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Coronary and Endocardial Fibroelastosis of the Ventricles in the Hypoplastic Left and Right Heart Syndromes

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Summary. In an autopsy material of 29 cases of the hypoplastic left heart syndrome coronary fibroelastosis was found in 1 case, endocardial fibroelastosis in 8 cases. Figures for 10 cases of the hypoplastic right heart syndrome were 6 cases of coronary fibroelastosis and 1 case of endocardial fibroelastosis. Age ranged from stillborn up to $11^1/_2$ months. Coronary and endocardial fibroelastosis seemed to be mutually exclusive localizations of congenital fibroelastosis since in our material they did not occur together in the same hearts.

In hypoplastic right hearts *coronary fibroelastosis* was either restricted to the right coronary artery (right circumflex and posterior interventricular branch), or it was found also in the left coronary artery (anterior interventricular branch), with the most serve affections always being situated in the right one. In the only case of coronary fibroelastosis among the hypoplastic left hearts the condition was limited to the anterior interventricular branch of the left coronary artery which communicated with the hypoplastic left ventricle by a fistula. Coronary fibroelastosis was exclusively found in branches supplying the hypoplastic right ventricle and/or in a branch connected by a fistula to the hypoplastic left or right ventricle. *Endocardial fibroelastosis* was generally found in hypoplastic left ventricles with either no outflow or with severe outflow obstruction.

A theory concerning the aetiology of both coronary and endocardial fibroelastosis of the hypoplastic ventricles is proposed. It is argued that development of fibroelastosis may in both localizations be caused or favoured by the coincidence of two factors: abnormal haemodynamic conditions and poor oxygenation of blood and tissues. Observations made in a reference material of 35 hypoplastic left and 24 hypoplastic right hearts were in accordance with this view.

Introduction

Fibroelastosis of the coronary arteries may accompany the hypoplastic left and right heart syndrome (Oppenheimer and Esterly, 1966; Bryan and Oppenheimer, 1969; Pyanov, 1972; Newton and Misugi, 1960; MacMahon and Dickinson, 1967; Nodzicka and Vortel, 1971; Le Tan Vinh, 1968; Ventura *et al.*, 1969). As an obstructing coronary disease coronary fibroelastosis is of clinical importance since it may contribute to cardiac malfunction. The combination of ventricular hypoplasia and coronary fibroelastosis is also of interest from the point of view of pathogenesis. The question arises whether in the hypoplastic left and right heart syndromes endocardial fibroelastosis, which may be present also, occurs independently of coronary fibroelastosis or, on the other hand, preferentially in

combination with or mutually exclusive from coronary fibroelastosis. The literature points to the latter relationship (Oppenheimer and Esterly, 1966; Bryan and Oppenheimer, 1969).

Furthermore, the question arises whether coronary fibroelastosis occurs in these hearts preferentially in the artery supplying the hypoplastic ventricle. This has mainly been answered in the affirmative (Oppenheimer and Esterly, 1966; Bryan and Oppenheimer, 1969; Pyanov, 1972), although some data throw doubt on this view (Newton and Misugi, 1960; MacMahon and Dickinson, 1967). Lack of consensus about this point may in part be due to the fact that generally both coronary arteries are involved in the blood supply of both ventricles, albeit with a broad spectrum of individual variations.

Also, it would seem of practical value for surgeons and pathologists to know whether the existence of coronary fibroelastosis can be predicted on the basis of the presence of meandering and/or other macroscopical changes of the coronary arteries supplying the hypoplastic ventricle (Klein, 1965).

Finally, it was attempted to define factors involved in the pathogenesis of both coronary and endocardial fibroelastosis. An autopsy material of 29 cases of the hypoplastic left and of 10 cases of the hypoplastic right heart syndrome from stillborn babies and patients up to 11.5 months of age allowed us to reinvestigate these questions. Our views could be checked using a reference material from the department of Anatomy and Embryology of the University of Leiden. It consisted of 35 cases of the hypoplastic left and 24 cases of the hypoplastic right heart syndrome from patients up to 26 months of age.

Material and Methods

Twenty-nine cases of the hypoplastic left heart syndrome and 10 cases of the hypoplastic right heart syndrome, found among 150 infantile hearts with congenital malformations, collected by us in recent years, were studied. All babies were born at term. The age range of the left hypoplasia cases was from 0 days (stillborn) up to 2 months and of the right hypoplasia cases from 1 day up to 11.5 months. From the total of 39 hypoplasia cases only 8 babies (21%), i.e. 5 out of the total of 29 with left and 3 out of the total of 10 with right hypoplasia, survived beyond the first month, whereas 21 babies (54%), i.e. 18 with left and 3 with right hypoplasia, died within the 1st week, Males comprised 27 cases (69%) of the total number, i.e. 24/29 of the hypoplastic left and 3/10 of the hypoplastic right heart group. Distribution of ages and sexes in the reference material of 35 cases of hypoplastic left and 24 cases of hypoplastic right heart syndrome, was similar.

The coronary arteries were dissected free in cases where thorough inspection of greater branches proved impossible. Tissue was taken whereever changes, such as meandering, localized thickenings, white spots and/or indurations were found. Material was also removed from standard sites in the proximal parts of the anterior and posterior interventricular branches. If the latter had already been partly removed at routine autopsy, tissue was taken more apically or from the terminal part of the right circumflex artery. Tissue samples always consisted® of a segment of an artery with adjacent myocardium. After formalin fixation and paraplast embedding the tissues were sectioned serially at 10 μ in a plane usually transverse to the arterial course. Staining was by the Lawson(elastine)-hematoxylin-azophloxin-saffran technique which proved useful both for overall histology and for demonstration of elastic fibres.

Similar tissue samples from 8 normal hearts of children of 0–24 months of age were processed in the same way and served as control material.

The criterion for coronary fibroelastosis was coexistence of: splitting up and fragmentatioa of the lamina elastica interna, proliferation especially of the elastic component in the tunica

media, and loss off clear distinction between tunica media and tunica intima (Fig. 2; MacMahon and Dickinson, 1967; Esterly and Oppenheimer, 1967; Neufeld and Vlodaver, 1971). Endocardial fibroelastosis was diagnosed on the basis of presence of a greater than normal amount of fibroelastic tissue between the endocardial lining and the myocardium (Andersen and Kelly, 1956; Pikiel, 1969). Cases in which diagnosis of presence or absence of fibroelastosis provep difficult were not encountered.

Observations

Using 3 parameters both the hypoplastic left and the hypoplastic right hearts were subdivided into 8 groups each. Two of these parameters characterized the condition on the hypoplastic side: 1 presence or absence of atresia of the atrioventricular canal; 2 presence or absence of atresia of the aortic or pulmonary ostium. The 3rd parameter was the presence or absence of a ventricular septal defect (Fig. 1 a and b). Ostia of normal size were never found on the affected side. They were in all instances stenotic in a degree corresponding to or more pronounced than the degree of ventricular hypoplasia.

As shown in Fig. 1, coronary fibroelastosis was found in 1 out of the 29 hypoplastic left hearts and in 6 out of the 10 hypoplastic right hearts. In 5 of the 6 cases of coronary fibroelastosis among the hypoplastic right hearts the right ventricle had only an inflow and no outflow, since a patent tricuspid ostium was found in combination with an atretic pulmonary ostium and an intact interventricular septum. It is also remarkable that of the 6 cases of coronary fibroelastosis among the hypoplastic right hearts 4 showed the lesions in the right coronary artery only, whereas the remaining 2 cases showed the lesions in both coronary arteries. However, fibroelastosis was less pronounced in these 2 cases in the left coronary artery and, in addition, was confined here to the anterior interventricular ramus (anterior descending branch). In one of these same 2 cases a fistula was present between the anterior interventricular branch and the hypoplasic right ventricle.

In the only case of coronary fibroelastosis among the left hypoplasia group, the left coronary artery alone showed lesions. Also in this case the lesions were confined to the anterior interventricular branch which communicated with the hypoplastic left ventricle via a fistula.

Coronary fibroelastosis was always found in places where local diverticulum-like thickenings, indurations and white spots had been noticed at macroscopic inspection; it was not found at sites where meandering of the coronary artery was the only macroscopic anomaly of the vessel.

In contrast to coronary fibroelastosis endocardial fibroelastosis of the ventricle occurred preferentially in hypoplastic left hearts, i.e. in 8 out of the 29 left hypoplasias versus in 1 out of the 10 right hypoplasias. A striking observation is that endocardial fibroelastosis of a ventricle occurred only in hearts which did not show coronary fibroelastosis and vice versa. On the other hand it was noted that endocardial fibroelastosis of the ventricle occurred in combination with a variety of anatomical abnormalities on the hypoplastic side of the hearts. However, all cases of endocardial fibroelastosis have in common that the hypoplastic ventricle probably has been functional due to the presence of open ostia and/or a ventricular septal defect (see Fig. 1 a: groups II, III and VIII; Fig. 1 b: group III). Endocardial

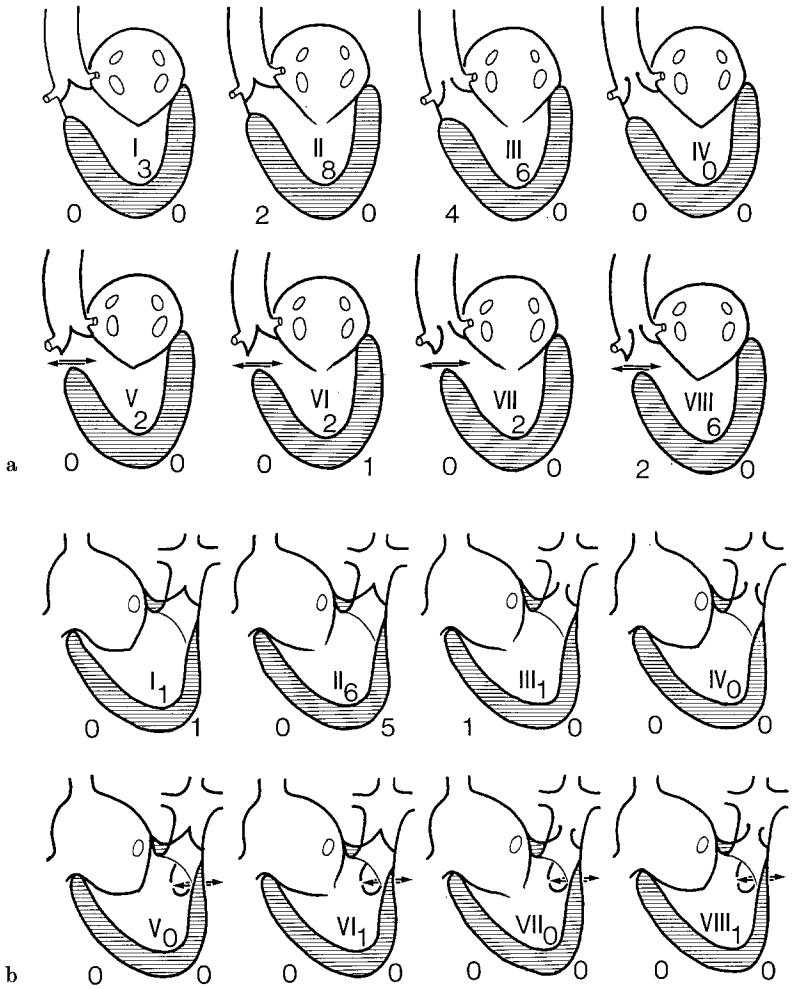

Fig. 1 a and b. Diagrams of subdivision of the hypoplastic left hearts (a) and the hypoplastic right hearts (b) into 8 anatomically different types of each. Roman numerals I–IV indicate the 4 types with intact ventricular septum, V–VIII the 4 types with ventricular septal defect (arrows). An open valve indicates a stenotic, a closed valve indicates an atretic ostium. Arabic numerals represent, in centre: the total number of cases of each type; bottom left: the number of cases with endocardial fibroelastosis of each type; bottom right: the number of cases with coronary fibroelastosis of each type

fibroelastosis was not observed in hearts in which the hypoplastic ventricle had neither inflow nor outflow (Fig. 1 a, b: groups I).

Discussion

The reported data allow the general conclusion that in the hypoplastic left and right heart syndromes coronary fibroelastosis and endocardial fibroelastosis

of the ventricles are mutually exclusive conditions. This view is in agreement with previous studies (Oppenheimer and Esterly, 1966; Bryan and Oppenheimer, 1969). Moreover, the hypoplastic left heart syndrome seems to predispose to endocardial fibroelastosis of the ventricle, as has been noticed previously by others (Noonan and Nadas, 1958). In contrast the hypoplastic right heart syndrome seems to predispose to coronary fibroelastosis.

The association between the localization of coronary or endocardial fibroelastosis and hypoplasia of the right, respectively left side of the heart showed only 2 exceptions in our material: one of the 9 cases of endocardial fibroelastosis occurred in right ventricular hypoplasia, and one of the 7 cases of coronary fibroelastosis occurred in left hypoplasia. The reference material showed this association equally clearly: 14 of the 35 cases of left hypoplasia versus 3 of the 24 cases of right hypoplasia showed endocardial fibroelastosis of the hypoplastic ventricle, whereas all 5 cases of coronary fibroelastosis occurred among the right hypoplasia group. Here also fibroelastosis in the coronary and the endocardiallocalization were never found together in the same heart.

On the basis of our observations and literature data the frequency of either localization of fibroelastosis can be expected to average 25% in the hearts with the type of hypoplasia which predisposes to either coronary or endocardial fibroelastosis. (C.f. the series of Noonan and Nadas, 1958: endocardial fibroelastosis of the left ventricle present in 20 out of 101 hypoplastic left hearts). In accordance with the indications of the rather large material of several authors (Oppenheimer and Esterly, 1966; Bryan and Oppenheimer, 1969; Pyanov, 1972), but in contrast to the view of others, based on smaller series (Newton and Misugi, 1960; MacMahon and Dickinson, 1967; Le Tan Vinh, 1968; Nodzicka and Vortel, 1971), coronary fibroelastosis, if present, is always found in the artery which roughly is the supplying artery of the hypoplastic ventricle. As has been described, the other artery may also show fibroelastosis but the artery supplying mainly the normal ventricle is never the only affected one. In gross anatomical terms, coronary fibroelastosis is as a rule limited to branches of both coronary arteries which supply the hypoplastic right ventricle (present findings).

From our experience it may be concluded that macroscopic features such as diverticulum-like thickenings, white spots and local indurations of coronary arteries do reflect the presence of coronary fibroelastosis and thus are to be regarded of predictional significance for its microscopic diagnosis. This does not hold for the presence of only meandering of coronary arteries.

Interestingly, coronary fibroelastosis was in our material nearly completely restricted to hearts in which the hypoplastic right ventricle had no outflow due to atresia of the pulmonary valve and/or infundibulum in combination with an intact ventricular septum (Fig. 1).

This suggested to us the following hypothesis concerning aetiologic factors involved in pathogenesis of coronary fibroelastosis.

A hypoplastic left or right ventricle which lacks an outflow contains stagnant and—in consequence—poorly oxygenated blood. A further abnormality, also caused by the lack of an outflow, is that ventricular pressure is raised above normal values in both the systolic and the diastolic phase. This has been demonstrated experimentally in dogs (Buckberg *et al.*, 1972) and by means of catheter-

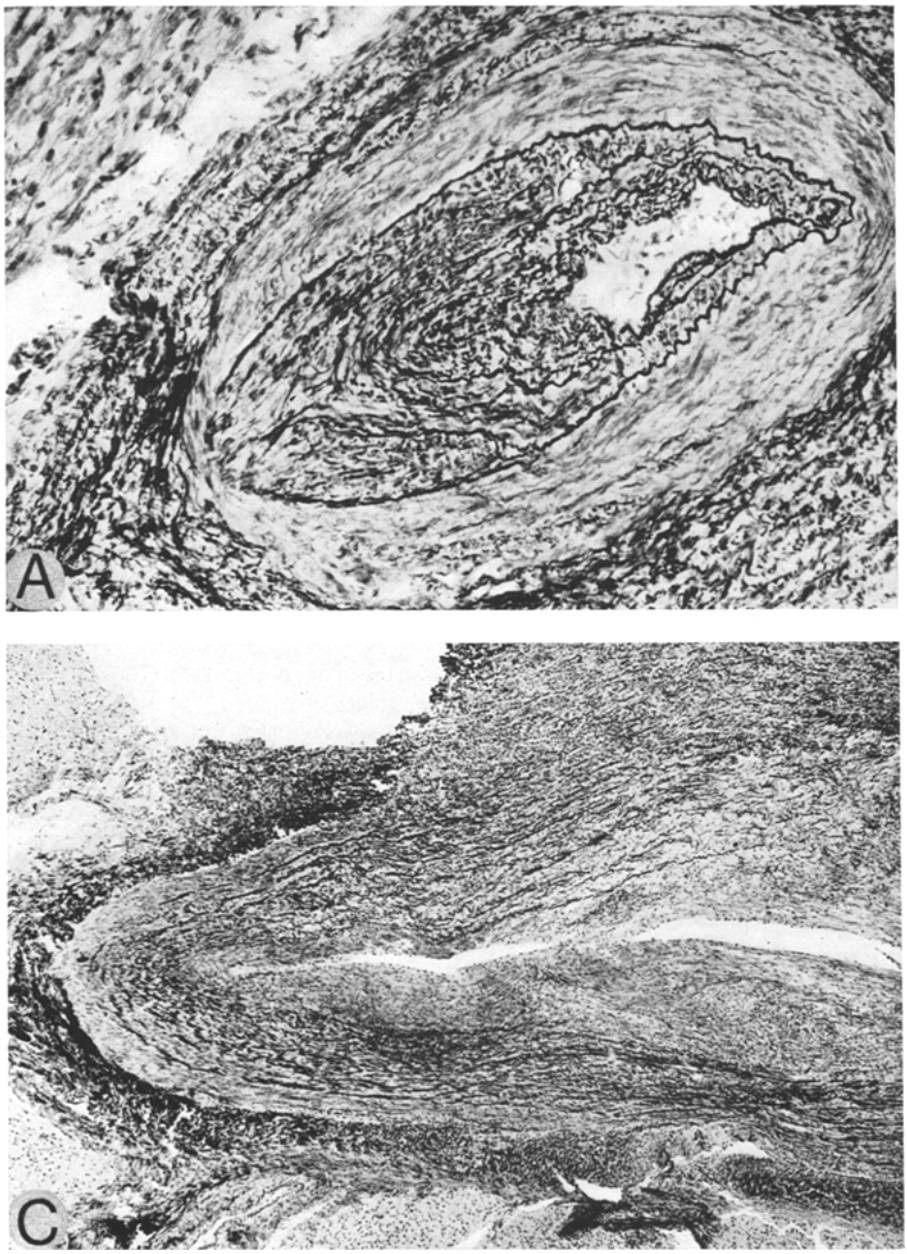

Fig. 2 A and C

Fig. 2 A—D. Photomicrographs of different histological pictures of coronary fibroelastosis in 2 cases of right hypoplasia: All sections 10 μ, stained with Lawson (elastin)-haematoxylin-azophloxin-saffran. (A) and (D) Right circumflex artery, no fistula; ♀ 23 days. (B) and (C) Right circumflex artery, no fistula; ♂ 7 days. (A) Formation of a musculoelastic layer with intact outer and fragmented inner elastic membrane (both derivates of membrana elastica interna) and an intimal zone of fibroelastic tissue. Severe excentric narrowing of the arterial

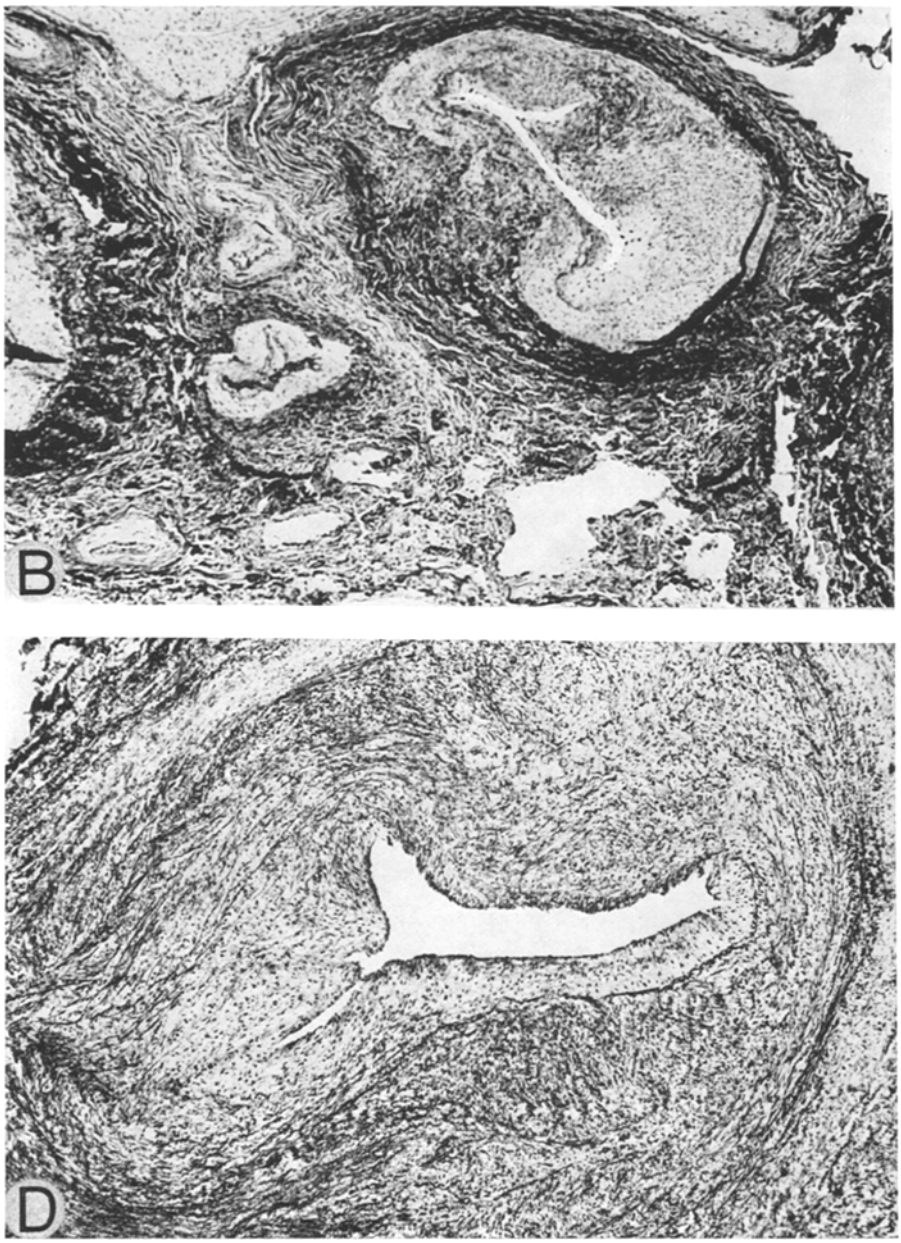

Fig. 2 B and D

lumen. Transverse section, ×185. (B) Fibroelastotic change of the tunica intima with local loss of definition of the membrana elastica interna. Severe narrowing of the lumen. Also note the involvement of smaller surrounding arteries. Transverse section, ×82. (C) Partial destruction of the architecture of the arterial wall. The clear definition of different layers is locally lost by fibroelastotic change. Oblique section, ×60. (D) Irregular narrowing of arterial lumen by fibroelastosis of greater part of both intimal and muscular layers of the arterial wall. Oblique section, ×75

ization of right ventricles in patients with pulmonary atresia in combination with intact ventricular septum (Freedom *et al.*, 1974). In hypoplastic right ventricles such outflow obstruction may lead to reserval of blood flow in the coronary arteries, since they may receive blood from the right ventricle by way of arterioluminal channels, defined by Roberts (1961) as "vestigial residuals of the vascular channels of sinusoidal nature . . . found on the inner part of the heart wall during early phases of embryonic development". These arterioluminal channels, connecting the coronary arteries and the cavities of the heart, are more numerous in the right ventricle than in the left ventricle (Lendrum *et al.*, 1945; Roberts, 1961). The combination of reversal and therefore insufficient flow in the coronary arteries on the one hand and the presence on the other hand in these arteries of poorly oxygenated blood entering via the arterioluminal channels from the hypoplastic right ventricle and of mixed blood entering from the aorta, could then be a causative or contributing factor in pathogenesis of coronary fibroelastosis in the hypoplastic right heart syndrome.

Crucial in this hypothesis is the role of the arterialuminal channels. Since they are less numerous on the left side of the heart (see above) our hypothesis can explain the nearly complete absence of coronary fibroelastosis on the left side. The single case of coronary fibroelastosis on the left side fits into our hypothesis since a fistula between the left coronary artery (anterior descending branch) and the hypoplastic left ventricle may here have been the functional equivalent of the arterioluminal channels on the right side of the heart. A similar case of left hypoplasia also supporting this theory was reported by Oppenheimer-Dekker and Gittenberger-de Groot (1971). The main difference with our case was the presence of a number of enlarged arterioluminal channels instead of a large fistula. Also, among 6 patients with a coronary artery fistula as the single congenital abnormality of the heart reported by Neufeld *et al.* (1961), in the only case examined histologically the branch with the fistula (right coronary artery-right ventricle) showed fibroelastosis.

Evidence for reversal of blood flow in coronary arteries, as mentioned above, has in cases with a fistula between a coronary artery and a ventricular cavity been provided by clinical angiocardiography and/or coronariography (Lauer *et al.*, 1964; Taber *et al.*, 1967; Singer *et al.*, 1973; Freedom *et al.*, 1974). A reversal of flow in a right coronary artery, connected to the right ventricular cavity by a fistula, had already been assumed by Williams *et al.* (1951) in a case of right hypoplasia. In this case also fibroelastosis was present in the branch of the right coronary artery leading to the fistula. The observation of coronary fibroelastosis in coarctation of the aorta (Singer, 1964; Vlodaver and Neufeld, 1968) is also in favour of our theory, since in those cases too systolic and diastolic pressures in the coronary arteries are raised.

Endocardial fibroelastosis which also occurs in conditions other than the hypoplastic left and right heart syndromes (Andersen and Kelly, 1956; Pikiel, 1969; Doerr, 1970) may in cases of congenital malformations of the heart, at least in part and under some conditions, be due to a similar combination of factors. The wall of the hypoplastic left ventricle, which both in our material and according to the literature is the main site of occurrence of endocardial fibroelastosis, may

have an insufficient blood supply. This, since the severely diminished or even interrupted flow through the left ventricle brings about that the coronary arteries receive blood mainly by way of the ductus arteriosus and a retrograde flow through the ascending aorta, now functioning largely or only as a common coronary artery. This may cause abnormalities in both the height and the rhythmic changes of blood pressure within the coronary arteries. Moreover these hearts, largely or wholly dependent on one ventricle only, are supplied with mixed blood. The development of fibroelastosis in the subendocardial area may then be caused or favoured by the fact that oxygenation of the left ventricular wall is poorest in the subendocardial layer (Buckberg *et al.*, 1972), which is supplied by terminal branches of the coronary arteries. Furthermore, these authors have pointed out that every change that diminishes the factor (Diastolic-Pressure-Time-Index/ Tension-Time-Index) diminishes subendocardial and deep myocardial perfusion. The most essential variables influencing this factor are shortening of duration of diastole and elevation of diastolic pressure, which both may happen in the hypoplastic left ventricle with absent or obstructed outflow in combination with intact or nearly intact ventricular septum. It is well established that already in normal hearts the innermost layer of the left ventricle has a marginal blood supply (Doerr, 1970). Therefore, when oxygenation of blood within the coronary arteries is lowered and when irrigation takes chiefly place during ventricular systole as is the case in left hypoplasia, tissue oxygenation probably becomes insufficient. Furthermore, there is in these cases no longer a possibility for the endocardium and adjacent myocardial fibres to profit from well oxygenated blood in the left ventricular cavity. We believe all this to support the anoxia theory of pathogenesis of endocardial fibroelastosis of Johnson (1952) in the sense that anoxia could be an important or contributing aetiological factor. In the hypoplastic left and right heart birth does not bring about any essential change in the circulatory conditions of the heart. Therefore the pathogenetic views discussed hold before as well as after birth. The preferred occurrence of endocardial fibroelastosis in left hypoplasia and of coronary fibroelastosis in right hypoplasia, demonstrated by the presented findings and possibly due to the mechanism discussed, probably explains that the two types of fibroelastosis occur mutually exclusive.

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