A LECTURE ON THE FUNCTIONAL CARDIAC MURMURS OF ANÆMIA.

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It is customary to include under the term functional murmurs all those cases in which the murmur depends upon temporary and curable conditions, or in which no distinct alteration in the valvular mechanism (it would, perhaps, be more correct to say in the valvular segments) can be detected after death. Functional murmurs are always systolic in time, and are generally heard at the base of the heart, most commonly to the left of the sternum, in the second interspace, *i.e.*, in the region of the pulmonary artery. They are sometimes also heard in the aortic, mitral, and tricuspid areas.

The exact significance and mode of production of the functional murmurs, which are audible at the base of the heart, have given rise to great debate, and are among the most unsettled points in cardiac pathology.

Walshe divides the so-called functional murmurs into two great groups, which he respectively terms hæmic and dynamic. Under the former, he places "murmurs originating within the heart and dependent upon an unnatural state of the blood;" while, under the latter, he includes "murmurs which result from abnormal action on the part of the heart," the proper valvular mechanism being healthy. And this division indicates the two great causes of functional murmurs, *viz.*: (1) *defective muscular action* allowing of mitral and tricuspid regurgitation; and (2) *an altered condition of the blood*, which probably aids, at least, in the production of the pulmonary and aortic murmurs which are met with in conditions of anæmia.

But it will be necessary to consider the subject in more detail. Anæmia, more particularly those forms of anæmia (such as chlorosis, progressive pernicious anæmia, etc.) in which there is defective formation of blood, is the chief clinical condition in which functional endocardial murmurs occur. Now, in cases of this description, the muscular fibre of the heart becomes fatty, the cardiac cavities become dilated, and the weight of the heart also becomes increased; in fact, there is a condition of combined fatty degeneration, dilatation, and hypertrophy, dilatation being, however, in excess of hypertrophy.*

Such a condition of the heart is just such as we would expect to produce "relative incompetence;" and, as a matter of fact, all careful clinical observers are, I presume, agreed that, in cases of this description, mitral and tricuspid regurgitation do ultimately occur. All observers are also, I suppose, at one in thinking that arterial murmurs (pulmonary and aortic murmurs) are usually present in advanced conditions of anæmia. The point which is still undecided, and which has lately given rise to so much controversy, is the nature and significance of the basic murmur, which is heard in the second left interspace, in the earlier stages of anæmia. Three theories, all of which have warm supporters, have been advanced to account for the condition, *viz.*:

1. *That the Murmur is Pulmonary.*—The exact manner in which the murmur is supposed to be produced in the pulmonary artery, has not been very clearly defined by the supporters of this view. The sudden propulsion of a large blood-wave of abnormal (spanæmic) composition into the vessel, which is, probably, in some cases at least, dilated, seems to me an efficient cause for its production; and we know, as a matter of fact, that the heart, in cases of chlorosis, is not only weak, but that it is unusually irritable; that even in the earlier stages the right ventricle is to some extent dilated; and that the blood is markedly altered in composition.

2. *That it is due to mitral regurgitation*, and that the murmur is conducted to the anterior wall of the chest, through the dilated appendix of the left auricle. (Dr. George Balfour's theory.)

* These changes are seen in the human subject in cases of progressive pernicious anæmia; and they have been shown by Beau to occur in the lower animals after venesection.

3. *That the murmur is produced in the pulmonary artery as the result of constriction of that vessel; the constriction being caused by the pressure of the dilated left auricle* (Dr. Russell's theory). Dr. Russell further believes that the systolic murmur heard in the second, third, and fourth left interspaces, in the later stages of such cases, is due to tricuspid regurgitation.

Before considering the arguments for and against these different theories, I may say that the question is still an open one; the balance of evidence, in my opinion, being in favour of the first or pulmonary view. I do not think, for the reasons to be presently given, that Dr. Balfour has conclusively proved his point; on the contrary, there seem to me to be grave objections to his view; and I do not see that anything which has as yet been advanced, conclusively negates the first or purely pulmonary theory.

Dr. Balfour's Theory.

The facts and arguments on which Dr. Balfour bases his theory, and the facts and arguments which seem to be opposed thereto, may be summarised as follows:

1. (*For.*) That in organic diseases of the mitral valve a systolic, basic, and apparently pulmonary murmur is often present. Admitted that this is so.

2. (*For.*) That this supposed pulmonary murmur is, as Naunyn was the first to suggest, not pulmonary at all, but that it is due to mitral regurgitation, and is conducted to the second left interspace through the dilated appendix of the left auricle.

(*Against.*) While granting that, if the appendix of the left auricle were dilated and were in contact with the chest-wall, the systolic murmur of mitral regurgitation would probably be conveyed to the second left interspace; and that, as Dr. Balfour points out, Naunyn's theory has been accepted as highly probable by many leading Continental authorities, it must be allowed that it has not been accepted as conclusively proved, more especially by many competent British observers. But, even granting that it is true for some cases of organic disease (*i.e.*, granting that cases of mitral regurgitation, in which the usual symptoms and signs of that condition, more especially a systolic murmur audible at the apex, are present, and in which the auricular appendix is dilated and is in contact with the chest-wall, do occur), it by no means follows that the chlorotic murmur with which we are now more particularly concerned is produced in the same manner.

The arguments with which Naunyn supports his theory are these.

(a) (*For.*) That the murmur has its point of maximum intensity at a point an inch and a half to the left of the sternum.

(*Against.*) Now, speaking for myself, I have in several cases of anæmia failed to satisfy myself that the position of the maximum intensity is so sharply defined as Dr. Balfour's statements seem to indicate. The murmur has seemed to me in several cases quite as loud half an inch to the left of the sternum as over the so-called auricular area, *i.e.*, an inch and a half to the left of that bone. Hayden, who is admitted to be a trustworthy observer, goes much further in this respect than I am disposed to do, for he states that the murmur is best heard at midsternum.

But even granting that the point of maximum intensity of the murmur, in cases of chlorosis, is in the position which Dr. Balfour describes it, it does not seem to me by any means to prove that it is not pulmonary. In the first place, Sibson has shown that "in the large majority of cases" which he examined, "the greater part (in twenty-five of forty-five instances), or the whole (in fourteen of forty-five instances), of the artery, bore to the left of the sternum, and presented itself behind the upper costal cartilages and their spaces from the first cartilage to the third space (Russell Reynolds's *System of Medicine*, vol. iv, p. 35); and further, that the average breadth of the vessel in Pirogoff's five front views of the healthy heart was an inch and a quarter (*ib.* p. 115). I see nothing improbable, therefore, in a pulmonary murmur having, in many cases, its point of maximum intensity considerably to the left of the sternum.

In the second place, there seems good reason to suppose, as Dr. Russell's observations seem to show, that in consequence of the dilated condition of the heart (of the right ventricle more especially), the relative position of the parts is somewhat altered, and that the pulmonary artery may be displaced forwards, and perhaps somewhat to the left of its usual position.

(b) (*For.*) That this point does not correspond to the origin of the pulmonary artery, but does exactly correspond to the spot where the appendix or the left auricle comes up from behind, just to the left of that artery, and that in cases of this description (*i.e.*, where the murmur is loudly heard in the second left interspace) the

appendix of the left auricle is dilated, and is closer to the chest, and therefore nearer to the ear than usual.

(*Against.*) It is not proved that the appendix of the left auricle is dilated in cases of chlorosis (with which we are at present more immediately concerned. On the contrary, there are, as we shall presently see, positive observations to the contrary; and there are, as Dr. Russell has pointed out, grave anatomical objections to the theory that the left auricular appendix is in closer contact with the chest-wall in cases of chlorosis than in health. This point will be again referred to.

But, granting for the moment that cases of mitral regurgitation do occur, in which the left auricular appendix is dilated, and in closer contact to the chest-wall than usual, and in which the systolic murmur produced at the mitral valve is heard as loudly in the second left interspace as at the apex, that is no reason for supposing that, in a case of anæmia, in which a murmur is heard in the second left interspace, and in which there is no murmur, be it observed, at the apex, and no other indication of mitral regurgitation present, that this apparently pulmonary murmur is of mitral origin.

The fact that the murmur is better heard over the auricular area than over the apex—the normal position of maximum intensity for mitral murmurs—Naunyn explains by attributing it to the better conduction of the murmur along the course of the regurgitating blood-current, the fluid veins producing sonorous vibrations louder at the point of impingement than at that of origin—a view which is adopted and endorsed by Dr. Balfour in support of his theory. He says: “the fluid veins formed in the early stage of chlorotic regurgitation are of low tension and but little force, hence the vibrations they originate are but slightly propagated to the left ventricle, and only with difficulty from it to the chest-wall in the mitral area, where they are heard as an impure first sound. But, on the other hand, these vibrations are readily communicated to the wall of the auricle on which these fluid veins impinge, and are easily transmitted to the chest-wall with which the auricular appendix is in contact, becoming audible in the auricular area as a distinct murmur.”

Against this view it may be argued that, if the fluid veins produce sonorous vibrations louder at their point of impingement than at their point of origin in one case, why should they not do so in all? In other words, if this theory be correct, we ought to hear the systolic murmur of organic mitral regurgitation much more frequently and much more loudly in the second left interspaces than is admitted by the majority of British observers, at all events.

Again, it may be asked if this explanation is correct in cases of chlorosis; and, if, as Dr. Balfour admits, “the auricle, at the moment of ventricular systole, is not only full, but somewhat tense from unusual dilatation,” and if the fluid veins produce sonorous vibrations louder at their point of impingement than at their point of origin, why is it that the murmur is not heard in the back, behind, *i.e.*, under the left scapula, as the murmur of organic regurgitation so often is?

3. (*For.*) That in some cases of mitral disease, the apparently pulmonary, but in reality auricular murmur, is occasionally actually louder than that audible in the mitral area, and that the murmur of mitral regurgitation is sometimes only to be heard in the first named situation, *i.e.*, in the second left interspace. Dr. Balfour, indeed, goes so far as to state that “a more or less distinct murmur in this (*i.e.*, the auricular) area, is one of the earliest indications of mitral regurgitation, from whatever cause” (second edition, p. 163).

(*Against.*) While admitting that some authorities—Rosenstein, for example—support this view, it is certainly contrary to the recorded opinion of almost all the best observers in this country; and I am not prepared to admit that a systolic murmur heard in the second left interspace is, *per se* (as Dr. Balfour states in the following passage), indicative of mitral regurgitation.

“In chlorosis,” says Dr. Balfour, “in which all these phenomena, to which I may now comprehensively refer under the head of cardiac dilatation consecutive to spanæmia, exist only in the very slightest degree, this pulmonary, or rather auricular, murmur is always present, and is often—so often as to constitute it almost invariably—the sole sign of mitral regurgitation in these cases” (*Diseases of the Heart*, second edition, p. 172).

4. (*For.*) Dr. Balfour claims “that Naunyn’s view is even more applicable in chlorosis than in any other form of heart-affection, because the essential cardiac lesion in chlorosis is muscular relaxation and progressive dilatation; hence, at a comparatively early stage of the disease, the dilated right ventricle has separated the left ventricle from the chest-wall, while the dilated appendix of

the left auricle has been, *pari passu*, brought into closer contact with it.”

“The peculiar position of the murmur is due,” says Dr. Balfour, “to the altered position of the heart..... This is due to the dilatation of the right ventricle, which dilates *pari passu* with the left ventricle, and, like a water-cushion, separates it from the chest-wall, leaving the dilated appendix of the left auricle the only part of the left side of the heart in contact with the chest-wall.”

Dr. Balfour further claims that the pulsation of the auricular appendix can be seen, felt, and graphically recorded by the cardiograph. He says: “In this situation, the dilated appendix not unfrequently gives rise to so distinct a pulsation, that its movements can be traced by the cardiograph; and the history of several such cases has been published, and their cardiograms figured, by my former resident, Dr. George Gibson; while the pulsation is so well marked and forcible in some of these cases, that the late Dr. Hughes Bennett sent me, on one occasion, a case of chlorosis as a case of aortic aneurysm.” (*Diseases of the Heart*, second edition, p. 175.)

Against these statements, it may be argued, *first*, that Naunyn’s explanation seems more particularly to apply to those cases of mitral regurgitation in which a systolic murmur is audible at the apex, *i.e.*, in the usual mitral area, as well as in the second left interspace; but that, in the earlier stages of chlorosis, the apparently pulmonary murmur is, as Dr. Balfour himself admits, usually the sole sign of mitral regurgitation; in other words, the usual evidence of mitral regurgitation, *i.e.*, a systolic murmur in the mitral area, is wanting.

Secondly, that, in the later stages of anæmia (chlorosis), a true mitral murmur, *i.e.*, a murmur audible at the apex—in the mitral area—does actually occur. This Dr. Balfour himself admits, and he explains it in the following manner, “By-and-by, as the regurgitation increases, and the ventricle hypertrophies, these fluid veins gain force sufficient to be communicated through the ventricle also; hence, in the later stages of chlorosis, we have a mitral murmur associated with the auricular one. It is, however, quite possible that this murmur in the mitral area is really tricuspid, due to the increased dilatation of the right ventricle, the apex of which may even occupy the mitral area. This not unfrequently occurs in mitral stenosis; it is not an improbable event in chlorosis; and it is of little consequence which explanation we accept, the actual truth probably embracing both conclusions, being sometimes due to the one cause and sometimes to the other.” (*Diseases of the Heart*, p. 177.)

Now I agree with Dr. Balfour in thinking that in many cases of advanced chlorosis the systolic murmur, which is heard at the apex, is due to mitral regurgitation, but I differ from him, inasmuch as I believe that it is a distinct murmur from that heard in the second left interspace; and I am unable to accept the theory which he advances to explain the supposed fact that a mitral murmur may, in the earlier stages of the condition, be confined to the base, while in the later stages it is heard at the apex. For is it not the fact that as cases of chlorosis (and more especially of progressive pernicious anæmia, in which the same sequence of events occurs), advance, that the degeneration of the heart-muscle increases, and that dilatation of the heart-cavities, with increased feebleness of action, rather than hypertrophy with increased force of contraction, occurs. If this is so, Dr. Balfour’s explanation obviously cannot hold good; and he himself states that “the essential cardiac lesion in chlorosis is muscular relaxation and progressive dilatation.”

The order of recovery, too, seems to be opposed to this view. If the hypertrophy of the left ventricle is the cause of the mitral murmur being audible at the apex in advanced stages of the case, and if the mitral and so-called auricular murmurs depend on one and the same cause, *i.e.*, upon mitral regurgitation, why, in cases which recover, should the basic murmur persist long after the apex murmur has disappeared? It can hardly be suggested that the left ventricle becomes weaker during the process of recovery; and if both murmurs depend on one and the same cause, *i.e.*, upon mitral regurgitation, and if the ventricle does not become weaker, both murmurs, surely, ought to disappear at one and the same time.

Thirdly, Dr. Russell has shown that in a case of progressive pernicious anæmia, in which both pulsation and *bruit* were present in the second left interspace during life, the auricular appendix was not dilated, and was not in contact with, but was far removed from, the chest wall, and that the pulsation, percussion-dulness and *bruit* were respectively seen and heard over the *conus arteriosus*. (*Edinburgh Medical Journal*, November 1882, page 408.)

Dr. Balfour’s counter argument against this, “that the

position of the auricle *post mortem* is no proof of its state during life, and where there is no mitral stenosis, an auricular appendix, beating in the second interspace during life, may very well empty itself and contract out of sight in the act of dying," is not, to my mind, a very convincing one. In the first place, our knowledge of the normal position of the appendix (including Naunyn's own observations, which Dr. Balfour quotes as one of his main arguments) has been largely acquired by the same means which were adopted in this case. And in the second place, in cases of progressive pernicious anæmia, such as this was, the heart is usually (I think I may say invariably) relaxed and flaccid after death. It seems unlikely, therefore, that in this case the appendix emptied itself and contracted out of sight.

Fourthly, that when the right cavities of the heart are dilated, as Dr. Balfour admits is the case, in *chlorosis*—the condition we are considering—the left auricular appendix is usually quite invisible from the front, a fact which Dr. Russell has also urged. I have had several opportunities of verifying this statement during the past session, two of the cases being typical examples of pernicious anæmia. In none of these cases was the appendix much dilated; indeed, in one of the cases of pernicious anæmia it was considerably smaller than usual. Dr. Russell says: "It is further recognised that, in debility, owing to dilatation of the right ventricle, the left is displaced outwards and backwards; or a change occurs which may be regarded as a rotatory movement of the heart round its longitudinal axis; and this must be conceded as having a displacing effect on the auricle analogous to what it has on the ventricle of the same side." Further, Dr. Russell argues that, since "the origin or root of the appendix is overlapped in part by the pulmonary artery, so, to reach the parietes, the appendix has to traverse a course equal to the diameter of that vessel. Any increase in the diameter of the artery from increase of its contents will thus place the appendix deeper in the chest." (*Edinburgh Medical Journal*, August 1882, page 131.)

I can from personal observation testify, as Dr. Russell's argument implies, that the pulmonary artery is dilated in (some) cases of pernicious anæmia; presumably, therefore, it is dilated in some cases of chlorosis.

It must be at once conceded that pulsation is frequently to be observed in the second left interspace in cases of chlorosis. I cannot, however, say that I have ever been able to satisfy myself that it was auricular; and, like Dr. Broadbent, I am not convinced that Dr. Gibson's latter tracings, to which Dr. Balfour particularly refers (see *Edinburgh Medical Journal*, October 1882, page 294), prove the pulsation to be produced by regurgitation into the ventricle from the auricle.*

Further, in common with Dr. Broadbent, Dr. Goodhart, Dr. Russell, and other observers, whose pathological experience is not inconsiderable, I have not met with any case of cardiac dilatation—certainly not in any case of anæmia, and I have had an opportunity of examining seven or eight cases, after death—in which the left auricular appendix was so markedly dilated "as to warrant the belief that it could have been the cause of the extensive pulsation claimed for it by Dr. Balfour in the second and third left spaces." (*BRITISH MEDICAL JOURNAL*, August 26th, 1882, page 354.)

Dr. Russell thinks that the left auricular appendix is more frequently thrombosed than dilated in mitral stenosis, and that this is an argument against Dr. Balfour's view.

My experience agrees with that of Dr. Russell, in so far as I have frequently seen thrombosis of the left auricular appendix in cases of mitral stenosis. I do not, however, lay any stress upon this point, for a thrombosed appendix may also be dilated; and if it be granted that a large appendix can come in contact with the chest-wall, the fact that it was thrombosed would rather favour than interfere with the conduction of sound, *i.e.*, of the murmur. A thrombosed appendix could hardly, however, be the cause of the extensive pulsation which Dr. Balfour describes.

Further, Dr. Russell claims to have frequently satisfied himself that the pulsation in the second interspace in cases of organic mitral disease, is due to the dilated right ventricle. (*Edin. Med. Journ.*)

I can corroborate Dr. Russell's statement in this respect, inasmuch as I have in several cases of right-sided dilatation—notably in a case of pernicious anæmia—found that a needle passed into the second left interspace transfixing the conus arteriosus of the right ventricle, and did not transfix the pulmonary artery, as it does under normal circumstances.

* The tracings published by Dr. Russell in the *JOURNAL* of June 2nd seem also opposed to Dr. Gibson's view.

A MEMORANDUM

ON THE

INFLUENCE OF VACCINATION IN THE PREVENTION AND DIMINUTION OF MORTALITY FROM SMALL-POX.

Presented to the Parliamentary Bills Committee of the British Medical Association.

By ERNEST HART,
Chairman of the Committee.

1. THE introduction of vaccination was followed by a marked decrease in the small-pox death-rate; and, concurrently with the diffusion of vaccination, the small-pox death-rate has further progressively diminished.

Prior to the introduction of vaccination, small-pox was an almost universal disease. Continuously present in all large centres of population, it assumed epidemic proportions at intervals of two to four years, while the smaller towns and rural villages—except such as were exceptionally isolated—were, as a rule, visited by an epidemic of the disease once in every three to six years.¹ No class of society was exempt from its ravages; and while most fatal in the filthy homes of the poor, yet it spared not the palaces of kings nor the mansions of the rich.² So common, indeed, was the disease that it was rare for anyone to reach adult life without having passed through an attack.³ The dread with which it was looked upon may readily be inferred from the eagerness with which inoculation was had recourse to towards the end of the eighteenth century. After the introduction of vaccination, the mortality from the disease underwent a marked diminution. How great this diminution has been in the case of London is shown by the following table.

Table showing the Small-pox Deaths per 1,000 Deaths from all Causes in London from 1631 to 1882.*

Period.	Small-pox Deaths per 1,000 Total Deaths.	Period.	S. P. Deaths per 1,000 Total Deaths.	Period.	S. P. Deaths per 1,000 Total Deaths.
1631-35	48	1741-50	73	1801-10	67
1651-60	59	1751-60	100	1811-20	41
1661-70	39	1761-70	103	1821-30	33
1671-80	66	1771-80	97	1831-40	23
1681-90	76	1781-90	92	1841-50	16
1691-1700	53	1791-1800	93	1851-60	11
1701-10	58	...	...	1861-70	11
1711-20	81	...	...	1871-80	19
1721-30	83	...	...	1881-82	17
1731-40	77	...	...	...	...

Preinoculation Period.

Inoculation Period.

Vaccination Period.

The table shows that the proportion of small-pox deaths to deaths from all causes, has undergone considerable diminution. It is, moreover, universally admitted that the total death-rate of the metropolis is now much lower than it was in the seventeenth and eighteenth centuries. Hence, as the small-pox death-rate has diminished in greater ratio, it is clear that the diminution in the small-pox death-rate must be enormous.

As regards England generally, it is equally certain that the mortality from small-pox is now infinitely less than in prevaccination days, although the absence of registration in the earlier period precludes any accurate statistical comparison. In registration times, however, the mortality from the disease has steadily declined, as the following table shows.

¹ Hillary: *Rational and Mechanical Essay on the Small-pox*. London, 1735.

² See Burnet's *History of William and Mary*, pp. 136, 304; Walpole's *Letters* (April 2nd, 1750); Pepys; Evelyn; St. Simon; Besenval; Vehse, etc.

³ Hillary: *loc. cit.*; Haygarth: *Sketch of a Plan to Exterminate the Natural Small-pox*. London, 1783.

⁴ Calculated from data in Marshall's *Bills of Mortality* and the Registrar-General's Annual Reports.