RECENT ADVANCES IN CARDIOLOGY

BY

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EATMENT

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PREFACE TO THE FIFTH EDITION

It was rather over thirty years ago that we first contemplated, with some diffidence, a review of the advances that had been made in the then young and hardly formed subject of Cardiology. What then was an "advance" and what was "recent" chiefly occupied our thoughts. To-day the vast flood of publications makes the question of recentness answer itself, while time soon shows what constitutes an "advance." It is not easy to keep abreast with this subject nowadays in all its many complicated and technical ramifications. The recent, like Time's winged chariot, is ever at our heels; if indeed one does not find oneself at its heels, faint yet pursuing. In the first edition, for example, there was no chapter on congenital defects; now this stone is become the head of the corner.

In this edition we have excluded arterial disease, pregnancy, diphtheria, and shock in some aspects. This has allowed room for treatment of other subjects on a larger scale; for it is now necessary to focus on certain selected matters where new knowledge has aroused special interest, rather than to try to review the subject as a whole. Of course, after ten years it has been necessary to write the book completely afresh and to include a number of new illustrations.

In preparing this edition we have had the advantage of being able to enlist the valuable help of Dr. Wallace Brigden of the Cardiac Department of the London Hospital and of the National Heart Hospital. His responsibility extends to the writing of Chapters 2, 3 and 5 and the section on syncope. The rest of the book is the combined responsibility of the original authors—for the last time.

The very up-to-date reviewer must remember, as we have pointed out before, that the time of literary gestation tends to increase so that many months must elapse between conception on paper and parturition in print.

In recent years the cardiac catheter has added enormously to our knowledge in the field of hæmodynamics; although in much it is becoming a question of confirming what has already been shrewdly suspected. Congenital defects are an ever-growing interest. The detailed surgical treatment is a technical matter outside the scope of this book. At the present moment all depends on giving the surgeon enough time in a bloodless field; then with the help of modern anæsthesia he will be able to do almost anything. The

derangements in the pulmonary circulation are of great importance now that pulmonary hypertension, dimly surmised twenty years ago, can be measured. In the field of myocardial disease there is much new pathological knowledge. The riddle of hypertension is still unsolved, but its effects are better understood and to some degree better mitigated. Disease of the coronary arteries is still obscure; more knowledge of its biochemistry will be the next step, but there is a long way to go. Heart failure is better understood in many aspects, and the knowledge of the causation and treatment of œdema has advanced a good deal.

As in former editions we have aimed at presenting in a practical way new knowledge; remembering, as always, that the academic of to-day is often the commonplace of tomorrow. Complicated techniques become simple once the secrets they reveal have been learned, so that it becomes easy to blend their information into the stock of common knowledge, when this is founded on the basic principles of physiology. Thirty years ago one stressed the importance of the myocardium as taught by Mackenzie. Now one goes back to Harvey and stresses the hæmodynamics of the circulation of the blood!

We wish, once again, to thank our publishers, always patient, helpful and hopeful, over many years.

T. E. C. W. C. B.
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ACKNOWLEDGEMENTS

ALTHOUGH most of the illustrations have been prepared from personal and hospital records of patients who have been under their care, the authors wish to express their appreciation of the ready co-operation they have received in connection with these and others appearing in this edition to—

Dr. M. Godfrey and the Editor of *King's College Hospital Gazette* for permission to reproduce Figs. 11 and 12; to Dr. William Evans for Figs. 57 and 58; to Dr. J. P. D. Mounsey for Fig. 43; to the Editor of the *British Heart Journal* for Figs. 31, 34, and 38 and the Editor of the *Lancet* for Figs. 28, 46, 47, 49 and 52–56 taken from articles by Dr. Wallace Brigden. Figs. 107 and 114 also from the *British Heart Journal* and Figs. 99 and 100 from the *Lancet* come from articles written by one of us (C.W.C.B.), and have appeared in earlier editions.

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CHAPTER 1

CONGENITAL CARDIAC DEFECTS

It is not possible to classify these complex and often multiple defects satisfactorily. Various attempts have been made on a physiological basis, but the difficulty is that for any given lesion the physiological derangements vary at different ages and stages. Hence the classification of Maude Abbott on the degree of cyanosis is not of much use nowadays. The relative volumes of the systemic and pulmonary flow has also been suggested, but these may vary too, also the volumes of the shunts. The best basis is anatomical or embryological. Here the difficulty arises that sometimes there are multiple defects. Many of these cases cannot survive, and are not of much clinical interest. Furthermore the reason of the derangement in development is usually obscure. Nevertheless, from the practical point of view of accurate diagnosis, which may offer some chance of surgical treatment, and also of prognosis, the anatomical basis seems best; and with the help of modern methods any case that seems likely to survive at all can usually be diagnosed accurately.

The contents of this chapter must be set forth in some sort of order. The following arrangement, based partly on anatomical, partly on physiological and partly on embryological features of these diverse and disconnected topics, is given for what it is worth.

ARRANGEMENT

1. Anomalies of Septal Formation. CARDIAC.

(*a*) *Cyanotic.*
Cor biloculare. Cor triloculare biatriatum. Cor triatriatum. Cor triloculare biventriculare.

(*b*) *Potentially cyanotic.* With pulmonary plethora.
Atrial septal defect.
Ventricular septal defect. Eisenmenger Complex.

2. Anomalies of Septal Formation. AORTIC

(*a*) *Cyanotic.* With normal or reduced pulmonary flow.
Persistent truncus arteriosus.

(*b*) *Acyanotic.* With pulmonary plethora.
Aortico-pulmonary communication.
(Aneurysm of sinus of Valsalva: may be no shunt.)

3. Anomalies associated with torsion of the Truncus Arteriosus and absorption of the Bulbus Cordis.

(*a*) *Cyanotic.*
1. Transposition of great vessels. With pulmonary plethora.
2. Tetralogy of Fallot. Ventricular septal defect. With pulmonary oligæmia.
3. Eisenmenger Complex. Ventricular septal defect. With pulmonary plethora.

(*b*) *Acyanotic or late cyanotic.*
Infundibular pulmonary stenosis: with pulmonary oligæmia.

(*c*) *Acyanotic.*
Subaortic stenosis.

4. Valvular Anomalies.

(*a*) *Cyanotic.*
Ebstein's deformity: with pulmonary oligæmia.
Tricuspid atresia: with pulmonary oligæmia.

(*b*) *Acyanotic or late cyanotic.*
Pulmonary stenosis (simple) and A.S.D. (often).

(*c*) *Acyanotic.*
Aortic stenosis.
Anomalies of aortic and pulmonary valves.

5. Anomalies of the development of the Aortic Arches.

Persistent right aortic arch: double aortic arch.
Coarctation of the aorta.
Anomalies of main branches.
Patent ductus arteriosus.
Anomalies of the coronary arteries.

6. Dextrocardia.

7. Miscellaneous.

Pulmonary arteriovenous fistulæ.
Anomalies of the pulmonary artery and its branches.
Congenital heart block.
Endocardial fibroelastosis.
Anomalies of great veins and pulmonary veins.

DEVELOPMENT OF THE HUMAN HEART

Although no recent advance is a feature here, some account of this complex process should help in understanding the nature and origin of the defects in its achievement.

During its development the mammalian heart passes through various stages which resemble the hearts of lower animals; in each individual the history of the development in various species is recapitulated. The congenital malformations of the human heart recall structures found in the hearts of fishes, amphibians, reptiles and birds.

The process of development aims at establishing a pulmonary circulation "in parallel" with the systemic, instead of "in series." But they must communicate with each other, so the two are also "crossed." The heart begins as a fusion of two straight tubes placed on either side of the body, which are brought together as the primitive ventral cleft closes in. In the 3 mm. embryo this simple tube is already becoming divided into four chambers, the sinus venosus at the tail end, the auricle, the ventricle, and the bulbus cordis at the head end. This stage represents the heart of some fishes. As the primitive cardiac tube increases in length, between two fixed points, it becomes kinked or bent, or twisted upon itself (Fig. 1). The ventricle grows downwards and forwards, the auricle upwards and backwards. The ventricular part becomes a loop bent on itself in a v-shaped manner, forming in the angle of the loop the bulbo-ventricular groove. A twist also develops, clockwise at the ventricular end, anti-clockwise at the venous end (Fig. 2).

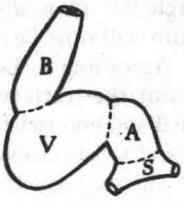


FIG. 1

A. Auricle.
B. Bulbus Cordis.
S. Sinus Venosus.
V. Ventricle.

(After Pichon)

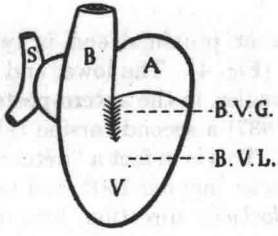


FIG. 2

A. Auricle.
B. Bulbus Cordis.
B.V.G. Bulbo-ventricular groove.
B.V.L. Bulbo-ventricular loop.
S. Sinus Venosus.
V. Ventricle.

(After Pichon)

Division of truncus and bulbus. In order to develop the two circulations in parallel, the truncus arteriosus must be divided. A septum forms across the truncus from two lateral ridges. At the distal end of the truncus, furthest from the heart, the plane in which they lie is at right angles to the antero-posterior plane of the body (Fig. 3). According to the theory of Spitzer (1929) two processes of torsion take place. In the first torsion, while the upper or distal part of the tube is turned through 180° in an *anti-clockwise* direction,

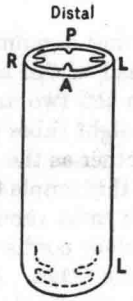
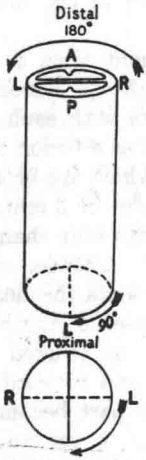


FIG. 3



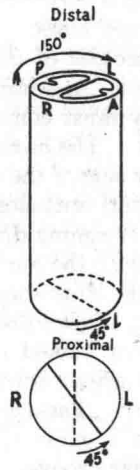


FIG. 4

the lower or proximal end is twisted through 90° in a *clockwise* direction (Fig. 4). The lower end of the septum will now be across, at right angles, in the antero-posterior plane. According to Lev and Saphir (1937) a second torsion takes place about the sixth week of fœtal life. This is in fact a "detorsion," for the distal end twists back in a *clockwise* manner 150°, and the proximal end twists back 45° in an *anti-clockwise* direction, towards the left. The incomplete interventricular septum is lying at an angle of 45° to the left of the antero-posterior plane (the right ventricle being somewhat to the right and in front). The bulbar septum can now join the interventricular septum and the partition is finally completed by the upper membranous portion.

This detorsion at the end of the tube proximal to the heart determines the relative positions of the pulmonary artery and

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aorta; the pulmonary artery arising rather in front and to the right and running upwards to the left and backwards; while the aorta comes from the left, forwards upwards and to the right. The two circulations are now "crossed" as well as "in parallel."

It can now be seen that if the "detorsion" is carried too far the aorta will be moved too far to the right, over the septum or even completely dextroposed over the right ventricle—while the pulmonary artery, if not small and deformed as in the Tetralogy of

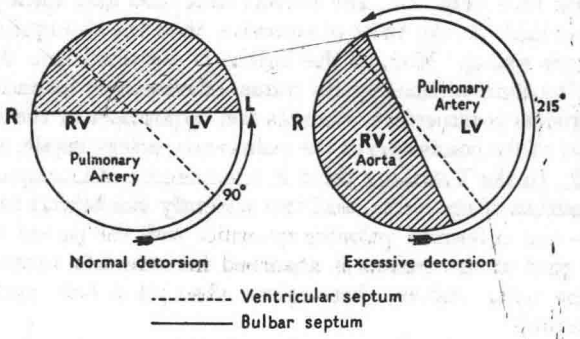

FIG. 5

Fallot, may arise wholly from the left ventricle (complete transposition). (Fig. 5.)

The septum of the truncus, called the aortico-pulmonary septum, becomes continuous with that end of the bulbar septum, which is distal or furthest from the heart. The distal part of the bulbar septum fuses with the proximal part of the bulbar septum. The two sides, right and left, with their efferent trunks, are thus divided by a partition.

The critical period of development lies within the fifth and eighth weeks. During this time, the septa of the auricles and ventricles are forming, and the rotation of the septum in the truncus and bulbus takes place; the cardiac septum fuses with the bulbar; and the bulbus cordis disappears. Many defects arise from the imperfect completion of these changes, and for this reason the defects are finally often multiple.

Abnormal rotation of the bulbus and truncus will result in a faulty position (transposition) of the aorta and pulmonary artery. A primitive single truncus may persist owing to the failure of the septum to develop. Dextroposition of the aorta may result from

this transposition, varying in degree according to the amount of excess detorsion above the 45° anti-clockwise movement needed to bring the bulbar and ventricular septa into line.

The absorption of the bulbus cordis. The bulbus persists in fishes as a separate chamber between the ventricle and the aorta. In the human heart the part embedded in the bulbo-ventricular groove, which forms the bulbo-ventricular ridge, ultimately disappears. It is at this spot that the 45° of detorsion or untwisting of the cardiac end of the bulbus occurs. This spot is called the bulbo-ventricular loop (Fig. 2). The correct detorsion and embedding is very important. If the twist is excessive, then the absorption of the bulbus goes wrong. Most of the bulbus is absorbed into the right ventricle to form ultimately its conus arteriosus or infundibulum. If this process is imperfect, stenosis and hypoplasia of the infundibular part of the conus and of the pulmonary artery result, as Keith supposed. In the Tetralogy there is some degree of transposition or dextroposition of the aorta, and this anomaly lies behind two other features—the deformed pulmonary orifice and the patent septum. A small part of the bulbus is absorbed into the left ventricle just below the aortic valves. Incomplete absorption here causes sub-aortic stenosis.

The atrial canal. Between the primitive atrium and ventricle lies the atrial canal. This is divided into two channels by the fusion of two endocardial swellings, which lie one in front and one behind, to form a partition. The musculature of the atria and ventricles, hitherto continuous, is divided by an ingrowth of connective tissue, so that only a few strands are left, which later become the bundle of His. This may be interrupted in congenital heart block. The septa of the atria and ventricles fuse with these endocardial swellings. If they fail to develop, defects in the septa and atrio-ventricular valves result.

Ætiology. Practically nothing has been known about the causation of congenital cardiac defects. They are often associated with abnormalities elsewhere. Fœtal endocarditis has had but little attention in recent years. The important evidence that rubella may play a part has revived interest in some such possibility. The incidence of congenital defects of various sorts, of which 57% were cardiac, in children whose mothers had rubella in early pregnancy, is very suspicious of some association. Investigation along these lines, with more information as to the type of defect may be valuable. Other virus infections may need consideration.

There is a very slight familial incidence. A woman who has had

one child with a congenital defect of the heart is rather more likely to have another than a woman who has had normal children.

ELECTROCARDIOGRAPHY

General features. The enlargement of one or more chambers of the heart may be indicated. Difficulties concern the right side. The bipolar leads, which are often biphasic, show deviation of the electrical axis in some cases. This should be over + 120° in hypertrophy. It must be remembered that in the first few months of life the right ventricle is normally as large as the left. This determines the normal at that age. The R wave in V1 and VR is of high voltage at birth, and gradually becomes smaller; at the end of the first year the S wave is much larger; Rs tends to become rS. A Q wave seen in V1 at this time indicates pathological enlargement of the right ventricle (*1*). The slightly delayed intrinsicoid deflection to 0·04 sec., bundle branch block being absent, is a point. The R/S or R/Q ratio in VR is over 1·0. The R/S ratio in V1 falls from 7·0 in the first three months to 2·5 at a year, while the ratio of R/S in V5 rises from 0·5 in the first three months to 0·8 at the end of 12 months. There is some overlap in V1 in the very young, but in V5 the large S wave is distinctive (*4*).

The T wave is normally negative from V1 to V4. In some 50% T may be negative to V6, but in a few weeks it usually becomes positive (*2*). In later childhood T may be negative to V4. The heart tends to be electrically vertical in children as shown by the patterns of the unipolar leads from the left arm and foot (*3*). The precordial leads often suggest clockwise rotation round the long axis of the heart. High voltages may be due to the thin chest wall.

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Abnormal types. AURICULAR ENLARGEMENT is shown by P waves of high voltage. These usually refer to the right auricle. The "pulmonary P wave" or "congenital P wave," appears as positive large bifid or prolonged waves in V1 and V2—and also in VF and in VR, where the wave is of course negative. This causes large P waves in standard II and III (*1*). Tricuspid atresia and the Ebstein deformity (*10*) are associated with these waves; and sometimes pulmonary stenosis and the Tetralogy of Fallot. They are seen

in transposition of the great vessels. Increased duration of the P wave may suggest that both auricles are enlarged (*1*), as may be the case in atrial septal defect. Abnormalities of the auricular waves are often best seen in the V3R position (*10*). Situs inversus of the heart of course produces a negative P in standard SI, due to the positive P in VR and the negative P in VL.

RIGHT VENTRICULAR ENLARGEMENT. This causes R waves in V1 and V2 (*2*). After the first year the curves are more reliable. It has been suggested that there may be two types (*3, 5*). These depend on whether the hypertrophy is due to obstruction, e.g. pulmonary stenosis (hypertrophie de barrage) or to overfilling (hypertrophie de surcharge) as in atrial septal defect, or anomalous pulmonary veins (*1, 3, 4, 5*). In the latter the curves are of the pattern of right bundle branch block, with a delayed intrinsicoid deflection in V1 and V2 (secondary R wave). In the former there is gross right axis deviation in the standard leads. QRS is not prolonged; the voltages of R in V1 and V2 tend to be high. If these waves are conspicuous a pressure of over 100 mm. Hg is likely to be present in the right ventricle (*10*). In V2, 3, 4 T tends to be negative. A dominant R wave is seen in VR. A vertical position, with QR pattern in VR, and Rs in V1 is usual (*6*). As time goes on these features become more conspicuous, taking some years to develop (*7*). The curves of right ventricular hypertrophy may be seen in the later years of ventricular septal defect.

LEFT VENTRICULAR ENLARGEMENT may be the cause of left axis deviation and large R waves in V5 and V6 in aortic stenosis: but the vertical position of the heart in early years prevents their appearance. The same applies to coarctation of the aorta. In tricuspid atresia the unusual combination of cyanosis and left axis deviation is seen. This may also occur in persistent truncus arteriosus and in the trilocular heart (*8*).

COMBINED PATTERNS may be seen in the later years of ventricular septal defect and patent ductus arteriosus (*9*), where usually at first the curves are normal. The relative degrees of right or left ventricular hypertrophy may decide the patterns of the cardiogram. But diagnosis is difficult. High voltage R waves may appear in V1 and V2 and also in V5 and V6. Mixed types are seen when the ventricular septum is grossly deficient. The pattern of the predominance or preponderance of one or other ventricle in the limb leads and the deviation of the electrical axis helps in the solution.

In addition to these types suggesting enlargement of one or other ventricle, curves indicating diseases of the myocardium may be

seen when there is an anomalous coronary artery arising from the pulmonary artery, and in fibroelastosis.

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CARDIAC CATHETERISATION

Cardiac catheterisation plays an important part in the diagnosis of congenital heart lesions. In all but the simplest types it is needed for a precise diagnosis. It gives information in three ways: the catheter may take an unusual course by passing through an abnormal orifice in the heart: the pressures in the chambers and vessels accessible to the catheter can be measured: the oxygen content of blood samples can be obtained, and left to right shunts can thus be recognised.

METHOD. A Cournand catheter of size 5–7 is connected by a three-way tap to a saline drip bottle and via a plastic tube to an electromanometer which transforms the pressures to electrical potentials, these can be recorded on an electrocardiograph. The level of the manometer should be brought to that of the right auricle which is taken to be 10 cm. above the level of the X-ray table on which the patient lies. If the level of the sternal angle is preferred, the pressures obtained will be approximately 5 mm. too low and, if reference is to the table itself, they will be 10 mm. too high.

The catheter is normally introduced into the median basilic vein. In young children it may be easier to use the femoral vein which is larger. It is passed up the superior, or inferior, vena cava, into the right auricle and thence through the right ventricle into the pulmonary artery and its branches. Finally it is wedged in a pulmonary arteriole. At this point pressures are recorded and a blood sample is taken. If the catheter is properly wedged in the pulmonary arteriole the oxygen content of the blood will be that of the venous

or oxygenated side of the pulmonary system, and the pressures will approximate to those of the left auricle. In practice it is often difficult to obtain satisfactory blood samples from the wedged catheter, nor do the venous pulsations always come through. The catheter is then withdrawn and samples and pressures are taken from the right and left branches of the pulmonary artery, and from the main trunk, from the infundibulum, and from the body of the right ventricle. The process is repeated with the right auricle and the superior and inferior venæ cavæ.

Sometimes, especially in mitral stenosis, it is helpful to know the pulmonary arterial pressures and oxygen content after exercise. The exercise is performed by the patient pedalling with his feet against resistance while lying on the couch. The oxygen consumption can be measured immediately after exercise so that the pulmonary flow can be calculated.

NORMAL PRESSURES. The normal pressures are as follows: right auricle $-2+2$ mm. Hg; right ventricle, systolic 15–30 mm. Hg, diastolic 0, mean pressure 10 mm. Hg; pulmonary artery, systolic 15–30 mm. Hg, diastolic 5–10 mm., mean about 14 mm. Hg. The pulmonary capillary pressures have a venous form with "a," "c" and "v" waves. The range is from 8–2 mm. Hg (*1*).

Blood samples. These are taken under oil so that the oxygen content can be measured. The slightest admixture with air during the drawing of the sample renders it valueless. The oxygen capacity, or the further amount of oxygen which the sample can take up, is obtained and from this the saturation is calculated. The oxygen contents are used to obtain the arterio-venous oxygen difference, and thus to calculate the pulmonary and systemic flows, and so the flow through any shunts that may be present. The normal oxygen saturation in the right auricle and right ventricle is about 70%. It is slightly higher in the superior vena cava than in the inferior. The different streams of venous blood may mix slowly and the pulmonary artery sample is usually the best to use. The oxygen content of the arterial blood is obtained by puncture from the femoral or brachial arteries.

PULMONARY BLOOD FLOW. The volume of blood flowing per minute through the pulmonary system is obtained by the principle of Fick, and is the amount of oxygen taken up by the lungs in cc. per minute (O_2 consumption) divided by the difference in the oxygen content per litre in the samples taken from the pulmonary vein and from the pulmonary artery. In practice the femoral artery sample is used instead of that from the pulmonary capillary bed as it is

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