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Research Review

The applied anatomy of the arterial blood supply to the heart in man

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INTRODUCTION

In man, as in all mammals, birds and reptiles, the arterial supply to the heart is achieved by two arteries which are the only branches of the ascending aorta. In each case these arteries branch in such a manner that they occupy the atrioventricular and interventricular sulci in the shape of a crown. Hence they are called the coronary arteries. This basic arrangement has been recognised since at least the time of Vesalius (Saunders & O'Malley, 1982), who, like his contemporary, Dryander, provided some early illustrations (Dryander, 1541; Allwork, 1976). Similarly, anomalous arrangements have long been recognised. According to Smith (1950), an example of a single coronary artery was reported by Thebesius in 1716.

Since the 1960s when cineangiography and coronary arteriography were developed, imaging of the coronary circulation in many thousands of people has demonstrated that there is a huge spectrum of variation in the disposition of the coronary arteries. Some of these variants are capable of causing impairment of cardiac function and some can be responsible for sudden death in young, apparently healthy people (Ogden, 1970). This article reviews some of these anatomical variants together with some other factors which can cause myocardial ischaemia.

Because the normal anatomical arrangements of the coronary arteries are so well known, they will not be described in detail here. However, the normal collateral circulation is perhaps less well known, and is included as its applied anatomy is of great importance, especially in acquired coronary artery disease. The ability of the heart to exploit its collateral circulation may determine prognosis in a given individual.

Dominance

The term 'dominant' coronary artery was introduced by Schlesinger (1940) who used it to indicate the areas of heart supplied by each artery. Although the left coronary artery always supplies a greater mass of myocardium than does the right, it is not usually 'dominant'. The dominant coronary artery is that which gives the posterior interventricular branch, traversing the posterior interventricular sulcus and supplying the posterior part of the ventricular septum and often part of the posterolateral wall of the left ventricle as well (Allwork, 1980).

The right coronary artery is dominant in approximately 70% of people. If the circumflex branch of the left coronary artery terminates in the posterior interventricular sulcus, left dominance is present. This occurs in about 15% of people. In

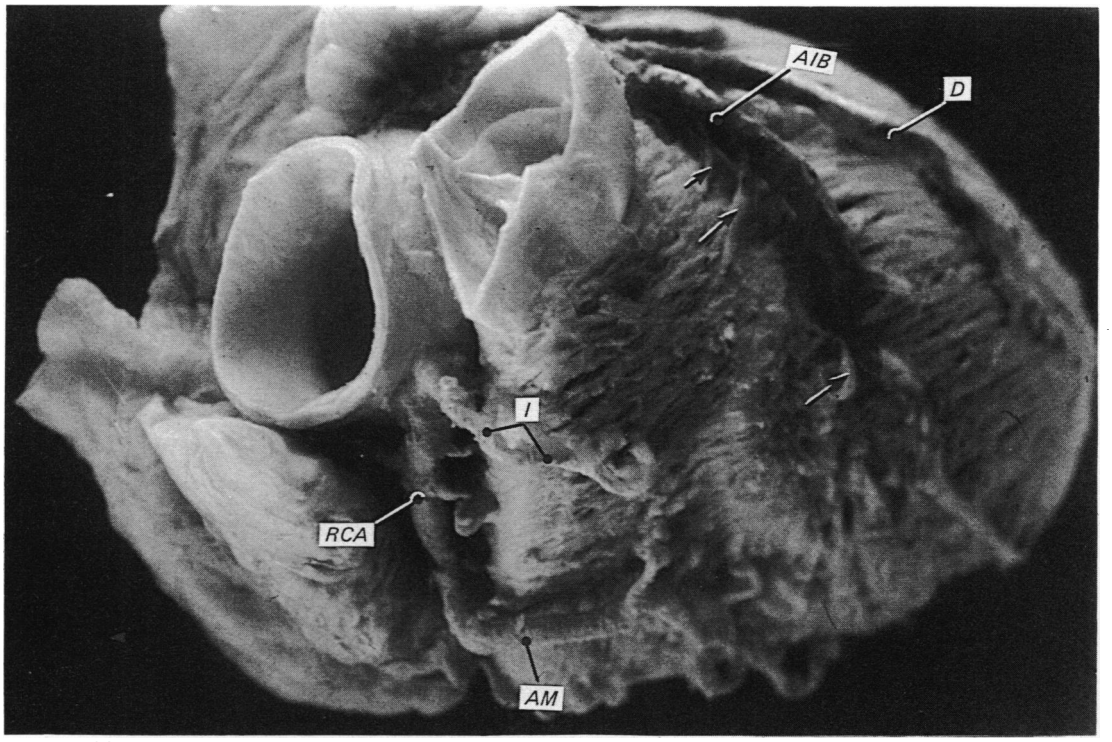

Fig. 1. Intercoronary anastomoses (arrowed). The heart is that of man of 80 without intraluminal disease but with calcification of the adventitia (so-called 'pipe-stem' coronary arteries). *AM*, marginal branch of right coronary artery; *I*, infundibular branch of right coronary artery; *D*, first diagonal branch of left coronary artery; *AIB*, anterior interventricular branch of left coronary artery; *RCA*, right coronary artery.

the remainder the posterior septum is vascularised either by descending branches from both the right coronary and the left circumflex arteries, or by a network of small branches from these two passing obliquely, so that there is no posterior interventricular branch. In such hearts the circulation is said to be 'balanced', as the posterior interventricular branch is either bilateral or absent (Allwork, 1980, 1986).

Dominance can be a significant determinant of prognosis in acquired coronary artery disease. In most individuals with left dominance the right coronary artery is usually small and often fails to reach the acute (right) margin of the heart (Raphael, Hawtin & Allwork, 1980) so that an acute, proximal occlusion could have disastrous consequences, as the potential for rapid development or re-opening of collateral vessels is likely to be diminished. Furthermore, cardiac rhythm may be deranged as the branch passing to the atrioventricular node usually arises from the dominant artery,

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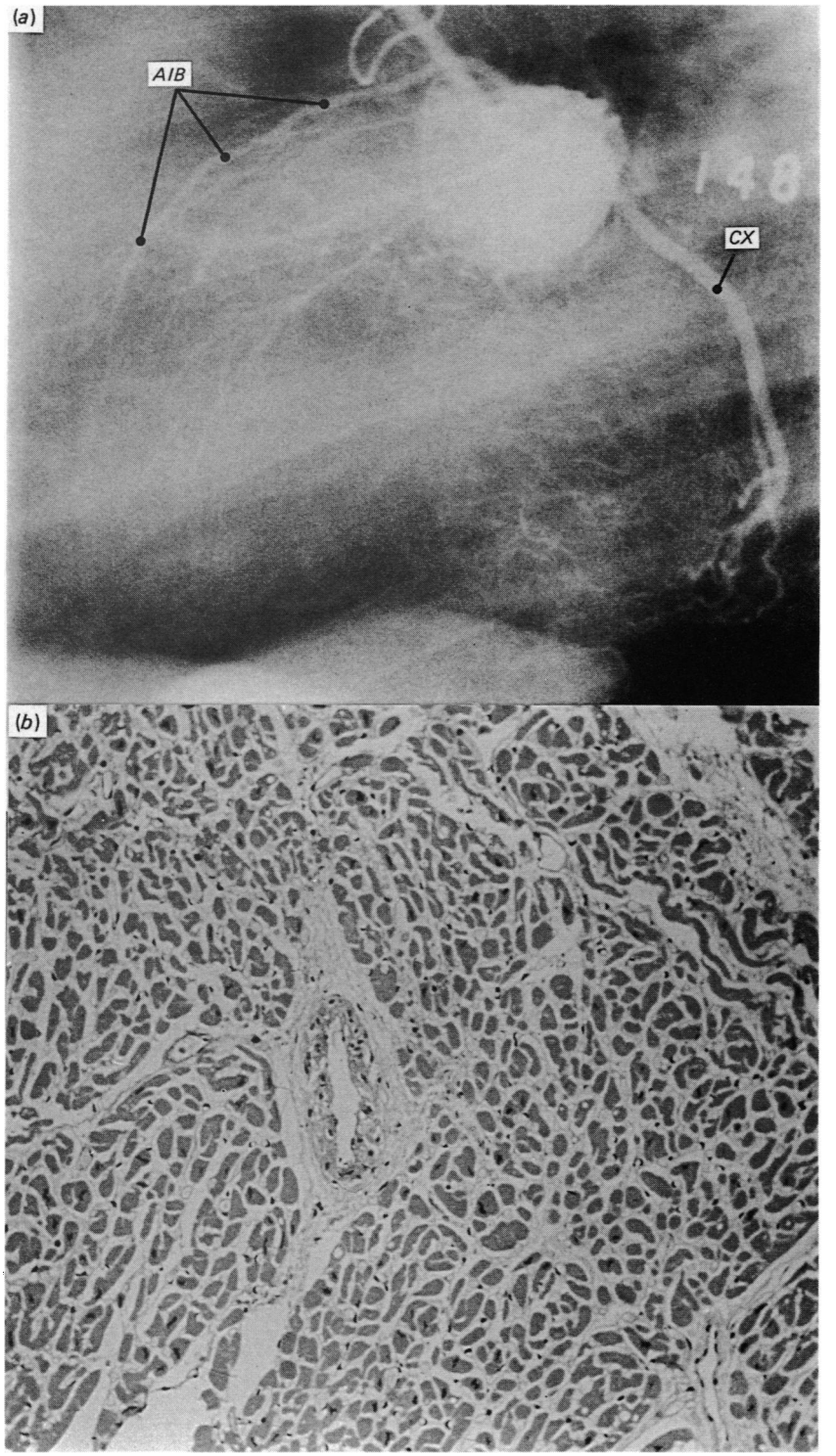
Fig. 2(*a–b*). (*a*) Left coronary arteriogram in a man of 47 who had had aortocoronary bypass grafts 7 years previously. (The loop at the top marks the position of an occluded graft. The opacified tube apparently passing through the loop is the angiographic catheter.) The native anterior interventricular branch is very small and there are numerous even smaller collateral channels. Similarly, multiple collateral vessels have developed in the area of myocardium supplied by the circumflex branch of the left coronary artery (*CX*). (*b*) Same individual. A thick-walled collateral coronary artery passes, without accompanying vein or nerve through the myocardial fibres. There is marked interstitial fibrosis and the wavy bands (top right) are characteristic of myocardial ischaemia. Haematoxylin and eosin. × 150.


(a)
AIB
CX
(b)

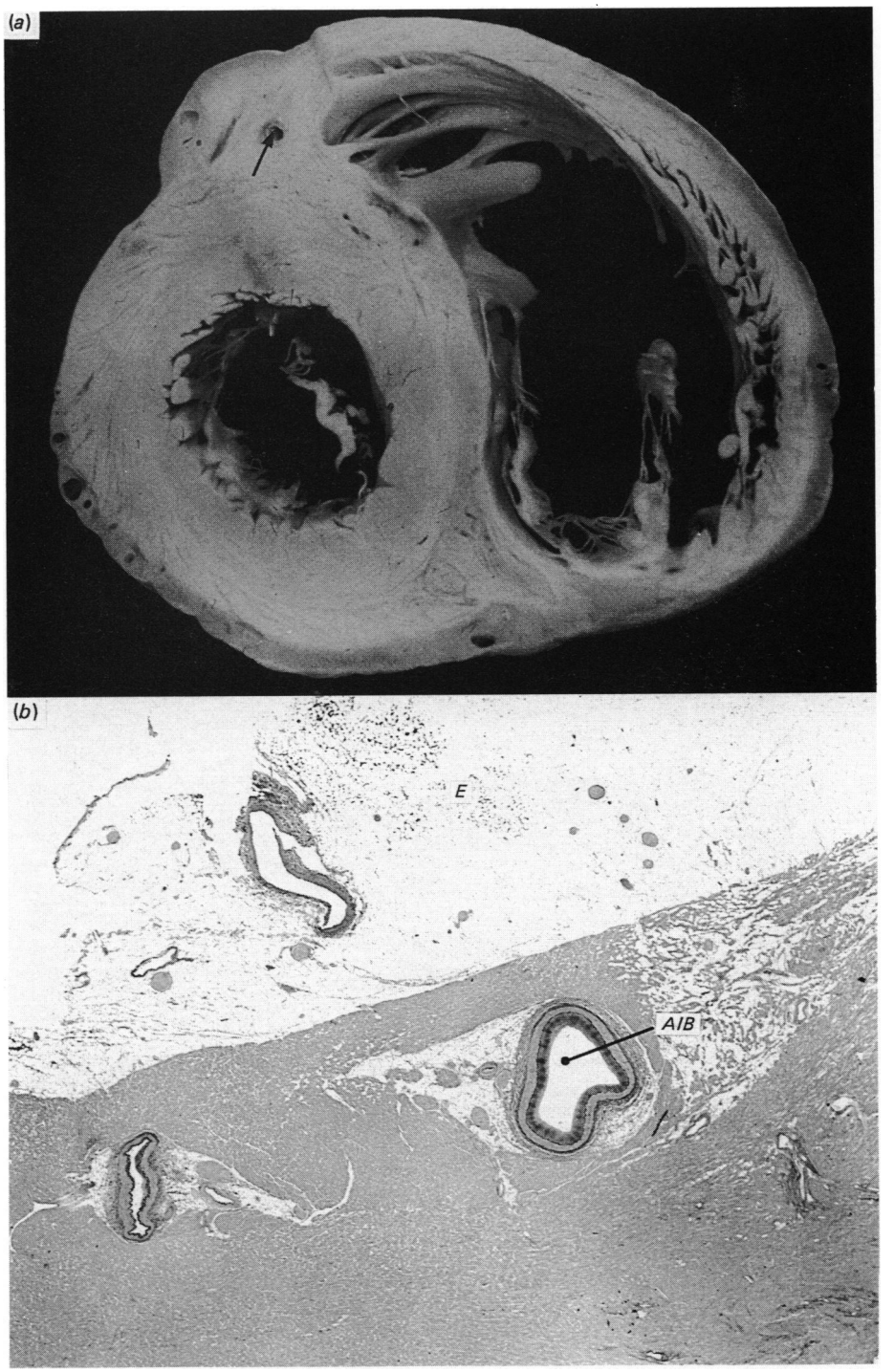


Fig. 3(*a*, *b*). (*a*) Intramyocardial anterior interventricular branch of the left coronary artery (arrowed) in a woman of 30 who died as a result of an accident. The vessel was 2 mm deep in the muscle and passed through it for 3 cm before regaining a subepicardial position. (*b*) Intramyocardial anterior interventricular branch (*AIB*) in a man of 70 with severe angina. The artery was small, and although there was some intimal hyperplasia, it was unaffected by the atheroma which was abundant in the remainder of his coronary circulation. Miller's Elastic Van Gieson. *E*, epicardial fat. × 12.

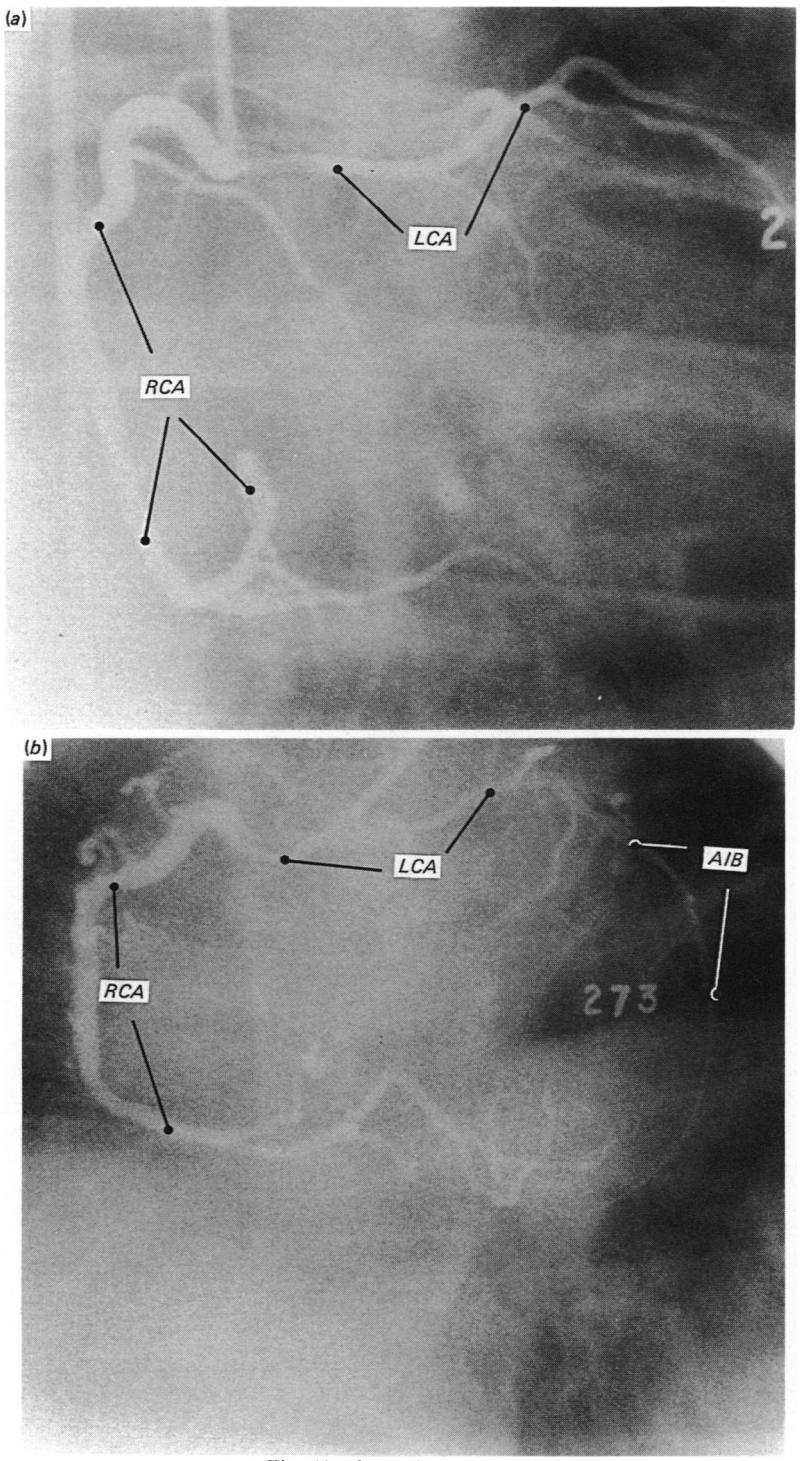
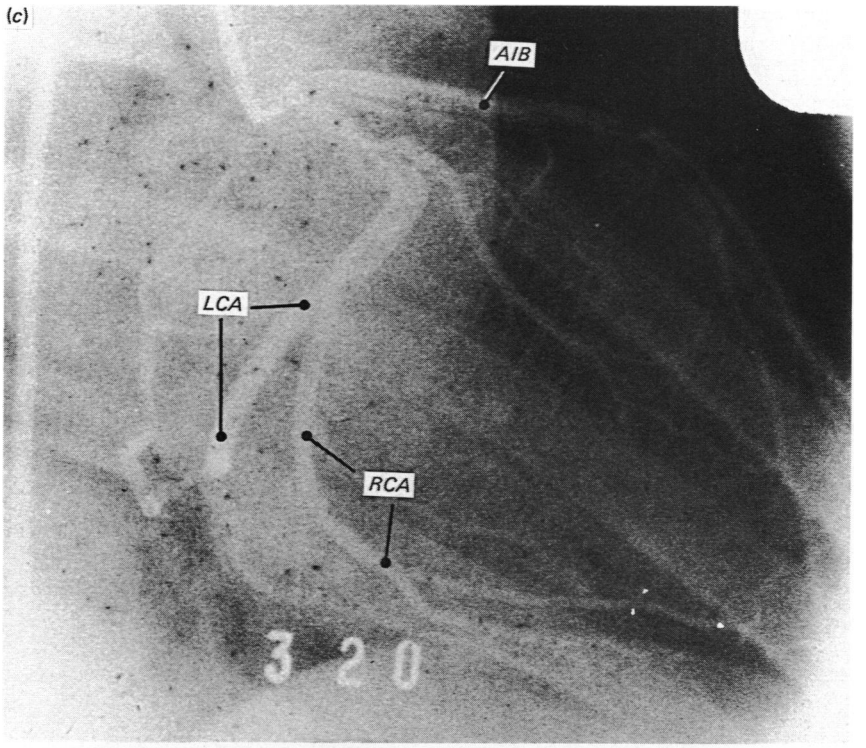


Fig. 4(*a–d*) For legend see page 7.


(c)
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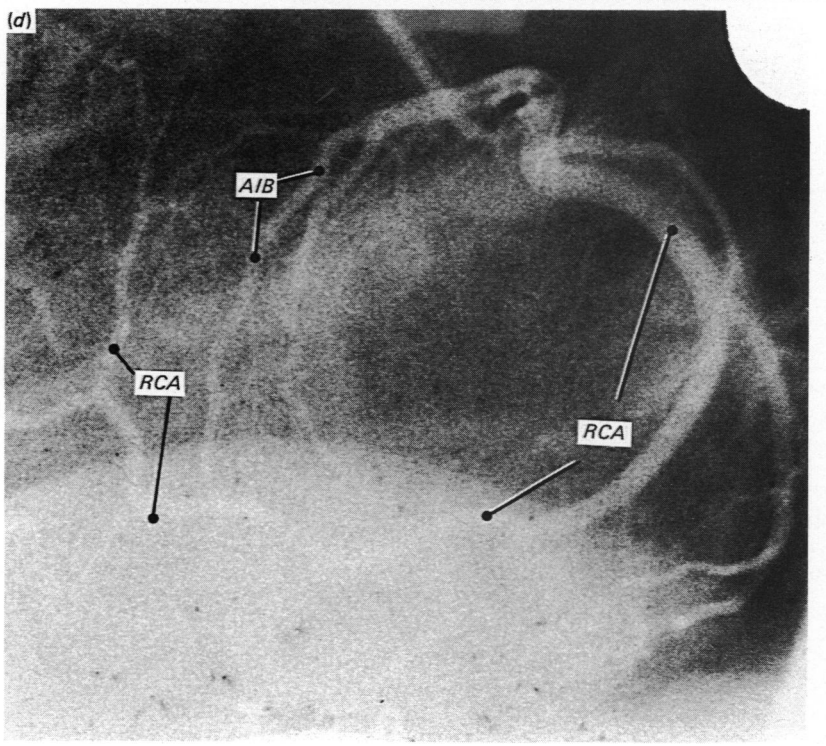
LCA
RCA
3 2 0


(d)
AIB
RCA
RCA

as does that to the sinuatrial node (Hutchinson, 1978). In hearts with left dominance the atrial branch which eventually supplies the sinuatrial node usually springs from the circumflex branch of the left coronary artery; atrial branches from the undivided, proximal left coronary artery are uncommon (Allwork, 1980).

Collateral coronary circulation

Intercoronary anastomoses are prominent in fetal life and persist until about the eighth postnatal month when they diminish in size (Castorina, 1957).

Collateral circulation was divided into two types, 'homocoronary' and 'intercoronary' by Baroldi, Mantero & Scomazzoni (1956). Homocoronary anastomoses occur everywhere except in the immediate subepicardial layer, while intercoronary anastomoses are more usually subepicardial (Fig. 1). When collateral channels expand in response to demand (e.g. hypoxia, coronary artery disease) they are usually easy to identify as they are unaccompanied by veins, and tend to pass parallel to or obliquely to the myocardial fibres (Fig. 2*a*, *b*).

The significance of this collateral circulation is that a potential exists for protecting the myocardium against the deleterious effects of ischaemia. However, there has been controversy about whether such protection actually exists: Harris *et al.* (1972) were unable to show that patients with a well-developed collateral circulation fared any better with respect to ischaemic changes on the electrocardiogram during exercise tests than those without. However, when ischaemic disease was treated with aortocoronary bypass grafts, those with a good collateral circulation all survived operation, but 5·4% of those without collaterals died at or soon after operation (Shimizu *et al.* 1986).

The author, in common with others, has also often observed that many individuals do not exploit their 'dormant' collateral circulation despite severe and chronic ischaemia (Blumgart, Schlesinger & Davis, 1940; Cox *et al.* 1975). It seems possible that factors additional to ischaemia may be invoked to redevelop the circulatory system which had been present at birth, but the natural history of the collateral circulation in man is still uncertain (Ambrose & Fuster, 1983).

Experimental studies have shown that the major determinant of the size of an acute infarction is the volume of flow passing through the collateral channels (Hirzel, Nelson, Sonnenblick & Kirk, 1976). Gregg (1974) demonstrated that in dogs which survived an acute occlusion, collateral flow to the endocardial layer doubled in the first 24 hours, and that even gradual additional stenosis induced over several days resulted neither in infarction nor death. Daily exercise enhanced development and enlargement of the collateral circulation in dogs (Lambert, Hess & Bache, 1977) but unfortunately perhaps, exercise does not induce this potentially satisfactory event in man (Nolewajka, Kostuk, Rechnitzer & Cunningham, 1979). (It should be noted that all

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Fig. 4(*a–d*). (*a*) Single coronary artery from the anterior aortic sinus. Coronary arteriogram, anterior oblique projection. The right coronary artery (*RCA*) is normal and pursues a normal course. A smaller artery also opacifies (*LCA*) but neither the origin nor its destination can be identified in this view. (*b*) Left anterior oblique projection. The left coronary artery (*LCA*) originates from the right artery and is small compared with it. The left artery appears to pass between the aorta and pulmonary artery to emerge on the anterior surface of the heart where it gives off the anterior interventricular branch and then continues its course. There is no intraluminal disease. (*c*) Single coronary artery from the left posterior aortic sinus. Right anterior oblique projection. The proximal coronary artery is short, dividing early into an anterior interventricular branch (*AIB*) and another to the left atrioventricular sulcus. This latter branch divides into two, one mimicking the continuation of a left coronary artery (*LCA*) and the other passing to the right (*RCA*). (*d*) Left anterior oblique projection. The morphology is more easily identified in this view, the larger of the two branches in the left atrioventricular sulcus passes to the right to occupy the right atrioventricular sulcus (*RCA*). It also gives branches to both the right atrium and right ventricle.

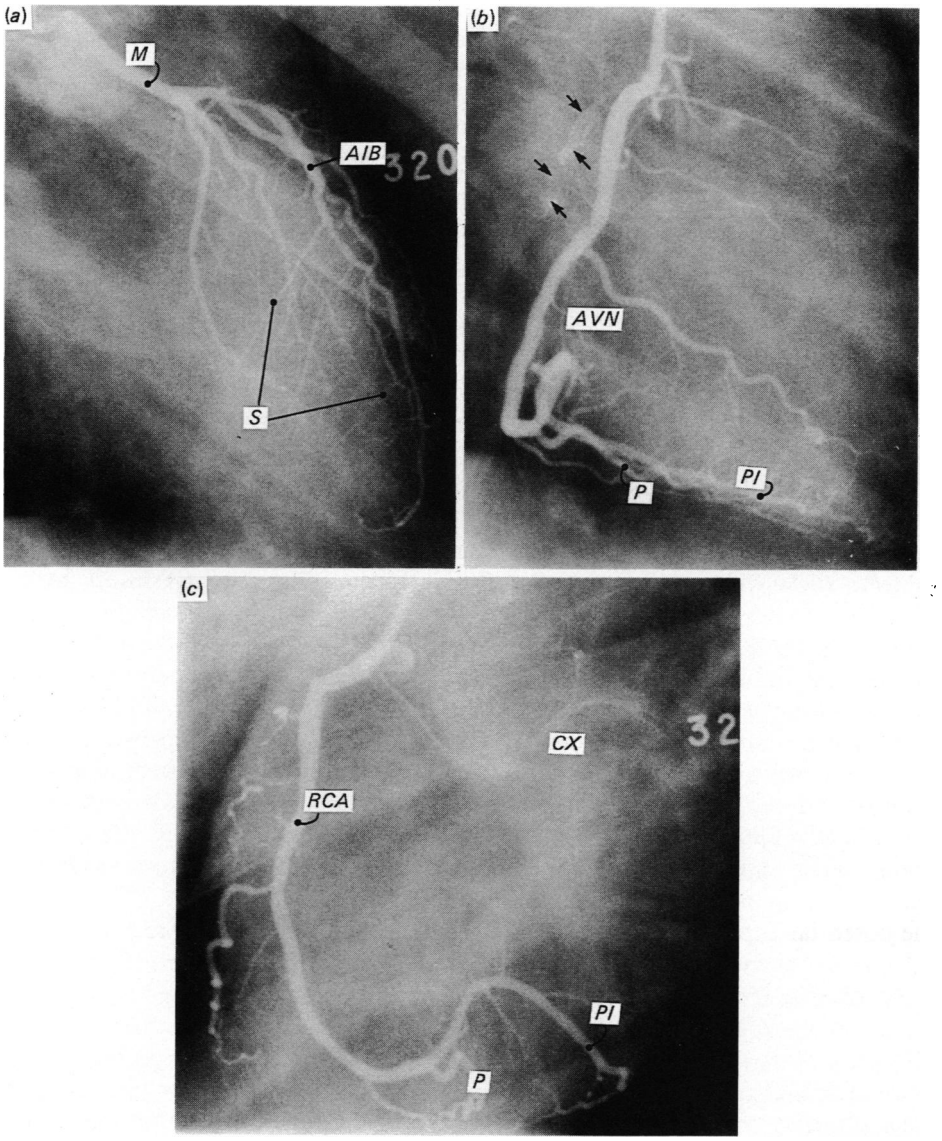

Fig. 5(*a–c*). (*a*) Aberrant circumflex branch. Left coronary arteriogram in right anterior oblique projection. The main stem (*M*) is unusually long in this individual; it divides into anterior interventricular and first diagonal branches and two additional diagonal branches. No branch passes posteriorly in the atrioventricular groove (to the left of the opacified arteries). There is a quite long narrowed segment in the most proximal part of the interventricular branch. Note also the septal arteries (*S*) arising at right angles from the undersurface of the interventricular branch to vascularise the anterior part of the ventricular septum. (*b*) Right coronary arteriogram. The normal right coronary artery pursues its course in the right atrioventricular sulcus. It loops into the myocardium at the crux cordis. A small branch (*AVN*) arises from the summit of the loop to supply the atrioventricular node. The relatively straight posterior interventricular branch (*PI*) is recognised by its numerous septal branches passing vertically upwards to vascularise the posterior part of the interventricular septum. The posterolateral (*P*) branch overlaps the posterior descending branch to some extent. The aberrant circumflex branch is just discernible (between the arrows) as it 'flash-fills'. Its course and destination cannot be identified in this view. (*c*) Right coronary arteriogram, left anterior oblique projection. The aberrant circumflex artery is clearly recognisable (*CX*). It loops at the base of the aorta and passes to the left atrioventricular sulcus. Its termination is unclear. The right coronary artery is 'opened-out' in this projection, and the posterior interventricular and posterolateral branches are easily distinguished.

dogs normally have a very large collateral circulation throughout life, so that with respect to myocardial perfusion, dogs are unsuitable subjects for comparison with man.)

Extracoronary collateral arteries

The possibility of a non-coronary collateral circulation was postulated by Thebesius in 1708 (Bloor & Liebow, 1965) in his description of 'openings' in the atrial and ventricular walls. These openings (venae cordis minimae or Thebesian veins) are an unlikely source of supply to the subendocardium because of inappropriate pressure gradients in both systole, and diastole (Archie, 1974). Furthermore, if they were a source, then the subendocardium, instead of being the myocardial region most vulnerable to ischaemic damage, would be the best protected (Griggs, Tchokoev & Chi Chen, 1972).

In addition, non-coronary collaterals sometimes develop in response to ischaemia. They arise as tortuous vessels from the mediastinal, pericardial and bronchial vessels and enter the heart through the pericardial reflections surrounding the pulmonary and systemic veins. They also sometimes develop from the vasa vasorum of the major arteries and veins in contact with both atrial and ventricular myocardium (Hudson, Moritz & Wearn, 1932; Kline, Stern, Bloomer & Liebow, 1956; Bloor & Liebow, 1956).

Flow from this non-coronary source, when present, is always small, but is greater in the atria (4·0–12·0 ml/100 g myocardium/minute) than in the ventricles (0·25–0·75 ml/100 g myocardium/minute) (Brazier, Hottenrott & Buckberg, 1975). Evidence of non-coronary supply to the heart is often observed during cardiac surgical operations, and although small, is probably sufficient to prevent complete anoxia during aortic cross clamping. The oxygen needs of the arrested heart at 37 °C are modest (approximately 2 ml/100 g/minute) (McKeever, Gregg & Canney, 1958) and fall markedly when myocardial temperature is reduced (Fuhrman, Fuhrman & Field, 1959).

The potential importance of this circulation is demonstrated in the few people who, despite total occlusion of all three major coronary arteries, maintain good ventricular function (Brazier *et al.* 1975).

Anatomical variants of coronary artery disposition

Although anomalies of these arteries are relatively rare causes of cardiac pathology, atypical position or branching may be found in up to about 2% of human beings (Ogden, 1970).

Intramyocardial arteries

Although the major branches of the coronary arteries are generally subepicardial, they are quite frequently contained in places by strands of myocardium ('myocardial bridges'). Mostly these strands are small and of no significance. According to Polacek & Zechmeister (1968, summarised by Boucek, Morales, Romanelli & Judkins, 1984) they occur in up to 60% of normal hearts. The bridges range in length from 9–69 mm, but are usually between 10–20 mm and up to 5 mm thick. They occur most often over the anterior interventricular branch of the left coronary artery and its diagonal branches; neither the right coronary artery nor the circumflex branch of the left artery is usually affected as the vessels occupy the atrioventricular sulci, but the right marginal and posterior interventricular branches of the right artery may be overlain by small (5–10 mm) 'loops' of muscle (Polacek, 1961).

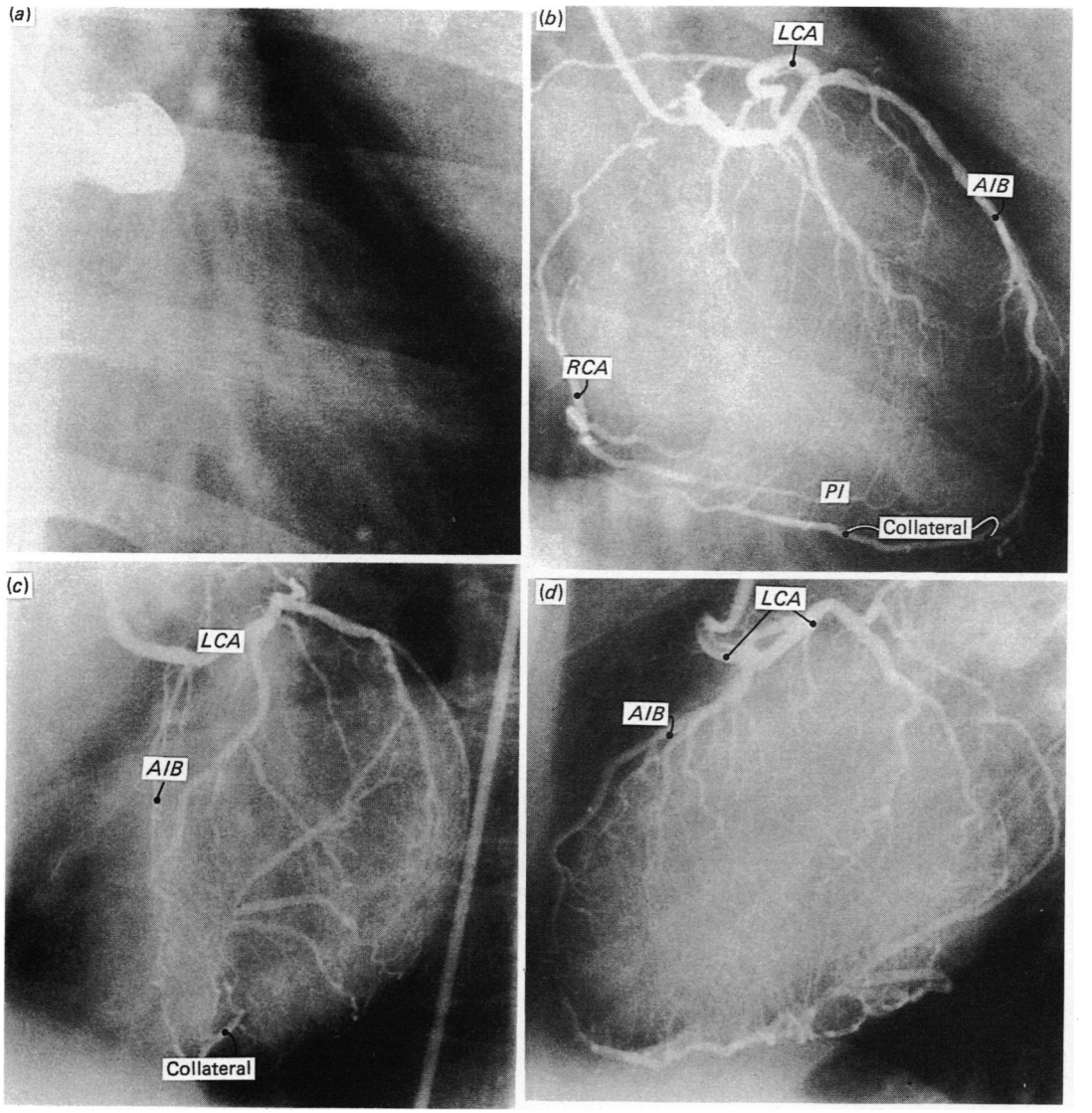

Fig. 6(*a–d*). (*a*) Origin of the left coronary artery from the anterior aortic sinus. Opacification of the left posterior aortic sinus does not outline a coronary artery. (*b*) Right coronary arteriogram, right anterior oblique projection. The right artery is severely narrowed. The uneven lumen of the anomalous left artery indicates atheromatous plaques in its proximal portion. It passes anterior to the pulmonary artery to give an anterior interventricular branch (*AIB*) before continuing in the atrioventricular groove. The right artery fills through a very large intercoronary collateral at the apex of the heart. *PI*, Posterior interventricular branch. (*c*) Steep left anterior oblique projection. The unusual morphology of the left artery coupled with the occlusion of the right artery make identification of all the branches difficult. (*d*) Lateral projection. The anterior position of the proximal left coronary artery is confirmed; the apical anastomosis is well seen. The numerous branches, many of them collaterals, are easier to distinguish.

The anterior interventricular branch occasionally passes through muscle for much of its length and Geiringer (1951) coined the term 'intramural' for this variant (Fig. 3*a*, *b*). An intramural left coronary artery surrounded by fibrous tissue has been found in babies who have succumbed to 'cot death' and has been implicated as a possible contributory cause of fatal ventricular arrhythmia (Boucek *et al.* 1984).

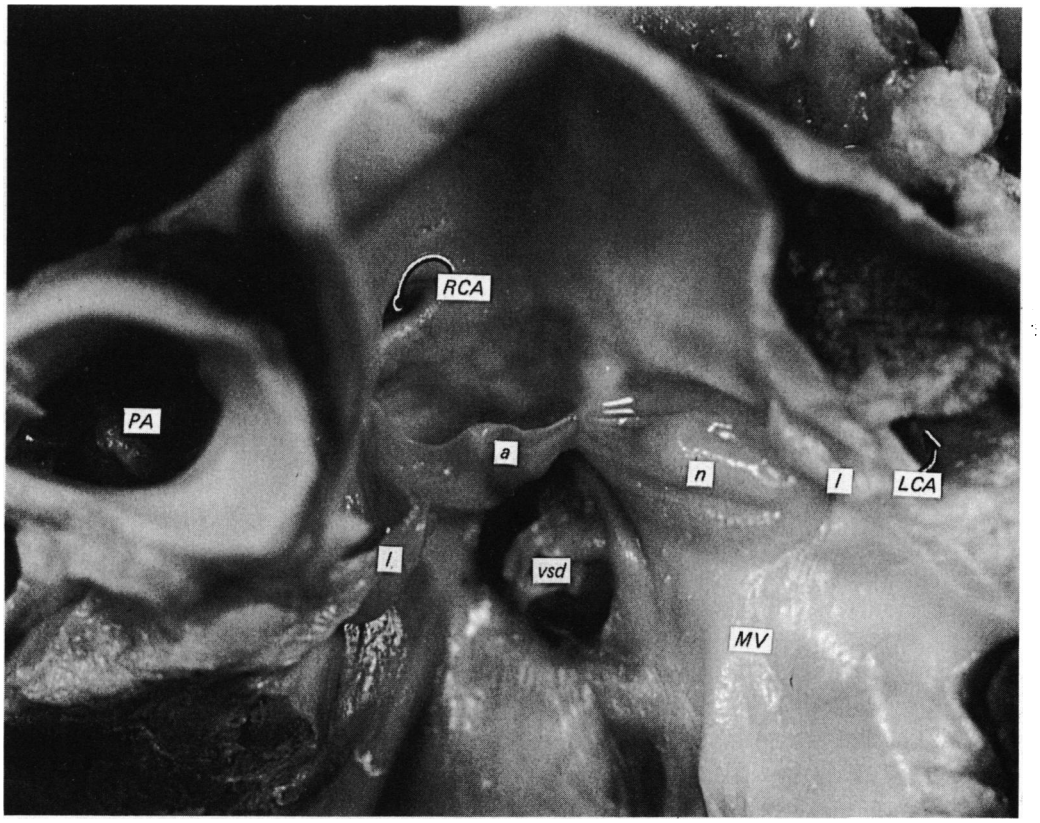

Fig. 7. Ectopic origin of the right coronary artery. The large right coronary artery originates a few millimetres above the anterior aortic sinus, while the left arises in the ordinary manner from the left posterior sinus. Child of 1 year with an interventricular communication (*vsd*) below the non-coronary (right posterior) aortic sinus. *l*, left posterior aortic sinus and leaflet; *n*, non-coronary sinus and leaflet; *a*, anterior aortic sinus and leaflet. *MV*, mitral valve; *PA*, pulmonary artery.

In the absence of intraluminal disease the intima of the artery is 30% thicker proximal to the bridge than it is in the subjacent segment (Polacek, 1961). When disease is present the proximal segments are more severely affected than those subjacent and distal to the bridge (Stolte, Weis & Prestele, 1977; Penther, Blanc, Boschat & Granatelli, 1977).

Bridging of the left coronary artery occurs with greater frequency in people with a dominant left coronary artery, and the bridges are wider and deeper than in the more usual right dominance (Polacek & Zechmeister, 1968). Thus not only is a dominant left system disadvantaged by its very existence but it is further at risk because of a common anatomical variant. The submerged part of the artery often demonstrates compression during systole. The degree of compression varies from negligible to severe, as expressed by a spectrum of pathology ranging from angina (Faruqui *et al.* 1978) through tachyarrhythmias and myocardial infarction (Endo, Lee, Hayashi & Wada, 1978) to sudden death (Morales, Romanelli & Boucek, 1980). Active exercise (swimming and jogging in particular) seem to be associated with sudden death in otherwise healthy young people with this anatomical variant (Boucek *et al.* 1984).

Single coronary artery

Single coronary artery is rare in normal hearts (Ogden, 1970; Allwork, 1979) but occurs with some frequency in congenitally malformed hearts. A single coronary artery may originate from either the left posterior or the anterior aortic sinus and it usually has the disposition of either a left or right coronary artery according to its origin (Fig. 4*a*,*b*), but it may branch in a manner resembling both normal arteries (Fig. 4*c*,*d*), or, more rarely in normal hearts, resemble neither. Single right arteries have greater morphological variability than left ones (Neufeld & Schneeweiss, 1983).

Single arteries may pass posterior to the aorta, or between it and the pulmonary artery. In the latter state, the coronary artery is at risk from compression. Single arteries may also pass anterior to the pulmonary artery. A right coronary artery anterior to the pulmonary artery is uncommon in normal hearts but it occurs with some frequency in congenital cardiac malformations that produce cyanosis.

Single coronary arteries are highly susceptible to atherosclerosis (Alexander & Griffith, 1956) and of course, intercoronary collaterals are absent, so that possession of a single coronary artery is disadvantageous.

Aberrant origin of the coronary arteries

Abnormal origin of the left coronary artery

As coronary arteriography has developed, a wide range of anomalies has been disclosed. All the variants considered here are slightly commoner in males. The commonest of these is an abnormal origin of the circumflex branch of the left coronary artery. This springs from the right coronary artery and usually crosses the posterior part of the aortic root and passes in the left atrioventricular sulcus (Fig. 5*a–c*) but if it passes between the aorta and pulmonary artery it is at risk from compression. The same applies to this artery when it originates from an ostium in the right posterior aortic sinus: this too is a relatively common variant. In a total of 213 adult patients with congenital coronary anomalies, the former pattern was noted in 32%, while the latter accounted for 13·6% (Boucek *et al.* 1984). Absence of the left coronary artery occurred in 21% of the same series, while anomalous origin of the anterior interventricular branch from the right coronary artery occurred in only 5%. (This too is fairly common in defects which cause cyanosis.)

Origin of the left coronary artery from the anterior aortic sinus

Individuals with this anomaly (Fig. 6*a–d*) are at risk from sudden death (Ogden, 1970; Liberthson *et al.* 1974). The anomaly can cause exertional pain, syncope and myocardial infarction, due presumably to either compression between the aorta and pulmonary artery or compression as the artery passes obliquely through the aortic wall. Proximal stenosis is common and, like single arteries, the anomalous arteries are susceptible to atheromatous disease at a young age (Liberthson, Dinsmore & Fallon, 1979).

Origin of the right coronary artery from the left posterior aortic sinus

This arrangement is no more benign than aberrant left coronary artery. The ostium may be narrowed to a slit, and the artery must pass obliquely through the aortic wall, thence between the aorta and pulmonary artery (Roberts, Siegel & Zipes, 1982).

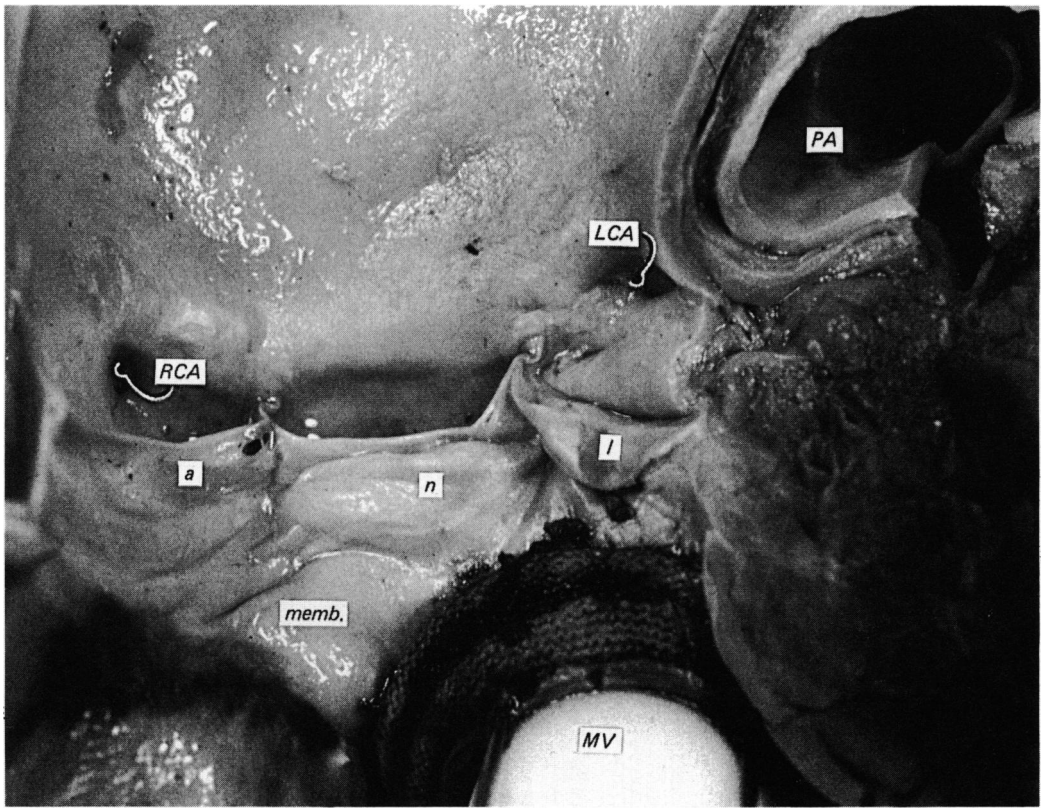

Fig. 8. Ectopic origin of the left coronary artery. Woman of 60 with rheumatic mitral valve disease necessitating valve replacement (bottom of picture). The right coronary artery was normally located. Neither had intraluminal atheromatous disease. *Memb*, membranous part of the ventricular septum.

Ectopic origin of the coronary arteries

Origin of the right coronary artery from the ascending aorta, usually 1 cm or more above the aortic sinus, is a fairly common variant (Anderson & Becker, 1980) (Fig. 7). The artery passes obliquely through the aortic wall, and its proximal portion sometimes tunnels through it. When this occurs the tunnel and the muscular artery may fuse and focal intimal hyperplasia can result (Boucek *et al.* 1984).

Ectopic origin of the left coronary artery (Fig. 8) is uncommon in otherwise normal hearts, but like the right, it may tunnel within the aortic wall or the muscular septum, and is thus exposed to the same risks of hyperplasia.

Coronary arteries arising from the pulmonary artery

When the left artery is affected a very severe congenital disease, often fatal in infancy, is present and is therefore outside the scope of this review. Anomalous origin of both arteries from the pulmonary trunk, usually incompatible with the survival of the baby for more than a few weeks, is also excluded.

Origin of the right coronary artery from the pulmonary artery however, although uncommon, is not usually a lesion that produces symptoms, being more often an incidental finding at arteriography or necropsy. Occasionally the collateral flow is so great that a left-to-right shunt occurs, necessitating surgical treatment (Achtel, Zaret, Iben & Hurley, 1975).

A very rare variant is the origin of the circumflex branch of the left coronary artery from the pulmonary artery (Ott, Cooley, Pinskey & Mullins, 1978). Of four cases reported, three had congenital intracardiac or aortic defects as well (Honey, Lincoln, Osborne & de Bono, 1975). The anomalous artery fills through collateral channels and a coronary 'steal' phenomenon may occur.

Origin of a coronary artery from the aortic arch or from the internal thoracic artery is almost completely confined to hearts with other major congenital malformations, for example, solitary pulmonary trunk (Allwork & Bentall, 1973). When it occurs, it may cause the same 'steal' phenomenon as in those originating from the pulmonary artery.

Abnormal communications

As the foregoing review has indicated, there is, in early life if not later, an elaborate collateral circulation, so that the coronary arteries are not anatomically end-arteries. Hence it is perhaps not surprising that this system may become over-elaborated so that coronary artery fistulae (often multiple) and aneurysms sometimes occur. Such fistulae are uncommon (Neufeld & Schneeweiss, 1983) in otherwise normal hearts but, like other coronary arterial anomalies, less so in congenitally malformed hearts (McNamara & Gross, 1969). Fistulae may communicate with any other coronary branch, with a cardiac chamber, or with the pulmonary circulation. Corono-cameral fistulae cause a 'steal' phenomenon and thus cause myocardial ischaemia (Ahmed, Haider & Regan, 1982).

Coronary artery fistulae rarely become apparent in early life, but may be an important factor aggravating intraluminal disease (Bosc, Grolleau-Raoux & Bertinchant, 1986).

SUMMARY

The applied anatomy of the coronary arterial and collateral circulations has been reviewed together with some of the more important variants of origin, branching and disposition.

There is a very wide range of variability among coronary artery patterns, and some of them can of themselves give rise to illness and even death. Others may exacerbate acquired disease. The increasing use of coronary arteriography and other forms of imaging will continue to reveal hitherto undescribed variants, while the study of coronary artery disease, so common in the Western world, will eventually elucidate the natural history of the coronary collateral circulation.

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