AUSCULTATION OF THE HEART*

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Advances in our knowledge of heart disease in recent decades have generally been the result of the introduction of new techniques, either diagnostic or therapeutic. The discovery of the string galvanometer by Einthoven and the pioneering work of Sir Thomas Lewis in the field of electrocardiography clarified many problems of cardiac physiology and helped particularly to elucidate the various disturbances in the mechanism of the heart beat. Later the work of F. N. Wilson enabled us to resolve many aspects of heart muscle injury and added greatly to the diagnostic accuracy of myocardial infarction and other types of cardiac pathology. The increased use of the X-ray obviously facilitated the study of heart disease by portraying the actual size, movements, and configuration of the heart, its various chambers, and the neighbouring blood vessels. A further advance was made mainly by Sosman when calcified valves were visualized in living patients. Quite recently the electrokymograph has been introduced and although its application in clinical medicine is in its infancy, it promises to reveal interesting data about the normal and abnormal function of the heart. During the past several years venous-catheterization of the heart has been employed; it is already clear that certain diagnoses, especially in the field of congenital heart disease, can be made by this elaborate method, that were impossible without it.

Apart from the above, there are other important measurements that are often made in studying patients suffering from heart disease. Amongst these are the determination of the arterial and venous pressure, the velocity of blood flow, the vital capacity of the lung, the blood volume, etc. Sometimes one or another of these various tests proves valuable and even decisive in guiding the physician to the correct

diagnosis. There are times when the serological test for syphilis or the determination of the basal metabolism prove to be the main clue in establishing a sound diagnosis and in directing effective treatment. Many of these examinations are time-consuming and expensive, but they are often indispensable. Diagnoses can now be made by the use of one or more of these procedures that were completely beyond the scope of our most learned predecessors. Nor has this advance been entirely theoretical or academic. There are numerous instances in which modern methods of study have enabled us to recognize and even to cure conditions that are amenable to effective treatment which were formerly either unrecognized or incurable. It is the function of the wise physician to choose from amongst all these procedures the one or more, preferably the simplest, that are essential in guiding his treatment.

During this period of rapid advance in technology and in therapeutics as it pertains to heart disease, one method of examination has suffered—auscultation of the heart. The interest and energy expended in these other fields, although extremely profitable, have detracted from the attention given to the use of the stethoscope. In addition, there was a more legitimate reason for attaching less importance to auscultatory findings. During the period before the first World War, physicians attributed too great importance to certain cardiac irregularities and murmurs. Largely as a result of the teaching of Sir James MacKenzie, the significance of the symptoms of heart failure (congestive or anginal) began to be appreciated. He convinced the medical profession that many cardiac murmurs and irregularities were benign and compatible with long and vigorous lives. Physicians were prone to administer digitalis and to

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restrict greatly the activities of patients, even confining them to bed, because of the detection of abnormalities that we now know were entirely harmless. The change in viewpoint that took place some thirty or forty years ago was a great step forward.

However, all this led to discrediting the use and value of the stethoscope. I recall hearing MacKenzie say that the stethoscope should be thrown away, because it had done more harm than good. To be sure, auscultation sheds very little light on whether the heart is failing or not. The only stethoscopic finding that indicates heart muscle incompetency is a diastolic gallop or possibly an alteration of ventricular systole. But what must not be overlooked is that many patients have organic heart disease, especially valvular disease, for many years before they have heart failure. They often want to know and need to know, during these early years, whether they do or do not suffer from heart disease. In some of them auscultation is the simplest and occasionally the only method of answering this question, which is important notwithstanding the fact that during the period of normal compensation one would not advise cardiac medication or unduly restrict activities if a faint murmur of rheumatic aortic insufficiency is found. Such a patient might need prophylactic penicillin therapy for the extraction of teeth to protect him against bacterial endocarditis if aortic insufficiency were present, and would not if the heart were perfectly normal. This and other matters of considerable importance to the health of the patient depend upon the accurate diagnosis of apparently minor abnormalities that auscultation may reveal.

It is surprising that so little direct experimentation has been carried on concerning auscultatory findings. Our interpretation of the significance of cardiac murmurs has largely depended upon the correlation of physical findings in the sickroom with observations carried out post-mortem. This method has been very valuable but has definite limitations. We are unable to trace back and study the anatomical and pathological changes that were present years before the patient died, and explain the findings that were present when the heart was well compensated. Furthermore, at the autopsy table we see the atonic heart and cannot readily picture what the position of the valve leaflets might have been when the heart performed its function with the existing pressure relationships. Valves that appear normal or essentially so when observed directly, may have been incompetent when functioning in the living heart. We know that the aorta can be considerably dilated when viewed fluoroscopically and yet show normal dimensions post-mortem. This has

been called the hyperdynamic aorta. May not similar changes be taking place within the chambers of the heart?

Another point about which accurate data are lacking is the relationship between the size of a defect and the loudness of a murmur that may result. Under what circumstances does a loud murmur represent a slight valvular defect and when is the reverse true? It would seem logical that a very loud systolic murmur of aortic stenosis would accompany a high degree of stenosis and a similarly loud mitral systolic murmur might indicate a slight mitral insufficiency, because in each case we would expect the intensity of the turbulence to be greatest when the blood stream suddenly flows through a very narrow orifice. The situation does not appear to be the same with regard to aortic insufficiency, for here it seems that a faint diastolic murmur is present with a slight regurgitation. Some of these questions could be readily answered by direct animal experimentation.

The exact position and movements of the valves, especially the auriculo-ventricular valves, during various phases of the cardiac cycle is of considerable importance in the discussion to be taken up later. Most physicians look upon the closure of the mitral and tricuspid valves as occurring in a hinge-like fashion, like the closing of a door. If that were true, it would appear that during the very early part of ventricular systole, as the valve leaflets were being approximated, there would necessarily be some regurgitation before the valve actually closed. Yandel Henderson (1912) reported some ingenious experiments many years ago, supporting the view that closure of the A-V valves took place in a manner that would prevent this possible reflux. He believed that as a result of a "jet effect" the A-V valves were drawn close together directly after auricular systole, and with ventricular systole they finally closed by a mechanism similar to the unrolling of a carpet. In this fashion there would be no regurgitation during ventricular contraction. Furthermore, it is a matter of some importance whether the valve leaflets are driven deeper into the ventricular cavity and spread more widely apart by auricular systole or whether they attain a higher position and are drawn nearer together. It may be debatable whether the experiments of Henderson can be translated directly to events that occur in the normal human heart and whether they apply with equal significance to abnormal states. In any case, there seems to be little doubt but that auricular contraction alters the exact position of the A-V valves and will have a determining effect on the position of these valves at the very moment ventricular systole occurs.

THE FIRST HEART SOUND

Although many points about the mechanism of cardiac sounds and murmurs are still vague and meagrely understood, there are some clinical correlations of auscultatory findings with practical application that are not generally appreciated or utilized. Amongst these is the significance of the intensity of the apical first heart sound. It is generally taught that the first sound has two components, valvular and muscular. Experiments performed by Dock (1933) led him to the view that the first sound is entirely valvular. Observations to be discussed here indicate that if there is a muscular component it is so faint that it is essentially inaudible on ordinary auscultation. For present purposes, therefore, it can be assumed that the apical first sound is valvular in origin.

There are many occasions on which the heart sounds are decreased in intensity. It is obvious that in patients who are dying or moribund, when the heart is extremely feeble, the heart sounds become very faint. Likewise, patients with much obesity, emphysema, or pericardial effusion may have very distant heart sounds. In fact, any condition that interposes excessive tissue between the heart and the skin will necessarily decrease the intensity of the sounds as heard with the stethoscope. In all those conditions, both first and second sounds will be diminished. Excessive amounts of air or tissue between the heart and the external thorax will not affect one sound without producing a similar effect upon the other. What is more important is that the first sound can be altered (increased or decreased) without a simultaneous change in the intensity of the second sound. It is this phenomenon that deserves particular attention, for its detection enables the physician to make certain bedside diagnoses by simple auscultation that formerly were regarded as impossible without elaborate graphic methods.

The loudness of the first sound depends upon several factors. The vigor of ventricular systole is one of these, but by no means an important one. Many patients have serious organic heart disease and even failing hearts with perfectly good heart sounds. Contrariwise, a decreased first sound is common in perfectly healthy subjects. Of greater significance is the quickness or abruptness of ventricular systole. Hyperactive or loud sounds, particularly the first sound, are often heard in hyperthyroidism (Fig. 1 and 2), anæmia (Fig. 3), after a brief exercise, during certain infections (particularly rheumatic fever), and with some emotional states. In most of these conditions, if not in all, the ventricles contract briskly. On fluoroscopic examination one can often see a "snapping or hyperactive type of beat." With this

there is likely to be an increase in the velocity of blood flow. Generally this is determined by measuring the circulation time from a vein in the arm to an artery in the head. What determines the effect on the loudness of the first sound, and for that matter on the production of cardiac murmurs, is not the total speed of flow but the velocity of the blood stream within the chambers of the heart and the neighbouring great vessels. It is quite likely that this may be accelerated even when the total speed of flow as measured in the ordinary way is little, if at all, affected. One would expect that the duration of mechanical systole would be slightly shortened in these hyperactive states, and the increased intensity of the first sound would result from the abruptness of closure of the A-V valves. Many years ago Samuel Gee (1908), in discussing murmurs, stated "The loudness of the sound depends upon the swiftness of the flow: the quality of the fluid and the size of the orifice are of import only inasmuch as they exert an influence upon the swiftness of the flow." This mechanism not only affects the intensity of murmurs, but also of heart sounds.

Another condition in which the first sound is notably increased in intensity is mitral stenosis. The cause of this peculiarity has been a matter of much speculation. One naturally suspects that the anatomical changes in the valve itself may influence the loudness of the sound. However, there are other influences involved. In mitral stenosis, the papillary muscles are hypertrophied and the chordæ tendineæ are shortened. The valve leaflets are probably deeper in the ventricular cavity than normally. Furthermore, the left auricle is generally dilated or hypertrophied, and with the obstruction of the valve, the filling of the ventricle occurs more slowly and gradually. Finally, the ventricle may contract with a slightly smaller volume and more abruptly. The result of these various factors is that the valve may be at an abnormal position the moment ventricular systole occurs and, as we shall discuss shortly, this influences the intensity of the first heart sound.

A final factor that determines the loudness of the first sound, and one that I believe to be the most important, is the exact position of the A-V valves at the instant the ventricles contract. It was stated above that there is some difference of opinion whether the A-V are driven deeper and wider apart or higher and closer together by auricular systole. Although I assume the first of these two premises, the fact that auricular systole changes the position of the valves can hardly be doubted. The observations to be discussed and the arguments involved are equally applicable on the basis of either theory, for the main point is that the first sound is different

FIG. 2.—Girl, aged 18 years. Diagnosis thyrotoxicosis. Upper tracing while BMR was +34. Lower tracing about one month later, after propyl thiouracil therapy. BMR +9. Note great decrease in intensity of first sound (S-1) and also of systolic murmur (SM).

FIG. 1.—Normal heart sounds. Normal male, 40 years old. Note first sound (S-1) is louder than second sound (S-2) at apex, but the relationship is reversed at the base of the heart. This is the customary finding in normal hearts.

FIG. 3.—Woman, aged 68 years. Diagnosis pernicious anæmia. Upper tracing: hæmatocrit was 22.8. Lower tracing: Several months later, hæmatocrit 45.0. Note decrease in intensity of first sounds (S-1).

if the valves are in one position from what it is if they are in another. It will be assumed that the first sound is louder if the valves are deep in the ventricles and wide apart and is fainter when higher up and closer together.

If this theoretical speculation is correct, it should follow that the relation between the time of auricular and ventricular systole will have a profound effect upon the loudness of the first sound. Normally the ventricles contract 0.16 to 0.18 sec. after the auricles. Auricular systole, it may be assumed, propels the A-V valves into the ventricular cavities, and as the ventricles partially fill, the valves move upwards to a mid-position and then are abruptly closed as ventricular systole occurs. If the ventricles contract 0.08 sec. after the auricles, the valves will be caught at a lower position and the snap that results with ventricular contraction will be louder. However, if ventricular contraction occurs 0.22 sec. after auricular, the ventricles will have had a longer time to fill and the valves will be higher and more closely approximated. Closure will then result in a more feeble sound. In a word, the loudness of the first

sound, according to this reasoning, ought to reflect to some degree the length of the P-R interval.

We have, therefore, a means of estimating the P-R interval by auscultation of the heart. If conditions cited above like mitral stenosis, hyperthyroidism, anæmia, etc., which may increase the intensity of the sound, can be eliminated, an accentuation of the first sound at the apex strongly suggests that the P-R interval is shorter than normal. It has been found that the sound is loudest when the interval is about 0.04 to 0.08 sec. (Wolferth and Margolis, 1930). Contrariwise, if the sound is fainter than normal, the P-R interval is full or unduly prolonged (0.20 to 0.24 sec. or more). It does not follow that the first sound becomes increasingly weak as the P-R interval lengthens more and more. The intensity may be decreased just as much or more when the P-R interval is 0.22 sec. as when it is 0.30 sec. In estimating an increase or decrease in the loudness of the first sound it is well to compare it with the second sound. None of the above inferences can be drawn if both sounds are much decreased. One should compare the

loudness of the first sound with what one would expect to hear in that particular case, considering the intensity of the second sound, the shape and thickness of the chest wall, and whatever other conditions may affect the heart sounds.

Many auscultatory findings are often missed because auscultation is not carried out methodically. One should spend several seconds deliberately listening to one point at a time. First, one should listen to the first sound for several seconds, dismissing everything else from one's mind. Then the same procedure should be carried out for the second sound. This is continued for the interval between the first and second sound (systole) and finally for the interval between the second and first sound (diastole), listening for additional sounds or murmurs. In no other way can fine points in auscultation be detected, except by concentration on one of these four components at a time. Normally, the first sound is generally louder than the second at the apex, and the reverse is true at the base of the heart (Fig. 1).

It is surprising how accurate one can become in guessing what the P-R interval will be on the basis of the intensity of the first sound (Fig. 4). Repeatedly I have been able to predict this interval to within 0.01 sec. This information, so readily obtained by simple auscultation, has considerable practical importance. There is no other way of eliciting it except by the use of graphic methods or other complicated techniques. It would require obtaining daily electrocardiograms to detect some of the transient changes in the P-R interval that occur in myocarditis of rheumatic or diphtheritic origin. Furthermore, many hearts with a slight delay of the P-R interval may otherwise appear quite innocent, so that the general practitioner is not tempted to obtain electrocardiographic records.

The following is an illustrative experience. A man about 50 years old came to the out-patient department because he had fainted a few times. He was otherwise well, having no breathlessness or pain in the chest. He could work and walk quite well. Physical examination revealed nothing abnormal.

FIG. 4.—Upper tracing shows loud first sound (S-1) with short P-R interval (0.08 sec.). Patient was a man, 24 years old, with essential hypertension. Lower tracing shows almost inaudible first sound (S-1) with only slightly prolonged P-R (0.22 sec.). Patient was a 24 year old woman with Gaucher's disease.

The blood pressure was normal, the heart showed no enlargement, arrhythmias, or murmurs, and the carotid sinus was not unduly sensitive. On careful auscultation the apical first sound was extremely faint, though the second sound was normal. This important finding was entirely overlooked by the physicians who first examined him. When the same physicians were asked to re-examine the patient, paying particular attention to the first sound, they quickly realized that it was strikingly decreased in intensity. Then followed a further misinterpretation of the significance of this finding. When asked what a decreased first sound signified, the reply was that it indicated heart muscle weakness. However, this patient seemed fit and gave no evidence of myocardial incompetency.

It is obvious that many patients have grave myocardial insufficiency with normal first heart sounds and may have poor or even inaudible first sounds with perfect heart function. The old concept that a poor first sound means a weak heart muscle is entirely fallacious. In the case cited above, the interpretation was made that the P-R interval was delayed, possibly to 0.21 to 0.22 sec. An electrocardiogram was then taken directly, which showed the interval to be 0.22 sec. The practical point of this experience is that a simple auscultatory finding led me to suspect that the previous attacks of syncope were due to Adams-Stokes disease. Only later did we learn that he had previously been observed in a hospital during one of these spells, and there showed transient complete heart block.

Similarly it is possible to suspect that the P-R interval is unusually short. If the common causes

for an accentuated first sound like mitral stenosis, hyperthyroidism, etc., can be eliminated from consideration, a snapping first sound is very likely to mean that the P-R interval is less than normal (Fig. 4). It may be only 0.14 sec., but more frequently 0.12 sec. or less. It has been somewhat surprising that the first sound in cases of the Wolff-Parkinson-White syndrome, though somewhat accentuated, is not as loud as one might have expected with P-R intervals as short as 0.08 to 0.10 sec. This may be due to the fact that in this condition early excitation occurs generally in the right ventricle, and that closure of the mitral valve (the more important one of the two A-V valves) takes place at a normal interval after auricular systole. In any event, there are other instances of short P-R interval that are associated with attacks of paroxysmal rapid heart action and, therefore, simple auscultation may lead the physician to recognize such cases.

There is abundant proof available that changes in the P-R interval *per se* have a profound effect on the intensity of the first sound. In an experiment in which the heart rate happened to remain unchanged after the intravenous injection of 1.0 mg. of atropine, the P-R interval was markedly decreased while the first sound became greatly accentuated (Fig. 5). In this experiment the microphone and stethocardiographic mechanism was not disturbed in any way, so that the loudness of the registered sounds could be accurately compared. A very feeble sound, which was present because the P-R interval was delayed (0.28 sec.), became quite loud fifteen minutes later when the interval shortened to 0.18 sec.

FIG. 5.—Upper tracing shows faint first sound (S-1) with long P-R interval (0.28 sec.). Lower tracing shows loud first sounds (S-1) 15 minutes after 1 mg. atropine i.v. when P-R interval was normal (0.18 sec.). Auricular sounds (A) were detected in upper tracing.

and at other times strikingly accentuated. This changing intensity of the first sound in a slow, regular beating heart is practically pathognomonic of complete heart block. Our medical internes have been sufficiently alert in recent years to make the bedside diagnosis of complete block by means of this sign, even in cases in which the ventricular rate was not 30 to 40 but 50 to 75, as occurs occasionally in acute coronary thrombosis. The marked diminution, and at times the complete absence of the first sound in occasional cycles when the P-R interval is delayed, afford additional proof that the muscular component must be of very little importance in the causation of the first sound. These cycles are just as vigorous, and are accompanied by a peripheral pulse of the same volume as those producing a loud sound.

One would predict that if there were complete heart block and the auricles were fibrillating the first sound would maintain a constant intensity. This would necessarily follow because, although the ventricles are beating independently, the auricles are not contracting, and therefore would not alter the position of the A-V valves at the time of ventricular systole. That this is the case is well illustrated in Fig. 6C.

PAROXYSMAL VENTRICULAR TACHYCARDIA

There are other conditions in which the contractions of the auricles should produce alterations in the intensity of the first sound. In most cases of paroxysmal ventricular tachycardia, the auricular rhythm is independent of the ventricular and controlled by the normal sinus pacemaker. As would be expected, the intensity of the first sound varies in different cycles (Fig. 7). This auscultatory sign

was described some years ago (Levine, 1927), and may enable the examiner to distinguish the auricular from the ventricular type of paroxysmal tachycardia. In the common variety of paroxysmal auricular tachycardia (without heart block) the heart sounds are not only rapid (150–250) and regular, but are constant in quality and intensity. In ventricular tachycardia, the rate is also rapid (150–250), generally regular (though frequently slight irregularities may be present) but the intensity of the first sound may vary considerably. In fact, obvious alteration in the first sound may be detected when the ventricular cycles are perfectly regular. This feature, together with the failure to influence the ventricular rate by vagal stimulation, helps to distinguish, on simple bedside examination, the ventricular from other types of paroxysmal rapid heart action.

AURICULAR FLUTTER

The character of the first heart sound has recently been investigated in auricular flutter. In many cases of auricular flutter before therapy is instituted, the ventricular rate is exactly one-half of the auricular. Under these circumstances every other impulse from the auricles is blocked. The rhythm of the auricles is perfectly regular though rapid (250–350), and the ventricular response is also perfectly regular (125–175). The interval between the auricular impulse that is not blocked and the subsequent ventricular contraction is constant from cycle to cycle. The result is that the first heart sound is constant in character, just as it is in paroxysmal auricular tachycardia or in normal sinus tachycardia (Fig. 8). However, there are instances of flutter in which the ventricular rate, though rapid,

FIG. 7.—Man, 63 years old. Diagnosis paroxysmal ventricular tachycardia. Note changing intensity of first sounds (S-I) with auricles and ventricles beating independently. Note loudest first sounds come with short P-R intervals.

is not absolutely regular. Slight differences may be detected in the interval between the auricular and ventricular impulses in different cycles. This would necessitate alterations in the intensity of the first sound (Fig. 8 and 9). Such changes have been observed, and have been shown to be due to the variation in the A-V relationships and not to the length of the diastolic pause (Harvey and Levine, 1948).

A further auscultatory finding in auricular flutter is the detection of auricular contractions. Not only may these faint sounds be audible during the long diastolic pauses when the ventricles have slowed, but they may be heard during systole (Fig. 9). Occasionally they are quite loud and confusing so that the observer may be misled into considering the mechanism as due to auricular fibrillation. A well known simple bedside method of distinguishing the irregularity of flutter from that of fibrillation is the effect of exercise. When the heart accelerates after a brief effort, the irregularity of flutter is likely to change to a more rapid but regular rhythm while that of fibrillation continues to be irregular though more rapid.

A peculiarity of auricular flutter that is less well known is the response to vagal stimulation. When the heart rate as auscultated over the præcordium is perfectly regular and rapid (140–175), it is often possible to slow the ventricular rate temporarily by carotid sinus pressure or other means of vagal stimulation, such as holding a deep breath. In auricular flutter, after a brief period of slowing, the original rapid rate is often resumed in a *jerky* fashion (Fig. 9). In paroxysmal auricular tachycardia without block, vagal stimulation will have no effect whatever or completely arrest the attack. In normal sinus tachycardia, if any effect is produced, there will be temporary slowing with a *gradual* return to the previous rate, and in ventricular tachycardia carotid sinus pressure will never alter the rate. The temporary slowing and the *jerky* return of the previous tachycardia is fairly characteristic of auricular flutter, and helps the physician to diagnose this condition by simple bedside examination.

The above auscultatory findings prove very useful, not only in the original recognition of auricular flutter, but in following its progress under treatment. While administering digitalis or quinidine, it is often important to increase, decrease, or omit the drug depending upon the changes in the arrhythmia. It might be necessary to take frequent cardiograms to know whether flutter is still present or whether the rhythm has reverted to normal or to fibrillation. A day or two after administering quinidine or digitalis to a patient with flutter, for example, the heart may

be found quite regular at a rate of 70. One might readily conclude that the mechanism is normal, but in point of fact flutter may still be present with an auricular rate of 280 and a pure 4:1 block. A brief exercise test is likely to clarify the diagnosis. If flutter is still present, the rate may quickly jump to 140 and remain regular for a short interval or become temporarily irregular and rapid (Fig. 9). If the original slow regular rate had been due to a normal sinus rhythm, temporary acceleration would have occurred smoothly and could only by accident have reached the rate of 140, which is an exact multiple of the original rate of 70. These simple guides may quickly enable the physician to make decisions, and will save considerable time and expense.

PULSUS ALTERNANS

Pulsus alternans is a phenomenon detected in the peripheral arteries in which the pulse, though perfectly regular, alternates in volume. It is generally believed to indicate a fairly grave disturbance in myocardial function. It is even more easily detected on auscultation below the pressure cuff when determining the blood pressure. Here the sounds will alternate in intensity, and when the condition is very marked, only the stronger of the alternate beats will be audible at the very highest levels of pressure. What has not been currently appreciated is that the same phenomenon may be detectable on auscultation of the heart. Not infrequently the intensity of the heart sounds or of an accompanying systolic murmur may alternate from cycle to cycle (Fig. 10). When this is heard, it has the same significance and is due to the same disturbance in contractility as obtained when pulsus alternans is observed in the peripheral arteries.

GALLOP RHYTHM

In identifying a gallop rhythm by auscultation, it is always important to distinguish the systolic from the diastolic type. Whereas a gallop in which the extra sound occurs between the second and first normal heart sounds is almost always a pathological finding and of grave significance, when the extra sound occurs between the first and second sounds, it is a benign phenomenon (mid-systolic click or gallop). It may be heard in some normal subjects, and when it accompanies organic disease, it does not add to the seriousness of the prognosis. There is a simple method of determining the fact that the sound is present in mid-systole. After identifying the three sounds at the apex of the heart, the stethoscope is rhythmically placed higher and higher over the præcordium. The middle one of the three sounds will be found to wane gradually and finally disappear on approaching the aortic

FIG. 11.—Systolic gallop disappearing at base. (Timing of gallop by “inching.”) Woman, aged 26, with question of epilepsy. No organic heart disease. Note benign systolic gallop sound (G) occurs between first (S-1) and second sound (S-2), loudest at apex and gradually disappearing on reaching base. S-1 decreased because of long P-R (0.24 sec.).

FIG. 10.—Upper tracing: Man, aged 61 years, with hypertensive heart disease. Note marked alternation of apical second sound (S-2). Lower tracing: Woman with calcific aortic stenosis. Note alternation of systolic murmur (SM). Loud = L. Faint = F.

area (Fig. 11). One will be left with the two normal sounds, making it clear that the sound that was audible at the apex and lost at the base of the heart occurred in mid-systole. This method of "inching" is helpful not only in identifying the types of gallop but also other events that occur in different parts of the cardiac cycle.

SYSTOLIC MURMURS

The interpretation and significance of a systolic murmur still remains a puzzling problem. It is quite true that many systolic murmurs (generally of slight intensity) are heard when there is no abnormality of the heart and, in fact, in those who have no structural disease whatever. In others, though there may be minor abnormalities; normal health and life expectancy is maintained. However, it is also true that many systolic murmurs detected in otherwise healthy people and regarded as benign or inconsequential, in later years have proved to be due to some structural abnormality. I look back at instances of this sort in which a congenital abnormality such as pulmonary stenosis or auricular septal defect was subsequently found by venous catheterization, which explained the "functional" basal systolic murmur. Others, ten or twenty years later, proved to have calcific aortic stenosis. Still others that had a slight apical systolic murmur developed unequivocal evidence of mitral stenosis or bacterial endocarditis. I believe it is fair to say that, whereas much too great emphasis was attached to the presence of a systolic murmur a few decades ago, too little attention has been paid to it in recent years.

It is not the purpose of this discussion to go into the mechanism or interpretation of the systolic murmur. However, I would urge that consideration be given to its detection and its possible significance, and that particular attention be paid to its intensity. Although faint systolic murmurs may be heard in otherwise healthy subjects, loud ones almost always denote some disease, generally of the cardiovascular apparatus. For this reason, I have become accustomed to grade the intensity of systolic murmurs from one to six. Grade one is the faintest and will generally not be audible until the examiner has listened for several cycles. Grade six is the murmur that is sufficiently loud to be heard with the stethoscope just removed from the chest wall. The intervening grades (2 to 5) are of intermediary intensity. Although at first glance this may seem a cumbersome terminology, it is surprising how quickly different observers will fall in line with each other, and rarely differ in their terminology by more than one gradation. The result of the use of such terminology is that one observer can more readily

understand and compare the findings of another with his own. Furthermore, it enables the physician to divide systolic murmurs into those that are likely to be associated with organic disease (grade three or louder), and those that commonly are found without structural disease of the heart (grade one or two).

There appears to be certain misconceptions in our current teachings concerning the transmission of murmurs. There is a prevailing opinion that murmurs are transmitted by or with the blood stream. One frequently is told that a particular systolic murmur heard at the base of the heart is transmitted to the carotid artery, or another at the apex region is transmitted to the axilla. These observations are made as added evidence that the murmur in the first instance is due to aortic stenosis, and in the second instance is due to mitral insufficiency. There is now abundant proof that the transmission of murmurs is mainly a function of its intensity, and that bony structures are the best peripheral conductors. Very loud murmurs, no matter what their origin may be, are propagated in all directions, and may be well heard over the carotid arteries or in the axilla. A loud systolic murmur of Roger's disease or pulmonary stenosis may be well heard in the carotid arteries (Fig. 12). Here the flow of blood producing the turbulence is within the heart or chest and not out into the peripheral circulation, and yet the resultant murmur is transmitted to the neck. In fact, very loud murmurs (systolic or diastolic) are easily audible over the olecranon process of the elbow, even when arterial flow to the arm is completely occluded (Fig. 13). This is convincing evidence that the propagation of murmurs is through bones. The conclusions from these observations is that the murmur of aortic stenosis is transmitted to the neck because the point of maximum intensity of the murmur is near the neck, and that the mitral systolic murmur is well heard in the axilla because it is near the axilla.

MISCELLANEOUS OBSERVATIONS

There are numerous other auscultatory findings of practical importance that can be easily elicited, which are beyond the scope of this discussion. Careful attention to the exact timing of the murmurs of mitral stenosis throws light on many otherwise baffling problems in diagnosis. The importance of the degree and duration of ventricular filling on the murmur of mitral stenosis has not been sufficiently emphasized. The presystolic murmur of mitral stenosis can disappear even when the auricles are contracting normally, if the diastolic interval is sufficiently long (Fig. 14). Not infrequently patients are observed who show no murmur whatever in

FIG. 13.—Murmur of patent ductus arteriosus. Woman, 23 years old, with patent ductus arteriosus. Note loud continuous murmur in upper tracing from pulmonic area. Middle strip shows systolic murmur (SM) at olecranon process with blood pressure cuff inflated to 250 mm. Same murmur readily heard at elbow with cuff uninflated (lower tracing).

FIG. 12.—Woman, 29 years old, with tetralogy of Fallot. Upper tracing shows loud pulmonic systolic murmur. Lower tracing shows same systolic murmur over left carotid artery.

FIG. 14.—Effect of long diastole on presystolic murmur of mitral stenosis. Woman, 26 years old. Diagnosis mitral stenosis. Note classical presystolic murmur (PM) disappears with long diastolic pauses following carotid pressure.

FIG. 15.—Diaphragmatic flutter. Woman 32 years old with mitral stenosis and diaphragmatic flutter. Upper tracing from apical region shows numerous sounds, a combination of normal heart sounds (S-1, S-2) and flutter sounds (F). This was at first clinically interpreted as auricular fibrillation. Note that flutter sounds (F) were very prominent in right axilla (middle tracing) and were entirely absent over right carotid artery.

diastole on most careful auscultation, and yet have unequivocal evidence of mitral stenosis, as shown by the finding of a calcified valve on fluoroscopic examination. Such cases are likely to be in the advanced stages of heart failure with auricular fibrillation. The absence of a diastolic murmur is then probably the result of the very slow velocity of blood flow and the large volume of residual blood in the various chambers of the heart.

Just to mention an occasional reward the physician may obtain as a result of intelligent auscultation, I may mention the following experience. In this particular patient who had obvious evidence of mitral stenosis, on one occasion very peculiar heart sounds were heard. They seemed baffling, and it first made one think that auricular fibrillation was present. On listening over different parts of the chest, it quickly became clear that regular sounds at a rate of about 120 could be heard far removed from the heart, even in the right axilla (Fig. 15). They were entirely independent of the pulse which was also regular, but 75 to the minute. At other times, these aberrant sounds were absent. The tentative diagnosis of diaphragmatic flutter was made. This

was confirmed by fluoroscopic examination, at which time regular rapid contractions of the diaphragm could readily be seen.

I hope I have commented sufficiently on various aspects of auscultation to lend some support to the proposition that this method of examination is valuable and simple. I also have reason to believe that much of the knowledge that is at present available is not being utilized to the full. I may urge that greater emphasis be placed on this subject in our medical schools, so that this valuable and inexpensive method of diagnosis be not neglected. It is also hoped that further interest and study will clarify many problems in auscultation at present poorly understood.

Finally, I wish to thank my British friends and physicians for the privilege and honour of coming here to address you. Many of the leading students of heart disease in the United States obtained their early inspiration and training here in your country, under your great teachers. This will always remain a debt quite difficult to repay. But in a broader sense there are no debts or credits for the medical profession is truly universal in its brotherhood.

REFERENCES

- Dock, W. (1933). *Arch. intern. Med.*, **51**, 737.
 Gee, S. J. (1908). *Auscultation and Percussion*, 6th ed., Oxford Medical Publication, London, p. 84.
 Griffith, T. W. (1912). *Heart*, **3**, 143.
 Harvey, W. P., and Levine, S. A. (1948). *Amer. Heart J.*, **35**, 924.
 Henderson, Y., and Johnson, E. (1912). *Heart*, **4**, 69.
 Levine, S. A. (1927). *Amer. Heart J.*, **3**, 177.
 Lewis, T. (1915). *Lectures on the Heart*, Paul B. Hoeber, 1915, p. 60.
 — (1925). *The Mechanism and Graphic Registration of the Heart Beat*, 3rd ed., Shaw & Sons, Ltd., p. 181.
 Strazhesko, N. D. (1906). *Russ. Vrach. S. Peterb.*, **5**, 603.
 Wolferth, C. C., and Margolis, A. (1930). *Arch. intern. Med.*, **46**, 1048.