Morphological study of coronary venous system in cardiac disorders

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An investigation into the state of the cardiac venous system in common cardiac disease was made by postmortem venography and by histology; 44 hearts including rheumatic, hypertensive, and ischaemic disease, and chronic cor pulmonale were studied and contrasted with 32 normal hearts of various ages. An appreciable smooth muscle hypertrophy with tortuosity affecting the coronary sinus and major veins in cases with pulmonary hypertension was detected, being maximum in chronic cor pulmonale. Intimal swelling and elastosis occurred more commonly in rheumatic disease. No changes were noticed in acute myocardial infarction. It is possible that this venous congestion in the heart contributes to cardiac failure, and it may be another mechanism causing left ventricular hypertrophy in chronic cor pulmonale.

Much investigative work has been published about the coronary arteries, whether normal or diseased. The coronary venous system, on the other hand, has received but little attention. Most texts on anatomy and cardiovascular pathology give brief descriptions of the normal gross anatomy of the veins, with rarely any reference to the histology (Hudson, 1965). Coronary venography has been carried out in the living human (Tori, 1952; Gensini *et al.*, 1965) while the pattern of regional venous drainage of the heart has also been studied by postmortem latex casts (Hood, 1968). Hitherto, however, the involvement of the cardiac veins in heart diseases has not been reported.

The present study concerns the morphological abnormalities, both gross and microscopical, in the cardiac venous system in some common heart diseases, as compared to the normal.

Material and methods

Seventy-six hearts were studied; 44 were diseased and 32 were normal controls. After removal from the body the hearts were injected via the coronary sinus with a mixture of barium sulphate and gelatin as used by Harrison and Wood (1949) for coronary arteries. After a few trials it was found that adequate filling of the venous system could be achieved by a brief initial injection pressure of 80-100 mmHg, lowered immediately and maintained at about 30 mmHg. Though injection through a single cannula into the coronary sinus

may be enough to fill the entire venous system, full visualization was more consistently achieved by using two cannulas, one in the coronary sinus and the other into the opening of the middle cardiac vein. After the injection a radiograph was taken in an ordinary anteroposterior position. Radiographs after opening and spreading the chambers were also taken initially but were not found to add any useful information. Subsequently, the hearts were inspected and the grossly visible veins identified by reference to the radiograph. Blocks for histology were taken from various sites, including the coronary sinus and major veins, and the mural venous channels of the ventricles and atria. Sections were stained by routine haematoxylin and eosin stain, and Weigert's elastic van Gieson.

Observations

The 44 diseased hearts comprised 13 rheumatic, 9 with infarcts (healed and fresh), 10 hypertensive, and 8 chronic cor pulmonale, as well as 3 cachectic hearts with systemic malignancy and one with systemic scleroderma.

Normal venograms and venous pattern

The intramyocardial venules migrate to the epicardial surface between muscle bundles. Around the apex of the heart, both anteriorly and posteriorly, a plexus of grossly visible channels is formed. These resolve into major named channels as described below. The *great cardiac vein* starts anteriorly (or at times posteriorly and curves in front) and ascends in

the interventricular groove by the side of the anterior descending artery. Curving left at the root of aorta, it travels in the atrioventricular groove with the left circumflex artery and is joined at the left border of the heart by the left marginal vein. On the posterior aspect of the left atrium it is joined by the oblique vein of Marshall and then continues as the coronary sinus to open in the right atrium. Two major trunks form on the posterior surface of the heart, the *middle cardiac vein* to the left of the posterior interventricular groove and the *posterior (or small) cardiac vein* to the right of the groove. The former opens into the coronary sinus near its ostium while the latter opens near the left end of the sinus.

The *anterior cardiac vein* and the *posterior vein of the right ventricle* are located in the respective positions on the right ventricle and open either into the posterior cardiac vein or directly into the right atrium. In addition to these major channels a few accessory tributaries may be seen. The larger channels named above form two overlapping triangles with the coronary sinus and great cardiac vein as common hinges. One triangle consists of the small cardiac vein, the great cardiac vein, and the coronary sinus and the other the middle cardiac vein, the left marginal vein, and the great vein as well as the coronary

FIG. 1 *Injected veins dissected off the surface epicardium to show extensive anastomosis among the major trunks. CS=coronary sinus; GCV=great cardiac vein; LMV=left marginal vein; PCV=posterior cardiac vein; OV=oblique vein (site shown by arrow). MCV=middle cardiac vein*

FIG. 2 *Postmortem venogram, normal pattern. The abbreviations are the same as in Fig. 1.*

sinus. There are free anastomoses between all these channels (Fig. 1).

The venograms show that except in the age group beyond 50 years, the veins are smooth-walled vessels (Fig. 2). In an occasional older individual, mild tortuosity may be seen. Atrial veins are rarely visualized by this contrast material. Finer channels were filled in adult and older hearts but not in younger ones. Numerous channels running at right angles, or obliquely to the long axis of the heart, were concentrated on the posterior surface of the ventricles, especially the left; the right ventricular shadow showed far fewer channels. All the venous channels named above were not always visualized and mild variations in their course were also seen. The left marginal, the oblique vein, and the right ventricular veins (anterior and posterior) were variable, while in some specimens, in place of two major posterior and one major anterior channels, a larger or lesser number of smaller tributaries were present. Valves in the veins were inconstant, though at the junction of the vessels with the coronary sinus, semilunar ridges or at times well-formed valves could be seen. The Thebesian valve on the coronary sinus could be seen in 40 per cent of specimens.

Histology revealed varying amounts of elastic tissue, smooth muscle, and collagen in the walls of the major channels. The coronary sinus near its opening is simply a prolongation of the right atrial endocardium and is surrounded by myocardial fibres. In children and young adults there is a thin interrupted layer of smooth muscle in the media and in older subjects muscle fibres are indistinct, but the walls are more elastic; still, older subjects show more collagen and no smooth muscle and little elastic tissue. A similar structure is seen in the major veins in their epicardial course. In the third order veins on the other hand, muscle is sparsely seen, and only a fine elastic coat makes up the wall. The intramyocardial veins when located in the intermuscular vascular bundles have a thin intima and fine elastic layer, while the smallest visible branches have a very thin flattened endothelial lining directly abutting against myocardial fibres. The contrast material used in this study rarely penetrated the veins beyond the inner one-third of the myocardium, nor did it leak into the ventricles. In an occasional specimen, leakage into the pectinate meshwork of the right atrium was noticed.

Veins in diseased hearts: rheumatic heart disease The 13 cases varied from 13 to 54 years of age and all had 2 or more diseased valves: mitral and aortic in 8, mitral and tricuspid in 2, and mitral, aortic, and tricuspid in 3.

The venograms were normal in 5 of the 13; in the 8 remaining, 5 showed tortuosity of the venous trunks, this being quite pronounced in 2 (Fig. 3). One other feature noticed in 7 hearts was the clear visualization of atrial veins, particularly the right, with their connexions both to the right ventricular as well as to the right atrial veins. Consequently, leakage of the contrast material into the right atrium was clearly visible. This feature was rarely seen in other cardiac diseases. On gross examination a consistent feature was the prominent, thick-walled, and gaping coronary sinus especially in its course through the posterior wall of the left atrium. Histology revealed a distinct smooth muscle coat in the coronary sinus as well as in the major veins (Fig. 4). This muscular hypertrophy was mild in 3 and moderate in another 2. In 5 instances there was thickening of the intima; this was intimal elastosis in 4, but in one it took the form of mucoid, rather basophilic swelling of the intima (Fig. 5). No relation was noticed between the venous wall musculoelastic hyperplasia and the type of rheumatic valvular lesion or size of various chambers. How-

FIG. 3 Postmortem venogram in healed rheumatic heart disease. Note the conspicuous tortuosity of channels. The anterior and posterior veins of the right ventricle and their connexions with the middle cardiac vein as well as the right atrial cavity are clearly seen.

ever, in 4 out of 5 cases showing intimal thickening the rheumatic process was active, as judged by the presence of Aschoff bodies in the myocardium and/or endocardium.

In 3 of the 13 instances, the gross and histological structures were normal. The tortuosity on the venogram showed correlation with muscularization, but not in all instances.

The intramyocardial venules were normal. The right atrium was dilated in all instances and also showed shaggy dull pericardium which, on histology, was found to be mild pericarditis.

Similarly, the left atrial wall was hypertrophied and dilated. The prominence of the coronary sinus seen on gross examination was due not only to smooth muscular hyperplasia but also to hypertrophy of myocardial fibres encircling the sinus.

Examination of the other organs in these cases showed the changes of established congestive failure; in particular, the lungs showed grade I to III pulmonary hypertension, according to the criteria of Wagenvoort, Heath, and Edwards (1964). However, no correlation

could be established between the severity of pulmonary congestive changes and the degree of coronary venous muscular hyperplasia.

Ischaemic heart disease The 9 cases included in this group were between 47 and 78 years of age. The infarcts were fresh (under 7 days) in 3, healed fibrous in 3, and recurrent in 3 cases.

In cases of fresh infarction, no abnormalities were found either in the venograms or in gross and histological studies. Among the remaining 6, mild tortuosity was noticed in 2. On histological examination, mild muscular hyperplasia of the coronary sinus and major veins was noticed again in 3 instances, and collagenous thickening was seen in 2. No abnormalities were seen in veins in or around the area of infarction, both fresh and healed. The patients with healed and recurrent infarction showed evidence of mild myocardial hypertrophy and dilatation, as well as passive venous congestion in systemic organs. Lungs in all instances showed established passive congestion with mild pulmonary hypertensive changes (grade 1).

Hypertensive heart disease The 10 cases ranged from 16 to 67 years, the youngest being a boy with malignant hypertension. All showed concentric left ventricular hypertrophy with or without dilatation; the older hearts also showed coronary atherosclerosis of varying degree.

The venograms showed wide, smooth outline venous channels in 4 cases, with mild tortuosity in another 3. Histological examination was normal, except for 2 cases with acellular thickening of the wall of the coronary sinus. Both these specimens had coronary artery atherosclerosis. As in previous groups, mild to moderate passive venous congestion in the lungs was also present.

Chronic cor pulmonale This group included 8 cases, ranging from 48 to 62 years. All 8 had chronic bronchitis, mild to conspicuous bronchiectasis, emphysema, and a varying degree of interstitial pulmonary fibrosis. All had cardiomegaly, mainly right ventricular but with mild left ventricular hypertrophy also. Mild to moderate coronary atherosclerosis was present in all.

Six cases showed variable tortuosity on the venograms. Histology revealed mild to marked smooth muscle hyperplasia, occasionally with minimal intimal elastosis.

A unique lesion seen in one heart was thrombosis of the middle cardiac vein near its junction with the coronary sinus, together

FIG. 4 Great cardiac vein showing smooth muscle hyperplasia in a case of rheumatic heart disease. (H. and E. $\times 100$.)

with thrombus in the right atrial appendage. As in previous groups, no abnormality was seen in the intramyocardial branches; occasionally filling of right atrial veins and leakage of contrast material into the right atrium were seen.

The last group comprised 4 cases, 3 of

FIG. 5 Swelling of the wall and of the intima of the coronary sinus with mild inflammatory reaction in a case of acute rheumatic carditis. (H. and E. $\times 240$.)

which showed atrophic, shrunken hearts associated with systemic malignancy (one each of oesophagus, lung, and breast) and one of cardiac involvement in systemic sclerosis. In the former 3, tortuosity of veins was seen on the venograms as well as on gross examination; the coronary arteries were also tortuous. On histology all 3 showed minimal patchy collagenous thickening of coronary sinus and major veins. No abnormality was noticed in the case of scleroderma.

Comments

This study of the cardiac venous system shows that normally these channels resemble pulmonary veins in their architecture and are quite different from the major systemic veins. The smooth muscle and elastic tissue decrease with age normally, but in many diseases, these components tend to undergo hypertrophy. Irrespective of aetiology, this muscular hypertrophy occurs in conditions associated with pulmonary vascular hypertension and passive venous congestion. It is most conspicuous in chronic cor pulmonale. The coronary venous hypertension is due to the back-pressure transmitted from the right heart into the systemic veins and coronary sinus, and gradually leads to the smooth muscle hyperplasia and to the tortuosity seen on the venograms. The counterpart of this lesion exists in the portal veins in portal hypertension and pulmonary veins in pulmonary hypertension. The isolated example of a thrombosed middle cardiac vein in a case of chronic cor pulmonale is also a part of such congestive venopathy. Such a state of passive venous congestion of the heart itself in cases of congestive cardiac failure therefore has a morphological basis. How much the venous stagnation interferes with heart muscle function and contributes to cardiac failure is a matter of conjecture. It is tempting to extend this conjecture a little further. Hood (1968) has established that 96 per cent of the left ventricular blood is drained into the coronary sinus; therefore, a coronary sinus distension with back pressure is likely to greatly hinder venous return of the left ventricle, and this may then be another factor

in the left ventricular hypertrophy in chronic cor pulmonale. It may operate by inducing anoxia and/or a mechanical overload to the left ventricle. Similar right-sided back-pressure with functional or organic tricuspid regurgitation, leading to right atrial dilatation, would explain the appearance of atrial veins and their openings on the venograms, especially in rheumatic heart disease. Lastly, whereas maximal venous muscularization and tortuosity is seen in cor pulmonale, intimal elastosis and plaque formation is maximum in rheumatic heart disease. The intimal reaction is possibly related to the inflammatory nature of the rheumatic process. This is borne out by the single case of intimal basophilic swelling without elastosis in a young patient with acute exacerbation of the rheumatic process, such intimal swelling being possibly a forerunner of intimal elastosis. Another outcome of this study has been the demonstration in normal hearts of progressive decrease of musculoelastic tissue of veins and its replacement by collagenous tissue in later adulthood. It is concluded that the cardiac veins undergo a musculoelastic hypertrophy in conditions associated with congestive cardiac failure, and pulmonary hypertension.

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