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ON SO-CALLED FUNCTIONAL HEART MURMURS.[1]

BY

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Among the signs of organic disease which perplex the diagnostician by appearing when the organism is in health, or at least upon its borderland, cardiac systolic murmurs take a prominent place. Their occurrence as significant of a purely functional disturbance without any underlying valvular lesion, is of course well recognised, but their frequency in this connection is perhaps scarcely appreciated by the profession. In the discussion on "The Prognosis of Cardiac Disease in its Bearings upon Life Insurance," led by Sir Wm. Gairdner at the recent meeting of the British Medical Association[2] systolic murmurs are barely mentioned among all the doubtful signs of cardiac disease complicating an "estimation of life." Yet it is chiefly in relation to such questions as those of life insurance that the subject is of special interest and assumes some proportions as a difficult problem demanding solution. Indicative on the one hand, of that form of organic disease which most seriously threatens longevity, yet, on the other, consistent with nothing more serious than a temporary lowering of the general tonus, a well marked systolic murmur is capable of plunging the conscientious medical examiner into most uncomfortable doubt. For the subject is unfortunately as obscure as it is important.

Not only is the murmur subject to all the variations governing the principles of sound, but further, one's conclusions can have but a relative worth, for they are rarely verified by autopsy, and can only be

[1] Read before the Montreal Medico-Chirurgical Society, Nov. 21st, 1898.

[2] British Medical Journal, Sept. 17th, 1898.

formed by continued observation of cases and by amassing an abundant material.

Some months ago, Dr. C. F. Martin suggested to me a statistical study of systolic murmurs as they occurred in the medical records of the Royal Victoria Hospital. I am indebted to the authorities of the hospital for their kind permission to carry out this suggestion and I have to thank Dr. Martin for much assistance in the arrangement and revision of the work.

It must be admitted that the term "Functional Murmur" as at present usually employed, is a misnomer. On the one hand, *all* murmurs are functional, whether due to organic disease of the heart or to a malady of the blood itself, inasmuch as they depend on an impairment of function of the valves, or the parts in their immediate vicinity, or else to eddies abnormally carried in the course of the circulation. On the other hand, what are usually called functional murmurs, are often, indeed very often, due to a degeneration of the heart muscle, be it in the cells of the wall itself or in the cells of the papillæ or trabeculæ.

It is difficult to define functional murmurs in any other way than as temporary murmurs occurring in a heart with no other signs of valvular disease, and which ultimately disappear. This will exclude the organic murmurs which from time to time cannot be heard at all even when extensive valvular vegetations occur, and will admit of the term being applied where chlorosis, typhoid fever, etc., occasion murmurs through the altered conditions of blood or heart muscle. Hence we would say that temporary parenchymatous degeneration of the myocardium produces functional (not organic) murmurs.

Leube[1] has recently analysed the various conditions under which systolic murmurs occur and the requirements for a differential diagnosis. His classification is, briefly, somewhat as follows:

1. *Accidental Murmurs:* Systolic, heard most strongly at the base; may or may not be transmitted; no pulmonary accentuation; no increase of cardiac area.

2. *Relative Mitral Insufficiency:* Pure systolic moderately loud murmur; a weak impulse; moderate pulmonary accentuation; moderately increased cardiac area to right and left; relatively small often irregular pulse (myocarditis or myo-asthenia); history.

3. *Acute Mitral Endocarditis:* Soft systolic murmur at apex; cardiac area slightly increased to the left; pulmonary second moderately accentuated; pulse and heart impulse relatively strong, coexistence of fever or of some infectious disease.

[1] Zur Diagnose der Systolischen Herzgeraüschen, Deut. Arch. f. Klin. Med. Nov. 5th, 1896.

4. *Chronic Disease of Mitral Valve:* Systolic murmur; generally louder and harsher; increased cardiac area; marked pulmonary accentuation; impulse moderately increased; pulse relatively strong and regular; frequent association with the signs of mitral stenosis; history of adequate cause for assuming organic cardiac disease.

Pure systolic murmurs then may be regarded as:

(a) *Valvular*, depending on an organic deformity of the mitral valve, or upon its relative incompetence due to so-called cardiac myoasthenia or to a myocarditis; and

(b) *Non-valvular*, accidental, or hæmic, heard best over the base of the heart and produced, according to most authorities, in the great vessels by lack of tone in their walls, or by lowered peripheral blood pressure. Such pure accidental murmurs when typical are supposed to present little difficulty in diagnosis for they are basal and unaccompanied by pulmonary accentuation or increase of the cardiac area, and claim differentiation only from aortic and pulmonary stenosis.

These latter are not easily confounded with basal functional murmurs. But with apical murmurs the matter is quite different and presents constant perplexity. Here it is necessary for prognosis to differentiate not only a relative incompetency from the true insufficiency of a diseased mitral valve; it is also most important to distinguish the permanent relative insufficiency of organic cardiac disease (primary dilatation with hypertrophy, myocarditis, etc.), from the temporary relative insufficiency of anæmic or febrile conditions where the valves fail to close simply from weakness of the papillary muscles and trabeculæ, or from dilatation due to lack of tone of the cardiac muscle itself.

From Jan. 1895, to Sept. 14th, 1898, the number of cases admitted to the wards of the Royal Victoria Hospital, were some 3,302; of these, I have examined the case reports of 2,780. All cases were rejected whose histories showed pure systolic murmurs known to be due to organic cardiac disease, whether pericarditis, myocarditis or endocarditis, and, as possibly organic, all cases where there was a record of previous or concomitant chorea, tonsillitis, acute and chronic rheumatism, arterio-sclerosis and acute and chronic Bright's disease. I retained for study 589 cases in which pure systolic murmurs occurred (21 per cent). After eliminating cases giving a previous history of rheumatism, nephritis, etc., or showing arterio-sclerosis from this number, there remained a total of 466 cases, or not quite 17 per cent. of pure systolic murmurs occurring in patients in whom there was nothing either in the previous history or in the general condition to suggest organic cardiac disease; with the excep-

tion of a possible old endocarditis based on a previous history of scarlatina which occurred in a certain proportion of cases, and which I noted but did not eliminate.

This (17 per cent.) is a high percentage, and it will be objected to it, that without doubt many of these are really cases of organic cardiac disease of insidious onset; but when one considers that in some of the many cases rejected because there was a bare suggestion of etiology, the murmurs may have been functional, and further, that, present as this sign often is when the patient is suffering from a malady that would never suggest it, the less marked murmurs must, even in these carefully kept records, have sometimes escaped observation, it is evident that this percentage of 17 per cent. must be pretty close upon the facts.

In the subjoined classification, various plans are adopted and the effort has been made to seek a diagnosis of the underlying physical condition. In functional murmurs, this is generally admitted to be an anæmia or a toxæmia leading, possibly through the nerve centres, to lowered vascular tonus and to consequent irregularities in the blood current.

I have divided all the conditions as follows:

1. Murmurs occurring in febrile and afebrile conditions.
2. Murmurs occurring in anæmic and non-anæmic conditions.
3. Murmurs occurring in pulmonary tuberculosis. This being kept separately as here the condition includes to a marked degree, fever with cachexia and anæmia.
4. Classification according to the nature of the sound with special reference to:

(*a*) Site of murmur.
(*b*) Transmission
(*c*) Condition of pulmonary second sound.
(*d*) Rhythm.
(*e*) Cardiac enlargement.

With reference to the *statistics* it is of note that:

In 466 cases with undoubted functional murmurs, 269 were in afebrile, and 163 in febrile cases.

Of the 269 afebrile cases, 185 showed anæmia; 74 none.

Of the 163 febrile cases, 85 showed anæmia; 78 none.

In 44 cases of pulmonary phthsis, 35 showed anæmia; 9 none.

The following *table* will indicate the main features it is desired to illustrate as suggested in a *few of the diseased conditions* with functional murmurs and the characteristics associated therewith. The relative frequency of murmurs in febrile and afebrile conditions and in pulmonary phthisis is also shown.

DISEASES.	Totals	Sub-totals.		Systolic Murmur at Apex.		Systolic Murmur at Pulmonary Area.		Systolic Murmur at Apex and Base.		Systolic Murmur at Aortic Cartilage.		Systolic Murmur Irregularly Situated.		Diastolic.	
		Anæmia.	No Anæmia.	Anæmia.	No Anæmia.	Anæmia	No Anæmia.	Anæmia.	No Anæmia.	Anæmia.	No Anæmia.	Anæmia.	No Anæmia.	Anæmia.	No Anæmia.
Anæmias.	87	87	....	24	....	21	....	36	....	5	....	1	....	4	....
Pernicious Anæmia.	16	16	....	9	....	1	....	5	....	1	....	....	....	..	....
Chlorosis	46	46	....	7	...	15	....	19	....	3	....	....	....	2	....
Leuchæmia	4	4	....	2	....	....	....	1	....	....	....	....	....	1	....
Hodgkins	3	3	....	1	....	1	....	1	....	....	....	....	....	....	....
Secondary to Hæmorrhage or C—a.	22	22	....	5	....	4	....	10	....	1	...	1	....	1	....
Typhoid Fever	78	31	47	9	18	12	9	5	15	3	3	2	2	...	....
Hysteria and Functional Nervous Disorders	64	30	34	13	14	11	8	5	5	....	1	1	4	....	2
Exophthalmic Goitre.	11	4	7	1*	2	1	...	2	3	....	1	....	....	....	1
Pulmonary Tuberculosis	44	35	9	11	4	12	4	8	....	1	1	2	....	1	....
Febrile Conditions	163	85	78	20	32	37	10	22	26	4	7	3	3	....	....
Afebrile Conditions.	269	185	74	67	36	47	16	49	7	9	6	7	5	6	4
Total number of Murmurs	466	305	161	98	72	96	30	79	33	14	14	12	8	7	4

* Tachycardia only.

It will be seen from the foregoing how frequently functional murmurs have been found when no apparent cause was discovered; neither anæmia, fever or other usually recognised cause; and further, that in site and rhythm of murmur some unusual conditions occur.

In the *true anæmias* are included, pernicious anæmia, chlorosis, Hodgkin's disease and anæmia secondary to hæmorrhage or carcinoma. These form 87 cases of which 24 have apex murmurs, 36 have murmurs at apex and base, while in only 21 were the murmurs mainly at the pulmonary cartilage.

The *origin* of the murmur cannot always be read from its situation; Dr. Heitler, in an article on "The Localisation of Systolic Mitral Murmurs"[1] makes an exhaustive study of this point. He states that his researches lead him to disbelieve the dogma that murmurs of different *timbre* are necessarily of different origin, even though they be heard less loudly in the interspace. He argues that murmurs are formed of a mixture of sounds which are not transmitted as a whole in any direction, but that certain parts are heard best where the conditions for transmission are most favorable, and he cites a case, seen post-mortem to be one of pure mitral regurgitation, where there was a loud musical murmur at the apex growing fainter towards the base and a loud murmur at the aortic cartilage of the same quality as that at the apex.

This is confirmed by other authorities in the case of regurgitant murmurs due to endocarditis where roughened surfaces combine with altered blood currents to produce a mixture of sounds, but in functional cases there are probably less complicated conditions. In these anæmias where the double murmurs which occurred, are often described as of different *timbre*, rough at one orifice, blowing at the other, they are frequently transmitted in two directions: into the axilla from the apex, and upwards from the pulmonary orifice, indicating that they really are the double murmurs of a relative mitral insufficiency and of a physiological pulmonary stenosis. Such at all events seems quite as plausible an explanation as any other.

Leube, in his article lays much stress on the site of the murmur. He states that in lesser degrees of blood alteration, accidental murmurs are produced in the great vessels at the base from lowered tonus of their walls through the action of impoverished nerve centres, while the more severe grades of anæmia and intoxication lead to a myo-asthenia or even a myodegeneratio cordis, and to the *apical* murmurs of relative mitral insufficiency.

The anæmic cases I have studied tend emphatically to confirm this

[1] Wiener Klin. Woch., No. 17, 1897.

dictum. In pernicious anæmia, representing the most extreme degree, we know that there do exist often extensive degenerative changes in the myocardium leading to weakening of the heart and the papillary muscles and to consequent dilatation. Here then are the conditions for a relative insufficiency, and here every murmur is apical. Of the 16 cases, 10 occurred alone at the apex and pulmonary cartilage, and the only one heard best at the pulmonary is transmitted into the axilla, indicating that it too is an apical murmur. In chlorosis, on the other hand, where there is a lesser degree of blood alteration, there are only seven cases at the apex against 15 at the pulmonary area, and 19 heard with equal force both at the pulmonary cartilage and apex. Of the seven cases with a murmur at the apex, five showed only slight chlorosis, scarcely sufficient one would think to produce a relative insufficiency; the thought is suggested that although mitral valvular murmurs are heard best at the apex, all apical murmurs are not necessarily valvular.

The apical murmurs of pernicious anæmia, however, do not always present the other cardiac conditions that we would expect to find in a relative mitral incompetency of some duration. The murmur is generally transmitted, but in only ten cases is there cardiac enlargement or pulmonary accentuation: but the absence of these signs is explained by the physical facts. We cannot expect much increase in area where the heart though frequently dilated, is atrophied rather than hypertrophied, and the right ventricle must often be too weak to give rise to pulmonary accentuation. In pernicious anæmia where pulmonary accentuation does occur (as in most advanced cases) it is probably dependent on the dyspnœa, due to the imperfect aëration of the tissues by the diminished number and power of the oxygen-carrying elements of the blood.

The cases of *exophthalmic goitre* illustrate the same point as do the anæmias. It is known that in this disease in cases where there is much vasomotor disturbance, there is apt to be dilatation of the over worked heart resulting in a relative mitral insufficiency. In the ten cases before me there is only one that does not show marked implication of the vasomotor system; in this one there is but a slight anæmia (erythrocytes 4,000,000, hæmoglobin (Fleischl) 70 per cent.) and the murmur is hæmic in its character being heard only at the pulmonary cartilage without pulmonary accentuation or cardiac enlargement.

In all the other nine cases there is vasomotor disturbance and the murmur is at the apex, generally well transmitted into the axilla. In five of the cases another murmur is also described at the pulmonary,

in two of these, this second murmur is probably hæmic in character for there is marked anæmia.

Typhoid fever may be taken as an example of a condition in which functional murmurs are extremely frequent, which contrasts strongly with anæmia. Here the more rapidly acting heart, the low tension pulse, the increased metabolism and evident intoxication of the system explain much more readily the strain on the cardiac system, of which the murmur gives evidence, than do the changes associated with a moderate anæmia. But, under such widely differing clinical circumstances, a common symptom makes one look for a common cause and the inference lies near, that in anæmia as in typhoid, a toxæmia rather than a hydræmia is at work in the one case acting slowly and insidiously, in the other, suddenly, acutely, and poisoning rather than impoverishing the nerve centres. Other facts suggest this idea also. Everyone knows that one meets with systolic functional murmurs often in apparent health, and in 78 of these 466 cases, there is present neither anæmia nor fever, but often a condition which suggests some form of intoxication. Instances of such conditions included among these murmurs are: 43 diseases of the digestive system (including six cases of cirrhosis), gout, acute alcoholism, morphinism, etc.

Out of about 298 cases of typhoid examined, a pure systolic murmur was noted in 78, making something over 29 per cent.

In this disease as in all infectious fevers, an apical murmur always suggests the possibility of an acute endocarditis: this is however, rare: Osler states that he did not find it in any of his cases and that it was present in only 11 of the 2,000 Munich autopsies Parenchymatous degeneration on the other hand is undoubtedly common, and yet though this is naturally often the main underlying cause for these murmurs, they often declare themselves functional in other ways, by disappearing during the active course of the disease.

Since the year 1896, minute daily records have been kept of the typhoid cases in the hospital. These "typhoid charts" reveal some interesting facts as regards the cardiac condition. When a murmur develops, although it frequently has a course parallel to the height of the fever, it often, too, appears and disappears quite irregularly without any apparent reference to this. In no condition of high continued fever are the variations in the cardiac condition more striking than in typhoid. The sounds vary in character and relative intensity; murmurs appear and disappear: signs of cardiac dilatation come and go, as the struggle between the reactive powers of the individual and the invading toxins of disease goes on in the organism.

The seemingly casual occurrence of these murmurs at the apex replacing those at the pulmonary and again a few days later giving place to them, differing too in no way from the basal murmurs in quality, makes one question Leube's statement that the hæmic murmur is always at the base and that a murmur at the apex is always due to a relative mitral insufficiency.

The transient murmurs of typhoid are best illustrated by a glance at two or three of the cases:

CASE I.—(Murmur corresponds with course of fever). A. B., admitted on 8th day of disease. High temperature until 25th day when defervescence began; normal temperature 41st day.

On admission, faint blowing systolic murmur at apex, not transmitted; louder murmur at pulmonary cartilage; faint murmur at aortic, accentuation of pulmonary 2nd sound. These conditions noted daily until 17th day when the basal murmur disappeared; that at the apex persisted; on the 24th day (commencing defervescence) this also disappeared. The heart was examined daily until the 62nd day and there was no recurrence of the murmur.

CASE II.—(Murmur ended with pyrexia but disappeared and reappeared again during its course.) C. D., admitted on 22nd day, temperature 105¼; high pyrexia until 42nd day; temperature slightly lower (102°) until 45th day, when defervescence began; temperature not normal until 95th day.

On entrance a blowing systolic murmur was heard at apex and pulmonary cartilage, noted for three days, on 27th day, no murmur at pulmonary nor at apex, but here lengthening of the 1st sound; on 33rd day, a faint blowing systolic murmur developed at apex, noted daily until 65th day when it disappeared; daily examination of the heart until 95th day revealed no return of murmur.

CASE III.—(Murmur at pulmonary cartilage during height of fever, interval with no murmurs; murmur developed at apex during convalescence). E. F., admitted on 27th day, defervescence began on 42nd day, temperature normal on 48th, no recrudescence.

On entrance, systolic murmur at pulmonary with pulmonary accentuation; this was noted every day until its disappearance on 36th day; on the 61st day (temperature normal; patient doing well in every way, still quiet in bed), a systolic murmur developed at the apex, heard as a rougher sound at the pulmonary, noted daily until the 69th day when it disappeared. On the 71st day (two days interval with daily note of no murmurs) murmurs were again heard at apex and pulmonary which persisted (daily note) until the 84th day. Patient discharged 87th day; pulmonary 2nd sound remained accentuated at exit.

Hysteria and functional nervous disorders.—In these, out of 62 murmurs 32 occurred in subjects who showed no anæmia; 14 of these murmurs occurred at the apex; eight at the pulmonary cartilage; five as double murmurs at both apex and base while one is heard at the aortic cartilage. Some of them bear all the characters of functional murmurs, being unaccompanied by other signs of cardiac disturbance, either enlargement or pulmonary accentuation. With no anæmia and no intoxication present, how are we to explain the temporary disturbance of the vasomotor system which the systolic murmur indicates? Is it possible that here, not poisoned nor impoverished, but disordered nerve centres are acting, and doing their part as inefficiently as do the higher inhibitory centres?

Lastly, with reference to the *nature of the sounds*, the following features may be of interest:

As regards the *site* of the murmur, it occurs at the apex in 170 cases, in 98 of which there is anæmia, in 72 none. It is at the pulmonary area in 126 cases. in 96 there is anæmia, in 30 no anæmia.

In 112 cases it is heard both at apex and base, sometimes with equal force, sometimes as a double murmur; in 79 of these there is anæmia, in 33 none.

In 48 cases the site is irregular (at the aortic or ensiform cartilages, etc.), 26 of these occur in anæmic and 22 in non-anæmic subjects.

In *character* the murmur is described as "soft, low, blowing," often "faint," sometimes "short" and "long," about 20 times; in three cases it is described as "musical," twice at the apex and once at the pulmonary area. It is described 42 times as "rough" or "harsh," and in all but 15 cases this applies to a murmur situated at the pulmonary cartilage.

Intensity is a point of some importance, for the murmur is in general low as opposed to the usually louder and harsher organic murmurs, but no hard and fast rule applies to anything about a functional murmur. "Loud" is applied to some 16 cases.

As regards *transmission*, murmurs heard at the apex are transmitted "towards the axilla" in 43 cases, and "into the axilla" in 73 cases. In 7 cases the murmur is described as heard "in the back" and "at the angle of the scapula." These cases are, 4 of chlorosis, 1 of lymphatic leukæmia, 1 of incipient exophthalmic goitre, and 1 of tuberculous meningitis. In none of these, except perhaps the latter, is there reason to suspect organic cardiac disease.

Pulmonary accentuation occurred in 191 cases, in 127 of which the pulmonary system was diseased. In about one-third of the cases, it was noted as "slight," more often it was present in anæmic subjects.

The *rhythm* of the functional murmur, with more special reference to the diastolic sound. Among the cases studied I have found 10 diastolic murmurs which I have not been able to reject as organic by any of the criteria I have used. They occur in the following diseases:

Chlorosis, splenic leukæmia, functional and cardiac disturbance, exophthalmic goitre, catarrhal jaundice, cerebral thrombosis, chronic bronchitis, and dementia.

So distinguished an authority as Strümpell states positively that he himself has observed a case of undoubted functional murmur which was diastolic in rhythm; and Sahli explains this (when occurring at the base) as a transmission of the *bruit de diable* from the neck. Leube, on the other hand, considers them so uncertain as to be better left aside in making any critical enquiry into the subject. He himself has never heard a diastolic murmur which he believes to be functional. Without discussing this disputed point, I quote a few of the cases:—

1. *Exophthalmic Goitre:* Blowing systolic murmur heard best at apex, heard also over præcordium and at aortic and pulmonary cartilages. Faint blowing *diastolic* murmur at pulmonary cartilage, heard down along left border of the sternum; pulmonary 2nd sound heard louder than aortic 2nd; dulness normal; apex beat normal position; 1st sound of heart heard at apex; pulse compressible.

2. *Phthisis:* Patient died 13 days after admission. Heart normal on entrance except for accentuated pulmonary 2nd sound, five days ante-mortem, systolic and *diastolic* murmurs were heard at 3rd right interspace, transmitted down right border of sternum; persisted until death.

3. *Splenic Leukæmia:* (Hæmoglobin 14 per cent.). Blowing systolic murmur at apex, transmitted to axilla; blowing systolic murmur at pulmonary transmitted upward; systolic and *diastolic* murmurs at aortic cartilage; pulmonary accentuation and 2nd sound at apex much accentuated; apex beat and dulness in left mammary line; pulse regular, tension low.

4. *Dementia:* Patient was a boy of 15, small and undeveloped; *diastolic* murmur at pulmonary cartilage; no enlargement of cardiac area or accentuation of pulmonary second sound.

Some *cardiac enlargement* was noted in 66 cases, and seemed gen-

erally to be consequent on a relative mitral insufficiency. In ten cases, relative dulness began at the right sternal border, in 30 cases it was noted at the midsternum.

General remarks: A functional murmur is usually, though not always, systolic in rhythm; while frequently at the base, it is very commonly situated at the apex; in the more severe degrees of anæmia or intoxication it is more common at the apex, in milder disturbances at the base. In intensity it is generally "low" and "faint"; in quality, "soft" and "blowing," especially when at the apex. Rough, harsh, functional murmurs are generally situated at the pulmonary cartilage. The murmur is often transmitted from the apex to the axilla, and, as due to a relative mitral insufficiency, may even be heard in the back. Moderate pulmonary accentuation is frequent. In those cases where the murmur is basal and appears to be accidental, it is generally associated with anæmia. Moderate enlargement of the cardiac area is fairly common, and points to a relative mitral insufficiency.

The cases studied illustrate especially the following points:

1 In cases of anæmia, pulmonary accentuation is often associated with a pure accidental murmur.

2. Functional murmurs frequently occur where there is neither anæmia nor fever. They are then often associated with some other condition suggesting intoxication.

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3. Diastolic murmurs have been noted which do not appear to have an organic origin.

4. Although accidental murmurs are generally heard at the base and those of relative mitral insufficiency at the apex, accidental murmurs are probably sometimes heard at the apex; (as in moderate anæmias where the murmur may occur at the apex unaccompanied by pulmonary accentuation or cardiac enlargement and disappear after a short time; or in high fevers where a murmur at the apex is replaced after a few days by one at the pulmonary cartilage of the same character.) On the other hand; murmurs produced at the mitral valve are occasionally, though rarely, heard best at the pulmonary cartilage.

Two conditions which it may be quite impossible to distinguish from each other by physical signs are:

A functional murmur at the apex with signs of moderate dilatation (relative mitral insufficiency), and an organic mitral murmur with signs of compensatory change. A decision can often only be reached

by considering the patient's general condition and the *persistency* of the murmur. This last is the clinching point and is the final criterion to which uncertain cases must be brought. In plain terms, *we must wait to diagnose the murmur until it is no longer there to diagnose.*

In this short paper such a large number of case reports have necessarily been dealt with in the most superficial way. Many points,—such as the persistency of the first sound of the heart—have not even been mentioned; and there remain the 123 cases where the previous history or present condition suggests organic cardiac disease; a comparison of these with the 466 cases glanced at in this paper where the murmurs were all apparently functional, would be very interesting. But a closer study of a subject such as this, is better combined with work at the bedside. The examination of these cases yields results which are useful chiefly as a basis for furthur study, and which become valuable when the conclusions drawn from them are confirmed by the prolonged observation of individual cases by a single observer. I hope at some future date, through the continued kindness of the authorities of the Royal Victoria Hospital, to be able to make further use of the material before me in a more thorough investigation of the subject.