Myocardial Fibrosis in Constrictive Pericarditis

Electrocardiographic and Pathologic Observations

By Harold D. Levine, M.D.

SUMMARY
It has been suggested that a tentative preoperative decision favoring a pericardial lesion, on the one hand, or a myocardial lesion, on the other, may be made from certain noninvasive procedures, including the electrocardiogram. An attempt was therefore made to detect associated myocardial fibrosis by electrocardiogram in 67 patients with constrictive pericarditis as proven at catheterization (63 patients), surgery (64 patients) or postmortem examination (12 patients). Seven of the 67 had electrocardiograms characteristic of, and 16 compatible with, old myocardial infarct. The electrocardiographic experience was otherwise typical of the literature with non-specific changes in the T waves or RS-T segments and/or low voltage in the remaining 44. All three autopsied patients whose electrocardiograms were interpreted as diagnostic of an old myocardial infarct and both autopsied patients with electrocardiograms compatible with that diagnosis showed myocardial fibrosis. In seven autopsied cases with non-specific T waves or low voltage, the myocardium was normal in three while four showed myocardial fibrosis. It appears that in a few cases right ventricular hypertrophy might have simulated infarct by inducing tall R waves over the right precordium, or R waves which decreased in amplitude as the electrode was passed from the right to the left precordium.

Pathologic evidence related myocardial fibrosis to: (1) direct subepicardial penetration by the inflammatory process or deposit of fat in the subepicardial myocardium; (2) compromise of coronary blood flow, as by (a) direct throttling of coronary arteries by scar tissue or (b) deficient irrigation of subendocardial layers due to rigidity of the pericardium; or (3) a concomitant myocardial and pericardial process (lupus, radiation fibrosis, rheumatoid). Independent pericarditis and coronary disease was surprisingly rare. This limited experience (1) suggests that, though myocardial fibrosis may be predicted in constrictive pericarditis if the electrocardiogram shows characteristic changes of myocardial infarction, non-specific T wave changes or low voltage may likewise be associated with myocardial fibrosis, and (2) emphasizes that the difficulty in determining the site of a constrictive process may be compounded by the co-existence in the same heart of both a pericardial and a myocardial process.

Additional Indexing Words:
Rheumatoid arthritis  Uremia  Arrhythmias  T wave  RS-T segment

In interpreting electrocardiograms at the Peter Bent Brigham Hospital, it was necessary occasionally to suggest the diagnosis, based on electrocardiographic records, of old myocardial infarction in patients known to have or suspected of having pericardial constrictive. This quandary suggested possible confusion between constrictive pericarditis and myocardial fibrosis as distinct entities, and the possible complication of constrictive pericarditis by myocardial fibrosis. A preliminary report of this experience was presented with Dr. George A. McLemore at the World Congress of Cardiology at Washington in 1954.1 To the 26 cases then reported 41 are added, culled from observations at the Brigham over the intervening 18 years. The most telling evidence for chronic constrictive pericarditis in the living patient, short of direct inspection at thoracotomy, is the demonstration at right heart catheterization of closely approximating pressures in the right atrium, right ventricle, pulmonary artery, pulmonary capillaries (wedge pressure) and, when the left side of the heart is also studied, in the left atrium. This same hemodynamic pattern, or “plateau of pulses,” may also be observed in chronic constrictive myocarditis2 (e.g.

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Table 1

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<th>Age at First Detection, Sex Distribution and Etiology in 67 Patients with Constrictive Pericarditis</th>
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replacement of myocardium by fibrous connective tissue, tumor, amyloid or other forms of cardiomyopathy or in chronic constrictive endocarditis (e.g., endocardial fibroelastosis or healed subendocardial infarct). In 63 of the 67 cases in this series this restrictive pattern of pressures was found by cardiac catheterization. In two patients with elevated venous pressure the diagnosis was established, in one by roentgen-ray demonstration of pericardial calcification and in the other on the basis of carbon dioxide bubble study. Sixty-four patients were operated upon at this hospital and in all of them the diagnosis of constrictive pericarditis was confirmed at pericardiectomy. In each patient the diagnosis was based at least upon either hemodynamics or surgery.

Methods and Materials

The Population Sample

The characteristics of the population sample studied are presented in table 1. In a number of respects the composition of this group differs somewhat from those of previous studies. There were proportionately more patients (1) whose constrictive pericarditis developed, or may have developed, as a complication of rheumatoid arthritis,2–10 (2) whose pericardial process was associated with valvular dysfunction,11–15 (3) whose constriction was associated with localized effusion pocketed in one or more loculi in the pericardial shell,16–20 or (4) whose pericardial lesion was the end result of uremic pericarditis.21

Electrocardiographic Observations

Of the 67 patients, all but 11 were studied electrocardiographically by the 12 leads in current use. The remaining 11 were studied by three, four or five bipolar leads. Thirteen patients had but a single set of tracings; the remaining 54 had two or more sets (average 4 sets of electrocardiograms). Excepting the notable feature to be elaborated below, electrocardiographic experience with these 67 patients was quite typical of that described in the literature for chronic constrictive pericarditis.16, 22–33 Thus, of the 67 patients, 57 showed nonspecific T wave changes (fig. 1). These consisted of low, flat, biphasic or inverted T waves where they were expected to be upright. In 12 of these 57 patients the RS-T segments were persistently or transiently depressed in the same leads as those showing abnormal T waves; though all 12 were receiving a digitalis preparation when the electrocardiogram was recorded, none showed the cupped contour characteristic of digitalis effect.

Thirty-five of the 67 patients showed low voltage of the QRS complex. Persistent first degree atrioventricular heart block was detected in six patients; in two of these this developed postoperatively. First degree block was transient in one patient during the preoperative period. In twelve patients the electrocardiogram was interpreted as at least suggesting right ventricular hypertrophy; in three of these the evidence was quite convincing (fig. 2), but in the remaining nine the tracings were no more than compatible with that diagnosis. In five patients the electrocardiogram was quite characteristic of left ventricular hypertrophy. Uremia was not present in any of these five patients.

Of 47 patients in normal sinus rhythm, 28 showed notched P waves. These were broad (duration of P wave > 0.10 sec) in nine and thus simulated "mitral" P

Figure 1

Changes in RS-T segment and T wave without QRS changes in 36-year-old housewife (Group III) who improved tremendously following pericardiectomy. A coronary cineangiogram showed, with each systole, dynamic kinking of a small segment of a distal portion of the right coronary artery.

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waves (fig. 3A); they were of normal duration (0.10 sec or less) in the remaining 19 (figs. 2 and 4A.) In one patient a broad downward second component of the P wave in lead V1 was the only electrocardiographic evidence suggesting left atrial enlargement.

Persistent atrial fibrillation was recorded in 19 patients; in two fibrillation was transitory. A number of patients exhibited arrhythmias other than atrial fibrillation. Atrial flutter was recorded in three. Three showed paroxysmal supraventricular tachycardia (fig. 5); in one of these the tachycardia was associated with atrioventricular block. One patient developed transient ventricular tachycardia during surgery. At one time or another, eleven demonstrated ventricular and three atrial premature beats. When in normal sinus rhythm one of the patients with previous or subsequent paroxysmal atrial flutter showed atrial premature beats, sinus bradycardia and junctional escape beats. One patient had sinus pauses at one time, variation of the pacemaker at another (fig. 6A). One had persistent, another transient, atrioventricular nodal rhythm and two had a short P-R interval (<0.12 sec).

The exceptional experience alluded to above was an unexpectedly high incidence of abnormalities in the QRS complex and the consequent implication of actual myocardial damage. The development of QR or Q-S complexes has been regarded as uncommon in chronic constrictive pericarditis. Yet, of the preoperative electrocardiograms of the 67 patients in this study, seven showed changes warranting an unequivocal diagnosis of old myocardial infarction (Group I) and sixteen demonstrated changes in line with, but not

**Figure 2**

*Changing electrocardiographic classification. Initial set of tracings (top two rows) recorded preoperatively at age 58 (11/19/51) considered as showing right ventricular hypertrophy and possible old myocardial infarct. Patient had rheumatoid and osteoarthritis and gave a vague history of chest discomfort on exertion; a bout of chest pain occurring two years before surgery possibly due to acute myocardial infarct. Surgery beneficial. Second set (bottom two rows) recorded at age 71 (9/22/64) showing right, and possibly left, ventricular hypertrophy. Old infarction not suggested at this time.*
diagnostic of, old myocardial infarct (Group II); the tracings of the remaining 44 patients in no way suggested myocardial infarction (Group III).

The discovery of these changes was often confusing, occasionally disconcerting; it compelled wholesale reassessment of the nature of the underlying heart disease. In figure 4A, for example, the notched Q-S complex in lead III, the prominent Q wave (QR) in lead AVF, and the decreasing height of the R wave from lead V1 to V4 constituted evidence quite convincing of old myocardial infarct (Group I), certainly diaphragmatic and possibly anterior as well. In this patient, as in one or two others, infarct was reported with some reluctance for it had been indicated on the electrocardiographic requisition that an initial, albeit only partially effective, operation had already been performed for a thickened pericardial scar. A second example of Group I, a 78-year-old man with right bundle branch block and old anteroseptal myocardial infarct, is described more fully below.

Figure 7 was included with Group II because the Q-S complex in lead AVF is only compatible with old diaphragmatic infarct and because, although the poor progression of R wave voltage over the right precordium is compatible with old anteroseptal infarct, left axis deviation is present and the diminutive Rs could be explained by left anterior hemiblock.

Group III is represented by the electrocardiogram shown in figure 1 which shows abnormal non-specific changes in the RS-T segment and the T wave but a normal QRS complex.

Pathologic Observations

Of the 67 patients, one refused surgery, one was operated upon elsewhere and one, at this writing, was yet to undergo surgery. Exploratory pericardiectomy in the remaining 64 showed in all a pericardial shell consisting of dense sparsely cellular collagenous tissue. This was generally quite distinct from the myocardium but in five cases the operative notes indicate that, because of intimate involvement of the myocardium in the epicardial scar, it was difficult or impossible to find a cleavage plane (fig. 4) and myocardial fragments were inadvertently removed along with the scar (fig. 5). In one patient a deep intraoperative myocardial biopsy was deliberately obtained.

Postmortem examination was carried out in 12 patients; in all but one of these surgery had been performed. Seven of the 12 had shown low voltage or non-specific RS-T or T wave changes but no electrocardiographic evidence of myocardial infarct (Group III). Two of the seven showed subepicardial fibrosis. In two cases the myocardial fibrosis was subendocardial or located in the zone intermediate between subendoocardium and subepicardium. In the remaining three patients myocardial fibrosis was not detected.

All three autopsied patients with electrocardiograms characteristic of old myocardial infarct (Group I) showed significant myocardial fibrosis at postmortem. The electrocardiogram of the first patient (fig. 4A) has already been described. Postmortem showed, in addition to constricitive pericarditis, an old infarct with aneurysmal thinning of the anterior apical part of the left ventricle, considerable subendocardial fibrosis (fig. 4B, C) and organized mural thrombosis; the coronary arteries were slightly atherosclerotic but showed no
evidence of significant narrowing or occlusion. The second autopsied patient had had a pericardectomy in 1947. His preoperative tracings (fig. 8A) showed right bundle branch block and an old anteroseptal infarct. He did extremely well, was subjected to a subtotal gastrectomy in 1953 and succumbed two years later, at 78, to carcinoma of the lung. Postmortem examination showed at the old operative site an area measuring 12 by 12 centimeters over the anterior wall where the heart was bare of pericardium. The remainder of the pericardium consisted of thickened fibrocollagenous tissue measuring 2 to 3 millimeters in thickness, in some areas inducing extrinsic compression of the coronary arteries. (fig. 8B). The free unencumbered coronary arteries showed some tortuosity and atheromatous change but their lumens were dilated to over twice their normal size ("coronary ectasia") (fig. 8C) and showed no narrowing or occlusion. Throughout, but chiefly subendocardially, there was marked focal interstitial fibrosis of the myocardium. The third autopsied Group I patient had a normal preoperative electrocardiogram but developed acute antero-lateral infarct in the postoperative period. At postmortem the coronary arteries were surrounded by a calcified ridge which partially occluded the lumen. The coronary branches were moderately atheromatous and narrowed. There was considerable interstitial fibrosis and endocardial thickening in atria and ventricles. Two patients in Group II were studied at postmortem. The heart of one, weighing 650 grams, showed, in addition to pericardial scarring, hypertrophic myocardial fibers replaced in a few areas by connective tissue. The second showed fibrosis and fatty infiltration in the subepicardium.

Thus all five autopsied patients with infarct-electrocardiograms (three in Group I, two in Group II) showed myocardial fibrosis; this was transmural in one, subepicardial in two and involved the subendocardium and extended further into the ventricular wall without involving the subepicardium in one.

Coronary cineangiography was performed in only two patients; neither showed intrinsic coronary artery disease. In one of these, a 36-year-old housewife with Group III electrocardiograms (fig. 1), a branch of the right coronary artery was caught up in the pericardial scar tissue; with each systole the lumen of the vessel was knuckled or considerably narrowed. In the other, the movement of the entire visible pericardial crown of vessels seemed sluggish and restrained, a change which was attributed to the catheterization team to the constrictive process. Exercise tests were not carried out in the present group of patients.

Discussion

A considerable number of published studies indicate that the electrocardiogram of chronic constrictive pericarditis is never, or only rarely, entirely normal. Abnormal direction or voltage of the T waves, depression of the RS-T segments, low voltage of the QRS complex, atrial fibrillation or flutter and abnormal P waves, alone or in various combinations, have been observed in a varying but noteworthy proportion of cases. Right ventricular hypertrophy, incomplete right bundle branch block, first degree ativoventricular block, and even left ventricular hypertrophy have been described. But other abnormalities of the ventricular complex have generally been considered helpful in differentiating other myocardial processes such as

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Figure 4

Fibrosis of myocardium (Group I electrocardiogram). A 41-year-old manual training teacher with tuberculous pericarditis, ascites, hydrothorax and edema. Pericardectomy in 1937 and 1938. Persistent elevation venous pressure and exacerbation of symptoms. (A) Tracings characteristic of old diaphragmatic infarct and show decreasing R V_{a}-V_{r}. Dissection discontinued because of intimate association of constricting pericardium with epicardium, only place of clearance being under epicardium. (Lead aV_{p} originally mounted upside-down. As remounted rightside-up that lead starts with the T wave of the preceding ventricular complex and ends after the succeeding QRS complex.) (B) Gross section through left ventricle showing constricting eschar (a), hypertrophied left ventricular wall (b), aneurysmal thinning of ventricle (c), organized mural thrombus (d), and subendocardial fibrosis (e). Coronary arteries not narrowed or occluded. (C) Photomicrographs showing extension of fibrosis into superficial bundles (left) and fibrous connective tissue intermingling with myocardial fibers (right).
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constrictive pericarditis, chronic fibrous myocarditis or scleroderma from pericardial constriction, rather than as signalling a myocardial component in and of chronic constrictive pericarditis. Evans and Jackson carefully emphasize that a significant Q accompanying a deformed T wave should be identified with myocardial rather than pericardial disease.

The possibility was considered that one or more of the characteristics of this particular group might somehow skew the data and invalidate any conclusions derived from them. But none of the four patients with rheumatoid arthritis showed electrocardiograms diagnostic (Group I) of myocardial infarct; one, clinically suspected of having had an infarct, showed tracings interpreted as compatible with that diagnosis. Furthermore, of the patients with sero-constrictive pericarditis (three proven at surgery, and two of them at postmortem as well), the electrocardiogram of two in no way suggested myocardial infarct (Group III); the tracing of the remaining patient (fig. 7) was interpreted as compatible with old myocardial infarct (Group II). None of the three patients whose constrictive pericarditis followed uremic pericardial effusion gave a history suggesting coronary artery disease and in none was the electrocardiogram diagnostic of, or even compatible with, myocardial infarction. It seems quite unlikely, therefore, that the extraordinary electrocardiographic evidence for myocardial involvement was in any way related to any of the factors thus far considered.

Constrictive pericarditis may be misdiagnosed as rheumatic mitral valve disease because of the pericardial knock, notching of P waves or atrial fibrillation. Furthermore, the clinical features of tricuspid regurgitation may be observed in constrictive pericarditis. Of the seven patients with abnormal venous pulsation, three showed Group II changes (small or decreasing R waves over the right precordium). This suggests that the appearance of infarct might be suggested because of valvular abnormality or dysfunction. It is well known, for example, that in right ventricular enlargement per se the R wave may be tall over the right precordium and may fail to increase or may even decrease in amplitude as the electrode is moved from right to left (figs. 2 and 4). The pulsatile phenomena, it would seem, generally reflect the hemodynamics of constrictive pericarditis and the burden it imposes upon the right side of the heart. Thus the only possible bias to simulation of infarct in this study would seem to be in the several patients with possible functional valvular impairment. But this would hardly account for the total experience.

Concomitant involvement of pericardium and myocardium could, it would seem, be produced by any one or more of four different mechanisms:

1. **Subepicardial penetration of a primary pericardial inflammatory process.** There is no rigid barrier at the epicardial membrane to prevent the intramyocardial extension of a bacterial or viral process arising in the pericardium. It has commonly been accepted, in fact, that the electrocardiographic manifestations of acute pericarditis are explained by an invariable process of coincident inflammation, extending into the myocardium immediately subjacent to the pericardial lesion. On healing, the inflammatory myocarditis is commonly replaced by fibrous connective tissue. This explanation is supported by logic and is currently accepted as the mechanism.

2. **Simultaneous involvement of myocardium and pericardium by the same pathologic process.** X-ray,43 and certain collagen diseases, notably rheumatoid arthritis,44 45 periarteritis nodosa46 47

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**Figure 5**

Constrictive pericarditis in atrial wall (Group III electrocardiogram). A 54-year-old man with tuberculous constrictive pericarditis and paroxysmal rapid heart action. Tracings showed low voltage and nonspecific T wave changes (A) Strip showing paroxysmal supraventricular tachycardia and artifacts about every sixth beat (arrows) presumably due to transmission of respiratory motions to heart by pleuro-pericardial adhesions. Venous pressure 250 mm. Dissection tedious, surgeon "unavoidably shaving off bits of heart muscle." Biopsy: giant cells; guinea pig positive. Excellent relief despite limited surgery, venous pressure falling to 100 mm. Later succumbed to reactivated pleural and pericardial tuberculosis, conceivably related to surgery. (B) Gross section showing operative window over right ventricle. Coronary arteries bound up in pericardial scar, some (arrows) showing periarterial cuffing. Endocardium thickened in some areas, not shown here. (C) Microscopic section showing epicardial thickening at left and fibrosis about an intramyocardial artery at center.
Figure 6

Variation of pacemaker in constrictive pericarditis. Housewife with chronic constrictive pericarditis and rheumatic heart disease (healed mitral, tricuspid and aortic calcultis) 50 years old at time of surgery. Staphylococcus aureus cultured from excised specimen. Venous pressure fell from 340 mm (preoperative) to 80 mm (postoperative). Died ten years later. (A) Preoperative tracings showing variation of pacemaker with periods of abrupt sinus slowing. (B) Photomicrograph (x25) at epicardial aspect of atrium showing larger blood vessel (x) caught up in pericardial eschar overlying pericardial fatty tissue, fibrosis extending into subepicardial myocardium and periarterial fibrous cuffing (arrows), anatomical changes conceivably related to the disturbance in rhythm.
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and disseminated lupus erythematosus may induce concurrent infiltration of at least these two of the three components of the heart wall.

(3) Impairment of coronary blood flow. Explicit description of the gross and microscopic anatomy of the coronary arteries and arterioles has been lacking in the literature on the pathology of constrictive pericarditis. Some have speculated that coronary blood flow may be altered by low blood pressure, the direct or indirect effects of compression resulting in increased external force upon the larger blood vessels, and by unusual transmural pressures affecting the smaller intramural coronary arteries. Very little has been said about entanglement of the surface coronary arteries in the pericardial process, about compression of coronary arteries by superjacent or adjacent scar tissue, or about intrinsic involvement of the walls or lumina by the same disease resulting in stenosis of the blood vessels. In the present study scarring was not limited to the outer layers of the heart; it often extended well into the middle layers of the heart wall and in many the endocardium was densely scarred. There is reason to believe that the

Figure 7
Group II electrocardiogram. Old myocardial infarct versus left anterior hemiblock in a 45-year-old man with involvement at all myocardial levels. Pericardial thickening suggested by air bubble in right atrium. 500 cc. of fluid in pericardium ("sero-constrictive" pericarditis) at postmortem. (A) Electrocardiogram showing left axis deviation, Q-S complexes in Leads V1 and V2, minute R waves in V1 and V2 and Q-S in Leads III and aVr. (B) Microscopic section (×150) showing pericardial fibrous thickening and subepicardial fibrosis. (C) Cellular infiltration of myocardium at intramural level. Inset (lower left) shows high power field demonstrating that the infiltrating cells are principally lymphocytes. (D) Subendocardial fibrosis and infiltration at inner layer of left ventricular wall.
subendocardium is particularly vulnerable to total impairment of coronary blood flow. Shock, hypotension and obliterative disease of the coronary ostia are among the pathologic entities that can thus curtail blood flow to this remote and, even under physiological conditions, relatively poorly inundated extremity of the coronary “watershed”; it seems reasonable to add chronic constrictive pericarditis to this list. One might speculate that such deficient irrigation could be due to an exaggeration of the normal pressure differential between endocardium and epicardium, to the low pulse pressure already referred to, or perhaps to the very rigidity of the pericardial eschar depriving the coronary vasculature of its normal compliance. A unique study of this problem has already been made in the laboratory animal. Isaacs, Carter and Haller,\textsuperscript{52} by fashioning a chemically inert asbestos shell in the dog's pericardium and, insofar as possible, sparing the myocardium, introduced an experimental preparation for the study of the isolated effects of a rigid pericardium. But even in this contrived pericardial constriction, with heart muscle known originally to be normal, these workers could not exclude the possible role of myocardial factors, other than the mechanical handicap of pericardial constriction, in the causation of hemodynamic abnormalities.

(4) Independent constrictive pericarditis and coronary artery disease in the same patient. The ubiquity of coronary disease hardly needs retelling. It is so very prevalent in middle age and beyond that one would anticipate a considerable overlap in incidence between coronary disease and any other process. It would not be unexpected, therefore, that in a considerable number of patients of a certain age group both coronary artery disease, on the basis of atherosclerosis, and constrictive pericarditis, from another cause, must occur together.

Only five in this group of 67 patients gave a history at all suggestive of angina pectoris or coronary insufficiency; in only two of these was this

Figure 8
The coronary vessels in constrictive pericarditis in a 78-year-old man who died eight years after successful pericardectomy. (A) Preoperative electrocardiogram showing right bundle branch block and old antero-septal infarct. (B) Thickened remnant of pericardial scar showing encasement of coronary artery branch (arrow) in fibrocollagenous tissue. (C) Dilated surface coronary artery (“coronary ectasia”) (×150).
diagnosis unequivocal. In one (fig. 2) the physician felt that the patient might well have suffered a myocardial infarct two years before pericardiectomy. The infrequency of chest pain or compression, it should be noted, by no means precludes the diagnosis of coronary artery disease. It is quite possible that the disability imposed by concomitant intermittent claudication or rheumatoid arthritis may prevent the patient from exerting himself to the point of inducing angina; similarly, the incapacitating dyspnea or fatigue induced by the pericardial constriction could act as a brake upon the patient's activity and thus suppress the symptom of angina.

As indicated above, coronary angiography was carried out in only two patients and exercise tests in none. Ramsey et al. were interested in coronary arteriography in a similar group of patients apparently intending not to demonstrate the presence or absence of coronary disease but, rather, to define the true edge of the heart shadow and thus to differentiate primary myocardial restriction from pericardial thickening. Jain and Sepaha did subject 15 subjects with constrictive pericarditis to Master's tests; none developed substernal pain during the test and in all the test was negative.

In the group of patients reported here, however, surgical biopsy and postmortem examination furnished surprisingly little evidence in support of this hypothesis. Of the twelve autopsied patients, for example, one showed moderate narrowing of atherosclerotic coronary arteries; one (fig. 8) had coronary atherosclerosis but the vessels, though tortuous, were actually dilated ("coronary ectasia"); one (fig. 4) had slight atherosclerosis but no narrowing or occlusion; and in the fourth the coronary disease was non-atherosclerotic and involved smaller vessels. In the latter patient fibrohyaline changes involved the intima and media of the smaller coronary arteries resulting in thickening of their walls and total occlusion of at least one vessel.

The view that concomitant atherosclerotic coronary artery disease accounts for myocardial fibrosis, though reasonable, is thus hardly supported by the experience described either in this study or in the pertinent literature. This discordant experience even suggests the diametrically opposite possibility that somehow constrictive pericarditis has a sparing effect upon the coronary arteries. Such a position would be conjectural.

Whatever the mechanism or mechanisms of conjoint involvement of epicardium and myocardium, it has commonly been considered that atrophy of heart muscle fibers (fig. 3B) or replacement of the latter by fibrous connective tissue explain myocardial impairment persisting after pericardiectomy, atrophy explaining delayed improvement and fibrosis accounting, at times, for incomplete long-run effectiveness of surgery.

The experience reported here of occasional difficulty in dissecting epicardium from myocardium corroborates that of Sawyer et al. and Chamblis et al. who found that the scar invaded the myocardium in 19 of their 61 cases, and of Louhimi, Koskelo and Laustela who had similar difficulties in 2 of 33 cases.

Acknowledgment

The substance of this communication was to have become an integral part of a comprehensive review of the subject of constrictive pericarditis by Dr. C. Sidney Burwell in collaboration with Drs. Eugene Robin, Albert C. Bickelman and myself. Owing to Dr. Burwell's death, this total overall experience was never completed. I owe a great debt of gratitude to Dr. Burwell as well as to Dr. George A. McLemore, who participated in the earlier phases of the study, to Drs. Lewis Dexter and Richard Gorlin, in whose laboratories the hemodynamic observations were made, to Drs. Gustav Dammin and the late Dr. S. Burt Wolbach for access to the files of the Department of Pathology, and to Drs. Dwight E. Harken, Robert E. Gross, Harrison Black, John J. Collins and the late Dr. Elliott C. Cutler, who performed the surgery in these patients.

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