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Organic cardiac murmurs have always been distinguished from the accidental and the functional. It is self-understood that the first named are caused by actual, mainly valvular, diseases, and will remain as permanent as the anatomical changes which produce them. Accidental[1] murmurs should be called those which, though they appear to be cardiac, do not result from actual cardiac disorders. They may even be extra-cardial, and sometimes require great attention and repeated examination before their true character can be ascertained. They depend on adhesions between the pleura and pericardium, with or without those between the two pericardial layers; are not transmitted through the blood-current, not always rhythmical, not quite synchronous with the contractions of the heart, and not of equal strength and audibility. Some, according to their origin, are superficial, some distant, grating (pericardial), or soft (pulmonary), increase during expiration, and may stop altogether when breathing is intermitted. They are seldom heard posteriorly.

Changes in the shape of the chest-wall, such as annoy the heart, alter the character of the cardiac sounds. In three cases of rhachitical infraction of ribs Hochsinger observed distinct

[1] Dr. George W. Webster, of Chicago, in a paper read before the American Medical Association of 1899, proposes to do away with the discrimination of "accidental" and "functional" murmurs and to employ the former adjective exclusively. He claims that "it commits us to no theory of causation, indicates no pathology, and avoids a discussion of the question whether functional disturbances occur with pathological changes." But it is exactly this discussion which is urgently required. Without it the difficulties surrounding the etiology and nature of inorganic murmurs will not be overcome.

cardiac murmurs; Steffen the same in the common forms of rhachitical deformities of the chest. These forty years I had many opportunities of publicly demonstrating, with or without apparent hypertrophies of the heart, more or less marked cardiac murmurs attributable to the triangular or quadrangular shape of the rhachitical thorax, the walls of which, being no longer elliptical, touch a large surface of the heart. These murmurs are not always the same. Within a few minutes the well-marked cardiac murmur of a baby sitting erect or bent forward may change into a muffled sound when the patient lies down—the best proof of its resulting from the mechanical annoyance on the part of the chest-wall. Indeed, this muffled sound and the murmur differ only in degree. The former may often be produced by the pressure of the stethoscope on the flexible ribs of the young. Such observations, as noticed by Henoch, and also by Hochsinger, who, however, speaks of a coarse heart-sound only, may readily be verified, provided the age of the patient is taken into account. They will be the more positive the younger the baby and the more flexible the ribs. Now and then the cardiac sound may be changed by pressure over the pulmonary artery.

"Functional" should be called all those murmurs which cannot be explained by some anatomical alteration of a valve or of the myorcardium. The causes, however, which are responsible for the exhibition of functional murmurs are altogether too numerous. To say that no single theory covers their etiology is not doing justice to the case. Indeed, there are but few conditions of the heart and bloodvessels to which "functional murmurs" are not traced back. Thus the imagination, or the diagnostic skill—or its absence—of the writer had always a great deal to do with their alleged nosogeny. Protracted diseases and convalescences, losses and abnormal condition of the blood, all forms of anæmia, chorea, poisoning by alkalies or by acids, acute intoxications and long-continued eruptive fevers, septic processes, irregular contractions of the myocardium, degeneration of papillary muscles, minute disturbances of valves or of bloodvessels, chronic

myocarditis, fatty degeneration, are all charged with causing "functional" murmurs. Nervous influences also come in for their share. Undoubtedly strong emotions, excitement, mainly in the young and those very impressible, influence the heart in the most various ways, from temporary palpitation to change of structure. Prolonged emotional strain certainly has that effect, even to the extent as to cause distention, dilatation, and hypertrophy, through prevention of complete systolic discharge equally with physical overexertion. Before and after violent exercises of athletes Schott could discover under the Röntgen rays the different degrees of distention. This momentary distention, when exertion is demanded of an enfeebled heart, though otherwise healthy (for instance, in chlorosis), may lead to persistent dilatation. When the heart is no longer healthy, however (for instance, after infectious diseases, or in fatty degeneration, or in the various degrees of other myocardial changes), both distention and dilatation are more readily established. Nor are pathological alterations required to facilitate their development; for here fatigue, physical, emotional, or mental, renders muscles more flaccid and favors distention. As far as the heart is concerned, its muscular labor depends, moreover, on the amount of support it finds in neighboring organs. The inability of lying on the left side, which is experienced by most healthy people, is caused in this manner. In that position the heart is more flaccid and requires more exertion to overcome resistance, a fact which is best shown by the increase in the number of respirations of from 50 to 80 per cent.

Functional murmurs are described as soft and low, short or long, not always blowing, and are frequently combined with, or are the terminations of, a more or less normal heart-sound. In almost every instance they are systolic; in the adult they are mostly aortic, and then audible in the carotid; in the child they are more frequently found over the pulmonary or over the pulmonary and mitral regions. In regard to the locality and extent of their audibility there have been many differences of opinion; still, there

appears to be unanimity in regard to their inaudibility posteriorly in almost every case. A functional murmur may persist for weeks and even months, but it has not the uniform quality of an organic murmur. It is more or less soft, or loud, or prolonged. Many disappear quite rapidly, or, after having vanished, return. In this respect they differ widely from organic murmurs, which are more persistent as far as time and character are concerned. It should, however, not be forgotten that organic valvular murmurs may disappear either through recovery from endocarditis or through the establishment of compensation. But in these cases they diminish in loudness and duration very gradually only. Nor should it be overlooked that an increased frequency of the pulse, with its frequent and insufficient contraction of the heart muscle, and thin bloodvessels, and shortened valve excursion, conceals a murmur which was present when the pulse was slow, or which returns when a cardiac stimulant reduces the number of heart-beats.

Duplicated sounds should almost never be taken to be either accidental or functional. They are nearly always organic and of more value than Leube appears willing to assume, both the splitting of the second sound (gallop rhythm—V V) and that of the first (V V—" rappel " of the French). The former is often observed in aortic stenosis, in chronic nephritis, sometimes also in conditions of utter exhaustion, and in bad cases of chlorosis; the latter in mitral stenosis, sometimes with oliguria followed by polyuria. Both of them are rare in infants and in the very aged. A fine specimen of the last anomaly has been under my observation (Charles G., ten years old, with the diagnosis of mitral stenosis and chronic myocarditis) in my division of Roosevelt Hospital.

Vascular murmurs should not easily be mistaken for cardiac; as a rule, they are transmitted. They are very rarely confined to the arteries of the neck, either in the adult or in the young. The relatively large size of the carotid in the young, mainly in the rhachitical young, with its lowered blood-pressure, may give rise to an occasional soft murmur.

This infantile condition of the carotid (and basilar) artery accounts for the murmur which is often audible over the open fontanelle, and was (rather erroneously) attributed by Fisher (Boston, 1835) to rhachitis only; it is quite possible that the irregular shape of the rhachitical carotid canal contributes to the murmur, which has always been mentioned among the "functional." If, however, the large size of the artery, with its consecutive diminution of blood-pressure, or an anatomical change in the carotid canal, or both, cause the murmur, to what extent should we be justified in calling functional the murmur which is due to such tangible anatomical causes?

Venous murmurs should never be mistaken for those originating in the heart. They are frequent, mostly about the chest and neck, and generally found in anæmic adults, less so in anæmic children, still less in infants, and never, it appears, in babies suffering from atrophy. The jugular vein is a frequent seat of murmur, particularly when the bulbus v. jugularis is large compared with the size of the vein; in these cases the murmur is explained by the formation of a vortex. The v. anonymæ also exhibit murmurs, which are combined with those of the jugular, are heard on both sides of the sternum, and are not isochronous with the sounds of the heart. When such murmurs are complicated with those of the apparently normal heart in adults, it is mostly safe to claim the latter as functional; when in children, and particularly in small children, as organic, for the number of very young children that develop other than organic murmurs is small. That is why while venous murmurs are frequent in pernicious anæmia, leucocythæmia, scurvy, and hæmophilia of the adult, even when cardiac murmurs are still absent or not marked, they are often missed in those of the young child. In fifty cases of infantile scurvy I do not remember to have ever met them. Why should this be so?

The heart of the young is comparatively large, heavy, and healthy. Its weight in the newly born is 0.89 per cent. of the body-weight; in the adult, 0.52 per cent. In the newly born the cavity, however, is small, 23 c.cm., compared with

100 c.cm. at the seventh and 140 c.cm. at the fifteenth year. Its muscle is massive, equally thick on the right and left sides, the contractions rhythmical and energetic and quite frequent. That is why the valves, which are small and elastic, vibrate easily and quickly. During the first five years there is an increase of the heart in bulk and weight, but none in circumference. That is why, while the area of dulness in early age is extensive, the impetus is quite marked. The cavities dilate rapidly only after the fifth year, and the large arteries, mainly the carotid and subclavian, lose their disproportionately large size only after the seventh year.

This condition of things prevents a predisposition on the part of the infant heart to murmurs of any kind. Indeed, they are very rare in the first four years. In regard to this fact, which was clearly stated by me in 1888,[1] the authorities do not always agree. Fifty years ago Charles West expressed the opinion that they were frequent, but it is very probable he mistook or meant vascular murmurs. Gerhardt thinks they are rare; Biedert and Steffen, however, frequent. Bouchut believed them to be very frequent, under the impression that what he described as a proliferating endocarditis ("endocardite végétante") in the newly born must necessarily cause murmurs. What he so denominated was, however, nothing but Albini's valvular nodes, or the "blood-cysts" of Luschka and of Parrot, recently again described by Giovanni Berti ("noduli ematici delle valvule cardiache," 1898)—that is, small elevations on the lower side of the valves containing or depending on minute hemorrhages. They are very frequent and liable to disappear, but do not always do so, for I have seen many a case, and followed it up to advanced age, in which those nodules must have been large enough to result in the systolic murmur observed by me which proved persistent. They are apt to be on the mitral valve, are found in the newly born, and persist, and suggest the diagnosis of intra-

[1] Brooklyn Medical Journal, March, 1888: "The heart exhibits functional murmurs but seldom. Whenever there are murmurs present in the infant, it is safe to attribute them to organic disease rather than to mere functional disorder."

uterine heart disease; but are found in the left cardiac cavity, contrary to the rule according to which fœtal inflammation or arrest of development occurs in the right side, and do not result in either dilatation or hypertrophy. Such cases, the like of which I have an opportunity to demonstrate in my clinic perhaps once a year, do not seem to have been observed by Hochsinger when he absolutely and positively denied the occurrence of cardiac murmurs within the first few years of life. Surely these murmurs when found cannot be called functional; they are organic.

Still, these cases are exceptional, and do not controvert the fact that the normal anatomical condition of the infant heart is too powerful to admit the presence of merely functional murmurs. There are but few cases of undoubted functional murmurs in the infant on record. Thiemisch[1] claims one. He tells of an anæmic rhachitic baby of six months that died of pneumonia. A distinct systolic murmur was heard at the apex for a week before death; it was surely not extracardial, for it was distinctly heard in the intervals of respiration, and at the autopsy no valvular lesion was discovered. That is why the murmur is called functional. Still, we are told that the heart was slightly large (may be, within normal limits), and that the muscle of the right ventricle was very pale and flabby. This latter condition means a myocardial anomaly, which is quite capable of rendering cardiac contraction incompetent and irregular when it is localized on one side only. It is more probable that such a limited localization has that effect, while we may imagine that if the myocardial change were universal and equable, the contraction, though feeble, would also be equable.

In more respects than for merely anatomical reasons, the first years of life are peculiarly immune in regard to some of the changes which in advanced life give rise to murmurs. Tobacco, alcohol, tea, coffee, gout, and uric acid have not had time to work; hereditary syphilis does not attack the heart so

[1] Jahrb. f. Kinderh., vol. xli.

often as the acquired form; erythematous and fatty degeneration of the heart and of the large arteries are exceedingly rare; brown atrophy of the heart is uncommon; the coronary arteries are normal; the myocardium, with the exception of thin deposits found on circumscribed parts of the pericardium, mainly near the insertion of the inferior cava and on the apex, does not suffer until some infectious disease has had an opportunity to affect it. After all, it appears fair to assume that the appearance of murmurs, no matter of what name, requires the presence of some changes in the cardiac structure which in most cases should be ascertainable.

Murmurs are caused by congenital heart diseases, which are frequent. Generally these prove fatal within a few years, with the exception of defects in the ventricular septum and of the occasional cases of subacute or chronic endocarditis, which are sometimes met with in pale and puny children without a history of traceable causes, that may have been unrecognized rheumatism or some other infectious disease. That is the more probable the less the symptoms of rheumatism are pronounced in the young. As early as 1875[1] I could point out what has been confirmed since, that local pain and swelling, even fever, may be less marked in the rheumatism of the young than in that of the adult, and still endocarditis is more sure to come and more frequent—indeed, sometimes the first and almost only symptom. Besides, rheumatism, whether ushered in by pharyngeal infection or otherwise, is more liable to be monarticular in children than it is in adults, and, therefore, liable to be overlooked. That is why many a case of rheumatic arthritis has been taken for traumatic, and *vice versa*. A girl of seven years entered my ward in Roosevelt Hospital with an old double, very coarse, and hard mitral murmur and a painful left shoulder; had a new attack of endocarditis, followed by pericarditis and pneumonia in the two lower lobes; was still kept in bed, when four weeks afterward the left shoulder was taken, and had not left the ward

[1] Seguin's Lectures, vol. i. No. 11.

when, three weeks after, the left foot was attacked with a new endocarditis. In the intervals she was free of pain and fever for weeks in succession. Thus three successive attacks of rheumatism, between which she appeared to be rapidly recovering, were monarticular.

What I mean to emphasize is this, that the absence of a history of rheumatism or some other infectious disease does not prove the non-existence of the latter. The variability of the symptoms, the difficulty of diagnosis, the absence of intelligence or experience in the parents, are just as many impediments to the correct appreciation of the present murmur. A systolic mitral murmur in a small child may easily be taken for functional when there is no history to explain it. The fact is, however, that while "rheumatism" is vehemently denied, "growing pains" are admitted, either with equanimity or with pride.

Murmurs are often, probably mostly, occasioned by an uneven pathological endocardial surface or by the incompetency of a valve. This incompetency may result from structural change or from faulty innervation. Bloodvessels, also, in order to facilitate the production of a murmur, should have an uneven surface; mere narrowness does not cause it; for in several cases of congenital chlorosis in girls with narrow, but probably smooth, arteries, I never found a murmur, nor was there one in the case of a baby, fourteen days old, with narrow arteries, that was described by A. J. C. Skene in the *American Journal of Obstetrics*, 1876. In two others, five and seven years old, I did find in the aorta and carotids murmurs which persisted as long as the children were under observation, without other anomalies, and without an opportunity to make a thorough diagnosis.

The occurrence of cardiac murmurs in abnormal conditions of the blood appears to admit of no doubt in adults, no matter whether they are due to them alone or to structural or functional changes in the heart and bloodvessels. The latter—*i. e.*, functional changes—should be doubted as long as the altered function may be explained by the altered structure. In chlo-

rosis, scurvy, hæmophilia, leucocythæmia, and pernicious anæmia of the adult a murmur is seldom absent; in the same conditions of the child, particularly of the infant and young child, it is rarely present. When we remember the superior development and the undisturbed condition of the young heart we feel obliged to attribute the absence and the presence of the murmurs, as the case may be, to the condition of the heart and not to that of the blood. Thus we should not be too anxious to claim any of the murmurs observed in the above-mentioned conditions as "functional," to the absolute exclusion of an organic origin. Similarly do we find that chorea, for instance, in the adolescent and adult that had many years to develop the rheumatic or other infectious form of valvular disease, is almost always attended with a murmur, while there are many cases of the same complex of symptoms in the young not so attended. It is only in those cases in which a murmur appears and rapidly disappears, and is again observed after an intermission of hours or days, that we are fully justified in believing it to be functional only; for though it be the result of incompetence, either of muscular strength or of innervation, the structural changes, if any there be, cannot be very radical. Such differences are, to mention another instance, exhibited in bad cases of masturbation, which when excessive will cause a cardiac murmur in the adolescent or adult, but never once in the forty years during which I observed many hundreds of cases in the very young. Another instance is that of rapid growth, which, in the very young, does not result in a sufficient disproportion between the heart and the body to cause a murmur, while adolescents mostly exhibit it as a symptom of cardiac incompetency.

In all these cases it appears that it is the condition of the heart which causes the murmur, but neither the blood nor an abnormal process of general nutrition or development.

In chlorosis the sounds of the heart are variously changed; there is frequently a systolic and now and then a diastolic mitral murmur, not always persistent; the second sound is duplicated in bad cases only. Nor is the presence of fully

developed chlorosis required to yield these alterations; neurasthenic young women may exhibit the same changes; they are also found in some cases of lead disease. In most instances the patients are pale and nervous, the pulse is small; nose-bleeding may be frequent, but dropsy there is none. It appears, therefore, that nervous influences alone are sufficient to cause temporary murmurs. Indeed, there are those who retain the existence of a spastic contraction of the mitral orificial ring without any organic alteration. According to H. Audeoud and Ch. Jacob-Descombes,[1] Revilliod teaches that there is a temporary mitral and orificial narrowness. Constantin Paul describes a murmur, either soft or rough, over the pulmonary artery, complicated sometimes with a jugular and mitral murmur, which he attributes to the co-operation of anæmia and of spasm. Huchard assumes the presence of a spasm of the coronary artery with symptoms of angina pectoris; he also speaks of "pseudo-angina" in nicotinized neurasthenics. Foville discriminates two kinds of spasm, one of which terminates in palpitations, the other in syncope. In all their cases and those of a few others there were some symptoms referable to the alleged changes; in some there was an increased area of dulness; in others there were either pulmonary, or mitral, or vascular murmurs, which would not persist, but disappear and return.

Dombrowski speaks of an organic and of a functional insufficiency and stenosis of the aorta and of the tricuspid. Drasche, Heitler, and Dombrowski report cases of functional mitral insufficiency caused by feebleness of the myocardium. Among others, Austin Flint described long ago cases of presystolic murmurs referable to mitral stenosis, when there was much aortic insufficiency, but at the autopsies nothing mitral. Thus both spasm and incompetency are believed to cause murmurs. Both may be explained by insufficient innervation, and murmurs thus produced deserve to be called functional as long as the anatomical condition of the nerves whose

[1] Les altérations anatomiques et les troubles fonctionelles du myocarde, 1894, p. 119.

physiological action is at fault cannot be calculated or even estimated. The complexity of the anatomy and physiology of the centres in the medulla and of the peripheral nerve branches is so great as to render every attempt at exactly weighing their abnormal action perfectly futile.

Leaving the field of neurology we are on much safer ground when considering the normal and abnormal action of the heart, mainly in regard to the origin of murmurs, in connection with the condition of the heart muscle, which has not had the attention it deserved bestowed upon it until a few years ago.

Endocarditis and pericarditis have until a short time ago attracted the principal attention of clinicians. Indeed, the lesions of the myocardium were extensively studied by pathologists before their results were utilized in the interest of diagnosis and practice. And still there is no period of life in which the muscle of the heart may not be of pathological interest. Even coarse lesions may be found in early infancy, beside those congenital chronic cases of endocarditis and arrests of development which lead to cyanosis. In Gerhardt's *Handbuch*, twenty years ago, Dusch published fourteen cases of acute and seven of chronic myocarditis, and seven cases of aneurism of the heart, five of which were in the ventricular system, two in the wall, in children. Diverticula of the heart have been found even in the newly born, also intertrabecular defects; they were sometimes of syphilitic character.

Of the latter variety was that found by J. Arnold[1] in a syphilitic female one and one-half months of age. Syphilis will probably be found in many more instances of congenital heart disease than have been hitherto recorded. If so the lesions will not necessarily be confined to the right side.

In advanced age myocardial changes are frequent. Their causes are numerous; indeed, most diseases affect the heart muscle. Not to mention the results of thrombotic and embolic processes which lead to anæmic necrosis, to septic infarc-

[1] Virchow's Archives, vol. cxxxvii.

tions, or to fibrous myocarditis, we meet with acute myocarditis mostly in infectious fevers, accompanied with swelling of the intermuscular cellular tissue, minute extravasations, and sometimes fatty degeneration. The same infectious fevers, also the presence of endocarditis or pericarditis, may give rise to granular degeneration of the parenchyma of the heart muscle to such an extent as to obliterate the striated structure. This is the condition which was formerly attributed to the influence of excessive body heat only. Fatty degeneration, mostly of the left ventricle, depends on the failing nutrition of anæmia, cachexia, age, or fevers, and complicates changes in the pericardium and in the coronary arteries. Fatty overgrowth of the pericardium or between the muscular striæ is often found beyond middle life; brown atrophy, with its pigmentation mostly about the nuclei, which follows valvular disease and is met with in the senile heart; amyloid degeneration of the connective tissue; amyloid or hyaline changes in the bloodvessels, with their influence on the nutrition of the organ; and calcareous deposits, small or large—all of them are frequent, and many are found in autopsies after no symptoms pointed to their presence. Still, there are often symptoms caused by them. There may be dyspnœa or angina pectoris; the pulse may be feeble, irregular, frequent, or slow; the cardiac rhythm galloping, the sounds replaced by murmurs. Indeed, murmurs are a frequent result of myocardial changes. I have seen them coming and slowly going, when they could be explained by nothing else. That parenchymatous changes in the heart muscle, and still more that interstitial inflammations of the connective interfibrillar tissue, should get well under the influence of tonics and rest and medication, can be denied only by those who have not seen the different stages in the same processes—invasions and recoveries—in other organs. The correct estimation of myocardial changes in the living, however, is beset with peculiar difficulties, mainly in this, that they may be local and not accessible to percussion. Even thorough and universal myocardial alterations need not change the size of the organ.

A few conclusions appear to be self-evident:

1. The diagnosis of deranged function in any organ is only a makeshift, and justifiable only as long as we are ignorant of the physical cause of that derangement. A functional heart murmur is one the anatomical cause of which we do not know. That is why a skilled diagnostician may recognize fewer functional murmurs than one who will not diagnosticate a heart disease unless he have all the symptoms, including dilatation and hypertrophy.

2. The same disorders of the blood and nervous system in which heart murmurs are observed in the adult do not cause them in the small infant. In the latter the heart is larger, more massive, and more powerful, and its contractions are more uniform and effective; its two ventricles are almost equally muscular, and the valves are smaller. Thus the greater frequency of murmurs in the adult is attributable to the physical condition of his heart, and should not be explained by a deranged function.

3. Even in the present limitation of our knowledge we should agree to call functional only those murmurs which are temporary, or intermittent, or variable in their character. They are met with in the neurotic and neurasthenic, in the (adult) anæmic, sometimes in syncope or in chorea minor, and occasionally in rheumatism. Even here they should be recognized either as myocardial or as neurotic.