

Original Articles

DIRECT FUNCTIONAL MURMURS, AND OBSTRUCTIVE SAFETY-VALVE ACTION IN THE HEART.

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IN a report of several cases of malignant endocarditis published in the *Medical News* of November 14, 1885, I dwelt upon the significance of a mitral, direct (præsystolic) murmur, which was proved by the autopsy, to have been unconnected with any lesion of the mitral orifice. The lesions found were those of an intense aortic regurgitation, and I concluded that the mitral direct murmur was due to the fact that, in default of the semi-lunar valves, the recoil of blood fell, during the diastole, upon the mitral valve, holding its leaflets tense and raised against the stream of blood coming from the auricle. The conditions were, therefore, recognized to be the same with those first described by Flint.¹ As far as I know, this is the first case recorded in support of the views held by Professor Flint. I regret deeply that we cannot have to-day the aid of his opinion to decide whether I am right or not in giving these views a much wider field of application. In the opinion of Professor Flint the existence of this direct functional mitral murmur was limited to a very small number of cases of aortic regurgitation; and indeed this limitation was held up as an argument against his views by Dr. Balfour, of Edinburgh. This observer claimed that Flint's murmur ought to be heard in all cases of aortic regurgitation, and that even in health the mitral cusps are floated up towards the auricle by the blood filling the ventricle in the normal process. He forgets, it seems to me, that in aortic regurgitation the leaflets are not floated upwards, but are actively driven against the auricular blood by the force of the general arterial tension. Now, it is one of the objects of this paper to show that the functional mitral murmur is by no means as rare as Dr. Flint believed it to be.

The diastolic murmur heard at the apex in aortic insufficiency is generally supposed to be a transmitted aortic murmur. Dr. Balthazar Foster claimed that this propagation is of diagnostic value as indicating a lesion of the posterior (mitral) aortic leaflet, because in such cases the regurgitant current trends towards the apex. I maintain that these propagated murmurs are in fact mitral obstructive murmurs, and that they are more apt to develop when the posterior aortic segment is affected, because in such cases the recurrent stream is brought to bear directly against the anterior leaflet of the mitral valve.

However this may be, the fact remains, I believe, that obstructive functional mitral murmurs are of frequent occurrence in aortic regurgitation. In three cases of this lesion that I have studied since the publication of my paper, the mitral murmur was readily detected. It is not difficult to separate the two murmurs. The diastolic fades perceptibly towards the end of the diastole, whilst, at the apex, a very distinct præsystolic accentuation precedes the imperfectly developed first sound. This feebleness of the first sound is also a result of the præsystolic tension of the mitral valves.

The following is an abstract from my notes of these cases:

¹ Treatise on Diseases of the Heart, second edition, p. 207.

CASE I. A. G., aged thirty-seven, colored. Physical signs: The superficial veins are prominent. The arterial pulsation is marked at the neck and the extremities. The apex beat is four-and-three-quarters inch below, and two to the left of the nipple. Palpation shows it to be a sustained impulse. During its slow collapse there is a smaller secondary impulse which corresponds with the recoil from a slight visible retraction which occurs at the moment of the systole.

Percussion denotes the existence of hypertrophy with dilatation. Auscultation. There is a loud double murmur heard at the second right interspace. The diastolic murmur which is louder and longer than the systolic, grows fainter towards the end of the diastole, so that there is a short silence between the ending of this murmur and the beginning of the systole. There is no aortic second sound. The murmur is loudly transmitted down the sternum, and can also be followed around the chest to the right axilla. At the second left interspace there is a distinct second sound. At the third left space near the sternum, the systolic murmur is lost, and a feeble first sound makes its appearance. At the third and fourth spaces, towards the apex, the feeble first sound disappears and a faint systolic murmur makes its appearance. The systolic and the diastolic murmurs can be followed around to the left anterior axillary line. At the apex the diastolic murmur is still the louder of the two, and is characterized by a lower pitch and a præsystolic accentuation. The latter feature is recognizable as far as the left anterior axillary line. The diastolic and præsystolic murmurs are intensified by exertion, whilst the first sound grows fainter and the mitral systolic murmur disappears. The effects of exertion, and the secondary apex beat should be noted as confirmatory of the views here maintained.

CASE II. D. S., aged thirty-three. Presents himself for treatment for acute dyspepsia. He is free from all symptoms of cardiac distress. Inspection shows well-marked cervical and infra-clavicular pulsation. There is no distinct apex beat, but a slight systolic tremor about the nipple. The apex beat is found by palpation to be indistinct, and situated behind the sixth rib in the line of the nipple. Percussion proves the existence of hypertrophy and dilatation of the heart. Auscultation. Diastolic murmur at the aortic cartilage and over the sternum, down to the xiphoid cartilage. The murmur commences with a booming second sound which is equally distinct at the pulmonary and aortic regions. The first sound is very weak and is accompanied at the base, with a short pulmonary artery murmur. Over the body of the heart, and as far as a point two inches beyond the apex, the diastolic murmur becomes higher pitched and presents a distinct præsystolic accentuation.

CASE III. H. A. aged fifty-eight. The physical signs in this case pointed to an enormous hypertrophy and dilatation of the heart, with dilatation of the arch, and obstructive and regurgitant lesion of the aortic valves. The double aortic murmur was sawing in quality, and could be heard all over the sternum. At the fourth, fifth, and sixth interspaces along the left border of the heart, there was a low-pitched, rumbling murmur, which was recognized by all those who examined the case as distinctly præsystolic in time.

Post-mortem. The heart weighed twenty-one and one-half ounces. The aorta was dilated. The aortic valves were short and thickened, especially about the

edges. The intercoronary segment presented a button-hole slit, with thick, hard edges, the result of sclerotic changes around a congenital fenestration. The mitral leaflets were opaque, large, and slightly thickened, but evidently competent, non-adherent, and smooth. The mitral orifice admitted the introduction of three fingers. The right heart was much smaller than the left, and its valves were healthy.

There was, in the same ward of the City Hospital, an Italian, thirty-nine years old, with a loud, aortic, regurgitant murmur. Marked cyanosis and intense dyspnoea were prominent features of this case. The heart was moderately enlarged. The pulse was small and irregular. There was a loud, præ-systolic, churning murmur, circumscribed to the region of the apex. These features were contrasted with the somewhat diffuse character of the murmur heard in the previous case, and a diagnosis of organic mitral stenosis was made. Both the mitral and the aortic lesions were found at the autopsy.

It will be readily admitted that a mitral stenosis, whether organic or dynamic, may increase the obstacles to the circulation. But this must not be considered as invariably deleterious. The safety of an over-distended ventricle must occasionally depend upon this check to the inflow of blood from the auricle. I have observed elsewhere that it may be said of this safety-valve action, as of many other compensatory modifications of the heart's action, "that there is no step tending to cardiac impotency which may not be, at some time, or within certain limits, compensatory and salutary."

But the existence of an obstructive safety-valve action in the heart will be more readily admitted in connection with the pulmonary artery. It is not, however, my intention to dwell much upon this problem, which must be of very difficult solution. I shall pass on to the consideration of some murmurs heard in the pulmonary region, principally with the object of calling attention to their great frequency, and of presenting for discussion the subject of their causation.

Pulmonary systolic murmurs are far more frequent than all other cardiac murmurs put together. I have noted the presence or absence of this murmur in one hundred consecutive individuals. This group consisted of all the patients in my wards at the Charleston City Hospital and in the United States Marine Hospital, together with the applicants to the out-patient department of the Marine Hospital Service. Among the last, besides the ordinary dispensary cases, were included a number of healthy individuals, who applied for a certificate of fitness for sea-duty. Now, I found that sixty-two out of the hundred individuals presented a systolic, pulmonary artery murmur proper, either during tranquil respiration, or during respiratory movements especially designed to produce changes of the blood-pressure in the pulmonary arteries. If account is taken, not only of the pulmonary artery murmurs proper, but also of all the peculiar bruits of cardio-pulmonary rhythm heard about this region, the proportion becomes still greater.

The region of the pulmonary artery has been called the region of romance, says Balfour, "because of the various interpretations which have been given to the murmurs having their position of greatest intensity in that situation." These various interpretations, however, are necessary to account for the variety of murmurs heard in this locality. The anatomical reasons

for the frequency of murmurs in this region are: the proximity of the pulmonary artery to the surface; the thinness of the walls of the bloodvessel; the nature of its surroundings; and finally, and most important of all, the proximity of the main trunk to the capillary distribution. It is particularly this last factor that I propose to discuss, and I shall consider it first in the healthy subject.

I have found that a systolic pulmonary murmur can be developed in the majority of healthy individuals, provided we exclude those who possess very thick chest-walls, and those who are not intelligent enough to modify their manner of breathing, according to directions, during the examination; and I further hold that the said murmur is generally a dynamic, obstructive, valvular murmur. It is produced by the action of changes of blood-pressure in the artery upon the play of the semilunar valves. After reaching a certain degree of pressure, the blood in the pulmonary artery evidently must commence to impede the opening of the semilunar valves. The resultant of the two forces, one propelling and the other impeding the onward flow, must be a slanting position of the valves, and, consequently, a narrowing of the orifice, and the production of a sonorous vein or whirl.

The fact that these obstructive dynamic murmurs are much less frequently developed at the aortic orifice, I consider as confirmatory evidence of the views just expressed. If we take into account the greater power of the left, as compared with the right ventricle, and the wide distribution of the systemic circulation, it will be apparent that the aortic semilunar valves cannot be influenced by changes of systemic arterial tension with the same facility as the pulmonary valves are influenced by changes in the pulmonic circulation. There are, however, cases in which an increase of the general arterial tension is expressed, not alone by the accentuation of the second aortic sound, but also by an aortic systolic murmur. I have heard such a murmur in incipient atheroma and Bright's disease, where there was no marked anæmia; and probably all of us have seen cases in which a post-mortem examination failed to confirm the diagnosis of aortic obstruction made during life.

Returning now to the pulmonary artery murmur, as it is heard during ordinary breathing in the healthy subject, I have to say that it is almost exclusively circumscribed to the expiratory act. I hear at once the objection that it is simply a pressure-murmur resulting from the uncovering of the anterior surface of the heart by the receding lung; but this argument can be easily disposed of, for the murmur is loudest, not at the end, but at the beginning of expiration. It is loudest at the moment in which the change from expansion to contraction of the chest sends a wave of arterial pulmonary pressure back against the valves of the pulmonary artery: in fact, in many cases, the murmur is heard only at this moment; namely, with the first beat that occurs during the expiration. It is at this moment (when the blood is retarded in the right heart and in the large veins, just after the beginning of expiration) that the fall of the respiratory curve of the systemic arterial tension commences.

In order further to develop this murmur, and to show the influence upon it of disturbances of the pulmonary circulation, it is only necessary to arrest the respiration. By so doing, the murmur is often developed when it cannot be found during tranquil breath-

ing. It is better to stop breathing during expiration, and especially at the end of normal expiration. A full expiration makes the murmur louder still, but then it sometimes becomes higher pitched and harsher, suggesting the possibility of pressure upon the artery by the stethoscope, or by the heart itself (Quincke's murmur). At the end of inspiration, it is much more difficult to develop the murmur, for several reasons: First, because it requires a longer arrest of the respiration at this stage to produce engorgement of the main trunk, the capillaries being then in a state of dilatation; second, because a prolonged inspiratory effort is soon accompanied by a noisy hum of the intercostal muscles, which very much obscures all other sounds; and finally, because the interposition of the anterior border of the lung interferes with the transmission of any murmur that may be present. Even against these odds, the murmur is frequently heard in inspiration, if the arrest of breathing is pushed long enough.

The most frequent form of this murmur, as heard in ordinary breathing, consists of a soft, short, systolic bruit, of medium pitch, heard with one or two beats during the expiration. It is often detected only during some excitement of the circulation, as at the beginning of an examination, or when the patient stands.

It is very interesting to watch the changes that may be wrought upon the pulmonary second sound by a prolonged arrest of respiration: first, there will be an accentuation of the sound, then a reduplication, then a murmur, and finally both the murmur and sound will become indistinct. Thus indicating the several stages of intensity of the pulmonary obstruction.

Now, in pathological conditions these different changes may be found as evidences of the pathological alterations, for instance, in mitral and aortic lesions, tuberculosis of the lung, fibroid phthisis, emphysema, asthma, pleurisy, pneumonia, embolism; in all of which conditions the valvular murmur is frequently encountered as a manifestation of the obstruction in the pulmonary circulation. A very significant example is found in a reported case of pulmonary embolism, in which the sudden closing of one of the large pulmonary branches was immediately followed by the development of a systolic murmur in the main trunk.

It is not rare, however, to meet in some of these lung lesions with other species of murmurs. One of these, first described by Quincke, is produced by a retraction of the lung, which causes the pulmonary artery to be pressed by the heart against the sternum during the systole. I have occasionally heard this murmur in cases of tubercular infiltration and adhesions of the left upper lobe; but it must be admitted that the evidences of retraction of the lung are very slight in some of the autopsies of cases published in support of this view. When cases of pregnancy and of pneumonia, are made to do service for this theory, under the supposition that the imperfect pulmonary expansion present in such conditions, must cause an uncovering of the pulmonary artery, I am so far from being convinced that I hold to exactly the opposite opinion, and it is this: That in many cases of collapse or disuse of some portions of the lung, such as we find in pleuritic adhesions, extensive bronchitis, and, above all, in phthisis, there is a compensatory activity of other portions of the lung which may include those that lie near the left border of the heart. As we speak of superior costal and of diaphragmatic

breathing, we may speak, in the present case, of mediastinal breathing. The puerile respiration which is produced in these portions of the lung is interrupted by the cardiac movement simulating thereby a pulmonary artery murmur. It is heard generally with the inspiration, disappears with the arrest of breathing, and covers a broader area than the murmurs of the pulmonary artery proper. It is further a very short, high-pitched sound, which appears to originate very close to the ear, and is frequently mistaken with exopercardial friction sounds. A murmur of the same nature is quite common in children and in persons with narrow chests.

Some murmurs heard in the pulmonary region have been supposed by Naunyn and others, to be mitral regurgitant murmurs propagated to the surface by the appendage of the dilated left auricle. I believe that the arguments advanced by Flint² against a general acceptance of these views are incontestable. The murmur heard over the pulmonary artery in mitral insufficiency is, as I have endeavored to demonstrate, an obstructive valvular murmur, and it belongs with the accentuated second sound and other evidences of obstruction of the pulmonic circulation. The auricular murmur of Naunyn is, in my experience, exceptional. I have recognized it when the following signs were present: auricular dilatation, a mitral regurgitant apex murmur, and a long systolic murmur of low pitch, having its point of greatest intensity to the left of the pulmonary artery. If influenced by respiration at all, I found the murmur to be louder during inspiration.

It will be readily seen that in all these conditions there is more or less obstruction to the pulmonic circulation, and the presence or absence of the pulmonary valve murmur will depend solely upon the different degrees of power of the right ventricle to overcome the obstruction.

Now, are we not justified in assuming that there is a safety-valve action in this attitude of the pulmonary valves? A safety-valve action, which, together with the leakage in the dilated tricuspid orifice would tend to prevent engorgement of the lung by retarding the blood in the systemic veins where, for a time at least, it is less likely to do harm? This is certainly a difficult problem in mechanics; but I am inclined to think that the form of the valvular sinuses must give the artery, under conditions of pressure, a relative advantage in resisting the propelling force of the right ventricle.

There is still another murmur-like sound mentioned by Gerhardt, that may be heard in this most interesting region. It is commonly met with in excited conditions of the circulation, and is produced as follows: The thud of the cardiac systole is often of the same tone or consonant with the respiratory murmur, the latter thereby receiving a rythmical intensification from the former, and giving rise to a sound which simulates very closely the murmurs of the artery proper. These murmurs by consonance are generally heard during inspiration, and they, of course, cease with the arrest of respiration. They can also be made to disappear by modifying the tone of the respiratory murmur, which can be done by inducing changes in the laryngeal element of that murmur.

Whatever tendency an anæmic state of the blood may have to further the production of sonorous veins,

² American Journal of the Medical Sciences, January, 1886.

it must be considered operative also, in the conditions that I have attempted to describe in this paper. But aside from the fact that the influence of the changes of the blood is questionable, I think that there is evidence enough to show that the murmurs of anæmia find a sufficient explanation in disturbances of the valvular apparatus.

The fact that some of the cases included in my series of pulmonary obstructive murmur were cases of anæmia, does not invalidate the interpretation I have given of them. A considerable proportion of the anæmic subjects who present a pulmonary artery murmur will be found to have also a mitral regurgitant murmur. The fact that an obstructive pulmonary murmur, or an accentuated pulmonary second sound is found in such cases is evidence enough to show that the mitral systolic murmur is not simply a ventricular murmur or a modification of the first sound, but is a manifestation of actual bicuspid insufficiency. Functional, mitral and tricuspid insufficiencies are as a matter of fact quite common in anæmia, being simply the result of ventricular contraction with a diminished volume of blood. The pulmonary murmur accompanying this insufficiency must be valvular obstructive. The views of Naunyn, already discussed in this paper, in connection with organic mitral murmurs, will be found even less applicable in anæmia. For if the transmitted murmur is often absent when we have distinct evidences of auricular dilatation, why should it be present at all when, as in anæmia, there is no evidence whatever of such a dilatation?

There is another mechanism by which murmurs are produced, in anæmia, throughout the vascular system. We have in anæmia a reduction of the volume of blood. The diminished calibre of the circulatory channels without lessening of the stream velocity gives rise to the formation of sonorous whirls, because the valves require a certain amount of expansion of the vessels in order to apply themselves smoothly to their walls, without giving rise to some obstruction. The normal rhythm of these murmurs shows that they are intensified during the partial collapse of the jugulars; and I have noted in two cases the complete disappearance of the cervical hum, in anæmic females, during the general vascular engorgement that is noticeable sometimes before menstruation.

I hold therefore with Ducheck that venous hums, and basic murmurs also, are of valvular origin, not as he believed, that the valves themselves are the starting point of sonorous vibrations, but that they act as obstacles to the circulation, and producers of sonorous whirls.

The valvular theory of venous hums received a decided check in the fact that the murmur was found in cases where the jugular veins proved to be valve-less. But I have no doubt that in such cases the murmur is due to regurgitation into the jugulars. I occasionally see a young sailor who has but slight anæmia and who presents on the right side the loudest jugular hum that I have ever heard. It differs from the ordinary anæmic murmur in the fact that it grows feebler and may even disappear during inspiration. It is further distinguishable as a murmur, not of diastolic but of præ-systolic intensification (cardiac time). It is loudest, therefore, at the moment of the auricular systole, corresponding with the negative pulse of the veins. I have concluded that there must be in this case some defect of the jugular valves.

THE TREATMENT OF PHTHISIS BY INHALATION OF ANTISEPTICS THROUGH COMPRESSED AIR-VAPOR. REPORT OF EIGHT CASES.

BY EDWARD O. OTIS, M.D.

At the present time, something more is being attempted in the treatment of phthisis—I do not include climatic treatment—than the old-time opium, cod-liver oil, and lies, accompanied, on the part of the physician, with skepticism and consequent lack of interest, and on the part of the patient, with hopelessness or self-deception as to the final result.

The new methods, first of all, have given the physician a deeper interest, or *some* interest, in his consumptive cases, and more eagerness and hope in their management. Without some ray of hope in the prognosis, the doctor is of little good to his patient, and the visits of a cheery friend will probably produce better results, or, at least, are more to be desired.

The new methods are those of pneumatic differentiation with the cabinet; compressed air-vapor, with antiseptics; gaseous enemata; direct injections of antiseptics into lung cavities, as practised by Dr. White, of New York; and, in some few cases, incision into a lung-abscess and drainage.

During the winter, I have treated eight cases of phthisis with compressed air-vapor and antiseptics at the out-patient department of the Carney Hospital, and although so few to report, still they may afford some reflection as to this method of treatment. The instrument used was one manufactured by Messrs. Codman and Shurtleff, and kindly presented to the hospital.

The inhalations were given every day, or as often as the patients would come, and for about ten minutes at a time. The patients were under treatment from a few weeks to two or three months. Only two formulæ were used, those first suggested, I think, by Dr. Evans. They are as follows:

No. 1. R Carbolic Acid 3 iss-3 iij.
Borax 3 ii.
Glycerine 3 ss.
Aque distill. ad 3 iv.
M. Filter.

No. 2. R Co. Tinct. Iodine m 7.
Tinct. Conium m 15.
Glycerine 3 iv.
Aque ad 3 iv.
M.

The patients were taught to take full, deep inspirations, and told to practice this at home and in fresh air. General hygienic rules of living were given, and in nearly all cases, the compound syrup of the hypophosphites was administered.

CASE I. L. M., thirty-five years, widow. Husband died of phthisis; no family history of tubercular trouble. Has had cough in the morning, only, for four years. Muco-purulent expectoration; loss of strength; shortness of breath.

Physical examination: Dulness of the summit of the chest on the left side, with subcrepitant râles. Tubular respiration in a circumscribed spot at about the third rib. Roughened respiration, with subcrepitant râles at the summit of the right side. A few moist râles at the left base. Weight, 99 pounds.

She was under treatment between two and three months, but not every day. Her weight increased to 101 pounds, and she said she felt stronger and could breathe better. The physical examination, however, indicated no change.