PRIMARY MYOCARDIAL DISEASE

SPECTRUM OF CARDIOMYOPATHY AND CURRENT CLASSIFICATION

By J. F. Goodwin

Before classifying cardiomyopathy it is necessary to define the term. The earlier definition of "A subacute or chronic disease of heart muscle of unknown or obscure aetiology often with associated pericardial or endocardial involvement but not due to atherosclerosis" has now been discarded on the grounds of a complex and unwiedly wording. The division into Primary and Secondary Cardiomyopathies has served a useful function but may now appropriately be abandoned. It has therefore been decided to define cardiomyopathies simply as a "Disorder of heart muscle of unknown cause or association" emphasising that in cardiomyopathy the myocardial disorder is a primary event. (Goodwin, 1971) (Oakley, 1971). The new definition excludes those diseases of heart muscle which, although rare are due to a known cause and associated with, or due to, disease elsewhere in the body. These conditions have previously been known as the Secondary Cardiomyopathies and it is now proposed that they should be redefined as: "Rare disorders of heart muscle connected with an associated disease elsewhere in the body, "each to be defined under its appropriate cause, such as, for example, haemochromatosis, sarcoidosis, diffuse systemic sclerosis, acromegaly, connective tissue diseases, Chaga's disease etc. The term "Cardiomyopathy" can now be used to describe those diseases primarily involving the heart muscle, of unknown cause.

The previous definition that was based on functional pathology remains an appropriate means of further classifying the Cardiomyopathies as now redefined (Goodwin, 1970). This classification describes three main contrasting types.

Hypertrophic (with or without Obstruction) notable for impaired ventricular distensibility and compliance.

Congestive notable for poor systolic ejectile function.

Obliterative notable for obliteration of ventricular cavity.

The type described as Constrictive Restrictive Cardiomyopathy has now been abandoned as this clinico-haemodynamic group is for practical purposes seen only in Specific Heart Muscle Disease and in our experience, although commonest in Primary Amyloid Disease, may also occur in leukaemic infiltration or polyarteritis nodosa.

Except in humid tropical parts of the world Obliterative Cardiomyopathy as typified by Endomyocardial Fibrosis is so rare as to be unimportant for practical purposes. The remaining type of Obliterative Cardiomyopathy, Loeffler's Fibroplastic Eosinophilic disorder, does occur in temperate zones but also is extremely rare. It is considered by some to be the temperate zone equivalent of Endomyocardial Fibrosis. Thus for practical purposes attention should be focused essentially on the two main contrasting types of Cardiomyopathy —The Hypertrophic and the Congestive. In the last 12 years in the Postgraduate Medical School my colleagues and I have seen 224 patients with Cardiomyopathy of whom 120 were Hypertrophic and 104 were: Congestive.

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HYPERTROPHIC CARDIOMYOPATHY (with or without Obstruction to Left Ventricular Outflow) (HOCM).

This form of Cardiomyopathy is also known as Idiopathic Hypertrophic Sub-Aortic Stenosis (IHSS) in North America. It was originally detected because of massive hypertrophy of the left ventricle with an asymmetrical distribution in the ventricular septum described by Teare (1958) as 'asymmetrical hypertrophy of the heart.' The asymmetrical bulge of the septum is frequently associated with a systolic gradient across the outflow tract of the left ventricle and it was the presentation of patients with these gradients mimicking aortic stenosis that led to the recognition of the disease (Braunwald et al., 1960; Goodwin et al., 1960; Cohen et al., 1964). There is a strong familial basis to the disease (Pare et al., 1961). In some families the incidence is extremely high and although a family history cannot be proved in many patients it is likely that the disease is genetically determined. No acquired cause for the disease has so far been discovered though the association of fixed outflow tract obstruction with Hypertrophic Cardiomyopathy that has occurred occasionally in our experience raises the question, still undecided, as to whether under certain circumstances Hypertrophic Cardiomyopathy may be secondary to other forms of outflow tract obstruction or damage to the myocardium. The cause of the disease remains unknown. The discovery by Meerschwam (1969) of shortening of the mean and polyphasic action potentials of skeletal muscle in patients with the disease together with abnormalities of peripheral muscle enzymes has suggested that Hypertrophic Cardiomyopathy might be part of a generalised myopathic disorder. Coltart and Meldrum (1970) detected prolongation of repolarisation time and of the maximal rate in the rise of action potential together with reduced follow-up in muscle resected from the outflow tract of the left ventricle in patients with Hypertrophic Cardiomyopathy. These findings would also support a generalised myopathic disorder. The wide age span of the disease from infancy to late middle age suggests that there might be a genetically determined disorder of muscle growth. The electron microscopical studies of Ferrans et al., (1972), which showed bundles of severely disorganised muscle cells running in different directions instead of in parallel with increased cellular branching and extensive side-to-side intercellular junctions suggests that his abnormal architecture may severely impair the normal contractile processes with groups of muscle fibres contracting against each other. This lattice work type of abnormality might also account for the increased rigidity and impaired compliance of the left ventricle.

The gross and the pathological changes, the histochemical findings and electron microscopical features will be discussed by my colleague Dr. Eckhardt Oslen, who, with our colleagues Pearse and Van Noorden has shown definite differences in histological features between Hypertrophic Cardiomyopathy and hypertrophy secondary to fixed outflow tract of the left ventricle.

In approximately two thirds to three quarters of the patients with Hypertrophic Cardiomyopathy there is a systolic gradient across the outflow tract of the left ventricle. This gradient occurs characteristically in the ventricle near the inflow tract and not at the apex. False gradients due to squeezing or trapping of the measuring catheter may occur. True outflow tract gradients are due to the apposition of the hypertrophi-

ed septum with the anterior cusp and anterior papillary muscle of the mitral valve in a 'pinch-cock' fashion (Goodwin, 1972). The highest pressure gradients may be seen in those patients with the predominant concentration of the disease in the septum, producing a diffuse massive bulge but can also occur in patients with widespread involvement of the left ventricle. The outflow tract gradient is increased by inotropic stimulation such as tachycardia or release of endogenous catecholamines, or by exogenous inotropic agents such as isoprenalin and digitalis.

It is now realised that the diastolic component of the disease is of extreme importance. Nearly all patients have an increase in left ventricular end-diastolic pressure (Goodwin, 1970), at rest or on effort. The rate of filling of the left ventricle is reduced (Stewart et al., 1968) and in our experience 20% to 30% of patients have no outflow tract gradient whatever, the disease being manifest entirely by gross hypertrophy and by impairment of left ventricular filling due to rigid inelastic greatly hypertrophied and poorly compliant muscle (Goodwin, 1970). On angiocardiography great distortion of the left ventricular cavity can be seen which is often slit-like in character and shows irregular masses of muscle projecting into it. Sometimes the distal apical portion of the cavity may be entirely cut off in systole by the contraction of the hypertrophied muscle.

In patients with outflow tract obstruction there is also mitral regurgitation which results probably from interference with function of the hypertrophied papillary muscle. It is likely that as the hypertrophied septum meets the anterior cusp of the mitral valve there is temporary dislocation of the papillary muscle mechanism so that regurgitation can occur.

Ventricular volume measurements are characteristically abnormal. End-systolic volumes and residual fractions are low while ejection fraction is high. The ratio of wall thickness to cavity size is much greater than in most other forms of hypertrophy and end-diastolic volumes are normal (Grant et al, 1968).

The high left ventricular end-diastolic pressure and slow filling of the left ventricle produces elevation of the left atrial pressure which is exagerated by tachycardia when the time available for filling of the left ventricle is reduced, with consequent considerable left atrial hypertension. Studies in our laboratory by Webb-Peploe et al., (1971), have shown that exercise and isoprenalin cause a rise in left ventricular end-diastolic pressure. This increase is not merely due to an increase in rate because atrial pacing does not produce the same degree of diastolic hypertension. Presumably it is due to sympathetic stimulation of the abnormal hypertrophied muscle. Beta adrenergic blockade in Hypertrophic Cardiomyopathy using Practolol in acute observations produces a fall in left ventricular end diastolic pressure, especially on effort. Beta blockade has an apparently unique effect in Hypertrophic Cardiomyopathy in producing an increase in ventricular volume with a reduction in left ventricular end diastolic pressure which suggests increased compliance of the left ventricle as a result of Beta blockade (Webb-Peploe et al., 1971).

The symptoms and physical signs in Hypertrophic Cardiomyopathy are readily explicable on the haemodynamic disorder. Thus; dyspnoea is due to the raised left atrial pressure secondary to the high end-diastolic pressure in the left ventricle. Angina is likely to be due to an excess of demand for blood over supply of the hypertrophied myocardium despite the presence of normal major coronary arteries. Syncope may have several causes. The original suggestion that it is usually due to the development of outflow tract obstruction may still be true in some patients. But it seems more likely that syncope is due to a fall in cardiac output as a result of difficulty of filling the incompliant ventricle particularly during periods of endogenous inotropic stimulation or tachycardia. The onset of atrial fibrillation with a consequent loss of atrial drive may cause a catastrophic fall in cardiac output and may also be responsible for syncope. Other arrhythmias appear to be unusual in Hypertrophic Cardiomyopathy. Sudden death is probably due to the same cause or causes as syncope.

The abrupt jerky ill-sustained arterial pulse is due to initial rapid ejection of the left ventricle with delayed onset of obstruction which causes sudden collapse of the pulse. The palpable left atrial beat is due to the powerful contraction of the left atrium under high pressure striving to fill the incompliant left ventricle. The ejection systolic murmur of sudden explosive onset and delayed in time and heard at the left sternal edge and apex is due both to left ventricular outflow tract obstruction and to the mitral regurgitation which accompanies it. Occasionally it may be produced by obstruction to right ventricular outflow by the massive septum. In patients without outflow tract obstruction the physical signs are more difficult to evaluate. In these patients the murmur is absent or trivial and signs may be confined to 3rd and 4th heart sounds and a palpable left atrial beat. The jerky pulse is less obvious then when the obstruction is present. Pulmonary hypertension results from the high left atrial pressure, and thus Hypertrophic Cardiomyopathy without obstruction may in some patients present as pulmonary hypertension of unknown origin.

The natural history of the disorder in our patients has been studied by Swan et al., (1971). The natural history of the disease takes three forms: patients may remain static symptomatically for many years, sudden death may occur unexpectedly and congestive heart failure may develop as the left ventricle becomes progressively fibrotic or the disease becomes more widespread throughout the ventricular musculature. The natural history of the disease is probably largely determined by the degree of elevation of the left ventricular end-diastolic pressure and the response to exercise. Some patients probably gradually develop outflow tract obstruction throughout their lives, but others have no obstruction at any time. Some having had obstruction, eventually lose this. In our experience the progression of the disorder is associated with spontaneous loss of outflow tract obstruction with progressive impairment of systolic contractile function without increase in systolic volume. Thus, a component of power failure develops which added to the failure of compliance, causes a significant fall in cardiac output. The onset of atrial fibrillation is liable to be followed by pulmonary oedema and systemic or pulmonary embolism and usually precipitates severe congestive heart failure. In our study spontaneous loss of outflow tract obstruction occurred in 8 of 42 patients who had been followed for over 5 years. The systolic murmur became less or disappeared in all and this was associated with increase in dyspnoea and often in heart size. Atrial fibrillation occurred in 15 patients and was accompanied by an increase in jugular venous pressure and by deterioration in symptoms. Systemic embolism occurred in 9 of these patients and heart failure in 12 including three who had pulmonary oedema. Five patients died in congestive heart failure. Sudden death could not be correlated easily with any single factor but appeared to occur more frequently in patients with a short progressive symptomatic history and a particularly high left ventricular end-diastolic pressure. Outflow tract obstruction did not appear to have any definite relation to sudden death appears to be commoner in children than in adults, occurring in 4 of 21 children as compared with 8 of 100 adults (Oakley, 1972).

Surprisingly, pregnancy is well tolerated, as shown by 18 patients supervised through 22 pregnancies by Turner et al., (1968). Infective endocarditis is well recognised and usually occurs on the mitral valve (Vecht and Oakley, 1968).

CONGESTIVE CARDIOMYOPATHY

Congestive Cardiomyopathy (COCM) is essentially a syndrome of multiple causes which results in severe ventricular failure due to poor systolic function. The force of contraction of the left ventricle is reduced, the heart is dilated and the residual volume of the left ventricle is increased. Necropsy examination reveals merely dilation of the left ventricle and often of the right ventricle with moderate degrees of hypertrophy much of which may be concealed by the dilatation which appears to be out of proportion to the degree of hypertrophy. The cardiac valves are structurally normal though dilatation of the ventricles may produce some atrio-ventricular valve regurgitation. The major coronary arteries are normal. The pathological appearances will be described by my colleague Dr. Eckhardt Olsen. It seems probable that the usual processes of compensatory hypertrophy which enable the heart partially to overcome the damage or increased load upon it has in some way been inhibited by the lesions that have produced the original damage. Patients with more than the usual degree of hypertrophy tend to live longer than those with less hypertrophy (Goodwin, 1970).

In the fully developed syndrome there is evidence of severe congestive heart failure with poor quality cardiac impulse, atrial and filling gallop rhythms, producing a sumation gallop due to sinus tachycardia. The jugular venous pressure is raised and there is often tricuspid regurgitation. The low cardiac output is manifest clinically by cool pale extremities with a tinge of cyanosis and small arterial pulses.

Electrocardiograms show non-specific appearances with low voltage QRS complexes and flat and inverted T waves. Left bundle branch block and atrial fibrillation may occur in about 20% of patients. Sometimes there is definite evidence of left ventricular hypertrophy suggesting that the disease has been of somewhat longer duration than usual. Appearances suggestive of extensive anterior myocardial infarction may be found in the presence of normal coronary arteries. These appearances may be the result of interference of conduction pathways by patchy myocardial cell damage and fibrosis. (Gau et al., 1972).

Haemodynamic studies confirm the low cardiac output and stroke volume suggested clinically and reveal elevated left ventricular end diastolic pressures with usually moderate elevation of pulmonary artery pressure, though occasionally there may be considerable pulmonary hypertension due to reactive vaso-constriction in the pulmonary arteriolar bed secondary to the left atrial hypertension. Angiocardiography reveals dilatation of the left ventricular cavity with generalised pulmonary hypertension. Angiocardiography reveals dicreased ejection fractions. Localised areas of dyskinesia or akinesia have not been found in our experience.

Systolic time intervals reveal a grossly increased ratio of pre-ejection period to left ventricular ejection time (PEP/LVEP) indicating a greatly reduced ejection fraction.

The clinical and haemodynamic picture therefore is one of severe myocardial failure and is not specific in any way.

The pathogenesis of Congestive Cardiomyopathy is unknown but a number of factors may be important and can be regarded as possible causes in some patients. These factors are; alcohol, pregnancy and puerperium, systemic hypertension, infections and possibly immunological disorders. In the majority of patients the aetiology is quite unknown. Of 74 of our patients in our initial series analysed by Kristinsson in 1969, heavy alcohol intake was noted in 16, history suggesting previous infection in 14. while in 7 the onset of the illness was in the pregnancy or puerperium. Otherwise there was not clues as to the cause. Endocardial fibroelastosis, Friedreich's ataxia or myocarditis due to Coxsackie B virus infection were present in isolated patients which probably should be regarded as having Specific Heart Muscle Disease rather that Cardiomyopathy.

Alcohol

If cobalt poisoning and beri-beri heart disease are excluded, alcohol appears to be rare as the sole cause of Congestive Cardiomyopathy, although it may well be a factor in the production of the disease. But there is well documented evidence that severe congestive heart failure occurs in subjects taking very large quantities of alcohol, remission occurring when alcohol is stopped, and recurrence developing when drinking is resumed (Tobin et al., 1968).

Cardiomyopathy in Pregnancy and the Puerperium. (Peripartral Cardiomyopathy).

The occurrence of Congestive Cardiomyopathy in the late stages of pregnancy or in the puerperium, especially in mal-nourished multiparous women is well documented by/and recently by Demakis and Rahimtoola (1971). The clinical and haemodynamic picture is one of Congestive Cardiomyopathy and the exact reason for its development is unknown. The suggestion has been made by Brockington (1971) that in the African peripartum cardiomyopathy may be a form of acute hypertensive heart failure as a result of post-partum hypertension. The sudden rise in systemic pressure is too great for the normal left ventricle to withstand, leading to left ventricular failure, a subsequent fall in cardiac output, followed by a fall in systemic blood pressure and heart failure in a minority of patients. Certainly hypertension may be one factor among others leading to congestive cardiomyopathy in the puerperium, but partially acts in association with unknown causes.

Hypertension

In a minority of patients with Congestive Cardiomyopathy systemic hypertension is a feature and in our experience has been present during the course of the disease in 19 of 74 patients. In those who were previously hypertensive before the onset of left ventricular failure it seems likely that heart failure might well be due to an unusual response of the left ventricle to the hypertension leading to dilatation and moderate hypertrophy rather than to marked hypertrophy without dilatation as is the usual response. Since epidemiological studies have shown that hypertension and Congestive Cardiomyopathy are often common conditions in the same populations hypertension may well be an important cause in some patients with Congestive Cardiomyopathy but the absence of hypertension at any time in the majority of patients makes it unlikely to be a leading aetiological factor.

Infection

The connection between previous virus infection of the heart and established Congestive Cardiomyopathy is somewhat tenuous. It is known that the Coxsackie B, Echo, and Influenza A viruses and possibly many others may cause myocarditis but this appears only rarely to progress to chronic myocardial disease, Bengtsson (1968). Definite evidence of an infective process in the myocardium of patients with Congestive Cardiomyopathy has not been obtained.

Immunity

Immuno-globul in binding in the hearts of patients with Congestive Cardiomyopathy has been described recently by Das et al., (1972). It has yet to be determined whether an autoimmune mechanism has any pathogenic significance or whether the immunoglobulin binding might be related to some associated process, perhaps infective.

Eosinophilia.

Eosinophilia occurs occasionally in Congestive Cardiomyopathy but more often in rare Specific Heart

Muscle Diseases such as polyarteritis nodosa or leukaemia where it may be associated with pericarditis. Eosinophilia is a definite though often not a striking feature of Obliterative Cardiomyopathy due to Endomyocardial Fibrosis but it is often a striking feature in Loeffler's Eosinophilic Fibroplastic Endocarditis where the obliterative process within the heart may possibly be a result of the reaction to eosinophilia, the cause of which is unknown. Drug sensitivity which can damage the myocardium may of course be associated also with eosinophilia.

OBLITERATIVE CARDIOMYOPATHY

The prototype of this group is Endomyocardial Fibrosis, a disease virtually confined to humid tropical zones of the world especially in East and West Africa, Parry (1964). There are obliterative changes in inflow tracts of both ventricles due to fibrous tissue with added thrombus.

In the right ventricle the disease characteristically involves the body of the ventricle and the posterior cusp of the tricuspid valve. Eventually the entire cavity of the ventricle except the outflow tract is obliterated and the tricuspid valve is rendered grossly incompetent, producing a giant right atrium. The obliterative lesion impairs diastolic filling of the right ventricle producing an early diastolic "dip and plateau" type of ventricular pulse pattern with raised central venous pressure typical of cardiac restriction. A large pericardial effusion is often present. Patients tend to be wasted, cyanosed and the high venous pressure may occasionally be associated with exophthlmus. Ascietes is common but oedema is usually not marked. The clinical picture may resemble constrictive pericarditis. When the left ventricle is involved the fibrosis occurs in the inflow tract and involves the posterior cusp of the mitral valve, producing in some patients severe mitral regurgitation and in others a restrictive disorder of left ventricular function. The clinical picture is that of sub-valvar mitral regurgitation and pulmonary hypertension. Systemic embolism is frequent and calcification may occur in the thrombus in the ventricles. Atrial fibrillation is common. The aetiology is unknown but the geographical localisation and preceding symptoms of intermittent fever and arrhythmia before the onset of heart failure suggest an infective origin.

Loeffler's Fibroplastic Eosinophilic Endocarditi is similar, but by no means identical to endomyocardial fibrosis. Large masses of the eosinophils form the main constituent of the obliterative material in Loeffler's disease but later the eosinophilia may become less. Thrombus is added to the eosinophilic mass and systemic embolism is not uncommon. It has been suggested by Brockington and Oslen, (1972), that the two conditions are basically the same, Loeffler's disease being the temperate zone equivalent of endomyocardial fibrosis.

RARE SPECIFIC HEART MUSCLE DISEASES

These conditions formerly known as Secondary Cardiomyopathies are out with the main scope of this paper but deserve a brief note. Many specific lesions may involve the heart muscle but the most important in this group are; Diffuse Systemic Sclerosis, Amyloid Disease, Sarcoidosis, Haemochromatosis, Acromegalic Heart Disease, Heart Muscle Disease Associated with Familial Neuromyopathic Disorders, and Endocardial Fibroelastosis. Connective tissue disorders such as systemic lupus erythomatosus, rheumatoid arthritis and messenchymal disorders such as Marfan's syndrome when they affect the heart tend mainly to involve the pericardium as systemic lupus and rheumatoid arthritis, or the aortic or mitral valves as in Marfan's syndrome.

Diffuse Systemic Sclerosis. The myocardium is replaced by fibrous tissue producing progressive cardiac failure often commonly associated with conduction disturbances, East and Oram (1947).

Amyloid Disease. Primary amyloid disease produces a stiff rigid myocardium which fails to relax and which has impairment of contractile force. The clinical picture stimulates constrictive pericarditis (Hetzel et al., 1953). Cardiac pain may be a feature and there may be no evidence of amyloid disease elsewhere. When this occurs it is found in the tongue, mucous membranes and sometimes in the rectus sheath.

Sarcoidosis. Myocardial involvement usually occurs in the presence of manifestation of sarcoidosis elsewhere in the body. Conduction disturbances and sudden death may occur (Porter, 1960). Myocardial failure and rarely acute papillary mucle dysfunction with mitral regurgitation can develop.

Haemochromatosis. Clinical evidence of haemochromatosis is usually clear cut and the picture is one of Congestive Cardiomyopathy. There may be a family history, but the exact way in which myocardial iron implantation damages myocardial function is not known.

Acromegalic Heart Disease. Cardiomegaly in acromegaly is well recognised but it is usually thought to be due to associated sytemic hypertension and in part to the generalised organ enlargement found in acromegaly. In our experience cardiomegaly and heart failure occurred in the absence of significant hypertension in 3 patients and appeared to be due to a specific effect of the acromegaly on the heart muscle. The coronary arteries were normal.

Heart Muscle Diseases Associated with Familial Neuromyopathic Disorders

Heart involvement may occur in Friedrich's ataxia, dystrophia myotonica and muscular dystrophies. Conduction disorders, arrhythmias, and heart block are common. Ventricular hypertrophy with fibrosis, degeneration of myocardial fibres with obliteration of small coronary arteries may occur, particularly in Friedrich's ataxia. The usual pattern is of Cardiomegaly, and in the late stages congestive heart failure with conduction defects and arrhythmias.

Endocardial Fibroelastosis. In this condition there is an intensive sclerosis involving the endocardium of the left ventricle which is covered with a thick layer of white fibrous tissue. This process severely impairs contractile force of the left ventricle so that heart failure and the picture of Congestive Cardiomyopathy, often with mitral regurgitation results. Endocardial fibroelastosis may occur in a primary form or in association with other cardiac lesions such as coarctation of the aorta or congenital aortic stenosis (Manning et al., 1964). The disease begins in infancy or early childhood and progresses to congestive heart failure. A familial incidence is common. The cause is unknown but lymphatic obstruction has been suggested (Miller et al., 1963) and mumps virus has been considered to be a cause (Sellers et al, 1964). Endocardial fibroelastosis should not be confused with the fibrosis of the endocardium which may occur in chronic, prolonged heart failure.

CONCLUSION AND SUMMARY

The Cardiomyopathies have been redefined as, "Disorders of heart muscle of unknown cause." The conditions formerly described as Secondary Cardiomyopathies, in which heart muscle disease is associated with disease elsewhere in the body have now been excluded from the definition of the Cardiomyopathies and redefined as Rare Specific Heart Muscle Diseases and designated according to the general system disease present.

The Cardiomyopathies as redefined are three main types: **Hypertrophic** with or without outflow obstruction with severe ventricular hypertrophy and reduced compliance; **The Congestive** with dilated ventricles and severe pump failure and modest hypertrophy; and **The Obliterative** with obliteration of the ventricular cavity by fibrosis and thrombi. These three tvpes have dis-

tinct haemodynamic and clinical features and differing pathologies.

The haemodynamic and clinical patterns are widely different and are described in detail with comments on aetiology in each group.

The Hypertrophic type of cardiomyopathy is a genetically determined disorder probably of heart muscle growth of a myopathic type while Congestive Cardiomyopathy appears to be a multifactorial condition leading to progressive congestive heart failure without any specific features. Obliterative Cardiomyopathy is typified by Endomyocardial Fibrosis in humid tropical zones and is also seen in temperate zones as a result of Loeffler's Eosinophilic Fibroplastic Endomyocarditis.

The rare specific Heart Muscle Diseases are briefly discussed with reference to Diffuse Systemic Sclerosis, Amyloid Disease, Sarcoidosis, Haemochromatosis, Acromegalic Heart Disease, Heart Diseases Associated with Familial Neuropathic Disorders, and Endocardial Fibroelastosis. Brief mention is made of heart muscle disease with Eosinophilia.

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