A LECTURE ON THE FUNCTIONAL CARDIAC MURMURS OF ANÆMIA.

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(Continued from page 1213.)

To the other points previously advanced in support of and against Dr. Balfour's theory, the following must be added.

5. That the pulmonary second sound is accentuated.

We all, of course, know, that in mitral regurgitation, the pulmonary second sound is accentuated in consequence of the increased blood-pressure in the pulmonary artery, which results from the mitral lesion. If, therefore, this accentuation occurs in the earlier stages of chlorosis, and if it cannot be satisfactorily accounted for in any other manner (than as the result of mitral regurgitation), we must, of course, admit that it is strongly corroborative of Dr. Balfour's theory, always premising that there is no fatal objection to that view.

Now, I at once admit that, in the later stages of anæmia, the pulmonary second sound is often intensified, and that the accentuation may be due to the mitral regurgitation which is often present in the advanced stages of that condition. In the earlier stages of chlorosis, too, I have frequently (though not invariably) found the pulmonary second sound louder than the aortic. I have been in the habit of regarding the accentuation, which occurs in the early stages of chlorosis, as apparent rather than real; in other words, I have supposed that the aortic second sound was diminished in intensity, while the pulmonary retained its normal loudness. Possibly, I may be mistaken in this view; and I am certainly not prepared to say that true accentuation of the pulmonary second sound does not occur, even in the earlier stages of chlorosis.

But even granting that the accentuation were always present and well marked, I cannot admit that it is sufficiently strong evidence to counterbalance the grave objections which have been advanced against the mitral regurgitation view. In particular, the objections, which Dr. Russell has so ably, and which I can not only corroborate, but also strengthen—(1) that in the most advanced stages of anæmia, as seen after death, the left auricular appendix is not dilated (in one of my cases it was actually smaller than normal); and (2) that when the right heart becomes dilated, as it does in conditions of anæmia, the appendix of the left auricle recedes from, rather than comes in closer contact with, the chest-wall—seem to me to negative Dr. Balfour's view. I feel obliged, therefore, to suppose, that if the pulmonary second sound is actually intensified, the accentuation must be due to some other cause than mitral regurgitation. Although I have made some inquiries, I have not as yet been able to ascertain whether the anæmic blood passes through the pulmonary capillaries with greater or with less facility than in health; and, until I have definite information on this point, I do not feel in a position to theorise on the matter.

Dr. Russell's Theory.

Let us now turn to Dr. Russell's theory. He believes:

(1) That the murmur heard in the second left interspace, in the earlier stages of chlorosis, is generated in the pulmonary artery.

2. That the murmur is due to a relative constriction of the vessel (i. e., of the pulmonary artery), produced by the pressure of the dilated left auricle, which is situated behind it.

Now, this theory necessarily presupposes that, during the ventricular systole, the pressure of the blood in the left auricle is greater than the pressure of the blood in the pulmonary artery; for otherwise the pulmonary artery could not, of course, be constricted by the pressure of the auricle. That this is so, Dr. Russell maintains; and he contends that the excessive pressure (if I may so term it) in the left auricle is due to a regurgitant current being propelled into it (the left auricle), by the left ventricle, through the mitral orifice.

The explanation which he gives is as follows: "Owing to the tension in the pulmonary vessels, the fulness of the auricle must at all times be increased; in fact, the blood coming from the lungs pours into it as rapidly as it empties itself into the ventricle, the result being that its cavity, although enlarged by sharing in the debilitating influences in existence, is already full before the ventricular systole not only prevents further relief to the tension in the pulmonary circuit, but throws back upon it (the auricle) the blood

embraced by the segments of the mitral valve as they swing to close the auriculo-ventricular orifice, as well as the column of regurgitant blood from the ventricle." (*Edinburgh Medical Journal*, August 1882, p. 134.) And again, "The question, then, arises as to what force may act through the left auricle, and is it greater than that of the right ventricle? Take mitral regurgitation: there a stream, at times of considerable magnitude, passes back into the auricle with all the force exerted by the systole of the left ventricle; and, as we cannot assume the left auricle to be a vacuum ready to receive this regurgitating stream, there must be a backward flow through it, nearly as much greater in force than the flow in the pulmonary artery as the systole of the left ventricle is greater than the right.....The next question to consider is, whether the tension in the left auricle is greater than that in the pulmonary artery at the moment of ventricular systole."....."When it is remembered that there is not only a column of blood sent with the force of the left ventricle into this tense auricle, but also that the mitral cusps, instead of being held well down into the ventricle and presenting a concavity towards the auricle, so as thereby to relieve the auricle and 'make room for the returning blood without hindrance,' must, on the contrary, owing to the enfeebled state of the papillary muscles, be allowed greater latitude of movement towards the auricle, it must be granted that the auricular tension bears a fixed relation to the strength of the left ventricle, and is therefore greater than that in the pulmonary artery." (*Ibid.*, November 1882, pp. 411, 412.)

In opposition to this theory, Dr. Balfour maintains that "it is absolutely impossible that the left auricle can ever so compress the pulmonary artery;".....and "that it is obviously impossible that the circulation should be carried on under these conditions." It is unnecessary, I think, to detail the facts and arguments with which Dr. Balfour supports these objections, and the facts and arguments which Dr. Russell urges in reply, for (granting for the moment that Dr. Balfour's objections are invalid) I maintain that, even if such excessive intra-auricular pressure could occur, it certainly is not present in the early stages of chlorosis—the condition which we are now considering. Such excessive intra-auricular pressure could only be produced by extremely free mitral regurgitation. In all cases of mitral regurgitation, there is probably a considerable direct obstacle (both valvular and muscular), in addition to the blood-pressure in the auricle, opposed to the force of the left ventricle; while there is no direct obstacle, but only the blood-pressure in the pulmonary artery, opposed to the force of the right ventricle. We cannot, therefore, with fairness say, because the left ventricle is much stronger than the right, it will in mitral regurgitation raise the blood-pressure in the left auricle higher than the right ventricle will raise the blood-pressure in the pulmonary artery. The right ventricle, though much weaker, is acting (more especially when the tricuspid is sound) at an immense advantage, and undoubtedly propels a much larger quantity of blood into the pulmonary artery than the left ventricle propels (in any ordinary case of mitral regurgitation) into the left auricle.*

Now, there is not sufficient evidence, I maintain, to prove that any regurgitation through the mitral valve occurs in the early stages of chlorosis and anæmia, much less the free regurgitation which would be required to produce the excessive pressure in the left auricle, which Dr. Russell's theory requires.

The only evidence which Dr. Russell produces in favour of mitral regurgitation in the early stages of chlorosis, is an accentuated pulmonary second sound. "It will not be seriously denied here," says Dr. Russell, "that in these affections there is a relative insufficiency of the mitral valve, the result of a debilitated and relaxed condition of the cardiac muscle, including the muscoli papillares. This insufficiency, however, is not always evidenced by a *bruit* at the apex, the point at which mitral *bruits* are ordinarily audible; but regurgitation is assumed† from the accentuation of the pulmonary second sound, and from the presence of a systolic murmur in the pulmonary area." (*Edinburgh Medical Journal*, August 1882, p. 130.)

I have already given in detail the facts and arguments which are, in my opinion, opposed to Dr. Balfour's view; and have previously stated that the presence of a systolic murmur in the second left interspace is not, in my opinion, *per se* (I may add, nor when conjoined with an accentuated pulmonary second sound), sufficient evidence of mitral regurgitation. And Dr. Russell himself maintains that this systolic murmur is in reality produced in the pul-

* The reader is referred to the original papers. See the *Edinburgh Medical Journal*, August and September 1882; and November 1882, p. 409.

† Dr. Russell is here speaking of Dr. Balfour's theory. He himself believing that the systolic basic murmur is not mitral, but pulmonary.

monary artery. According to his own showing then, the only evidence of mitral regurgitation which remains is accentuation of the pulmonary second sound. This, I maintain, is insufficient evidence, and I feel obliged therefore to dissent from his theory.

Since this lecture was written, Dr. Russell seems to have modified the view which he originally proposed, inasmuch as he no longer insists that the increased tension of the left auricle (which his theory necessarily supposes is present during the first part of the ventricular systole) is due to mitral regurgitation. In the passage quoted in the foot-note, from his instructive paper in the *JOURNAL* of June 2nd, he suggests that the increased tension of the left auricle may be due to the incomplete emptying of the left ventricle during its systole, and consequent imperfect relief to the full auricle.*

But if this view be correct, should we not expect to hear a systolic pulmonary murmur as a necessary accompaniment of all cases of organic mitral disease, in which the pulmonary second sound is accentuated? We can hardly suppose that the distention of the left auricle is great in the early stages of chlorosis; and if a small amount of distention is sufficient to constrict the pulmonary artery and to produce a systolic murmur, should we not *a fortiori* expect to have a systolic pulmonary murmur produced in those cases of mitral stenosis, for example, in which we may legitimately conclude that the distention of the auricle is still more considerable? It may, of course, be argued (1) that a systolic murmur is sometimes heard in the pulmonary area in cases of mitral stenosis; and (2) that in those cases of mitral stenosis in which a systolic pulmonary murmur is not present, the left auricle is not dilated. In support of the latter proposition, it may be urged that in mitral stenosis the cardiac muscle is not degenerated, as it is in the cases of anæmia, and that the left auricle, for a time at all events, is able to resist the excessive blood-pressure in its interior, and does not dilate. But looking at the matter from the broad ground of clinical experience, most observers will, I think, agree that cases (such as cases of mitral stenosis, mitral regurgitation, fatty heart, etc.) are frequently met with in which the left auricle is quite as much distended as we can legitimately suppose it is in the earlier stages of chlorosis, and in which there is no pulmonary systolic murmur. If this general proposition be granted, we must of course conclude that the pulmonary murmur met with in the earlier stages of chlorosis, is not produced by the pressure of the distended left auricle upon the pulmonary artery, but that it is due (either wholly or in part) to some other condition or conditions.

For these reasons I am unable to accept Dr. Russell's theory; and there are (as I have already pointed out in detail), in my opinion, grave objections to Dr. Balfour's view. I am compelled, therefore, by the method of exclusion, to fall back upon the purely pulmonary theory and to ask whether there is any conclusive reason why the murmur should not be generated in the pulmonary artery itself, irrespective of any constriction by the pressure of the auricle, such as Dr. Russell's theory implies.†

Dr. Balfour argues that the murmur cannot be pulmonary:

(1.) Because "there are—in chlorosis—no causes of murmur operative at the pulmonary orifice which are not at least as active at the aortic opening, so that a pulmonary murmur would certainly be accompanied by an aortic murmur also, and the latter would, of course, be propagated along the course of the aorta, and more or less distinctly into the carotids." (*Edinburgh Medical Journal*, October 1882, p. 294.)

This argument does not, however, appear to be conclusive.

In the first place, we might as well say, in opposition to Dr. Balfour's own view—the auricular theory—that the murmur cannot be mitral, because there are no causes of murmur operative at the

mitral orifice, which are not at least as active at the tricuspid opening, so that a mitral murmur would certainly be accompanied by a tricuspid murmur; and since it is generally admitted, and as Dr. Balfour himself allows in the passage quoted below, that the tricuspid murmur is of later occurrence than the mitral murmur, Dr. Balfour's auricular theory falls to the ground. Dr. Balfour says: "Shortly after the appearance of the primary hæmic murmur, a tricuspid murmur and jugular undulation are found to be developed. This is naturally accompanied by a pulmonary and also by an aortic systolic murmur, the active cause in the production of both these murmurs being the large blood-waves sent on by the dilated and hypertrophied ventricles, as was first, I believe, pointed out by Beau." (*Edinburgh Medical Journal*, October 1882, p. 295.)

In the second place, I am not prepared to admit, unconditionally, that there are no causes of murmur operative at the pulmonary orifice, which are not at least as active at the aortic orifice. It appears to me quite possible that such causes may exist in the respective conditions of the two ventricles; in the respective resistances which the arterial blood meets with at the orifice of the aorta and in the systemic circulation, and which the venous blood meets with at the orifice of the pulmonary artery and in its passage through the lungs; and in the respective physical conditions (thickness of coat, relationship to the chest-wall, etc.) of the aorta and pulmonary artery.

In the third place, I believe that aortic murmurs are sometimes present in the early stages of chlorosis. Possibly they would be more frequently audible in such conditions, if it were not for the fact, that they are so soft and faint as to be obscured at the base by the pulmonary murmur, and of such low tension, and of such little force, as to be inaudible over the course of the aorta and in the carotids.

(2.) Because the point of maximum intensity of the murmur is not over the pulmonary artery at all, but from one to two inches to the left of the sternum in the second interspace. (*Edinburgh Medical Journal*, October 1882, p. 294.) This argument also fails to convince me, for the reasons already detailed. (See p. 1213.)

(3.) Because no murmur of strictly pulmonary origin could possibly be referred to all four orifices in turn, as has been the case with the hæmic murmur; and second, because however singular a murmur of mitral regurgitation in this position may seem to be, its causation is by no means difficult to understand. (*Diseases of the Heart*, second edition, p. 137.)

Neither of these reasons seem to me to exclude the pulmonary hypothesis. The mitral origin of the murmur, which Dr. Balfour supports, would be as effectually excluded by the first reason, as he argues the pulmonary origin is, for no murmur of strictly mitral origin could possibly be referred to all four orifices in turn. The only legitimate conclusion to be drawn from the first reason is, that in chlorosis, murmurs may be generated at more than one orifice, a conclusion which we all allow. The second reason, even if admitted, does not exclude the pulmonary hypothesis, but only shows that a mitral murmur can be heard in the neighbourhood of the pulmonary artery.

I feel compelled, therefore, to differ from Dr. Balfour, for I do not see that any argument which has been as yet advanced, conclusively negatives the purely pulmonary theory.

As I have previously stated, the sudden propulsion of a large blood-wave of abnormal (spanæmic) composition into the vessel, which is possibly dilated, seems to me an efficient cause for the production of the murmur; and we know, as a matter of fact, that in cases of chlorosis these conditions are actually present—the chlorotic heart is unusually irritable, and contracts with unusual suddenness; even in the earlier stages there is some dilatation of the right ventricle; the blood is spanæmic, and in some advanced cases, *i.e.*, fatal cases of pernicious anæmia, the pulmonary artery is dilated.

Dr. Balfour himself states, in the passage I have quoted above, that aortic and pulmonary murmurs do occur in the later stages of chlorosis, and that the active cause in the production of both is the large blood-wave sent on by the dilated and hypertrophied ventricles. Now, if aortic and pulmonary murmurs can be produced by this cause in the later stages of chlorosis, and after, as Dr. Balfour argues, tricuspid and mitral regurgitation have occurred, should they not, *a fortiori*, be produced in the earlier stages, before, as I maintain, there is sufficient evidence of mitral and tricuspid regurgitation, for would not the presence of a leak at the tricuspid and mitral orifices diminish the size of the blood-wave, and so, other things being equal, be likely to interfere with the production of rather than to cause such murmurs?

* "I shall now endeavour to explain the clinical phenomena. The accentuation of the pulmonary second sound, if no lung-affection be present, must be taken as indicating an abnormal accentuation of blood behind the mitral orifice. Whether this be due to the incomplete emptying of the left ventricle during its weakened systole, and consequent imperfect relief to the full auricle, or, from the first, due to a certain amount of regurgitation, or to both these causes, it is unnecessary to discuss here. The fact of accumulation of blood in the pulmonary circuit, including the left auricle, is sufficient for our present purpose, and is warranted by the evidence given by pulsation at the root of the neck, by the course of the external jugulars becoming visible, and perhaps by pulsation appearing over the right ventricle, that a like accumulation is taking place in the right chambers of the heart and the large vessels leading to it.

† There seems to me to be no sufficient evidence to justify the belief, that the murmur heard in the second left interspace, in the early stages of anæmia, is due to tricuspid regurgitation, as Parrot supposed. The mere presence of a murmur in that position, in the absence of the usual signs of tricuspid regurgitation, is insufficient evidence to justify such a belief. It is, however, quite possible that, in the later stages of anæmia, a tricuspid murmur may be heard in this situation, as Dr. Russell supposes.