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Ever since auscultation of the heart has been practised it has been known that heart murmurs frequently occur, which are not dependent upon any organic disease of the cardiac valves. Lænnec, the father of auscultation, described these murmurs as follows: "I have known a considerable number of persons to die of different diseases, acute and chronic, who have presented the 'bellows' murmur very distinctly during life, sometimes during several months, as well in the heart as in different arteries, and upon examination of their bodies I could discover no organic lesion coinciding constantly with the phenomena, which are not constantly met with in subjects who had never exhibited anything of the kind during life."

Since that time these murmurs have been the object of much investigation, and every one practically agrees as to their frequent occurrence, but, nevertheless, they are often apt to lead to mistakes involving great hardship to individuals who may thus be prevented from entering the services or from insuring their lives, or may be forced to live a restricted and semi-invalid life with the dread belief always present that they are suffering from heart disease. Sir William Broadbent, in an address[1] delivered before the Northwest London Clinical Society on October 20, 1897, alluded to this point, and said that young men are sometimes rejected, on totally inadequate medical grounds, after having obtained a place on the list at Woolwich or Sandhurst. He describes this class of cases so clearly that I give his description *in extenso:* "The candidate has usually been spending long and late hours in study with restricted exercise and limited fresh air and with possibly unlimited tobacco. He presents himself for medical examination in a state of extreme nervous excitement. His pulse is rapid and perhaps irregular, his cardiac impulse violent, and may be diffused even beyond the right sternal

[1] Lancet, Nov. 13, 1897.

border. Murmurs may be heard at one or more orifices. . . . It would take a great deal to make me reject the captain of a football team of a large school. . . . I have known such bruits to be looked upon as indicative of valvular disease requiring treatment by digitalis and demanding all sorts of precautions in the matter of exercise." All of us have seen examples of this class. But these murmurs may also occur in the apparently healthy, who have not been undergoing any debilitating process such as students do on the eve of examinations. Thus last week I saw the following case:

Case I.—A young man of healthy appearance and good build complained of palpitation on emotion but not on exertion. He was a civil engineer and lived a typically healthy, out-of-door life, and neither drank nor smoked. The condition had troubled him more or less since he first entered the University several years ago. As a student he was a good boxer and could stand a great deal of knocking about without distress, but while waiting to begin a boxing match, or in fact any physical or mental test, he was much troubled with palpitation, which, however, always wore off as soon as he got well into the struggle. In the neck a well-marked *bruit du diable* was present. The pulse was 120, but usually about 80, and varied greatly with posture. He noted this point himself and found that his pulse was 70 while lying down and 96 when standing. In the standing posture no murmur was present, but if he lay down a well-marked systolic murmur was audible in the second and third left intercostal spaces. He was not anemic.

One might describe such cases almost to any number, but this would serve no purpose. The subject may perhaps be best dealt with from three points of view: (1) A description of the murmurs which occur; (2) a short summary of the views held as to the physical causes of these murmurs, and (3) the diagnosis.

DESCRIPTION OF THE MURMURS

Inorganic or functional murmurs may occur in any of the cardiac areas, but by far the most common position is from the second to the third left intercostal spaces close to the sternum or a little external to it. While heard loudest at this point, these murmurs may be heard over most of the precordium, as far down as the apex and even to the right of the sternum. In an individual having such a murmur there will usually be present also a *bruit du diable* in the

veins at the root of the neck, and also murmurs in the large arteries; but with these vascular murmurs we are not now concerned. There is early and marked accentuation of the pulmonary second sound and such accentuation usually precedes the murmur.

In a well-marked case one may sometimes detect four distinct systolic murmurs over the precordium, one at each of the four cardiac areas. Functional cardiac murmurs are always systolic in time, and the importance of this point can scarcely be overestimated. In spite of an occasional statement found in literature to the contrary, it is extremely unlikely that a murmur occurring in any part of the cardiac cycle other than that occupied by the ventricular systole is of a functional nature, and the few cases placed on record in which diastolic murmurs are explained as functional must be considered as open to doubt. Functional murmurs accompany rather than replace the first sound of the heart and vary from mere impurities of that sound up to loud rasping bruits, but as a rule they are of a soft, blowing nature. The ones occurring away from the base of the heart may be distinctly post-systolic in time, that is, they occur during the short pause of the heart when the ventricle is still contracting, but the first sound has ceased.

Functional murmurs vary very much from time to time, being usually more marked when the heart is acting vigorously. They are much affected by the posture of the patient, being as a rule only slightly marked or even absent in the vertical posture and much louder in the horizontal. They are considerably affected by respiration and are louder during expiration than at other times. They are not propagated so extensively away from the point of their production as are murmurs due to organic disease. These functional murmurs are of extremely common occurrence, and it is surprising, if the heart be carefully and systematically auscultated in a series of individuals who are lying down and who are not supposed to have heart disease, how often one or more of these murmurs may be detected. I found them present in 60 per cent. of the inmates of the surgical wards of the Sick Children's Hospital, and Mr. W. S. Lemon (fourth year student) found them in 50 per cent. of patients taken at random in the General Hospital. Their ages varied from 5 to 84 years and in many cases they had been horizontal for weeks or months. In these cases the apex is usually slightly displaced to the left and upward. The cardiac dulness is not as a rule extended

laterally to any extent, but is enlarged upward, reaching sometimes to the second rib,—a point of importance which will be again referred to. An unusual amount of pulsation is generally visible in the left intercostal spaces near the sternum.

These murmurs occur very frequently in cases of anemia, whence the name hemic or anemic bruits often applied to them.

Case II.—A girl, aged 18 years, complains of fainting attacks and shortness of breath. Her blood count shows the red corpuscles to number about 3,000,000 to the cubic millimeter, while the hemoglobin is only 40 per cent. There is a loud *bruit du diable* in the neck and a well-marked systolic murmur heard chiefly on the left side of the sternum about the third costal cartilage. Under rest and treatment with iron she completely recovered.

But it is a great and yet common mistake to consider that these murmurs are limited to such individuals, and the clinician soon discovers murmurs in cases in which no abnormality of the blood exists.

Case III.—A young woman who suffers from well-marked exophthalmic goiter. Functional murmurs consisting of vascular ones in the neck and a well-marked systolic one in the pulmonary area are present, and yet the blood count shows the red corpuscles to number almost 5,000,000, and the hemoglobin to be 80 per cent.

Case IV.—A. B., a medical student, aged 22 years, complains of palpitation of some weeks' duration. He has been working hard at his books and is feeling run down and losing weight. Two weeks ago he felt faint and consulted a medical man, who told him he had heart disease with enlargement of that organ, and gave him tablets containing digitalis and nitroglycerin to take frequently. He has been distinctly worse since then, and the palpitation has been very troublesome. He has no special shortness of breath and no swelling of the feet. He does not smoke or drink. Present condition: He is a pale, anxious, thin youth with cold extremities; has lost twelve pounds in the last year. His pulse is rapid and slightly irregular. The cardiac impulse is markel all over the precordium and a good deal of pulsation, which troubles him much, is present in the epigastrium. The apex beat is one inch below the normal and half an inch outside the nipple line. There is a loud *bruit du diable* in the neck. At the apex the first sound is impure, but there is no conduction of the impurity into the axilla. At the base there is a loud systolic murmur and accentuation of the pulmonary second sound. The blood

is normal; the tongue foul. A diagnosis of functional heart trouble was made. He was put on a mixture containing strychnin, and, when his digestion improved, on malt and cod-liver oil, and later on plain cod-liver oil. He steadily improved and in six months all the murmurs had disappeared and he had gained eleven pounds in weight. That was three years ago, and he has not relapsed in spite of hard work.

Yet one author [2] writes thus: "Having found that there is a murmur and *from the absence of anemia* that it is due to organic cardiac disease, it is now necessary to determine at what orifice, etc." The italics are ours. Here the reader must assume that if he can exclude anemia in a given case of heart murmurs, the disease must be organic.

Further, it is a common experience to meet with cases of even pernicious anemia in which no impurity of the cardiac sounds can be detected.

Case V.—Mrs. C., a widow, aged 68 years, is suffering from a chlorotic condition, in that her hemoglobin keeps between 40 and 50 per cent., but the red corpuscles average 4,000,000. There is no leukocytosis. The spleen is much enlarged. The patient is so frail and weak as to be confined to bed most of the time. There are no murmurs present over the heart, even in the horizontal posture.

Case VI.—E. F. is a male hospital patient, aged 35 years, suffering from profound anemia, probably of pernicious type, although the diagnosis is not absolute. No murmurs are present over the heart or in the neck.

In all kinds of lowered general health occurring in the wake of some acute disease, or perhaps being nothing more than a "run-down" condition, these murmurs are apt to appear. Students working hard for examinations, women worried out of good health by domestic affairs, youths following indoor occupations and perhaps indulging too freely in tobacco, and in other ways possessing habits which tend to lower their general health, are especially prone to have these murmurs. They may also occur when no flaw in health can be detected as in Case I above mentioned. They are not common after middle life.

Even in the absence of anemia these murmurs may be associated

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[2] H. A. Hare, Practical Diagnosis, p. 289.

with symptoms referable to the heart, such as shortness of breath, palpitation, dizziness, and faintness, but there are seldom symptoms of real breaking down in compensation such as edema, cyanosis, and venous engorgement of the liver and other organs, and on physical examination the signs of marked dilatation of the heart are absent. On the other hand, the vaso-motor tone is usually lowered and the arterial blood-pressure is consequently low. There is generally a vaso-motor instability with a tendency to bounding aorta and throbbing of the carotids, and the extremities tend to be cold.

Case VII.—B. C., aged 20 years, a lithographer, has been complaining of palpitation, flushing, sweating, and trembling for about one year. He has grown rapidly recently; works nine hours a day at his very close occupation; easily gets out of breath. He is a pale, nervous youth, and weighs 126 pounds. A loud *bruit du diable* is heard in the neck, and a pulmonary-systolic murmur is well marked. Less marked ones are heard over the other three areas. A good prognosis of eventual complete recovery was given. He was put on Blaud's pills, and recommended to be in the fresh air as much as possible. The murmurs gradually disappeared, and two years later I made the following note: "Patient has been working hard all summer and has had no holiday; feels run down but no murmurs are now present."

Compensation for gravity in such patients is usually imperfect, and the pulse beats more rapidly than it should do in the vertical posture, rising perhaps 30 or more beats instead of the normal 10, as compared with what it is when the individual is horizontal. It has been commonly noted that debilitated people, for example convalescents from typhoid, have no cardiac murmurs while laid up, but as soon as they begin to go about and exercise themselves these develop.

When individuals possessing functional heart murmurs die, the chief thing found post-mortem is a dilatation of the right ventricle, this being largely confined to the conus arteriosus. The pulmonary artery is also dilated and the pulmonary valve is carried upward and outward, perhaps as high as the second left costal cartilage. Foxwell [3] quotes reports of 20 cases given by different observers, in

[3] Causation of Functional Heart Murmurs, Lancet, Nov. 4, 1899.

which the pulmonary valves lay on the average behind the second costal cartilage.

CAUSATION OF THE MURMURS

It may be taken for granted that the physical conditions necessary for the production of the mitral and the tricuspid murmurs will be the same, and further that the pulmonary and the aortic murmurs will similarly be due to the like physical causes, so that we need only discuss the causation of one of each kind of murmur, say the tricuspid, and the pulmonary.

Tricuspid Murmur.—Let us look at the tricuspid murmur first. The only physical condition which will produce a murmur at the tricuspid orifice during the systole of the ventricle is one allowing of regurgitation of the blood through the orifice into the auricle. The cusps which close the orifice are normal (if abnormal then we would not be dealing with functional heart disease), and therefore it must be the orifice itself which has become too large to admit of its closure by normal cusps. The part played by the papillary muscles is here ignored. The function of these muscles seems to be to steady through the chordæ tendinæ the cusps and prevent these being everted toward the auricles. A lessening of this action of these muscles might tend to allow of such an accident, but could not well give rise to a regular systolic leakage and murmur. The size of the orifice depends upon the muscular sphincter which surrounds it, and the part which the sphincter plays in the closure of the orifice is very important. Dr. Clifford Allbutt[4] mentions some experiments done by Dr. D. McAlister which showed that the auriculo-ventricular sphincter normally so nearly closes the orifice during ventricular systole that "we began to wonder whether valves were not luxuries rather than necessities; for the sphincter fibers contracting during the systole of the ventricle seemed to reduce the orifice almost to an imperceptible chink."

The leakage may be of two kinds: (1) The sphincter may be stretched along with the rest of the heart muscle, it being merely a part of the ventricular wall, or, (2) it may alone be enlarged. Stretching of the ventricular wall occurs acutely in the athlete after some violent exertion. It occurs more chronically in obstruction to the outflow of the blood from the ventricle, as in emphysema. In

[4] Clifford Allbutt's System of Medicine, vol. v. p. 507.

either case it leads to leakage at the orifice, the normal cusps not being able to close the abnormally large opening. But in functional heart murmurs signs of dilatation of the heart are largely, although not entirely, absent, and it seems probable that the leakage which occurs at the auriculo-ventricular orifice is not due to stretching of the sphincter from general dilatation of the ventricular wall, but rather to the relaxation of the sphincter,—hypotonus of the muscle associated and probably due to the same causes as the general relaxation of the vascular musculature. When dilatation of any hollow viscus having muscular walls, for example, the stomach, occurs, it arises from one of two causes, either an increased internal pressure, or a decreased tone of the muscular coat of the organ. Now in the case of the athlete's heart the right ventricle becomes enlarged and the tricuspid valve leaks because the intraventricular pressure is abnormally high, just as the normal stomach may become dilated from the imbibition of a large quantity of fluid. On the other hand, in certain weakened conditions the ventricles, and especially the sphincters guarding the auriculo-ventricular orifices, may become dilated *not* owing to increased intracardiac pressure, but rather to decreased tonus of the muscular wall, just as very frequently the stomach becomes dilated from decreased tone in its wall without any distention by contents. It goes almost without saying that if both factors be present, dilatation of a hollow viscus will occur with exceptional ease. Thus, if an individual with his heart in a condition of hypotonus run a race or otherwise exert himself, that heart will very easily dilate. Thus it is that in certain anemic or otherwise debilitated individuals functional mitral or tricuspid murmurs occur either without, or certainly with, only a slight amount of exertion.

Lænnec, strange to say, attributed the functional "bellows" murmurs produced at the mitral and tricuspid orifices to a spasm of these orifices. He does not seem to have noted the murmurs at the base of the heart at all.

Pulmonary Murmur.—Looking next at the functional murmurs heard over the base of the heart, we find that two distinct systolic murmurs occur here, one, a comparatively rare one, in the aortic area, and the other, a very much commoner one, in the pulmonary area. This latter is by far the commonest functional cardiac murmur that occurs. We may probably assume, as already mentioned,

that the physical conditions will be the same in either case, and so will only discuss the common or pulmonary bruit. There is much difference of opinion as regards the causation of this murmur, and some of the theories advanced are so fanciful that Balfour has somewhat cynically called the pulmonary area the "region of romance." One may classify the theories regarding this murmur into two groups: (1) Those asserting that the murmur is produced at or about the pulmonary orifice; and (2) those setting forth that the murmur is not of pulmonary origin at all, but arises at one or other of the auriculo-ventricular orifices and is merely conducted toward the pulmonary area. It might be mentioned in passing that Potain believed that *all* functional heart murmurs were cardio-pulmonary, that is, were produced in the lung by the movements of the heart.

Balfour and Naunyn believed that this murmur heard in the pulmonary area was really due to mitral regurgitation. In favor of this view it was urged that a pulsation could frequently be observed in the second left intercostal space farther out than the normal position of the pulmonary artery, and that frequently the murmur was louder over this outer pulsation than elsewhere. It was considered that this pulsation was due to the left auricular appendix, which, being dilated by the leakage through the mitral orifice, was thrust forward against the chest wall. To my mind conclusive arguments can be used against such a theory. In the first place, when a non-functional mitral incompetency exists, that is, one due to organic disease, the systolic murmur is best heard near the cardiac apex and is transmitted toward the axilla. When such a regurgitation exists, pulsation in the outer part of the second left intercostal space is not observed. Again, in cases of debility a loud murmur is frequently heard in the pulmonary region and another in the mitral area, and as the individual improves in health the latter disappears while the former persists for some time. I have again and again observed this clinically. Evidently for a time in such cases there is some mitral leakage which produces a murmur in the ordinary position, and as the mitral sphincter improves in tone this leakage stops and the mitral murmur in consequence disappears, and yet for some time longer the murmur in the pulmonary area persists.

A second theory is that the murmur is due to tricuspid regurgi-

tation, but this is not a very popular view and it is hard to understand why a tricuspid murmur should, in case of debility, be heard in the pulmonary region rather than in its own position, and further when debilitated patients develop tricuspid incompetence, as they often do, a murmur develops in the tricuspid area, that is, over the lower part of the sternum, and is accompanied by a true venous pulsation in the neck.

Thus it is most probable to my mind that the murmur is produced somewhere near the pulmonary orifice. If this be the case, what causes it there? It may be taken as proved that an altered condition of the blood will not *per se* produce a murmur at a normal orifice. Perhaps the belief in the hemic production of murmurs is the most commonly held one that exists, and yet it has been proved again and again both experimentally and clinically that a watery state of the blood does not, other conditions being normal, cause any vascular or cardiac murmur. Foxwell,[5] in the Bradshaw lecture for 1899, gave most convincing experimental proof that alterations in the character of the blood passing through a normally shaped heart would in no case cause a murmur, and as already stated and illustrated, we have all seen cases of profound anemia without murmurs, and, on the other hand, cases of well-marked functional murmurs without anemia. Skoda wrote in 1839: "It is not true that a watery state of the blood is a cause of murmurs, because in many cases one does not find it." In order to understand the pulmonary murmur it is necessary to look for a moment at some of the physical conditions which govern the production of murmurs anywhere, and here I must acknowledge my indebtedness to Professor J. C. McLennan, of the Physical Department of Toronto University, for kindly help given.

(1) Fluid of any kind flowing at any speed through a cylindrical tube will not cause a murmur, even if the tube be curved, so long as it retain its cylindrical form.

(2) Fluid flowing from a cavity into a cylinder will similarly produce no sound. This explains why no murmurs normally exist at the pulmonary and aortic orifices. Here the blood flows from a cavity into a cylinder, there being no constriction normally at the arterial orifices.

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[5] Lancet, Nov. 4, 1899.

(3) Fluid flowing from a cylinder into a cavity *may* produce a sound, but it is not likely to do so unless the flow be very rapid. Probably the murmur heard frequently over an aneurism is often dependent on the physical grounds of fluid flowing from a cylinder into a cavity.

(4) The figure *par excellence* which will most easily give rise to a murmur is one in which the fluid must flow through a constriction. This constriction sets up eddies and fluid veins in the blood which cause sound vibrations.

It is easy to understand now how a true stenosis of an orifice gives rise to a murmur, for here we have a cavity (the ventricle), a constriction (the stenosed orifice), and a cavity again (the normal artery beyond). But can we apply the same explanation to the inorganic murmur in the pulmonary region? I think we can. All that is necessary in order to produce the hour-glass figure that we require is that the pulmonary artery be dilated while its orifice remains of normal size,—but to this point we will return. Curiously enough Fagge, in discussing the functional murmurs which occur at the base of the heart in anemia, says: "The trunks of the two main arteries are supposed to be unable to retract, in correspondence with the diminished volume of the blood, to the same extent as the orifice through which the blood enters them." It is hard to understand how a fibrous ring like that at the orifice could retract, and furthermore the bulk of blood in anemia is not as a rule lessened.

Russell, of Edinburgh, believes that the pulmonary murmur is caused by the bending of the pulmonary artery round a dilated left auricle. He points out that in cases in which the murmur exists the conus arteriosus is enlarged upward so that the pulmonary orifice is carried upward and to the left, with the result that the pulmonary artery tends to be bent, as its distal end is a fixed point; and this bending is facilitated by the enlarged left auricle. Now it has been proved beyond all doubt by Foxwell and others by post-mortem results that the pulmonary orifice *is* displaced upward by enlargement of the right side of the heart from any cause and may even reach to the level of the second costal cartilage, but there is no reason to suppose that the left auricle is distended, much less that it is so distended that the pulmonary artery could be actually bent by pressure from it. In order to produce such a result the pressure in the auricle

would have to be higher than that in the pulmonary artery, and such a condition is extremely unlikely to occur. As a matter of fact at the moment of systole of the ventricle the auricle is probably empty or nearly so. Furthermore, bending of a cylindrical tube, as already stated, will not cause a murmur unless it be so acutely bent as to destroy its cylindricity.

A common belief is that a dilated conus arteriosus can by itself produce a murmur, but unless the pulmonary artery be also dilated we merely have a cavity opening into a cylinder, which, as already stated, will not give rise to a murmur. Foxwell thinks that it is a dilated conus arteriosus plus a dilated pulmonary artery which is the cause, but although probably such is the actual physical condition existing, the conus arteriosus need not be dilated in order to give a murmur, for its diameter is normally greater than that of the pulmonary orifice. If then the pulmonary artery alone be dilated, this, along with a normal conus arteriosus and pulmonary orifice will give us the hour-glass figure required. It is likely, however, that, as Foxwell points out, it is the dilatation of the conus arteriosus which leads largely to dilatation of the pulmonary artery. By its enlargement it moves the pulmonary orifice upward and this relaxes the strain on the artery and allows it the more easily to dilate. This dilatation of the pulmonary artery has been shown experimentally to take place six times as easily as that of the aorta, after due allowance has been made for the different tensions at which they work. (Foxwell).

Chauveau showed experimentally, many years ago, that a stream passing from the heart into a dilated vessel produced a sound, and such an experiment was easily repeated. A rubber tube 20 feet in length was introduced through the tricuspid orifice into the right ventricle of a bullock's heart and firmly secured there. A similar tube was tied into the pulmonary artery, which was cut as long as possible. The near end of the first tube was connected with a water tap and the far end of the second tube was partially closed. The tap was then turned on and the pulmonary artery was auscultated. It dilated gradually under the pressure and a roaring murmur was heard over it which increased in loudness with the dilatation. This experiment shows that a murmur occurs from a dilated pulmonary artery and that the murmur is louder in proportion to the amount of dilatation. The same experiment was repeated on the left side of

the heart, but the aorta scarcely dilated at all and practically no sound was produced. Taking everything into consideration it seems to me most probable that the common functional murmur, that is, the one heard chiefly in the second and third intercostal spaces, is produced at the pulmonary orifice and is caused by, and is therefore a sign of, a temporary dilatation of the root of the pulmonary artery.

DIAGNOSIS OF THE MURMUR

It is scarcely necessary to emphasize the importance of clearly differentiating these functional murmurs from those produced by organic disease, inasmuch as in most cases the prognosis and treatment are so different. As a rule no difficulty exists. Given an overworked, neurasthenic youth with a systolic murmur heard loudest about the third left costal cartilage, associated with a well-marked venous hum in the neck, and one can scarcely think of anything except functional and curable disease. But some cases are very puzzling if not quite undiagnosable, and in these we require to make use of every known test in order if possible to reach a right conclusion. I venture to give here a categorical list of such tests, compiled partly from literature and partly from my clinical observation, which may perhaps be of use.

(1) Functional murmurs most commonly occur during adolescent and early adult life.

(2) They are more common in males than in females, although there are many exceptions to this, and chlorotic girls are very prone to have them.

(3) They always occur during the systole of the ventricles, either accompanying or immediately following the first sound of the heart; that is, they are always *systolic* in time. Certain diastolic murmurs have been described by Cabot and others in which no organic lesion was present, but such are so rare as to be of no practical interest. It should take a great deal to make us diagnose a diastolic murmur as functional.

(4) While functional murmurs may occur over any of the cardiac areas, by far the commonest site is the pulmonary area and a little below this, say about the third left costal cartilage. A murmur occurring away from this point and unaccompanied by one here, should not be diagnosed as functional unless for some very special reason.

(5) A pulmonary systolic murmur due to organic disease is very rare except when of congenital origin. When due to organic disease, other signs, such as cyanosis, stunted growth, clubbed fingers, etc., are usually present, and the pulmonary second sound is not accentuated.

(6) The *bruit du diable* and arterial bruits heard in the neck (except in aneurism) are always functional; hence when a cardiac murmur is associated with such vascular ones there is considerable reason for believing that it too is functional. On the other hand there is no reason why organic valvular disease should not be associated with functional disease, and one often finds this to be the case. The functional element may clear up in time while the organic one persists.

(7) Functional murmurs are, as a rule, soft in character and accompany rather than replace the first sound. They may, however, be loud and rasping, and the pulmonary one is especially apt to be harsh in character.

(8) Functional murmurs are not so widely conducted as are organic ones and are seldom heard in the axilla.

(9) Functional cardiac murmurs vary more under different conditions than do organic ones. They are louder after exertion and during expiration, and they are markedly increased by the supine position, and in fact may only be heard while the patient is lying down.

The importance of posture as affecting cardiac murmurs was well emphasized by W. Gordon,[6] and I fully endorse his conclusion "that in describing and discussing murmurs, which posture modifies, the patient's position should always be stated." Zeehuisen[7] also emphasized this point. Foxwell writes thus in this connection: "The murmur in the pulmonary region is much more evident in the supine than in the erect posture, especially if it be listened for immediately upon the patient's lying down before the circulation has been able to accommodate itself to its new relation to gravity. So much is this the case that it is doubtful if it be not the dynamic rather than the static change in position which is the more important element in its intensification." That the horizontal posture in itself

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[6] British Medical Journal, March 15, 1902.

[7] Centralblatt für innere Medicin, March 11, 1899.

is an important factor is, however, shown by the murmur occurring so well in the children mentioned who had been for months horizontal.

(10) The pulmonary second sound is early accentuated, and this sign may occur before any murmur is audible. In true pulmonary stenosis no such accentuation is present.

(11) In functional murmurs there is usually little sign of hypertrophy or dilatation of the heart and the apex beat is not much displaced. A certain amount of cardiac dilatation and displacement of the apex beat is, however, quite common, the apex being usually displaced a little upward and to the left. Wybauw [8] has pointed out that some dilatation of the heart is very common in chlorosis and anemia, and Byrom Bramwell mentions the same thing.

(12) Cardio-respiratory sounds are sometimes mistaken for cardiac murmurs. They are produced in the adjacent lung by the cardiac movements and largely disappear when the patient holds his breath. As a rule they give rise to no difficulty.

(13) Functional murmurs tend to disappear as the patient improves in general health. This is not the case with organic murmurs, which are apt to become louder as the heart's action strengthens.

(14) Signs of breaking down of compensation are rare in functional cases, and such breaking down should always suggest organic disease of the valves or heart muscle.

Here it should be mentioned that the term "functional heart murmur" may be misleading in that, although nothing be actually wrong with the valves themselves, a great deal may be organically wrong with the rest of the heart. Thus in a case of fatty degeneration of the heart muscle with consequent dilatation, a mitral murmur may occur from enlargement of the mitral orifice. Such a case could not be called one of valvular disease, and yet the term functional heart murmur would scarcely suggest the serious condition present. Theodore Fisher,[9] in a paper read before the Bristol Medico-Chirurgical Society, states his belief that in most cases of even rheumatic valvulitis in which, at the post-mortem, vegetations

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[8] Journ. Med. de Brux, March 15, 1900.

[9] Lancet, July 18, 1896.

are found about the mitral valve, the leakage occurring during life was not due to the valvular disease, which often is evidenced chiefly by a row of small vegetations which could not possibly prevent the closure of the cusps, but was rather due to the associated dilatation of the mitral sphincter. In other words, we have a functional condition complicating the true endocarditis, and if care be taken not to strain the heart during convalescence the valve will again become competent in spite of the vegetations on the surface of the cusps. Dr. Fisher continues: "Dr. Caton treated several patients suffering from rheumatism, over whose hearts cardiac murmurs were audible, by rest in bed and blisters over the precordial region. Forty patients were kept in bed on an average of 41 days and in 29 the murmurs disappeared. Dr. Caton attributes the disappearance of the murmurs to the treatment of the endocarditis by the blisters, but it seems far more reasonable to suppose that the prolonged rest in bed allowed the dilated hearts to recover, and the murmurs depending upon the dilatation were consequently noted to have disappeared while the patients were under observation."

(15) Seeing that functional murmurs are so often found by accident, so to speak, as, for example, in examining for life insurance, it follows that a great many individuals have these murmurs unknown to themselves or their physicians. If now such individuals be attacked by rheumatic fever, scarlet fever, chorea or any of the other conditions in which endocarditis is apt to occur, we may be led to diagnose the more serious condition, although by close attention to the characters of functional murmurs we may generally avoid such an error.

(16) Fevers are very apt to give rise to functional murmurs. La Salle[10] found that these murmurs occurred in 66 per cent. of cases of scarlet fever in females between the ages of 15 and 25 years. He also noted that vasodilators, such as trinitrin, tended to increase these murmurs, which is evidence in favor of the theory that they are due to a relaxed state of the muscular wall of the heart and vessels. In rheumatic fever they are also apt to occur. How, then, are we to distinguish them from murmurs due to endocarditis occurring in these conditions? This cannot always be done, and in many cases we must wait and watch, in the meantime,

[10] Thèse de Paris, No. 9, 1898–1899.

of course, treating the patient as if it were of the more serious nature. But an important point here is that endocarditis usually occurs, if at all, during the first ten days of the rheumatic or other fever, while functional murmurs are apt to occur later when the tissues have become relaxed by the prolonged fever. Thus, the earlier in the case the murmur occurs the more likely is it to be due to organic disease. There are very many exceptions to this rule, especially in the direction of functional murmurs occurring earlier.

(17) No mention has been made so far of the effect of pressure by the stethoscope in altering murmurs. Some writers put considerable weight upon this and believe that functional murmurs are more easily affected than organic ones by this pressure. Sewall[11] states that all non-organic murmurs at the base of the heart can be stopped by pressure with the stethoscope. But I am not convinced that this is the case, nor indeed that pressure has any marked effect upon any cardiac murmur.

I have purposely avoided lengthening this paper by giving the details of many individual cases. Any practitioner can, I am sure, think of so many in his own experience that it seems unnecessary to give them.

In conclusion, I would express the belief, (1) that we all are too apt to conclude that the heart is organically diseased because murmurs are present, and (2) it may be added, that we too easily assume that the heart is organically sound because murmurs happen to be absent. Either error leads to bad prognosis and treatment.

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[11] Stethoscopic Pressure in Physical Examination of the Heart, New York Medical Journal, Dec. 4, 1897.