

PROCEDINGS OF THE BRITISH CARDIAC SOCIETY

A special AUTUMN MEETING of the BRITISH CARDIAC SOCIETY was held at the Royal Society of Medicine, London on October 29, 1948. Chairman: JOHN HAY. The Chairman took the chair at 9.30 a.m.; 89 members and 38 visitors were present.

CHEST LEADS

TERENCE EAST moved the adoption of the report that had been prepared by Curtis Bain, I. G. W. Hill, Paul Wood and himself at the request of the Council (see page 103).

The following members then took part in the discussion.

WILLIAM EVANS. Since the introduction into this country of unipolar chest leads they had been accepted as ideal on theoretical grounds. Doubtless in time things would sort themselves out, but in the meantime we must decide what helped us most when a patient with cardiac pain came to us for diagnosis. Leatham had made a valuable contribution to this subject and we could see more clearly what we should do in the future. The following points which had been the main outcome of this study and of the application of the three chest leads, CR, CF, and V, in actual practice.

(1) Since the T wave was so often inverted in CF leads in health, this lead should be discarded.
(2) CR1 depicted the P waves better than did V1, and for this reason it was a better lead to use in arrhythmia. (3) Since the R wave was better portrayed in CR1 than in V1, it proved a better lead for the diagnosis of septal infarction. (4) Since the T wave was so often inverted in V1 in healthy adults, CR1 was a better lead to use in the diagnosis of right heart preponderance, for the T wave was seldom inverted in this lead in healthy adults. (5) Again, since the T wave in V leads was usually small at the chest stations 6 and 7, sometimes becoming flat or inverted in health, the CR lead was superior in that the T wave in CR7 was always upright in health.

For these reasons anyone who customarily used CR leads need not change to unipolar chest leads in the hope that they would gain additional information in clinical diagnosis, for they would be disappointed.

PAUL WOOD. A discussion on which chest leads are the best is not profitable, because they are all facets of the same thing: thus conclusions drawn after comparing CR, CF, and V leads are obvious if unipolar limb leads have been taken with any chest lead. The time had surely come to adopt unipolar leads in general.

The question of how best to mount the records might be trivial, but it seemed to be a problem that needed solution. The obvious way was to mount the chest leads around a diagrammatic section of the heart in the positions from which they were actually taken, and to set Einthoven’s triangle about them, the unipolar limb leads appearing at the apices and the standard leads along the sides. This, however, was admittedly impracticable. The next step was to make a more schematic diagram of the heart in the centre of the triangle, so that the chest leads appeared in a horizontal straight line. This was also too laborious. The diagram of the heart could be omitted with advantage, the leads being placed as before with reference to Einthoven’s triangle. Finally, the triangle could be dropped, and lead VF brought into line between leads II and III. The result was neat, and the individual leads could be easily picked out according to their geographical relationship to an imaginary Einthoven’s triangle. Figures illustrating these suggestions were shown.

K. D. WILKINSON. Being old, I am not yet convinced that multiple electrocardiographic leads offer us greater security in the diagnosis of coronary occlusion. These leads, if taken, should be recorded in the simplest possible form, and it seems to me that we shall only reach a definite conclusion when, by combination, we have records of a large number of cases complete with electrocardiograms and the findings of autopsies. In the meantime, I want to stress the importance of taking a good history in every case.

MORGAN JONES. It is not claimed that the “unipolar” limb leads are precisely unipolar, so it is perhaps best to call them “extremity potentials.” Goldberger’s “augmented unipolar” limb leads have two advantages over the original Wilson leads: (a) the lead connections necessitate only one electrode on each limb, and (b) the potential changes are conveniently large. They omit the Wilson resistances, however, and Bryant and Johnston have
shown that this omission introduces significant error in a small percentage of cases. So, although Wilson adopted the Goldberger technique, he preferred to retain the resistances and this appears to be the most acceptable method for general use.

In recording either the precordial or the extremity potentials, it is not correct to say that the resistances are unnecessary if the skin resistance is low; the purpose of the resistances is to prevent an appreciable flow of current when there is a considerable potential difference between points coupled together—they are therefore more necessary if skin resistance is very low. In practice, the resistances are naturally more advisable in recording extremity than precordial potentials. The Wilson resistances have the same effect as a high skin resistance in increasing the tendency to pick up alternating current. To reduce interference when recording the classical limb leads, it has always been the custom to earth the "spare" limb through the instrument but, when either the central terminal or the Goldberger technique is used, all three limbs are required, so it is impossible to earth any of them. Therefore, to reduce interference, all switches designed for recording "unipolar" leads should incorporate a connection to the right leg which is constantly earthed.

The importance of using a considerable number of precordial leads has been underestimated. These leads reflect principally the potential changes of a small part of the ventricular muscle adjacent to the electrode, so records from three or less positions may fail to show a lesion confined to part of the anterior ventricular wall. This is especially important in the recognition of anterior infarction, for only one of the six usual precordial leads may reveal diagnostic abnormalities. In addition, multiple precordial leads give valuable information concerning the extent of the infarct. Further, as the electrode is moved across the precordium, the progressive changes in the ventricular complex form a regular pattern in normal subjects, and disturbances of this pattern from disease cannot always be recognized when the form of the complex is known only at two or three points. Finally, it cannot be over-emphasized that the right precordial potentials rarely influence the extremity potentials, so that anteroseptal infarction is usually associated with a normal limb lead electrocardiogram.

After further discussion the Society then approved the publication of the Report as an expression of the present views of the majority of its members. [The Report is published on page 103 of this number.]

**SHORT COMMUNICATIONS**

**A CLINICAL COMPARISON OF CR, CF, AND V LEADS IN HEALTH AND DISEASE**

**By Aubrey Leatham**

*(Introduced by William Evans)*

On 500 patients, including 100 controls, leads CR, CF, and V 1–7, standard limbs leads and unipolar limb leads have been taken. Analysis of the chest leads on a purely practical basis has led to four main conclusions.

Since the CF electrocardiogram so often appears abnormal in health it is best to forego it in routine practice.

In ventricular preponderance, bundle branch block, posterior cardiac infarction, and in most cases of anterior infarction, a comparison of the CR and V electrocardiograms showed that there were no significant differences between the QRS complexes and T waves.

In 30 patients with anterior or antero-lateral cardiac infarction showing slight cardiographic changes, the abnormalities were seen more plainly in the V leads, although in only one were they absent from the CR leads.

On the other hand, the T inversion commonly found in V1 in health and occasionally in V7 might be considered a disadvantage. In practice, therefore, neither electrocardiogram has shown much superiority over the other.

**A COMPARISON OF STANDARD LEADS, UNIPOLAR LIMB LEADS; AND PRECORIAL LEADS**

**By D. R. Cameron**

*(Introduced by J. H. Wright)*

Standard limb leads, unipolar limb leads (Wilson and Goldberger techniques) and precordial leads 1–6 of CR, CL, CF, and V (Goldberger) types were taken in 30 normal and 30 abnormal subjects. The site of the remote electrode may have considerable effect on the form of the precordial cardiogram. Any component—P, QRS, S–T or T—may be affected.
In the normal case, differences are usually most obvious in the extreme right (1 and 2) or left (5 and 6) positions, where the amplitude of the deflections is small and therefore more liable to noticeable distortion. They are also more obvious in P and T because (a) deflections are smaller, and (b) duration is longer than in QRS.

In certain abnormal cases, the differences may be most obvious in the transitional zone (3 and 4). Prediction of the differences in CR, CL, and CF is possible from a study of any one of them in conjunction with the S limb leads (of Wolfirth and Wood).

The V leads always occupy an intermediate position between the extremes of the other precordial leads, i.e. distortion is reduced to a minimum. Consideration of the unipolar limb and precordial leads renders possible a fairly accurate prediction of the CR, CL, and CF leads. In the normal cases the variations depend mainly on the position of the heart. In the abnormal cases they depend also on the nature and site of the lesion.

Preliminary study (6 cases) indicated no significant differences in V (Goldberger) and V (Wilson) precordial leads.

The augmented Goldberger technique for unipolar limb leads seems in some cases to give larger amplitudes than could theoretically be expected, though the form of the complex is unaltered.

**UNIPOLAR ELECTROCARDIOGRAMS IN CORONARY THROMBOSIS**

*BY ANNE C. AITKENHEAD*

(Introduced by J. H. WRIGHT)

The present contribution deals with 150 cases mostly of coronary thrombosis studied by unipolar leads over the past three years. In most early cases, serial tracings were taken every few days and cases studied at the beginning of the investigation have been recalled for check-up. The cases have been classified according to localization of infarct after the method of Wilson and the relative frequency of the different sites has been noted.

While many fit into the well recognized groups, a considerable number are atypical, e.g. a typical T III electrocardiogram in standard limb leads may be associated with a small R in leads over right side of precordium and not the usual large R. The coexistence of left ventricular hypertrophy is a complicating factor in leads over left side of precordium but the height of R and the level of S–T help to differentiate.

Serial tracings have shown the regression of an infarct from its edges inwards; thus an antero-septal and antero-lateral infarct shows most rapid signs of healing in V6 and later V5 and also in V1 and later V2 while V3 or V4 may be the last to return to more or less normal. We have used V7 frequently and find it particularly useful in postero-lateral infarcts where it may be the only lead which overlies the infarct directly (albeit only its edge). Leads V5 and V6 are not infrequently redundant.

**CARDIAC PAIN WITH RECOVERY OF THE T WAVE**

*BY TERENCE EAST AND S. ORAM*

Published in full, Brit. Heart J. (1948), 10, 263

**THE EFFECT OF POSTURE UPON NORMAL AND ABNORMAL ELECTROCARDIOGRAMS**

*BY A. MORGAN JONES, H. K. HELLERSTEIN, AND HAROLD FEIL (introduced)*

The effect of posture upon the standard limb leads, the extremity and precordial potentials has been studied in 100 cases, 20 normal, 20 right, and 20 left bundle branch block, 20 with isolated right ventricular enlargement, and 20 with isolated left ventricular enlargement. Electrocardiograms were recorded in the supine, sitting, right and left lateral positions except in the cases with bundle branch block, in which the sitting posture was not studied.

In normal subjects the axis could be changed within wide limits in a considerable proportion of cases, sometimes over almost the whole normal range from +90° to 0°, but in no case did the axis fall outside normal limits in the postures studied. The axis was usually farthest to the left in the sitting posture and to the right in the left lateral position.
The changes were due to reversal of the form of the extremity potentials of the left arm and left leg; the precordial potentials remained of the same general form, but the transitional zone rotated to the left when the axis in the standard limb leads shifted to the right. T wave changes were associated with the QRS changes.

In left bundle branch block very striking changes occurred, especially on turning into the left lateral position. In this posture abnormal right axis deviation appeared in 4 cases, and the limb lead cardiograms simulated right bundle branch block; the precordial potentials remained substantially unchanged and in all positions were characteristic of left bundle branch block. The changes were much less striking in right bundle branch block, the limb leads being substantially of the same form in all positions.

In left ventricular enlargement even more striking changes were present, again most extreme in the left lateral position. In that position abnormal right axis deviation appeared, and ST-T changes appeared in lead III instead of in lead I, owing to reversal of the form of the extremity potentials of the left arm and left foot. The appearances in this position thus often simulated those of right ventricular enlargement, but the precordial potentials remained characteristic of left ventricular enlargement. In cases with right ventricular enlargement the changes in the limb leads were very slight and did not simulate left ventricular enlargement in any position.

The striking postural changes in left ventricular enlargement and in left bundle branch block were compared with the slight changes in right ventricular enlargement and right bundle branch block. If the amount of axis change is taken as an indication of the extent of the changes in pattern, there is a statistically significant difference between the extent of the changes in left ventricular enlargement and in normal subjects on the one hand, and between normal subjects and right ventricular enlargement on the other. Possible reasons for this difference were discussed.

**Subacute Bacterial Endocarditis**

**By K. D. Wilkinson**

The Birmingham centre has treated 63 cases and has had 12 relapses. The *Streptococcus viridans* is the infecting organism in over 90 per cent of cases. Fifty-eight per cent of all cases have been cured and are alive, many at full work as before their infection.

When the centre began we used a dosage of 0.25 mega units daily and met 4 cases with infections that could not be controlled. With a dosage of 1 mega unit daily for 28 days the results have been better. For a relapse 2 mega units daily for 6 weeks is the usual dose, but for one highly resistant *Streptococcus D* a dosage of 11.5 mega units daily for 6 weeks resulted in a cure. There have been no failures to cure the infection with the bigger doses.

The initial symptoms of a relapse may be very slight: as patients are under supervision the relapses are detected early and treated efficiently and early. It is probable that the early symptoms of most cases of infective endocarditis are slight, and it seems most important to point out the early symptoms so that all cases may be investigated and brought under treatment as early as possible.

A change in health is the first thing noticed. Fever, seldom high; sweating, especially at night; malaise, lassitude, and vague pains in back or limbs which is commonly diagnosed as influenza. Some anorexia and loss of weight occur, but these symptoms tend to be slight and vague. Even those who have had a similar illness before tend to say "I don't feel well."

The physical signs may be almost as slight and vague. Splenic enlargement and changes in the heart murmurs are among the most definite. Major embolic manifestations are unusual but intermittent albuminuria with blood cells and casts in the urine are quite the most frequent significant sign.

There were 2 cases of hemiplegia, 1 of aphasia with no other definite nervous lesion, 2 of repeated pulmonary embolism, and 1 with glycosuria which disappeared as the infection was brought under control.

The best results are obtained in those who come under treatment early. The mouth is far the most important source of infection: in eight of this series the extraction of teeth was related to the onset of symptoms so definitely that there can be no doubt that the operation acted as a trigger, but gingival infection is at least as important as apical abscesses. The patient with the resistant *Streptococcus D* whom I have mentioned as cured by 11.5 mega units daily for 6 weeks began to respond after an extensive excision of swollen infected gingival margins.

Teeth and gingival infection can be treated while the patient is on the penicillin course, and this is
very important if relapses are to be avoided. As might be expected, the blood urea gives some indication of the severity of renal damage. Those patients with raised blood urées do less well as a rule. Hæmoglobin estimations are important. Bramwell stated in his recent paper that the rate of hæmoglobin recovery was slow: recalling a few individual cases I doubted this, but on working out the recovery curves of all our cases I find that the observation is perfectly correct. The blood recovery rate contrasts most remarkably with the results of iron therapy in low colour-index anemias or liver treatment in typical Addisonian anemia. In cases of infective endocarditis who had no hematinics or transfusion in the first month the average gain of hæmoglobin is 3-5 per cent, i.e. from 76 to 79 per cent. In the second month all the records tend upwards, the average gain being 8 per cent, i.e. from 79 to 87 per cent; while in the third month the rise was 7 per cent, i.e. from 87 to 94 per cent.

Mitral Stenosis in Later Life

BY HAROLD COOKSON

Results of observations on 36 cases with mitral stenosis ranging in age from 51 to 77 years were reported. Women outnumbered men by 3 to 1, and most of the patients were seen in private practice. A clear history of rheumatic fever or chorea was given by 13, and in a further 8 heart disease or a valve lesion had been diagnosed in early life. In 4 cases the first known attack of rheumatic fever occurred at the age of 34 or later.

All patients had lead normal active lives up to the sixth, seventh, or eighth decade, and 15 of the 21 married women had had children. When first seen 32 patients had auricular fibrillation, and 2 auricular tachycardia, and the onset of an arrhythmia seems nearly always to coincide with the first appearance of symptoms. Hypertension was present in a high proportion.

The criteria of diagnosis were given. The X-ray appearances of the heart and great vessels, which differ in some ways from what has been regarded as the characteristic picture of mitral stenosis, were described. Prognosis, cause of death, and necropsy findings (3 cases) were dealt with. The etiology of the valve disease was considered to be rheumatic in all cases, and the reasons for the latency of the lesion until late in life was discussed. Reasons were given for thinking that the diagnosis of mitral stenosis in later life is often missed.

An Artificial Circulation

BY R. J. S. McDOWALL

This artificial circulation is a robust piece of apparatus which has been in use by medical students for a year. By means of it, the main mechanical features of the circulation of the blood may be shown.

The pump has the special feature that its output can be shown to depend both on its input and to a limited extent on the frequency of its stroke. It consists essentially of a piece of flattened bicycle 1-inch inner tubing 4 inches long which is compressed by a moving plate controlled by a cam. A large inlet "valve" of little resistance is provided by the closure of the inlet by a narrow plate driven by another cam on the same shaft just before the main stroke of the pump. A very robust and simple exit valve is provided by a piece of inner tubing stretched over a piece of brass tubing into the outer end of which is inserted a wider flat piece of bakelite. A visible flow indicator in the system is provided by a bent tube inside a small Kjeldahl flask, the flow being directed against the side of the flask to avoid frothing.

The rate of the pump can be altered by changing the starting resistance of the motor driving the camshaft and the effects of tachycardia shown.

The "arterial" pressure taken on a mercury manometer can be altered up to 200 mm. Hg by changing the filling of the heart, its rate within limits, and by varying the resistance of the system. Increased filling of the heart is produced by raising the hinged part of the system to the horizontal or by compressing the "blood depot" which is a piece of inner tube which can be rolled up. The air pressure can be adjusted in the bottle so that the changes in "heart" output become visible.

The "venous pressure" taken on a manometer (a narrow burette with a fountain-pen cap as a piston), can be shown to be affected by the amount
of circulating fluid, by the capacity of the system, which can increase by unrolling the "blood depot," by the peripheral resistance, and by the output of the pump. If the pump is slowed or stopped, the "venous pressure" rises markedly as in cardiac disease.

A reduction of the elasticity of the system to imitate hardened arteries provided by the inverted bottle is effected by pinching the tube and is shown to increase the pulse pressure and render the flow in the flow-meter intermittent.

A lever system from the rubber valve chamber may be added to record the rapid changes in the "arterial pressure" better than the mercury manometer.

For making original forms of the pump and the exit valve I am particularly indebted to Dr. A. E. Schuster.

THE EFFECT OF DIGITALIS ON THE VENOUS PRESSURE

BY PAUL WOOD AND JOHN PAULETT (introduced)

Published in full, Brit. Heart J. (1949), 11, 83

HEART FAILURE AND TRICUSPID INCOMPETENCE

BY W. BRIGDEN AND E. P. SHARPEY-SCHAFFER

It was shown previously that in cases of tricuspid incompetence mean right auricular pressure was higher than the mean pressure in a peripheral vein, a phenomenon that can be reproduced in a simple mechanical model. In cases with tricuspid incompetence it may be difficult to decide to what extent the high venous pressure results from incompetence or from heart failure. Manometric curves of right auricular pressure show a high systolic curve and indicate the diastolic pressure level. Continuous recording of pressure waves on rapid withdrawal of the catheter from auricle to peripheral vein show damping out of the systolic pulsations in the peripheral vein. The presence or absence of heart failure may also be demonstrated by measuring the response of the forearm flow to changes in posture. In the normal subject the forearm vessels constrict on tipping into the upright posture, while in cases of left heart failure, with or without high right auricular pressures, constriction occurs in the supine position. Some cases of tricuspid incompetence show the same response of the forearm vessels as the normal subject to changes in posture, although systolic and mean right auricular pressures are high.

ANGIOCARDIOGRAPHY IN CONGENITAL HEART DISEASE

BY MAURICE CAMPBELL AND T. H. HILLS (introduced)

The apparatus consists of a special trolley over which an X-ray tube is located at a suitable height. Just below the trolley top is a 15 by 16 inch fluorescent screen and the image produced at this plane is recorded by a large roll-film camera.

Exposures are made at the rate of one a second on a film whose width is such that the finished negatives are about 5 by 5 inches square. As many as fifty consecutive exposures could be obtained but in practice the number is normally limited to fifteen.

The opaque medium used is a 70 per cent solution of diodrast in water, and a preliminary sensitivity test is carried out.

Success depends on the intravenous injection of from 30 to 50 ml. of the dye in a maximum of 2 seconds. It is usually necessary to expose a vein in the arm and insert a wide gauge canula. A preliminary injection of 20 ml. of saline demonstrates the suitability of the vein and the speed with which the injection can be made. About five seconds after the injection the patient feels a sensation of heat which reaches its maximum rapidly and fades more slowly within two minutes.

It is not practicable to carry out this investigation in small or nervous children without an anaesthetic, and this must add to the risk. The best anaesthetic is probably the combination of cyclopropane and oxygen. Oxygen should be continued till normal conscious respiration has been restored.
In Fallot's tetralogy the interpretation of the films can be considered under two headings.

Firstly, the volume shunt of blood from right to left is estimated by the degree of filling of the aorta which is generally seen as early as two seconds after the injection. The presence of dye in the subclavian artery, the abdominal aorta, and the kidneys gives additional direct evidence of the proportion of dye taking this route.

Secondly, the pulmonary arteries usually show some filling at about two seconds but the degree of stenosis cannot be assessed on this time basis alone. If there is much flow of dye through the lungs their general density will show a steady increase to a maximum in about eight seconds. This estimation of the changing density of the lung fields would appear to be a more valuable observation than the actual time and degree of filling of the pulmonary arteries.

The first film of the series will often show some venous backflow due to a temporary rise of venous pressure. Traces of dye may be seen in the inferior vena cava and the internal jugular vein. This abnormal pressure must be taken into account when estimating any volume shunt but is not likely to persist after the first two seconds.

In cases that appear to be typical instances of Fallot's tetralogy evidence of a right to left shunt has been obtained regularly. In one case thought to be Fallot's tetralogy no right to left shunt was found and post-mortem there was high grade pulmonary stenosis with a small auricular septal defect without the other features of Fallot's tetralogy. A moderate right to left shunt has sometimes been suggested in cases that were not obviously cyanotic.

Our early clinical impression is that the method is perhaps less sensitive in detecting pulmonary stenosis, presumably in cases where this is not so severe. This differentiation may prove of value in estimating the degree of success following a systemic-pulmonary anastomosis or in choosing cases suitable for pulmonary valvulotomy.

In addition, a high proportion of angiocardiograms show points of interest that were not suspected—a double superior vena cava, a large pulmonary artery on one side with obstruction on the other side, and a widespread venous anastomosis on the right side. In one case regarded as tricuspid atresia with non-functioning right ventricle the angiocardiogram provided good supporting evidence.

Circulation Times in Congenital Heart Disease

By K. D. Allanby
(Introduced by Maurice Campbell)
To be published in full, Brit. Heart J., April, 1949

Apical Diastolic Murmurs in Severe Anaemia

By H. E. S. Pearson

A murmur closely simulating that of mitral stenosis can occur occasionally in cases of severe chronic anaemia in the absence of valvular disease. Numerous references to this are found in late nineteenth century continental textbooks and articles on pernicious anaemia, and Cabot (1896) recorded 9 such murmurs in his series of 857 cases of this disease. Gunewardene (1933), dealing with hookworm anaemia, describes 4 cases in whom a confident diagnosis of mitral stenosis was disproved either by autopsy or by the disappearance of the murmur on treatment, and Klinefelter (1942) obtained graphic records of apical presystolic murmurs in patients with sickle-cell anaemia.

Three cases presenting this physical sign are described, and the apparently arbitrary appearance and fugitive nature of the murmurs are shown by means of charts. This murmur has several features in common with the transient diastolic murmur that can occur in cases of rheumatic fever (Carey Coombs, 1924) and in discussing its possible cause certain explanations that have been put forward for the latter are admissible here for consideration.

With these inclusions, the following factors have been held responsible by various authors.

1. Decreased viscosity of blood (Sahli, 1895; Garb, 1944).
2. Increased speed of blood flow (White and Wood, 1923) with rapid filling of atomic left ventricle (Bland, Jones, and White, 1935).
3. "Relative mitral stenosis" (White, 1937), or combination of these (Luisada, 1948).
4. Pressure of dilated pulmonary artery on mitral orifice (Kerr, 1936).
5. Pressure of dilated right and left ventricles on mitral orifice (Weinstein and Lev, 1942).
It is felt that the facts observed in these three cases cannot be satisfactorily explained in any of the above ways, although the first factor may play a part. The suggestion is made that the apical diastolic murmur in severe anaemia is caused by incomplete opening of the mitral valve, associated, in the way to be described, with loss of passive tone in the papillary muscles.

The stream of blood, entering the ventricle axially, recoils radially and upward towards the base of the heart, thus tending to press the mitral cusps together. Closure is prevented chiefly by the lateral pressure of the entering stream but also by the passive tension of the papillary muscles on the chordae tendineae. Only minor pressures are involved and failure of the papillary muscles to exert their normal light "spring-loading" action on the valve allows the cusps to lie so sharply curved towards one another that a murmur is produced in the narrowed commissure. Fatty change, when present, is commonly maximal in the papillary muscles, and may lead to stretching.

This paradoxical movement and its control by the papillary muscles can be demonstrated in the cadaveric heart by a simple experiment.

**REFERENCES**


**THE GRAPHIC REGISTRATION OF BASAL DIASTOLIC MURMURS***

**BY BERTRAND G. WELLS**

(Introduced by GEOFFREY BOURNE)

The vibrations of the diastolic murmur of aortic regurgitation are of high frequency and very low energy value. Other cardiac events, however, cause at the same location vibrations of lower frequency and many thousands times the energy. If the phonocardiogram were to register deflections of amplitude directly proportional to the energy of these vibrations the tracing would have to be hundreds of metres wide in order to show the diastolic murmur as deflections of 1 mm. in width. If the amplitude of the low frequency waves is attenuated logarithmically with regard to their frequency, we are enabled to show the 1 mm. diastolic vibrations clearly while the low frequency waves are all reduced in amplitude by this process of filtration, so that they can be confined to paper a few centimetres wide. Workers in phonocardiography have almost always used unsuitable apparatus, and have not been able to register this murmur satisfactorily. We have used a Sanborn phonocardiogram with stethoscopic and logarithmic registration, and interchangeable chest pieces, and have, with due attention to technique, been able to register every murmur audible clinically.

An analysis of 50 consecutive cases with audible murmurs of aortic or pulmonary regurgitation showed much variation in pattern. The frequency of the vibrations varies from moderately low to moderately high. The murmur usually persists throughout most of diastole. The configuration is that there is usually an early crescendo phase before the longer decrescendo phase. This phenomenon is sometimes audible, but much more frequently registered, and failure to hear the early crescendo phase may be explained by study of the characteristics of the human hearing mechanism. Examples of all types of basal diastolic murmur are shown, excluding the continuous murmurs of patent ductus, A-V aneurysm, and venous hum. Occasional tracings are found where diastolic vibrations are registered when no murmur is audible. The significance of these vibrations is obscure, but their

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evaluation is helped by a comparison of their pattern with that of the audible diastolic murmur. Finally, since there is often difference of opinion among cardiologists as to whether a diastolic murmur is present or not, we consider that phonocardiography with proper technique may be of value in settling questions of this kind.

* This study was made while on a Cabot Fellowship, and with the assistance of Mr. Maurice Rappaport, and Dr. Howard Sprague of Boston, Mass., U.S.A.

**SIMPLE CALIBRATED PHONOCARDIOGRAPHY**

**BY G. J. AITKEN (introduced)**

Knowledge of the frequency response of a phonocardiograph is necessary before records can be adequately interpreted. Even then, comparison with records of other instruments is difficult. Lack of this knowledge resulted in disagreement among earlier investigators. With instruments introducing attenuation of low frequency vibrations, heart sounds appear shorter, and the third sound and possibly important low frequency components of murmurs are recorded less frequently than with instruments that register low frequency vibrations well. The higher frequency murmurs, however, are more easily recorded and their moment of onset defined with just such attenuation of confusing high intensity low frequency cardiac vibrations.

A simple calibrated phonocardiograph is described. Basically it is a stethoscopic phonocardiograph with electrical filters enabling records to be obtained of vibrations in a "low frequency band" of 20 to 200 cycles a second, and in a "high frequency band" of 140 cycles a second upwards. It offers a fairly satisfactory solution to many difficulties in interpretation of single recordings. The precise frequency characteristics of the basic phonocardiograph are less important. Whatever the likely degree of attenuation of the low frequency components of the cardiac vibrations in the unfiltered phonocardiogram, they are easily recorded at suitable amplitude in the low frequency band, in the absence of all high frequency vibrations.

In the band embracing frequencies greater than 140 cycles a second the predominantly higher frequency of most systolic and diastolic murmurs allow them to be recorded uninfluenced by the high intensity low frequency heart sounds. The first heart sound is represented by sharply defined overtones of its second and third components, more suitable points from which to time auricular or ventricular systolic murmurs than any so far described.

Examples illustrating the value of simple calibrated phonocardiography are given.

**A CASE OF POLYARTERITIS NODOSA SHOWING MULTIPLE CORONARY ANEURYSMS**

**BY JOHN R. H. TOWERS**

The case described is one of polyarteritis nodosa. For years the presence of palpable nodules was regarded as an essential diagnostic criterion in this condition, but recently it has been recognized that palpable lesions of this type are the exception rather than the rule. In this case the diagnosis was not so difficult but the clinical and autopsy findings were so striking as to be of interest.

The patient, a motor mechanic of 39, had no important illness until 1943, when serving in the 6th Airborne Division, he developed a duodenal ulcer, which gave rise to intercurrent dyspepsia and finally to his discharge in 1945. He had intermittent dyspepsia over the last two years, but remained at his work until May, 1948, when he suddenly developed pains in both legs and difficulty in walking, and he had to go to bed. Symptoms were first noticed whilst getting into a motor car. These pains were present from the knees to the feet. A few days later the pain became generalized; his hands and feet and testicles became slightly swollen and painful. There was in addition, swelling of the left breast which was very tender.

A fortnight after the onset he was free from pain and feeling better. A week later, however, he noticed his stools were black and he developed epigastric pain with some vomiting. This pain was worse before meals and relieved by food, but liable to occur at any time of the day or night and to last for several hours; at times it was very severe.

At this stage, i.e. three weeks after the onset, he had an attack of diplopia lasting half an hour; there was no previous attack and none since. After this there was a return of generalized aches and pains in the limbs and back; his appetite diminished and he lost much weight. His skin, normally rather
deeply pigmented, became darker in colour and he developed a sallow complexion. His sleep was disturbed by pain. Bowels a little constipated; micturition normal. These symptoms continued unaltered until his admission to the Leeds Infirmary in July, approximately ten weeks from the onset.

Examination on admission. He was cachetic with pigmentation of the skin, rather suggestive of Addison's disease. He had obviously lost much weight. He was pyrexial up to 101° for four days and thereafter there was no rise of temperature. His pulse rate averaged 100 until a day or two before his death. He had what were taken to be shotty glands in the neck, axillae, and groins, and the spleen was just palpable. The blood pressure was 130/100. The urine contained a trace of albumen, but no red cells. The heart was not enlarged.

The blood count showed an anemia without eosinophilia—Hb. 71 per cent; red blood cells, 3,700,000; white cells, 9,600; basophils, 5 per cent; polymorphs, 74 per cent; eosinophils, 2 per cent; lymphocytes, 20 per cent.

The X-ray of stomach and duodenum showed an appearance consistent with duodenal ulceration. The stools were negative for occult blood. The Wassermann reaction was negative.

Extensive investigation was carried out with no helpful finding, but shortly we discovered that the so-called glands extended down the arms, along the line of the vessels and also to a lesser extent, down the inner aspect of the thighs, and a hard, shotty mass, the size of a pea was felt through the anterior rectal wall. Biopsy of one of the masses from the arm showed it to be a thrombosed aneurysm arising from a small muscular vessel, the wall of which showed extensive mucoid degeneration. The diagnosis of periarteritis nodosa was then made. The patient went gradually downhill, became emaciated, and died in September, 1948.

Autopsy. This was performed by Dr. Carmichael and showed a striking picture. A very large number of aneurysms ranging in size from 1 cm. to 2 cm. were seen in association with most of the principal arteries in the body, with the exception of those in the lungs and brain. The lesions have all relatively thick fibrous walls and are filled with dense thrombus, mostly of dark red colour but there are also masses of pale greyish-white clot or laminated grey and red clot clinging to the walls of many of the sacs. Few of the aneurysms are attached to vessels large enough to be opened up with scissors, i.e. they apparently spring rather from relatively small unnamed branches than from the larger trunks.

Impressive examples were as follows: a number of aneurysms adhering to the outer surface of the thoracic aorta and related to the intercostal arteries; a small vessel on the surface of the diaphragm at the attachment to the pericardium, where six close-set aneurysms, each 3 mm. in diameter were concentrated in 3 cm. of the vessel's length; a "beading" of the anterior aspect of the bony spine by aneurysms; and numerous aneurysms along the length of the spermatic arteries.

There are many large aneurysms on the right and left gastric arteries to the gastroepiploic artery. The mesentery also shows dozens of aneurysms of a size varying from 2 to 3 mm. to nodules almost 1 cm. in diameter. The aneurysms related to the stomach measure 1-5 cm. and more. The pelvic mesocolon shows only a very few small aneurysms and the position is similar with most of the arteries supplying various parts of the colon. Right, relatively large branch of hepatic artery bears a large aneurysm, 1 cm. in diameter, which is on the main stem, but there are several other similar sized aneurysms in this neighbourhood. There are several aneurysms of 2 cm. diameter and smaller within the head and body of the pancreas and at least two of these have ruptured, but the effused blood does not extend far into the adjacent tissue. There are few aneurysms in the vicinity of the abdominal aorta. The aneurysms are present on the branches of the splenic aorta in the hilum and substance of the spleen, and of branches of the renal aorta within the venal substance; also in considerable number of small size in the very large portal tracts. No aneurysms on main splenic and renal arteries.

The spleen contain an aneurysm fully 1 cm. in diameter and one or two smaller nodules and an infarct at one pole. The kidneys contain numerous thick-walled aneurysms approximately 3 to 6 mm. in diameter and located chiefly in the region of the boundary zone or farther in towards the hilus. There is also much recent infarction, and much irregular coarse pitting on the outer surface, probably due to scarring from an old infarction. The renal tissue otherwise is mottled, mostly showing a dusky congestion; other parts are pale. Large tracts of tissue are partially fibrosed.

The stomach was normal apart from well-marked engorgement at the crest of the mucosal folds in places and some hypertrophy of the pylorus and large area of scarring centred over the pylorus and lesser curvature, and spreading into the stomach and duodenum.

The brain and lungs, however, were normal, showing no gross change in their substance or vessels.

There are numerous aneurysms connected with the coronary branches, particularly the arteries supplying the line of the interventricular sulcus, and the anterior and lateral surface of the right ventricle.
At least 30 to 40 aneurysms of varying sizes are present, varying from a few mm. to 2.5 cm. in diameter. There is a thin layer of fibrinous exudate scattered over the whole epicardial surface. The chambers of the heart are all slightly dilated, but the myocardium has a healthy appearance and texture and shows no evidence of infarction.

**Electrocardiographic Studies in Cretinism**

**By Bernard Schlesinger and Bernhard Landtmann (introduced)**

*To be published in full, Brit. Heart J., July or October 1949*

**A Case of Pheochromocytoma with Sustained Hypertension**

**By Leslie Cole**

This case is described to emphasize the fact that tumours of the adrenal medulla may cause sustained hypertension in the early stages, without giving rise to paroxysmal symptoms, or paroxysmal hypertension; to show the clinical picture in the early stages; and to comment on the use of certain diagnostic aids.

The patient was a farmer's wife aged 35, with three children, and she developed sudden headache three weeks after a severe antepartum haemorrhage. This persisted daily almost without remission until her death thirteen months later. It was marked in the early morning, grew worse month by month and latterly was associated with early morning nausea, vomiting, profuse sweating, cold hands and feet, shakiness, nervousness of an unusual sort and "peculiar sensations"; and during the last three weeks, muscular weakness and exhaustion. The blood pressure was first found to be raised seven months after the onset (180/130) and remained steadily at this level until just before death. Repeated clinical examination was otherwise negative and thorough investigation failed to show any cause. A single paroxysm of hypertension seven days before death (260/150) revealed the probable diagnosis, but came too late to save her life. It was followed by a severe attack of right-sided abdominal pain and paralytic ileus which necessitated laparotomy and cæcostomy. At operation, a tumour was found at the site of the left suprarenal gland which subsequently proved to be a typical pheochromocytoma. This was removed a week later but the patient succumbed. Autopsy showed that the right-sided pain was due to a large infarct of the right kidney.

Diagnosis is difficult and involves recognition of the syndrome produced by hypersecretion of adrenalin and localization of the tumour or tumours. For the first, it is essential to realize that sustained hypertension is indistinguishable from the essential or malignant type and without paroxysms may be present in the early stages, and also to appreciate the significance of the symptoms illustrated by this case. In this and many others recorded there are unique clinical grounds for suspecting the diagnosis once the clinician is aware of the syndrome. Goldenberg's benzodioxane test then appears to be of great value in confirming it, but this patient died before his paper had been published. To localize the tumour, an intravenous or retrograde pyelogram may be sufficient, but perirenal insufflation may be necessary, or even exploration. Here, although the symptoms and signs pointed to a right-sided lesion, the tumour was on the left. Early diagnosis and accurate localization are extremely important because successful removal may be expected to give complete cure.

**The Q-T Interval in Acute Rheumatic Carditis**

**By D. G. Abrahams**

*(Introduced by Paul Wood)*

*To be published in full, Brit. Heart J., July or October 1949*