little smaller.

2. Double Aortic and Mitral systolic—78 cases. In 43 of these, there were found what we may call the expected lesions, viz. a damaged aortic valve with an incompetent mitral : in 5

more a damaged aortic was found with a stenosed mitral: on the other hand, in 21 the aortics alone shewed any lesion: arranged in classes, we have—

Class I. 43 or 55.1 p.c.
Class II. 9 or 11.5 p.c.
Class III. 25 or 32.0 p.c.
Class IV. 1 or 1.2 p.c.

It is possible that in the 25 cases of Class III, relative incompetency may have existed at the mitral and tricuspid orifices during life, and have been indistinguishable after death.

3. Double Mitral bruit only—41 cases. In 14, mitral stenosis was present alone, that is with other valves healthy: in 7, it was incompetent only: in 5 more the aortics were incompetent also, in tabular form we get—

Class I. 14 or 34 1 p.c.
Class II. 12 or 29.2 p.c.
Class III. 15 or 36.6 p.c.
Class IV. nil.

4. Aortic systolic and Mitral systolic—34 cases. In 5 cases only was the exact lesion found without addition: but in 10 more the method of estimation of incompetency and stenosis would allow the bruits to be present: tabulated we have—

Class I. 5 or 14.7 p.c.
Class II. 17 or 50 p.c.
Class III. 12 or 35.3 p c.
Class IV. nil.

5. Diastolic aortic and mitral systolic—26 cases. In 10 the lesions corresponded accurately with the diagnosis, in the whole of the remainder the bruits indicated only a portion of the damage.

Class I. 10 or 38.4 p c.
Class II. 16 or 61.6 p.c.
Class III. nil.
Class IV. nil.

6. Double aortic—23 cases. In 16, the bruits must be admitted to have correctly diagnosed the lesion: in 6, they

omitted a portion of their duty, and in the other one they quite failed: in table—

Class I. 16 or 69.6 p.c.
Class II. 6 or 26.1 p.c.
Class III. nil.
Class IV. 1 or 4.3 p.c.

7. Double aortic and double mitral—20 cases. In only five can the bruits be said to have given a true insight into the cardiac condition: in five more they came so near to it that we must put 10 into Class I: in one they indicated much more than was present: and in nine they indicated less: we have then—

Class I. 10 or 50 p.c.
Class II. 9 or 45 p.c.
Class III. 1 or 5 p.c.
Class IV. nil.

8. Diastolic aortic—19 cases. In eight the diagnosis was exactly correct, though it is to be noted that I have allowed vegetations in other cases to give rise to systolic bruits, and we should certainly expect some of these eight to have done the same. In eight the bruits did not indicate the full extent of the damage, and in three the indication was absolutely wrong: in classes then—

Class I. 8 or 42.1 p.c.
Class II. 8 or 42.1 p.c.
Class III. nil.
Class IV. 3 or 15.7 p.c.

9. Mitral presystolic—18 cases. In 13 the diagnosis was absolutely correct: in four mitral stenosis was present with other lesions not suggested by the bruits: and in one only was uncompetency the sole fault: in tabular form—

Class I. 13 or 72.2 p.c.
Class II. 4 or 22.2 p.c.
Class III. 1 or 5.5 p.c.
Class IV. nil.

10. Mitral and tricuspid systolic murmurs—16 cases. In only two was the diagnosis absolutely correct. The large figures in Classes II and III are, I suspect, to be accounted for by the

difficulty of estimating incompetency after death which has probably been only relative during life.

Class I. 2 or 12.5 p.c.
Class II. 7 or 43.7 p.c.
Class III. 7 or 43.7 p.c.
Class IV. nil.

11. Double mitral bruit and tricuspid systolic—9 cases. It is not a little curious that in each of these cases the post-mortem revealed a different lesion, in no less than eight of them, however, was mitral stenosis present: arranged in classes they are—

Class I. 1 or 11.1 p.c.
Class II. 7 or 77.7 p.c.
Class III. 1 or 11.1 p.c.
Class IV. nil.

12. All those bruits which were heard less than nine times—52 cases. Amongst these one very interesting autopsy is included in which aortic, mitral, and tricuspid stenosis and incompetency were correctly diagnosed during life by the bruits. Another point worthy of remark is that out of 25 cases where a mitral presystolic bruit had been heard, mitral stenosis was present in 14 of them. On the other hand in four cases a pulmonary systolic bruit had been heard, but in not one of them was a lesion of the pulmonary valves found. In tabular form we have:—

Class I. 9 or 17.3 p.c.
Class II. 17 or 32.7 p.c
Class III. 24 or 46.1 p.c.
Class IV. 2 or 3.9 p.c.

13. No bruits heard at all—34 cases, In 24 of these, only one valve either aortic mitral or tricuspid was diseased; in 6 both the aortic and mitral had distinct lesions: in one the mitral and tricuspid were both found incompetent: and in 3, all three—aortic mitral and tricuspid—were found incompetent. All these cases must obviously go into Class IV.

Having now proceeded in detail through the various bruits, me may sum the totals up when we find in—

Class I. 196 or 36 p.c.
Class II. 209 or 38.3 p.c.
Class III. 86 or 15.8 p.c.
Class IV. 54 or 9.9 p.c.

LESIONS. PRESENT.	Pulm. / Tricuspid / Mitral / Aortic	Total No. of Cases.																																No Bruits heard.
	Pulm.		..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	S.	..	..	..	..	..	S.	..	..	..	..		S.	
	Tricuspid		..	..	..	..	..	..	S.	..	S.	..	S.	..	..	..		..	S.	..	S & P	..	..	S.	..	..	P & S	S.	S.	P.	S.	..		
	Mitral		..	..	..	S	P.	S & P	..	S.	S.	P & S	P & S	P.	P.	S.	S.	P & S	P & S	..	S & P	S.	P & S	S.	P.	S.	P & S	P & S	S.	P.	P.	S.		
	Aortic		D.	S.	D & S	..	..	..	..	D.	D.	D.	D.	D.	S.	S.	S.	S.	S.	S.	S.	D & S	D & S	D & S	D & S	D & S	D & S	..	..	..	..	S.		
A. Inc. M. Inc.		135	7	1	3	42	..	4	..	11	3	1	..	..	1	10	..	..	..	..	..	36	5	2	1	2	..	1	2	..	..	..	3
M. Inc.		99	..	1	1	61	1	7	1	1	..	1	..	..	..	4	1	..	..	..	..	3	1	..	..	..	..	..	7	..	..	..	9
A. Inc.		88	8	4	15	11	..	..	..	8	..	..	..	2	..	3	1	..	..	1	..	18	5	3	1	1	..	..	..	..	..	1	6
M. Sten.		60	1	..	..	20	13	14	..	..	..	..	..	..	..	3	..	1	..	..	..	..	..	..	..	..	..	1	1	..	..	..	6
A. Inc. M. Sten.		39	..	..	1	13	..	5	..	1	..	3	..	..	..	1	1	3	..	.	..	3	3	1	1	..	..	1	..	..	..	..	2
A. Inc. M. Inc. Tr. Inc.		23	..	..	2	4	..	2	1	2	..	..	..	..	..	1	..	..	..	..	..	5	..	1	..	..	..	..	1	1	..	..	3
M. Inc. Tr. Inc.		13	1	..	..	5	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..	1
A. Sten.		13	1	1	2	2	..	..	..	1	..	..		1	..	1	..	..	..	..	..	2	..	1	1	..	..	..	..	..	..	..	..
A. Sten. M. Sten.		12	..	1	..	2	..	..	..	1	..	..	1	..	..	2	..	..	..	..	..	2	2	..	..	..	..	..	1	..	..	..	..
A. Sten. M. Inc.		12	..	..	..	1	..	..	..	..	..	..	..	..	..	4	..	..	..	..	.	5	..	..	..	..	..	..	1	..	..	..	1
A. Inc. M. Sten. Tr. Inc.		11	..	..	..	2	2	2	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	2	..	..	..	..
A. Sten. M. Sten. Tr. Sten.		8	..	..	..	2	..	1	..	..	..	1	..	..	..	2	1	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..
M. Sten. Tr. Inc.		8	..	..	..	4	1	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..
A. Inc. M. Sten. Tr. Sten.		7	..	..	..	1	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	1	1	..	..	..	..	..
M. Sten. Tr. Sten.		5	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	1	..	..	..	..	..		..	1	..	..
Tr. Inc.		3	..	..	..	..	..	..	..	..	..	.	..	..	..	..	..	..	.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3
A. Inc. M. Inc. Tr. Inc. P. Inc.		3	..	..	1	1	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
A. Inc. M. Sten. P. Inc.		2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..
A. Inc. M. Sten. Tr. Sten. P. Inc.		1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..
A. Inc. M. Sten. Tr. Inc. P. Inc.		1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Tr. Sten. P. Inc.		1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
M. Sten. Tr. Sten. P. Inc.		1	..	..	..	..	..	..	..		..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..
		545	18	8	25	173	18	42	2	27	3	6	1	3	1	32	4	4	2	1	..	75	19	9	4	3	1	9	17	1	2	1	34

Interpreting these figures into clinical language they mean—that out of every hundred hearts we listen to, in 36 the bruits will give us exact information: in 10 they will lead us absolutely astray: and in 54 they will give us information either in excess or defect of the truth. This can hardly be regarded as a masterpiece of scientific accuracy, and its lesson is certainly one that I have already hinted at—viz. to rely less on our mechanical aids to accurate diagnosis, and more on observation by visual sense.

We may now proceed to investigate the figures in Table VI, where the post-mortem conditions are taken as fixed points: discussing them seriatim in order of numerical frequency, we have—

Group I. Aortic and mitral valves incompetent, or with vegetations on them—135 cases. That this combined lesion is the commonest, will I think, agree with the experience of anyone who has done much post-mortem work, but at the same time we must call to mind that the most frequent bruit is one that indicates mitral incompetency alone: this contrast would by itself justify the investigations here set forth. In only 11 out of the 135 cases were the exactly corresponding bruits present alone, but in 36 more, they were present with an additional aortic systolic, a condition of things which is easily understood: in 42, a mitral systolic was heard alone, the incompetency of the aortic valves not revealing its presence, and in 10 additional ones, two systolic bruits were present: while in 3 cases no bruits were heard at all. As in Section I of this Problem we may divide the murmurs into four Classes, thus, Class I, bruits heard, those precisely corresponding to the anatomical details: Class II, those suggesting a part only of the lesion: Class III, those suggesting either the whole or part of the lesion, and suggesting also something which was not found: Class IV, those totally irrelevant to the diseased condition. Classified thus we have—

Class I. 57 or 42.2 p.c.
Class II. 53 or 39.3 p.c.
Class III. 22 or 16.3 p.c.
Class IV. 3 or 2.2.

Group II. Mitral Incompetent—99 cases. This lesion is the one which from our present point of view, gave rise to the

most constant results, no less than 61 cases presenting a mitral systolic bruit only: in 26 more, the bruit was present with others: but in 12, the bruit was quite misleading: classified the cases are—

Class I. 61 or 61.6 p.c.
Class II. nil.
Class III. 26 or 26.3 p.c.
Class IV. 12 or 12.1 p.c.

Group III. Aortic valve incompetent, 88 cases. In only 27 did the bruits heard give the exact state of the valves: in 38 more the expected murmur was heard, but others were also present: while in 11 the murmurs indicated a mitral lesion only: we have in classes—

Class I. 27 or 30.7 p.c.
Class II. nil.
Class III. 44 or 50 p.c.
Class IV. 17 or 19.3 p.c.

Group IV. Mitral valve stenosed—60 cases. In 13 cases a presystolic bruit alone was present: in 16 more it was heard with others: in no less than 20 cases, a systolic mitral was the only bruit, and in 6, no bruits were heard at all: we thus get—

Class I. 27 or 45 p.c.
Class II. 20 or 33.3 p.c.
Class III. 6 or 10 p.c.
Class IV. 7 or 11.7 p.c.

Group V. Aortic incompetence with mitral stenosis—39 cases. No less than 14 different combinations of bruits were heard for this combined lesion, the largest individual number being 13, which represented the cases in which a mitral systolic bruit only was heard: the remainder is divided into the other 13 combinations. We have in classes—

Class I. 10 or 25.6 p.c.
Class II. 24 or 61.5 p.c.
Class III. 3 or 7.7 p.c.
Class IV. 2 or 5.1 p.c.

I have here allowed all cases to go into Class I in which a mitral presystolic was heard with an aortic bruit, but it is a curious circumstance that in not one single case was a diastolic

aortic bruit heard with a mitral presystolic, without the presence of a third murmur.

Group VI. Aortic, mitral and tricuspid incompetence—23 cases. Here again no less than 11 combinations of bruits are represented, but the precisely expected one is absent entirely: in one case only was it present with others: in 3 no bruit at all was heard. In classes we may arrange them thus—

Class I. 1 or 4.3 p.c.
Class II. 16 or 69.5 p.c.
Class III. 3 or 12.9 p.c.
Class IV. 3 or 12.9 p.c.

Group VII. Mitral and tricuspid incompetency—13 cases. Two cases gave the exactly corresponding bruits, and in the remainder more or less, false information was given by the associated murmurs: they permit of classification thus—

Class I. 2 or 15·4 p.c.
Class II. 5 or 38·4 p.c.
Class III. 4 or 30.8 p.c.
Class IV. 2 or 15·4 p c.

Group VIII. Aortic stenosis—13 cases. It is not a little remarkable that this lesion is so comparatively rare: but I presume the explanation is in the fact that the contracting aortic valves almost always distort, and render incompetent the neighbouring mitral valve. No less remarkable is the rariety of exact correspondence between the lesion and the bruits heard, for only 3 cases can honestly go in Class I: thus—

Class I. 3 or 23.1 p.c.
Class II. 1 or 7.7 p.c.
Class III. 7 or 53.8 p.c.
Class IV. 2 or 15.4 p.c.

Group IX. Aortic and mitral stenosis—12 cases. To include these, no less than 8 combinations are represented, and only one of them approximates to the true one, viz. 2 cases in which a double aortic and double mitral bruits were heard: classified the figures are—

Class I. 2 or 16.7 p.c.
Class II. 8 or 66.6 p.c.
Class III. 2 or 16.7 p.c.
Class IV. nil.

Group X. Aortic stenosis and Mitral incompetency—12 cases. Only five combinations of bruits occurred with this lesion, but no less than 9 cases gave practically the correct one, we have consequently in

Group I. 9 or 75 p.c.
Group II. 1 or 8.3 p.c.
Group III. 1 or 8.3 p.c.
Group IV. 1 or 8.3 p c.

Group XI. Mitral stenosis with aortic and tricuspid incompetency—11 cases. In these seven combinations of bruits were heard, not one of which represented in its entirety the lesions found. All the cases go into Class II and we have—

Class I. nil.
Class II. 11 or 100 p.c.
Class III. nil.
Class IV. nil.

Group XII. All those lesions which had not ten representatives—40 cases. Amongst them may be mentioned three in which tricuspid incompetence was the only lesion found, no one of which had caused any murmurs. Nine had vegetations on the pulmonary valves, but not one of them had had a pulmonary bruit of any kind to indicate that the valves were injured. They may be divided into classes as follows—

Class I. 2 or 5 p.c.
Class II. 32 or 80 p.c.
Class III. 3 or 7.5 p.c.
Class IV. 3 or 7.5 p.c.

Summing the totals up as we did in Section I, we have—

Class I. 201 or 36.9 p.c.
Class II. 171 or 31.4 p.c.
Class III. 121 or 22.2 p.c.
Class IV. 52 or 9.5 p.c.

Translated into words as before, these figures mean that in 100 cases of heart disease, in 37 we may trust the bruits absolutely: in 31 they will tell us half the truth: in 22 they will tell us more than the truth: in 10 they will lead us completely astray.

For reference the foregoing figures possibly possess some scientific value, but for clinical working purposes we may consider what we have written from another point of view. We may take each valve and consider what clinical evidence we have of its lesions (independently of any associated injury to other valves), translating incompetency by recent lesions (vegetations, ulcers, &c.) or failure under the hydrostatic test, and stenosis by vegetations or old contraction. As a result of this method of investigation, we arrive at the following.

A. If the Mitral valve has vegetations on it or fails under the hydrostatic test, then in no less than 76.5 per cent. of the cases, the lesion will reveal itself by a mitral systolic bruit: in 1·7 per cent there will only be heard a presystolic, this fact most clearly indicates, that for the production of the presystolic bruit an ordinary L. Auricle will suffice without introducing any question of hypertrophy and dilatation, for in calculating incompetency, I was most careful to exclude old lesions of the valve. In 8·5 per cent there will be present a double mitral bruit, *i.e.* systolic and presystolic, still further evidence of the truth of the above statement. Finally, 13.3 per cent. of the cases will give no ante-mortem auscultatory evidence of a diseased mitral.

B. If the mitral valve be stenosed by old lesions, then the clinical evidence of the condition will be much less conclusive, for in no less than 46 per cent of the cases, there will be heard a systolic bruit only, in connection with the valve; this fact alone must make us very cautious in denying the presence of mitral stenosis, and also teaches us to look around for other evidence in cases where sex and age would lead us to expect constriction of the orifice. In 35 per cent. of cases a stenosed mitral is also incompetent as judged by a double bruit, due no doubt to the stiffening of the curtains. In 12 per cent. a presystolic will be heard alone, and in 7 per cent. there will be no murmur heard at the mitral valve suggestive of disease.

C. If the Aortic valve affected by a recent lesion, such as vegetations, or fails to respond to the hydrostatic test, some very curious and startling results appear. In a similar division to that used for mitral bruits we get 38·4 per cent. of the cases without any bruit indicating disease of the aortic valve. In 37·1 per cent

the valve is stenosed as well as incompetent to judge by bruits, or more correctly in this proportion of cases, we shall be unable by bruits alone to distinguish recent vegetations from old constricting lesions. In 15·5 per cent the valve is simply rendered incompetent, the recent lesions not revealing themselves as causes of obstruction: and in 9 per cent. the vegetations while acting as causes of obstruction do not render the valve incompetent at all.

D. If the aortic orifice be constricted by old lesions, then in 33·3 per cent of the cases, it will be incompetent also as judged by the bruits heard: in 26·6 per cent. there will be simple obstruction, the valve being still able to prevent regurgitation: in a precisely equal percentage, we shall have no evidence whatever of the lesion: and in 13·3 the stenosis will, to use a hibernicism reveal itself only by incompetency.

A table shewing these results is as follows:—

Table VII.

For the Mitral Valve.

	Number.	Syst. Bruit.	Presyst.	P. & S.	No Mitral Bruits.
Incomp.	285	218	5	25	37
Stenosis	155	71	19	54	11
Or per cent.		76·5	1·7	8·5	13·3
		46	12	35	7

For the Aortic.

	Number.	Syst. Bruit.	Diastolic.	D. & S.	No Aortic Bruit.
Incomp.	310	28	48	115	119
Stenosed	45	12	6	15	12
Or per cent.		9	15·5	37·1	38·4
		26·6	13·3	33·3	26·6

Using Table V, for a similar clinical deduction it appears that—

A. If a mitral systolic bruit appears to be heard in 64·3 per cent. of such cases, the mitral valve will be affected by a recent lesion of some description, or will fail under the hydrostatic test. In 20 per cent. it will be stenosed by old cicatrising deformities. This statement emphasizes the fact we have already ascertained in connection with stenosis, that nearly half the cases give no presystolic bruit. In 15 per cent. we shall be wrong entirely in attributing the bruit to a defective mitral.

B. If it be thought that a presystolic bruit be heard, then in 71·4 per cent. an old constricting lesion will be found, thus shewing that of all bruits this is the most reliable as a guide to the condition of its supposed originating valve. In 18 per cent. only recent lesions will be found, the question of whether vegetations can cause sufficient obstruction to produce a presystolic bruit is hardly touched by this statement, though it seems certainly to point in the direction of an affirmative answer. In 10·6 per cent. the apparent bruit will be quite delusive, the mitral valve being quite healthy.

C. When both a presystolic and systolic bruit originating at the mitral valve are thought to be heard, the evidence in favour of a mitral lesion is very strong. In 30·6 per cent. of the cases only recent lesions or simple incompetency will be found, and in 61·2 per cent. an old constricting lesion will be found in addition. In only 8·2 per cent. will the mitral valve be found quite healthy.

D. If a bruit be diagnosed as an aortic systolic, the figures shew that the evidence of aortic mischief is by no means conclusive. In 54 per cent. there will be only recent vegetations: in 24 per cent. there will be old cicatrising trouble in addition or by itself: while in no less than 22 per cent. the other valves will be responsible for the murmurs, the aortic semilunars being quite healthy.

E. If we believe that we hear an aortic diastolic bruit, we may with a considerable degree of certainty, assume that the valve is incompetent. In 82·4 per cent. of cases there will be recent lesions, such as vegetations, or the valve will not be watertight, and in an additional 7 per cent. there will also be an old

stenosis. In only 10·5 per cent. will the bruit originate in some other valve.

F. Even where it is believed that a double aortic bruit is heard, the evidence in favour of the valve being diseased, is not yet absolutely conclusive. In 84·7 per cent. (the highest point of correct results) recent vegetations will be found, or valves are not water-tight, in a further 9·5 per cent. an additional old stenosis will be found. In only 5·8 per cent. of cases shall we be wrong in assuming any pathological condition of the aortic valves.

Expressed in tabular form these results appear thus :—

Table VIII.

FOR THE MITRAL.	Recent Lesions.	Old Stenosis.	No Lesion whatever.	No. of Cases.
Systolic Bruit	223	69	55	347
Presystolic Bruit	5	20	3	28
P. and S. ,, ...	26	52	7	85
Or percentages.				
	64·3	20	15·8	
	18	71·4	10·6	
	30·6	61·2	8·2	
FOR THE AORTIC VALVE.				
Systolic	29	13	12	54
Diastolic	47	4	6	57
D. and S.	116	13	8	137
Or percentages.				
	54	24	22	
	82·4	7	10·5	
	84·7	9·5	5·8	

Problem V.

At what age is cardiac valvular disease most rife?

Table IX.

CLASS	Grand Total of Subsection.		Total of Subsection.		Grand Total of Class.	Total of Class.		5 and under.		5–12 including 12.		12–15 including 15.		15–20		20–25.		25–30.		30–35.		35–40.		40–45.		45–50.		50–55.		55–60.		60–65.		65 70.		Over 70.	
	M	F	M	F		M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
I No Rheum and no other Lesion	..	..	..	..	57	34	23	1	..	..	..	..	2	1	2	2	2	2	3	4	5	..	..	4	2	1	2	1	2	1	3	1	2	..	..
II With " " "	205	113	92		101	55	46	..	..	2	3	1	8	14	8	6	5	4	5	1	2	2	2	9	5	3	2	1	1	3	1	1	1	..
III ? " " "	..	..	..		47	24	23	..	..	..	1	4	3	2	1	..	1	3	..	2	6	4	2	4	5	2	2	1	1	..	1	..	..	..	1	..
IV No Rheum but with secondary cardiac lesion	..	..	..	..	66	39	27	..	..	3	..	4	4	2	5	1	..	6	2	4	2	6	6	5	2	4	2	1	1	1	..	..	..	1	..
V With " and " " "	280	127	103		124	64	60	1	..	6	9	5	6	12	9	6	8	7	6	5	10	9	10	18	8	12	10	14	8	3	3	3	2	2	2	..
VI ? " " " "	..	..	..		40	24	16	..	..	2	1	1	6	5	..	..	2	3	3	1	2	..	3	3	4	3	4	4	1	..	1	..	..	2	1	1	..
Totals of those who died of Cardiac Disease ..	435	240	195	195	435	240	195	..	..	13	14	15	29	36	25	15	18	25	20	23	23	33	29	24	23	33	29	26	17	22	15	9	7	5	3	..
" in decennial periods.....	..	..	..	..	..	..	..	12 & undr.		12 & undr.		12–20	..	12–20	54	40	37	..	..	58	39	30–40		40–50												over 70	
VII No Rheum but with Ind. Lesion	..	..	..	..	163	91	72	1	..	1	1	1	..	5	8	4	2	6	7	5	10	9	10	18	8	12	10	14	8	3	3	3	2	2	2	..
VIII ? " and " "	..	..	..	..	144	85	59	..	..	1	1	1	3	9	13	7	7	17	6	6	8	14	9	14	2	6	4	3	4	4	1	..	..	2	1	..
IX ? " " " "	499	290	209		152	90	62	1	..	4	1	1	4	2	5	2	7	11	6	11	4	10	7	15	8	4	7	10	5	9	4	6	2	2	1	2
X Cardiac Cases with Con. Gran. Kid.	..	..	..	..	40	24	16	..	..	..	..	..	..	1	..	1	..	..	1	2	1	..	3	4	3	4	1	4	3	2	..	3	3	2	..	1	1
Totals of those who died of Ind. Lesion	499	290	209	209	499	290	209	2	..	6	3	8	7	17	26	14	16	34	20	24	23	33	29	51	21	26	22	31	20	18	8	17	8	8	4	6	2
" in decennial periods	..	..	..	..	..	..	..	12 & undr		12 & undr.		12–20	..	12–20	33	20	36	..	..	57	52	30–40		40–50												over 70	
XI Cardiac Cases of interest not under above headings	22	14	8	8	22	14	8	..	3	2	..	..	..	4	..	..	..	1	2	1	1	..	1	1	1	..	1	2	..	1	..	1	..	..	..	..	..
Gross Totals.....	956	544	412	412	956	544	412	4	3	21	17	18	36	57	51	29	34	60	41	51	47	65	46	78	38	49	38	42	27	27	13	23	12	10	7	9	2
" in decennial periods	..	..	..	..	..	..	..	12 & undr.		12 & undr.		12–20	..	12–20	87	75	89	..	..	116	93	30–40		40–50												over 70	

Table IX affixed to this Problem gives an analysis of the ages of all the cases I have collected. My reason for sub-dividing the ages under twenty in the manner in the Table is that 12 is taken, I believe, as the limit of age in children's hospitals, and I wished my facts to be comparable with those derived from other souces, of which children's hospitals would, I think, be most interesting.

In the next problem I have endeavoured to deal with some questions concerning the average age at death, and will here consequently merely draw attention to a few interesting points in regard to the absolute numbers at various ages.

In the classes of which the individuals succumbed directly, the 5 years from 15–20, contain absolutely the largest number of cases; this fact is at first sight in startling contrast with the results we had arrived at in Problem II, but it has to be remembered that a large proportion of the cases here noted would probably be such that the patient progressed steadily from the beginning downwards, allowing no time for a period of complete compensation, and scarcely therefore giving rise to any real question of prognostication that had not an only too obvious answer at hand. From 20, the numbers again increase up to 35 years of age, remain nearly steady to 40, decline gradually to 50, and then occurs a very great drop; from 50 onwards the decline is more gradual.

Of those who died of some intercurrent affection but little needs to be said. Under 20 the numbers are small compared with those just mentioned. After 20 the numbers steadily increase up to 50; the greatest point of distinction between this group and the previous one lies in the gradual decline after 50, instead of a sudden drop over that age: this is precisely what we should expect from Problem II, and another strong confirmation of the deductions made there, lies in the fact that we find 8 patients in this group attaining the full age of threescore years and ten, against the 3 only in the cardiac group.

Class XI will not be further alluded to in this connection.

Children.—When heart disease occurs in a child, it is evidently a very serious matter, with a most unfavourable prognosis,

for of my cases no less than 32 out of 45, or 71 per cent., died directly from the valvular trouble, and two more in Class XI died of phthisis, probably induced by congenital pulmonary stenosis. It is also worthy of remark that 21 out of 29 who died of the heart disease had some secondary lesion, shewing how very prone children are to such occurrences.

From 12–20 the prognosis is also but little better than in children, for (omitting the 4 cases in Class XI in whom no valvular trouble was found) out of 158 cases, 105, or 66.45, died directly through the heart.

After 20 the prognosis becomes decidedly better, in the first two decades about the same percentage (viz. 47) died of the cardiac disease, whereas in the third decade (viz. from 40–50) only 40 per cent. so died, and in the succeeding decades the percentages are 27.3, 27.4, 27.2, respectively.

Comparing the sexes in one or two particulars, some curious facts become apparent. Under the age of 20 there are absolutely a larger number of females die than males: I thought this might possibly be due to the fact that chorea and rheumatism are commoner in females, but, as far as my limited numbers go, on comparing the males and females in Classes I, IV and VII with those in II, VI and VIII, I find that this is not the explanation of my figures at any rate, for I get in the non-rheumatic cases 18 males to 22 females, and in the rheumatic ones, 52 males to 60 females, and to have the same relative proportion in the rheumatic cases, there should be 64 females. My impression is that the explanation lies in the fact that, from birth to death, women have a greater tendency to die *directly* of heart disease than men. That this is so, is born out by the figures in almost every individual decade, except that between 30 and 40, the actual proportions over the whole table being as follows.

	Under 20.		20–30		30–40		40–50		50–60		Over 60		Total all ages.	
	M	F	M.	F.	M	F	M.	F.	M	F	M	F	M.	F
	70.2	65.4	45.4	50.4	50.4	43	38.4	42.6	25.8	30	24.4	33.3	45.3	48.3
Difference against women	— 4.8		+ 5		— 7.4		+ 4.2		+ 4.2		+ 8.9		+ 3	

Problem VI.

What is the average age to which patients affected with cardiac valvular disease attain?

To make this problem of much scientific value, we want of course a very much larger supply of particulars, we want the Registrar General's returns of the average duration of life of the whole population, and we want also returns for other diseases that we may correctly estimate the heart's influence on the death rate of the community. Such, however, as the materials are, they afford one or two interesting points of comment, by comparison amongst themselves.

The completed Table shews—

Table X.

	NUMBER.			AVERAGE AGE.			AVERAGE AGE OF SUBSECTION.		
CLASS	M	F	Total	M	F	Together	M	F	Together
I	34	23	57	40.56	38.61	39.77			
II	55	46	101	33.55	28.09	31.06	35.58	32.49	34.19
III	24	23	47	34.69	35.13	34.89			
IV	39	27	66	33.62	32.33	33.09			
V	64	60	124	27.77	26.45	27.13	30.32	27.83	29.20
VI	24	16	40	31.79	25.38	29.23			
VII	91	72	163	43.42	39.81	41.82			
VIII	85	59	144	35.87	31.32	34.01			
IX	90	62	152	41.77	37.55	40.05	41.21	37.44	39.62
X	24	16	40	49.71	48.63	49.28			
XI	14	8	22	...	...	...	...	...	...
Gross	544	412	956	...	...	...	...	...	...

I may be allowed to recapitulate the meaning of the classes; the first six include those cases where death was actually due to valvular disease of the heart, I and IV non-rheumatic in origin, II and V certainly rheumatic, III and VI rheumatic factor unknown; classses VII to X include all the cases where some independent lesion was found, sufficient in itself to cause death,

in VII rheumatism had certainly not caused the valvular lesion, in VIII it certainly had done so, and in IX the point was doubtful: Class X includes all cases, rheumatic or not, in which cirrhotic kidney was present as the sole independent lesion; class XI contains some interesting cases which are not here considered.

The first point to which I may draw attention, is the fact that, whether we compare them in Classes, or in Subsections, women always die at a younger age than men. When I first realized this, I was most extremely astonished, for I had always held that the laborious lives to which men were exposed, more commonly than women, must inevitably lead to a more rapid onset of fatal symptoms; and I expected to find that this would reveal itself by a smaller average age at death. It may be objected that I have not shewn the *duration* of the disease, this is true, but it holds for both sexes, and its influence would certainly tend to be extinguished by large numbers. The difference is roughly about 3 years in each subsection, this, with our present table before me, I should expect would be increased if all classes of the community were included, for it must be remembered that in the class of hospital patients, the work of the women is far harder, physically, than it is among the upper classes, and thus tends much more to eliminate sex differences. It would be most interesting, and possibly of no inconsiderable importance, to compare my figures given above with those of a similar collection taken from the middle and upper classes of society, amongst whom the physical exertion of the women is certainly much less than that of the men, and of lower class women. I say of importance, for if the hard work of the labouring class prolongs their lives beyond that of the women, it behoves us to be cautious how far we condemn our patients to lives of indolence, or sedentary occupation when they come to us with cardiac lesions, asking our guidance in the manner of their lives. The extremes of difference are met with in Classes II, VI, VIII and XI, pointing to a rheumatic origin as having some influence in determining the sex ratio. Whether the ages at death of men and women throughout the world accounts for the above differences, I do not know, but as we are taking one disease only, and

comparing the sexes under equal conditions, I presume that we must look for something in the course of the disease itself to account for the fact under consideration.

Another point of interest to note is, that if secondary cardiac lesions occur—such I mean as infarcts in various organs—death takes place usually at a much earlier age than where such lesions cannot be proved to exist. This gives us a slight hint in forming a prognosis in certain cases, thus, if a pulmonary, or renal infarct had once occurred in a patient, the ultimate prognosis would be so much the worse, independently of whether the occurrence was of immediate gloomy significance. It might be objected that the younger age of the secondary-lesion-class is accounted for by the effects of these lesions, in directly producing death, but infarcts are, I think, very rarely fatal in themselves, except occasionally, when they take place in the lung and set up a fatal consolidation: and even if they were fatal, this is only begging the question, for it still has to be shewn why they occur on an average at a comparatively early age.

Again, it is to be noted that the average age of those who died of some independent malady is greater by some years than that of the other two subsections. This indeed is only what might have been expected *a priori*, as such patients have battled with their valvular trouble, and, so to speak, overcome it, succumbing at last to a "complication of disorders"; moreover, in a fair proportion of this class, the cardiac trouble was probably only developed in the last few days or weeks of the fatal illness in, which case the age expected is merely that of the concomitant disease, a statement well illustrated by taking the figures of Class X, for 48 to 49 is about the average age at which chronic interstitial nephritis proves fatal.

Lastly, I may draw attention to the fact that the average age of the rheumatic classes is always some years less than that of the distinctly non-rheumatic. This agrees very well with the well recognised clinical experience, that the younger the patient with acute rheumatism the more likely is his heart to suffer: and that atheroma of the valves, as a primary affection, is rarely found before middle age.

Problem VII.

What cardiac lesion gives the worst prospect of prolonged life.

This is of course a very different question to what is the immediate prognosis—as determined by the lesion—in any given case of heart disease; with this latter we have nothing here to do, it is a pure clinical question, only to be answered by the materials which are present at the bedside—by the symptoms, and their severity and duration, by the appearance and surroundings of the patient, &c. The figures and tables that follow are only of use in that class of cases in which we are asked our opinion as to occupation, marriage, &c., when the interested person is the possessor of a cardiac valvular lesion still latent, as it were, or fully compensated.

Table XI.

LESIONS.	No. in Classes I–VI.	MALES.		Average Age.	Gross Total.	Average Age.
		Average Age.	No. in Classes VII–X.			
M Inc.	16	28	41	33.95	57	32.28
M Sten.	6	27.16	19	41.74	25	38.24
Total $\bar{c}$ mitral lesion only.	22	27.77	60	36.42	82	34.09
A Inc.	39	38.28	49	47.26	88	43.27
A Sten.	5	39.2	8	39.12	13	39.15
Total $\bar{c}$ aortic lesion only ...	44	38.39	57	46.10	102	42.74
A Inc M Inc.	41	33.27	69	40.69	110	37.93
A Inc. M Sten.	8	25	20	38.65	28	34.75
A Sten. M Sten.	7	16.86	4	34.25	11	23.18
A Sten. M Inc.	4	48.25	5	49.6	9	44
Total c aortic & mitral lesions	60	31.25	98	40.47	158	36.97
M Sten. Tr. Inc.	1	31	...	...	1	31
M Sten. Tr. Sten.	1	17	1	24	2	20.5
M Inc. Tr. Inc.	1	46	8	57.12	9	50.55
M Inc. Tr. Sten.	...	...	...	...	...	...
Total $\bar{c}$ mitral and tricuspid lesions	3	31.33	9	48.11	12	43.92
A Inc. M Inc. Tr. Inc.	6	30.83	9	47.22	15	40.66
A Inc. M Sten. Tr. Inc.	3	17.33	1	72	4	31
A Inc. M Sten. Tr. Sten.	...	...	...	...	...	...
A Sten. M Sten. Tr. Sten.	1	16	...	...	1	16
A Sten. M Inc. Tr. Inc.	1	14	...	...	1	14
Total $\bar{c}$ aortic mitral and tricuspid lesions	11	24.27	10	49.7	21	36.38

375 out of 382.

Problem IV proved pretty conclusively that, for accurate data on the condition of the valves, the bruits heard during life are not sufficient: I have consequently discarded all those cases where the condition was not accurately ascertained after death, leaving myself 665 autopsies from which to draw conclusions.

Tables Nos. XI, XII, XIII, contain these 665 cases. No. XI giving the males arranged according to the valve affected, proceeding from the simplest lesions to the most complicated; No. XII the females in similar arrangement; and No. XIII the totals irrespective of sex. From these complete Tables, I have

Table XII.

LESION.	No. in Classes I–VI.	FEMALES. Average Age.	FEMALES. No. in Classes VII–X.	Average Age.	Total Gross.	Age.
M Inc.	16	24.56	48	36.08	64	33.2
M Sten.	21	32.09	28	41.68	49	37.57
Total $\bar{c}$ mitral lesion only ...	37	28.84	76	39.46	113	35.1
A Inc.	6	30.5	17	42.53	23	39.39
A Sten.	...	...	1	33	1	33
Total $\bar{c}$ aortic lesion only ...	6	30.5	18	42	24	39.13
A Inc. M Inc.	20	25.95	33	35.36	53	31.81
A Inc. M Sten.	6	14.33	11	31.91	17	25.71
A Sten. M Sten.	2	24.5	...	...	2	24.5
A Sten. M Inc.	3	17.33	1	55	4	26.75
Total $\bar{c}$ aortic and mitral lesions	31	22.77	45	34.96	76	29.99
M Sten. Tr. Inc.	3	22	4	31.5	7	27.43
M Sten. Tr. Sten. ...	2	30.5	1	26	3	29
M Inc. Tr. Inc.	3	28.33	3	48	6	38.16
M Inc. Tr. Sten. ...	...	...	...	...	...	...
Total $\bar{c}$ mitral and tricuspid lesions only	8	26.5	8	37	16	31.75
A Inc. M Inc. Tr. Inc.	4	15	12	41.5	16	34.88
A Inc. M Sten. Tr. Inc.	3	42	7	34.14	10	36.5
A Inc. M Sten. Tr. Sten.	3	23	5	37.6	8	32.13
A Sten. M Sten. Tr. Sten.	4	30	4	34	8	32
A Sten. M Inc. Tr. Inc.	...	...	...	...	...	...
Total $\bar{c}$ aortic mitral and tricuspid lesions	14	26.79	28	37.89	42	34.19

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deduced the three succeeding ones, No. XIV, in which the 15 commonest lesions are represented in order of the average age at death; *A* gives the results in the case of those who distinctly died of heart disease, *B* of those in whom it was only a contributory factor (often a very small one) in the fatal issue, and *C* of both classes taken together: in these three latter tables no account is taken of sex. We will first pay a little attention to the information which Table XIV affords, turning to the others for additional suggestions or for corroboration of deductions.

Table XIII.

LESION.	MALES AND FEMALES TOGETHER.					
	No. in Classes I–VI.	Average Age.	No. in Classes VII-X.	Average Age.	Gross Total.	Average Age.
M Inc.	32	26.28	89	35.10	121	32.77
M Sten.	27	31	47	41.70	74	37.80
Total $\bar{c}$ mitral lesion only	59	28.44	136	37.38	195	34.68
A Inc.	45	37.24	66	46.03	111	42.47
A Sten.....	5	39.2	9	38.44	14	38.71
Total $\bar{c}$ aortic lesion only ...	50	37.44	75	45.12	125	42.04
A Inc. M Inc.....	61	30.87	102	38.97	163	35.94
A Inc. M Sten.	14	20.43	31	36.26	45	31.33
A Sten. M Sten.	9	18.55	4	34.25	13	23.38
A Sten. M Inc.	7	35	6	50.5	13	42.15
Total $\bar{c}$ aortic and mitral lesions	91	29.35	143	38.73	234	34.70
M Sten. Tr. Inc.	4	24.25	4	31.5	8	27.87
M Sten. Tr. Sten. ...	3	26	2	25	5	25.6
M Inc. Tr. Inc.	4	32.75	11	50.27	15	45.6
M Inc. Tr. Sten. ...	...	...	...	...	...	...
Total $\bar{c}$ mitral and tricuspid lesions	11	27.82	17	42.88	28	36.96
A Inc. M Inc. Tr. Inc.	10	49	21	43.95	31	37.68
A Inc. M Sten. Tr. Inc.	6	26.66	8	38.87	14	34.93
A Inc. M Sten. Tr. Sten.	3	23	5	37.6	8	32.13
A Sten. M Sten. Tr. Sten.	5	27.2	4	34	9	30.22
A Sten. M Inc. Tr. Inc.	1	14	...	...	1	14
Total $\bar{c}$ aortic mitral and tricuspid lesions	25	25.68	38	41	63	34.92

Table XIV.

A CLASSES I–VI.					B VII–XI.					C ALL CLASSES.				
A	M	Tr.	No.	Age.	A	M	Tr.	No.	Age.	A	M	Tr,	No.	Age.
Sten.	Sten.	.....	9	18.55	.....	Sten.	Sten.	2	25	Sten.	Sten.	.....	13	23.38
Inc.	Sten.	.....	14	20.43	.....	Sten.	Inc.	4	31.5	.....	Sten.	Sten.	5	25.6
Inc.	Sten.	Sten.	3	23	Sten.	Sten.	Sten.	4	34	.....	Sten.	Inc.	8	27.87
.....	Sten.	Inc.	4	24.25	Sten.	Sten.	.....	4	34.25	Sten.	Sten.	Sten.	9	30.22
.....	Sten.	Sten.	3	26	.....	Inc.	.....	89	35.10	Inc.	Sten.	.....	45	31.33
.....	Inc.	.....	32	26.28	Inc.	Sten.	.....	31	36.26	Inc.	Sten.	Sten.	8	32.13
Sten.	Sten.	Sten.	5	27.2	Inc.	Sten.	Sten.	5	37.6	.....	Inc.	.....	121	32.77
Inc.	Sten.	Inc.	6	29.66	Sten.	.....	.....	9	38.44	Inc.	Sten.	Inc.	14	34.93
Inc.	Inc.	.....	61	30.87	Inc.	Sten.	Inc.	8	38.87	Inc.	Inc.	.....	163	35.94
.....	Sten.	.....	27	31	Inc.	Inc.	.....	102	38.97	Inc.	Inc.	Inc.	31	37.68
.....	Inc.	Inc.	4	32.75	.....	Sten.	.....	47	41.7	.....	Sten.	.....	74	37.8
Sten.	Inc.	... ..	7	35	Inc.	Inc.	Inc.	21	43.95	Sten.	.....	.....	14	38.71
Inc.	.....	.....	45	37.24	Inc.	.....	.....	66	46.03	Sten.	Inc.	.....	13	42.15
Sten.	.....	.....	5	39.2	.....	Inc.	Inc.	11	50.27	Inc.	.....	.....	111	42.47
Inc.	Inc.	Inc.	10	49	Sten.	Inc.	.....	6	50.5	.....	Inc.	Inc.	15	45.6
			235					409					644	

When I had completed my figures in this problem, I turned to several authorities to see how far I could corroborate or differ from their opinions; I was met by an astonishing want of agreement on the point under discussion. Drs. Peacock, Bristow and Wilks (in Fagge's medicine) give the relative danger to life of the simple lesions as—Aortic Stenosis least dangerous, and then in order, Mitral Stenosis and Mitral Incompetency, with Aortic Incompetency as most dangerous. Strümpell, on the other hand, almost inverts this order, and asserts that Aortic Incompetency is the least dangerous, Mitral Incompetency next, then Aortic Stenosis, and most dangerous of all, Mitral Stenosis. Dr. Byrom Bramwell takes an intermediate position between the two, and would thus arrange them—Aortic Stenosis least dangerous, then Aortic Incompetency; but he declines to decide between Mitral Incompetency and Stenosis. On this particular point my tables shew:—

From CLASSES I–VI.			*From* CLASSES VII–X			*From* ALL CLASSES.		
No.	Lesion.	Average Age at Death.	No.	Lesion.	Age.	No.	Lesion.	Age.
32	M Inc.	26.28	89	M Inc.	35.10	121	M Inc.	32.77
27	M Sten.	31	9	A Sten.	38.44	74	M Sten.	37.80
45	A Inc.	37.24	47	M Sten.	41 70	14	A Sten.	38.71
5	A Sten.	39.2	66	A Inc.	46.03	111	A Inc.	42.47

It will be seen that my figures do not agree exactly with any of the views mentioned, but those taken from all classes agree with Strümpell's in two important particulars; they make Aortic Incompetency give the longest life, and Mitral Stenosis a shorter one than Aortic Stenosis. They also agree with Byrom Bramwell in making Aortic lesions of both kinds less serious than any Mitral one, and decide for him that Mitral Incompetency is more serious than Mitral Stenosis.

In discussing these figures from the point of view of a direct answer to our original Problem, a very obvious and ready criticism occurs. It is a well known fact, that what we may call primary atheroma of the aorta and its valves is a lesion far more frequent after 40 than before, and consequently, that what my figures shew is simply this fact expressed in a different manner. I felt this

difficulty so strongly, that I took the trouble to tabulate the cases I had in the two distinct classes—viz. the rheumatic and the non-rheumatic—in order to see whether my conclusions given above would stand this test: I assumed that in the rheumatic class the aortic lesion was *per se* as likely to occur at an early age as a mitral one; and in the non-rheumatic cases, I assumed that a mitral lesion was *per se* as likely to occur late as an aortic one. Classified in this way, and arranged as before, the figures are:—

NON-RHEUMATIC.			RHEUMATIC.		
No.	Lesion.	Age.	No.	Lesion.	Age.
38	M Inc.	36.39	46	M Inc.	26 48
8	A Sten.	40.62	4	A Sten.	32
21	M Sten.	41.62	27	M Sten.	32.66
46	A Inc.	42.13	23	A Inc.	38.26

It is true that the numbers here are almost too small to draw very general conclusions, especially as regards absolute ages, but such as they are, they agree absolutely with my others, shewing Mitral Incompetency as the lesion with the shortest expectation of life, and Aortic Incompetency with the longest.

Thus, however much it may conflict with preconceived ideas, facts seem to prove conclusively:—

(*i.*) That Mitral Incompetency gives, amongst single lesions, the worst prospect of prolonged life.

(*ii.*) That Aortic Incompetency gives the best prospect.

(*iii.*) That the question between Aortic Stenosis and Mitral Stenosis depends on other factors to such an extent that the isolated lesion sinks into a position of doubtful value for prognosis.

Dr. Bristow further remarks that, speaking generally, Stenosis in a simple form should, in any case, give a better prognosis than Incompetency, alleging as a reason for the statement, the power of hypertrophy possessed by the cavity whose exit aperture is stenosed. As regards the mitral orifice, his view is borne out by my tables, but not so as regards the aortic

orifice. My supplementary Tables No. XIV., A, B, C, allow themselves to be divided very simply into three groups, each containing 5 lesions. Group (*i*) contains cases of mixed stenosis and incompetency, group (*ii*) pure stenosis, group (*iii*) pure incompetency, and in the matter of age they arrange themselves thus

88	Cases of	mixed Sten. and Inc.	give an average	at death of	33.28
115		pure Sten.	,,	,, ,,	35·17
441		pure Inc.	,,	,, ,,	37·17

A result contrary to expectation, and to Dr. Bristow's statement, and one which proves, beyond doubt, that the particular form of lesion is but one, and probably only a small factor, in determining whether compensation will or will not take place to a satisfactory extent.

Having considered the relative importance of the simple lesions, we may now proceed to discuss the more complicated ones. It is usually asserted, in text books of physiology, that the valves of the heart are so constructed, that, under certain circumstances, they may allow blood to regurgitate through them, in a direction the reverse of the normal; this constitutes what is known as their safety valve action. Have we any evidence from disease, that such an action can he brought into play, and that it can prolong life. For calculating absolute ages, my numbers are far too small, but evidence of a certain weight is forthcoming to shew that such action can take place, and that life is prolonged by it. The evidence that I can bring is not very conclusive, and refers only in reality to chronic cases of endocarditis: whether there is any proof of such action on the part of the valves, in cases of sudden emergency in an otherwise healthy heart, I am unable to say, but I should imagine that it was very unlikely to occur.

On referring to Table XIV., Div. B., it is seen that—

	5	Cases of Aortic Sten.	gave an average of 39.2 years.
while	7	,, A. Sten. and M Inc.....	35 ,,

This apparently goes against such safety valve action, but it has to be remembered that these cases come from those in which the heart killed, and were therefore presumably acuter in their nature, and it is fair to assume that the incompetence of the Mitral valve

occurred in a ventricle that was rapidly dilating. Taking the figures from the next table, where the cardiac trouble was in general more insidious, and presumably slower in progress with better managed compensation, we find a very different result, for—

9 Cases of A. Sten. gave an average age of 38.44
while 6 ,, A. Sten. and M. Inc... ..... 50.5

a difference of a little over 12 years in favour of those with mitral regurgitation. This is certainly a very noteworthy piece of evidence; but again the numbers are very small, and consequently the absolute ages proportionately unreliable. Table of gross results shew us:—

14 Cases of A. Sten. with an average age of 39.71
and 13 ,, A. Sten., M. Inc. 42.15

a balance in favour of the latter of about $3\frac{1}{2}$ years, which is probably not far from the truth.

With regard to Mitral Stenosis associated with Pulmonary Incompetence I have no data to bring forward, nor yet with regard to Pulmonary Stenosis and Tricuspid Incompetency; but looked at in the light of the next paragraph, it is probable that no benefit can be derived from the association.

Tricuspid Incompetency, as we might expect, rarely seems to be of advantage, but must always be taken as of very unfavourable omen, thus comparing simple Mitral Stenosis with the same associated with Tricuspid Incompetency, in all three tables (XIV, A, B and C) there is a serious curtailment of life—$6\frac{3}{4}$, 10.2 and 10 years respectively. On the other hand, if we take an incompetent aortic and stenosed mitral valve, we gain 3 years by combining with them an incompetent tricuspid, and if both aortic and mitral valves be incompetent, nearly two years are gained by allowing an overflow through the tricuspid orifice. This evidence in favour of tricuspid safety action is, however, so much discounted as to become practically of no value, when we reflect on the absence of proof of the duration of the tricuspid lesion, and we shall probably act more wisely if we decline altogether to listen to such an argument.

Besides safety valve action, there appear to be certain regulative mechanisms at work in valvular disease which have some power in the direction of prolonging life. Thus in Table

XIV. C, we see that stenosis of the aortic and mitral orifices is a very fatal lesion, but if the tricuspid be also stenosed, the patient gains on an average about 7 years of life; a difference that points strongly to the lungs as the weak point in circulatory disturbances, for the additional tricuspid stenosis can only spare the lungs from over-severe congestion.

We may now briefly pass in review the double lesions in order of severity.

(*i.*) A. Sten., M. Sten. This appears to be the most formidable valvular disease, carrying off its victims at an age between 23 and 24 as a rule. There is a remarkable difference of nearly 16 years, when it is the main and when only a contributory cause of death: the difference I presume depends on the rapidity with which contraction takes place; if slowly advancing it would be comparatively innocuous, the larger proportion of cases in Table XIV. A, tends to prove that it usually advances rapidly and kills quickly.

(*ii.*) M. Sten., Tr. Sten. This appears also to be a very severe lesion, killing usually between the ages of 25 and 26, the numbers are very small.

(*iii.*) M. Sten., Tr. Inc. The slight advantage that this lesion possesses over (*ii*) lies undoubtedly in the fact that the right ventricle is here in a better position for sparing the lungs.

(*iv.*) A. Inc., M. Sten. With moderately large numbers we get here again a curious difference between the age in Tables XIV. A. and XIV. B.; it would appear that in moderately acute cases we can best help an incompetent aortic valve by allowing a large volume of blood to be sent through it, plenty being cut to waste so to speak; but if the condition be more slowly developed, then the amount returned through the valve would appear to be less, and so compensation is brought about.

(*v.*) A. Inc., M. Inc. The relative position of this lesion is apparantly the same, whether occurring as the main or subsidiary cause of death, the absolute ages naturally differ considerably. We have already seen that aortic incompetency is the best possible lesion, if other disturbance must occur, it would appear that an additional incompetent mitral is the best possible interference, though this robs the victim of seven years of probable life.

(*vi.*) A. Sten., M. Inc. This we have already sufficiently discussed in connection with safety valve action.

(*vii.*) M. Inc., Tr. Inc. The position of this double lesion, as the absolutely best which it is possible to have, is so anomalous compared with mitral incompetency alone as the worst single lesion, and its relative position varies so much in tables XIV. A. and XIV. B., that I had rather reinvestigate its data than venture to make any dogmatic statement about it.

Lesions in which three valves are involved. Chronic pulmonary lesions are so rare, that I have been unable to obtain sufficient data for including them under this heading. Table XIV. appears to shew that when three valves are affected, the worst balance of circulation is obtained by having them all stenosed: it is better to have an incompetent aortic with a stenosed mitral and tricuspid: better still to have the tricuspid also incompetent and the mitral alone stenosed: but, curiously, it appears to be best to have all three merely incompetent. This result again seems broadly to point to the lungs as the most important organs to preserve from over congestion, but I suppose so much would depend on the relative severity of the three lesions, and also on their respective duration, that no safe conclusions can be drawn. It is certainly worth noting that no less than three out of the four commoner single lesions give a better prognosis than any triple one, and consequently as soon as more than one valve becomes affected, the prognosis *ipso facto* becomes more unfavourable.

As far as the rarer lesions and combinations are concerned, I have very little evidence to give bearing on our Problem.

I have 11 cases in which the pulmonary valve was affected. In six of these, the valve was fringed with small vegetations only, evidently formed during the last illness; these cases must be obviously excluded in all questions dealing with age at death. The remaining five I will give at length that they may be recorded, but I make no deductions from them.

No.	Sex.	Age.	Condition of Valves.
1.	M.	11.	A Inc and $\bar{c}$ rec. veg. P. Inc. from old thick.
2.	M.	18.	A Sten, M Sten, Tr. Sten, P Sten.
3.	M.	16.	A thick & Inc. M Sten & thick, Tr. Sten & thick, P thick & Inc.
4.	F.	34.	A thick & Inc, M thick & Inc, Tr. very thick, P thick & Inc.
5.	M.	52.	Tr. thick ossified & Sten, P thick & Inc. from old endoc.

It is interesting to note that in case 1, a systolic bruit was heard all over the chest, but in none of the others was a pulmonary bruit diagnosed. Case 5 certainly proves that after old lesions of the tricuspid and pulmonary valve, the patient is by no means certain to die early, he, as a matter of fact, died of some independent intercurrent illness.

I have also 5 cases in which the tricuspid was the only valve affected. Three of these were cases of ulcerative endo-carditis; one was a case of chorea with small recent vegetations only: the remaining one died at the age of 38, when it was found that thickening and puckering had attacked the tricuspid valve alone.

Problem VIII.

What is the order of relative frequency of the affections of the several valves?

Table XV.

CLASS.		Aortic and Mitral.		Mitral.		Aortic.		Aortic, Mitral and Tricuspid.		Mitral and Tricuspid.		Tricuspid.		Aortic, Mitral, Tricuspid and Pulmonary.		Aortic, Pulmonary and Mitral.		Aortic and Pulmonary.		Tricuspid and Pulmonary.		Mitral, Tricuspid, and Pulmonary.		Total.		Gross Total.
		M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	
I		3	1	2	...	10	...	...	2	...	...	...	...	...	...	...	...	...	...	...	...	...	...	15	3	18
II		8	7	4	3	7	...	1	...	...	1	...	...	1	...	...	...	...	...	...	...	...	...	21	11	32
III		7	...	1	2	5	3	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	14	6	20
IV		11	7	6	8	10	1	3	2	...	...	...	...	...	1	...	...	1	...	...	...	...	1	31	20	51
V		26	13	7	18	8	...	6	7	1	6	...	...	1	...	2	...	...	...	...	...	...	...	51	44	95
VI		6	4	2	6	5	1	...	3	2	1	..	...	..	...	...	...	...	...	...	...	...	...	15	15	30
VII		21	12	15	28	27	5	5	2	4	5	1	3	...	...	...	...	...	...	...	..	...	...	73	55	128
VIII		38	17	19	20	9	3	2	13	1	1	...	...	1	...	...	...	...	...	...	...	...	...	70	54	124
IX		27	9	22	24	15	8	3	8	4	...	1	1	...	...	...	...	...	...	...	...	...	...	72	50	122
X		10	6	4	3	7	2	...	4	...	1	..	...	...	...	...	...	1	...	1	...	...	...	22	16	38
XI		...	1	2	1	...	...	...	1	...	1	...	...	...	...	...	...	...	...	...	...	...	...	2	4	6
Total		157	77	84	113	103	23	21	43	12	16	2	4	3	1	2	...	1	...	1	...	...	1	386	278	664
		234		197		126		64		28		6		4		2		1		1		1				

In the previous three problems dealing with the ages of patients, I have included only those cases which occurred with a tolerable frequency. The following table includes all the autopsies, of which I have the records.

From these figures, it would appear that it is commoner to get both the aortic and mitral valve affected, than either one by itself, which is probably to be accounted for in many cases by direct extension from one to the other. The tricuspid valve is affected in a very fair proportion of the cases: no less than 104 out of 664. Divided into right and left sided lesions, the following is the grouping:—

557 or 83.88 per cent. are pure left sided lesions.
100 ,, 15.06 ,, mixed right and left lesions.
7 ,, 1.06 ,, pure right sided lesions.

I thought it of some little interest to compare the figures of the rheumatic cases with those of the non-rheumatic. I found that in the rheumatic classes the figures were 109, both mitral and aortic, 71 mitral alone, and only 27 aortic alone, whereas in the non-rheumatic cases the aortic cases rose to an equality with the others nearly, the figures being 55, 59 and 53 respectively, in the previous order, illustrating very forcibly the curious preference that rheumatism has for the mitral, and atheroma for the aortic valves, respectively.