Treatment of congestive cardiomyopathy

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Summary
Although the majority of patients with cardiomyopathy are in the category of primary or idiopathic cardiomyopathy, for which therapy is symptomatic and non-specific, there are a number of secondary forms of cardiomyopathy for which specific therapy is available, thus giving impetus to prompt and accurate diagnosis. Among inflammatory lesions, brucellosis, psittacosis and toxoplasmosis are examples. Treatable metabolic causes include thyrotoxicosis and thiamine deficiency, the latter as well as calorie-protein malnutrition are also preventable. There is presumptive evidence that the cardiomyopathy of haemochromatosis is benefited by repeated phlebotomies. Symptomatic relief of obstructive cardiomyopathy is achieved by \( \beta \)-adrenergic blockade, although resection of obstructing myocardium still has a place.

The therapeutic approach to the vast majority of cases of congestive cardiomyopathy is non-specific, comprising controlled activity, sodium restriction, digitalis and diuretics. Vasodilators and, occasionally, \( \beta \)-adrenergic blockade may be beneficial. Pacemakers may be life-saving, whereas the place of anti-arrhythmics remains uncertain. Transplantation warrants further application. Valve replacement has little to offer.

Primary prevention, comprising balanced nutrition, vaccines and genetic counselling, merits wider application. In individuals at risk or already afflicted, programmes of secondary prevention should include good nutrition, abstinence from alcohol and protection from drugs and toxins.

The approach to the patient with congestive cardiomyopathy
When the contemporary, well educated physician faces a patient with fever, he is quite likely to perform a thorough clinical and laboratory examination in order to find the cause of the fever, identify the specific infectious organism or inflammatory process responsible, and select the appropriate therapeutic regime. A diagnosis of fever is generally considered unacceptable.

The corresponding presenting symptom of congestive cardiomyopathy is congestive heart failure. What is the usual approach of the practising physician to congestive heart failure? A prevalent approach consists of making the diagnosis of congestive heart failure and selecting a therapeutic regime comprised of digitalis and diuretics. Striking relief of symptoms is usually obtained and, unless symptoms persist or recur, further diagnostic evaluation and search for possible specific therapy often is not undertaken. Here, then, the very effectiveness of therapy on the one hand and, on the other, the complexity of diagnostic studies that may have to be employed to establish an aetiological diagnosis become impediments to early diagnosis of the underlying heart disease. Not only may this approach prevent the early application of specific therapy, but it may also preclude steps toward secondary prevention.

The recommended approach to a patient with congestive heart failure must include (1) the acceptance of the need to make every effort to establish an early aetiological and anatomical as well as functional diagnosis, (2) the consideration that, with few exceptions (e.g. infectious or toxic myocarditis), congestive heart failure is a manifestation of chronic heart disease, (3) the recognition that chronic heart disease warrants continuity of care to prevent further cardiac reserve, and to facilitate rehabilitation of maximal self-care and function within family and society.

Once congenital, valvar, hypertensive, ischaemic and pericardial heart disease have been ruled out, the diagnosis of cardiomyopathy may be entertained. The first question to be answered then must be ‘are we dealing with a secondary cardiomyopathy?’ Although the likelihood that one is dealing with a disease for which there is specific therapy is small, it always exists. Furthermore, the diagnosis of a
specific disease often permits a more accurate prognosis, a responsibility of the physician well recognized by Hippocrates.

The therapy of secondary cardiomyopathies

Inasmuch as almost any recognized disease process may at one time or another involve the myocardium, a full list of secondary cardiomyopathies would be very large indeed. When one reduces the list to diseases which have been associated with heart failure and also are known to respond to specific therapy, however, the list becomes manageable.

Table 1 is an attempt to do just this. Recent advances allow one to expect that before long there may also be anti-viral agents available for treatment of viral myocarditis if recognized early (Pavan-Langston, Buchanan and Alford, 1975).

**Table 1.** Secondary cardiomyopathies for which there is special therapy

<table>
<thead>
<tr>
<th>Infectious</th>
<th>Rickettsial infections</th>
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<tr>
<td>Brucellosis</td>
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<tr>
<td>Histoplasmosis</td>
<td>Toxoplasmosis</td>
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<tr>
<td>Pneumococcal pneumonia</td>
<td>Trichinelliasis</td>
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<tr>
<td>Mycoplasma pneumonia</td>
<td>Tuberculosis</td>
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<tr>
<td>Psittacosis</td>
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<tr>
<td>Inflammatory – Infiltrative</td>
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<tr>
<td>Haemochromatosis</td>
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<tr>
<td>Lupus erythematosus</td>
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<tr>
<td>Sarcoidosis</td>
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<tr>
<td>Endocrine – Metabolic</td>
<td></td>
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<tr>
<td>Calorie-protein malnutrition</td>
<td></td>
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<tr>
<td>Thiamine deficiency</td>
<td></td>
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<tr>
<td>Thyrotoxicosis</td>
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One must now address a hidden assumption: 'Myocardial disease or dysfunction, associated with a systemic disease for which there is specific therapy, itself is benefited by this therapy.' This remains largely an open question. The answer is surely in the affirmative for some conditions, such as thyrotoxicosis. Increasing evidence also points to improvement of congestive heart failure with treatment of haemochromatosis by repeated venesections (Easley, Schreiner and Yu, 1972; Skinner and Kenmure, 1973).

One other consideration is worthy of mention. The systemic disease or disorder underlying a secondary cardiomyopathy may aggravate the associated cardiomyopathy by increasing cardiac load. A few examples may serve to highlight this point. A patient with a viral or bacterial infection may have fever, which by increasing metabolic rate and need for heat elimination increases cardiac output and hence preload. A patient with lupus erythematosus and renal failure may have an increased blood volume, associated with increased cardiac output and hence preload. Preload is also increased by anaemia, as encountered, for instance, in sickle cell disease or neoplastic disease. Generalized infections may be associated with tachycardia, increasing cardiac work. Renal disease may be accompanied by hypertension, confronting the left ventricle with an increased afterload. Similarly, acute or chronic pulmonary disease associated with pulmonary hypertension may increase right ventricular afterload. A pertinent example here would be pulmonary sarcoidosis. Thus, therapy of systemic disease underlying a cardiomyopathy may benefit the heart by reducing the cardiovascular load, even when there is no direct effect of therapy upon the heart.

The non-specific therapy of congestive heart failure

In general, congestive heart failure in patients with cardiomyopathy responds to non-specific therapy, especially when acute or early in the course of the disease process (Goodwin, 1973). There is a tendency for recurrent episodes of congestive failure to respond less and less well, especially if the recurrence is a manifestation of the natural history of the disease rather than attributable to an acutely supervening increased cardiac load or the patient's non-compliance with the previously prescribed therapeutic regimen.

Non-specific therapy of congestive heart failure associated with cardiomyopathy has four cardinal objectives, to (1) strengthen the function of the pump; (2) spare the pump by reducing the work load to the extent compatible with the body’s need for blood flow and perfusion pressure; (3) prevent secondary damage to the myocardium as well as other organ systems; (4) promote optimal healing and recovery of function of the myocardium.

1 Control of activity

It is well recognized that pathophysiological and clinical manifestations of myocardial failure are brought out or precipitated by increasing the work of the heart. Although the increase in cardiac output associated with increased bodily activity or exercise constitutes largely a volume or preload, and hence is relatively well tolerated, it is often associated with an increased afterload as well. The increased venous return and ventricular filling pressures result in ventricular dilatation and hence increased wall tension. In the case of acute viral as well as trypanosomal myocarditis, it has been clearly shown experimentally that myocardial damage is increased by exercise (Gatmaitan, Chason and Lerner, 1970; Elson and Abelmann, 1965).

Furthermore, the increased sympathetic tone associated with congestive heart failure may raise arterial blood pressure not only at rest but especially during exercise. A good case can thus be made for resting the heart by restricting activity of patients.
with heart failure. Clinical experience indicates that this indeed is a valuable therapeutic approach, although therapeutic trials of rest alone have not been reported. The chief proponent of rest therapy, George Burch, obtained considerably better results with hospital controlled bed rest in patients with alcoholic cardiomyopathy than in patients with other forms of cardiomyopathy, raising the question of the role of environmental and nutritional effects of prolonged stays in hospital (Burch and Giles, 1972). Strict bed rest may not be required; actually, both heart size and stroke volume in the supine posture are greater than in the upright posture. Furthermore, controlled activity may be tolerated better psychologically than absolute bed rest. Psychological stress may be associated with tachycardia and/or increased peripheral resistance, increasing cardiac work. Alcoholic cardiomyopathy often responds well to abstinence and good nutrition. Careful analysis of a patient's life style may reveal ways and means by which unnecessary physical exertion and stress may be avoided without totally incapacitating the individual.

2 Caloric restriction

Body weight is directly related to heart rate and cardiac output. The demand for oxygen transport, and hence blood flow, for a given bodily activity is a direct function of body weight. Therefore, reduction of body weight may increase cardiac reserve and thus raise the threshold for symptoms of heart failure. Weight reduction is mandatory for overweight patients and may be helpful in others. In the natural course of chronic cardiomyopathy, many patients develop anorexia, lose weight, and may actually improve symptomatically. One must be alert, however, for the development of cardiac cachexia, which itself may be deleterious.

3 Sodium restriction

The value of dietary sodium restriction in the therapy of congestive heart failure is well established. It is worth pointing out, however, that since the advent of potent diuretic agents, physicians tend to be less forceful in prescribing restriction of sodium intake. While this is understandable psychologically, in that it substitutes giving for taking, it should be remembered that the value of sodium restriction lies in the reduction of need for potent diuretics.

4 Diuretics

The aim of diuretic therapy is the excretion of sodium and water, effecting a reduction of circulating blood volume and hence preload. The resulting decrease in ventricular volumes will also decrease wall tension and afterload; the net effect will be improved cardiac function. Ejection fraction, stroke volume and cardiac output may actually increase. One must be alert, however, to several risks associated with diuretic therapy. One danger of diuretic therapy lies in the possibility of excessive depletion of circulating blood volume, resulting in a fall of ventricular filling pressures below the level required to maintain an adequate cardiac output. Another danger lies in the excessive depletion of tissue potassium and magnesium, which may enhance myocardial damage and delay healing; loss of these electrolytes may also lower the threshold of digitals toxicity. These undesirable effects of diuretic therapy are especially likely when the powerful diuretic agents ethacrynic acid and furosemide are used. Therefore, whenever possible, management of patients with congestive heart failure should prevent the advent of congestive stages of a severity requiring these potent diuretics. Thiazide diuretics, in association with moderate restriction of dietary sodium, should be used to prevent accumulation of fluid and to maintain close to dry weight. Potassium supplementation in the form of fruit or potassium chloride should be remembered. Aldosterone inhibitors, such as spironolactone or triamterene, are most effective when combined with thiazides.

5 Oxygen therapy

Pulmonary oedema may result in decreased saturation of arterial blood for oxygen by decreasing pulmonary diffusion and increasing venous admixture. In addition, low output cardiac failure is characterized by a wide arteriovenous oxygen difference, resulting in further lowering of the partial pressure of oxygen in the tissues, and especially in the myocardium. The resulting tissue hypoxia may enhance tissue damage in cases of toxic or infectious cardiomyopathy, as has been shown in experimental viral myocarditis (Pearce, 1960).

By means of intermittent inhalation of high concentrations of oxygen, the oxygen-carrying capacity of blood, and hence tissue oxygenation, may be significantly increased. Improved myocardial oxygenation often reduces ventricular irritability. It is important to keep this in mind, inasmuch as the biventricular failure of congestive cardiomyopathy often protects the lungs and is not accompanied by dyspnoea or orthopnoea, the usual indications for oxygen therapy.

It should be pointed out that even mild anaemia causes a significant reduction in oxygen-carrying capacity and should be corrected.

6 Digitalis glycosides and other positive inotropic agents

Congestive cardiomyopathy represents the purest form of failure of the muscle pump. This should be the ideal situation, then, for effective use of agents with positive inotropic action, among which the
digitalis glycosides remain most important. Indeed, congestive cardiomyopathy is characterized by marked depression of ventricular function, which, in general, responds favourably to digitalis glycosides (Yankopoulos et al., 1968). Stroke volume tends to rise, even while ventricular filling pressure falls. Exceptions may be seen in patients with marked endocardial fibrosis and occasionally in end-stages of severe myocardial fibrosis, constituting what has been called restrictive cardiomyopathy, the physiological equivalent of constrictive pericarditis. There is, however, a tendency for progressive loss of myofibres and increase in fibrosis as the natural course of cardiomyopathy progresses, with the result that the compliance of the myocardium decreases, the heart becomes stiffer, and digitalis glycosides become less effective.

The improved blood pressure, pulse pressure and cardiac output caused by digitalization alone may initiate an effective diuresis, even when diuretic agents have become ineffective.

Clinical experience also holds that digitalis toxicity may be more prevalent in patients with cardiomyopathy, especially in infiltrative forms and in amyloidosis. Controlled studies, however, are not available. When either underdigitalization or overdigitalization is suspected, plasma digoxin (or digitoxin) levels may be helpful in determining the proper dosage of the drug (Duhme, Greenblatt and Koch-Weser, 1974).

In acute, severe exacerbations of congestive heart failure, especially when associated with hypotension, more powerful positive inotropic agents such as norepinephrine, isoproterenol or dopamine have been used and may be of value. This is especially the case if the episode has been precipitated by intercurrent illness which is treatable or self-limited. The newer agent dobutamine appears to be especially useful in patients with cardiomyopathy because of its predominant positive inotropic activity without significant vascular, chronotropic or arrhythmogenic actions (Akhtar et al., 1975). Loeb, Bredakis and Gunnar (1977) recently compared the acute haemodynamic effects of dobutamine and dopamine in thirteen patients with chronic low output failure, secondary to cardiomyopathy in eight. At dosages adjusted to achieve comparable increases in cardiac output, dobutamine reduced left ventricular filling pressure from 25 to 17 mmHg, while dopamine increased it to 30 mmHg. Mikulic, Cohn and Franciosa (1977) were able to show that the effect of dobutamine could be enhanced by combining the drug with the vasodilator nitroprusside.

7 Vasodilator therapy

It has long been known that increased peripheral vascular resistance and increased aortic impedance constitute an undesirable increase in left ventricular afterload, likely to precipitate or increase left ventricular failure. Furthermore, the peripheral vasodilatation associated with thiamine deficiency allows a prolonged state of high cardiac output without failure, whereas treatment with thiamine, restoring peripheral vascular tone, may precipitate acute ventricular failure (Akbarian, Yankopoulos and Abellmann, 1966). The author and his colleagues have also been aware that, in the absence of valvular heart disease or arrhythmia, thyrotoxicosis associated with peripheral vasodilatation tends to be quite well tolerated by the heart, as is anaemia with its lowered peripheral vascular resistance.

Nature's experiments have only recently been translated into the therapeutic approach of left ventricular unloading by lowering aortic impedance. Cohn and his colleagues first applied this therapeutic approach to congestive heart failure secondary to ischaemic heart disease as well as to cardiomyopathy (Franciosa et al., 1972). A recent study of the circulatory response of twelve patients with congestive cardiomyopathy to the intravenous infusion of sodium nitroprusside, at a rate of 51–100 μg/min, exemplifies the striking effects of this method of unloading the heart (Rossen, Alderman and Harrison, 1976). Systemic vascular resistance fell from 22·3 to 13·9 resistance units, associated with a 48% increase in cardiac index from 2·1 to 3·1 l/min/m², whereas the mean arterial pressure fell only 16% from 86 to 72 mmHg. These changes were associated with decreases in pulmonary arterial, pulmonary wedge and left ventricular end-diastolic pressures of 35, 38, and 37% respectively. These effects must be attributed primarily to the action of nitroprusside as an arteriolar vasodilator, although this drug is also known to decrease preload by dilating peripheral veins. Nitroprusside, however, must be given intravenously, and continuous monitoring of arterial and left ventricular filling pressures is indicated. Thus, its usefulness is restricted to intensive care areas in hospitals.

More recently, it was shown by Franciosa, Pierpont and Cohn (1977) that the same principles of reducing pre- and afterload may be applied to the ambulatory treatment of patients with congestive cardiomyopathy. A single oral dose of 50–100 mg of hydralazine was given to sixteen patients with left ventricular failure, due to cardiomyopathy in nine. The pulmonary wedge pressure was decreased by 5·5 mmHg and the mean arterial pressure by 7·8 mmHg, while the cardiac index increased by 0·95 l/min/m². Studies of central venous pressure, forearm venous capacitance and tone revealed no significant changes after hydralazine, confirming that this drug acted primarily by reducing left ventricular afterload.
Massie et al. (1977) explored the combination of systolic unloading of the left ventricle by hydralazine with reduction of preload by means of nitrates, administered either sublingually or orally as sorbide nitrate, or topically as glyceryl trinitrate ointment. Their group of twelve patients with severe chronic heart failure included five with cardiomyopathy of 3–15 years' duration. They demonstrated that, whereas nitrates reduced the filling pressures of both ventricles without affecting cardiac output, hydralazine increased cardiac output without altering filling pressures. Combining the two drugs resulted in a 36% decrease of left ventricular filling pressure and a 58% increase in cardiac index. This and other recent studies suggest that oral vasodilator therapy may indeed represent a major addition to the management of chronic congestive cardiomyopathy. The study of Aronow et al. (1977), investigating the vasodilator trimazosin, which acts on resistance as well as capacitance vessels, merits special mention, inasmuch as its sixteen cases included eight with primary and one with alcoholic cardiomyopathy, and because it was controlled, improved exercise duration was shown.

8 Antiarrhythmic agents and pacemakers

Sinus tachycardia, sinus bradycardia, atrial and ventricular arrhythmias, as well as disturbances of conduction are commonly encountered in patients with congestive cardiomyopathy. Essentially no controlled studies of the prognostic significance of individual arrhythmias and of the therapeutic efficacy of individual antiarrhythmic agents are available. Thus, in general, the therapeutic approaches have been those used in the face of rheumatic or arteriosclerotic heart disease. A comprehensive review of these approaches does not fall within the scope of this review. However, a few comments are in order.

The haemodynamic consequences of arrhythmias in patients with low output cardiac failure deserve emphasis. As a result of the depressed myocardial function, a high ventricular filling pressure is required to maintain even a low stroke volume. These patients are thus especially vulnerable to reduction in the diastolic filling period such as that caused by tachycardia or premature contractions. On the other hand, stroke volumes are also limited, so that excessive bradycardia also reduces cardiac output. One goal of antiarrhythmic therapy, then, is to assure optimal cardiac output for the given state of the myocardium. These considerations may constitute the primary indications for drug therapy of frequent premature beats or for cardioversion of atrial tachy-arrhythmias. In cases of excessive bradycardia, an artificial pacemaker may be indicated.

Until controlled studies of the efficacy of individual anti-arrhythmic agents in cardiomyopathy are available – and it is conceivable that individual variations may preclude general conclusions – the choice of drug for each patient must be made largely on an empirical basis. Random sampling by examination or routine ECG, however, should be replaced by long-term monitoring, preferably during the patient’s routine day and night.

Changing atrial and especially ventricular arrhythmias, as well as unstable conduction disturbances, especially in a previously unknown patient, may signal an active inflammatory cardiomyopathy and constitute an indication for close surveillance and monitoring such as is available in coronary care units.

In cases of persistent sinus tachycardia, β-adrenergic blockade has been reported effective in improving ventricular function as assessed non-invasively, in reducing heart size, and in increasing working capacity (Waagstein et al., 1975).

In certain secondary cardiomyopathies, such as sarcoidosis and Chagas' disease, the frequency of sudden death in patients with conduction disturbances represents an indication for early prophylactic permanent pacing (Duvernoy and Garcia, 1971).

9 Anticoagulant therapy

Thrombo-embolic complications are frequent in patients with chronic congestive cardiomyopathy. A proved thrombo-embolic episode or demonstration of intracardiac or peripheral venous thrombosis by means of angiography or radionuclide portrayal constitute indications for chronic anticoagulation, usually with a coumarin derivative. In patients with chronic, intractable congestive heart failure, prophylactic anticoagulation may be well advised.

10 Corticosteroids

Despite experimental evidence of their deleterious effect in viral and parasitic myocarditis (Kilbourne, Wilson and Perrier, 1956), corticosteroids may have a place in fulminant myocarditis, non-responsive to conventional therapy. However, the evidence is anecdotal, and corticosteroids should be tapered promptly as the patient improves.

The main usefulness of these agents is in the therapy of cardiomyopathy associated with collagen diseases known to be responsive, such as lupus erythematosus or periarteritis nodosa, and perhaps in myocardial sarcoidosis, inasmuch as the myocardium tends to heal with fibrosis.

11 Surgical treatment of mitral and tricuspid insufficiency

Severe ventricular dilatation often causes incompetence of the atrio-ventricular valves, resulting in significant increases in preload, further cardiomegaly and increased wall stress. In the case of the
mitral valve, significant regurgitation superimposed on cardiomyopathy carries an especially poor prognosis. Fortunately, the treatment modalities already discussed often effect a decrease in heart size and reduction or even elimination of valvar regurgitation. When this is not the case, patients may come under consideration for surgical replacement of the mitral and/or tricuspid valve. In the author's experience, results have been uniformly disappointing. These patients constitute formidable surgical risks and usually represent end-stages of myocardial disease. He has, however, seen moderately good results when pre-operative medical therapy was suboptimal and the operation brought the patient under a strict therapeutic regimen.

12 Circulatory assist devices

The most widely used circulatory assist device is the intra-aortic balloon, which by means of systolic unloading reduces left ventricular afterload and by means of diastolic augmentation increases coronary blood flow. In low output states, significant increases in cardiac output at decreased ventricular filling pressures may be effected. This device often is of clinical value in intractable cardiogenic shock. Because it cannot ordinarily be used for more than a few days, it is of limited value in patients with cardiomyopathy. The device should, however, be considered in the therapy of acute fulminating, intractable myocardial failure, when the process is thought to be self limited or reversible, e.g. in severe toxic or infectious myocarditis. The intra-aortic balloon has found its greatest application in the cardiac surgical patient, whose acute or chronic myocardial depression does not provide an adequate circulation after discontinuation of cardio-pulmonary by-pass (Buckley et al., 1973). In practice, these are likely to be patients with ischaemic or valvular heart disease.

When the residual myocardial function is so poor that the support given by intra-aortic balloon pump does not suffice to maintain adequate tissue perfusion, partial cardiac by-pass or an artificial heart device may be considered (Litwak et al., 1976; Radvany et al., 1978). At this time, such devices are largely experimental and suited only for temporary use.

A non-invasive approach to systolic unloading and diastolic augmentation, known as external counterpulsation (Soroff et al., 1969), warrants further trials in patients with cardiomyopathy.

13 Cardiac transplantation

Primary congestive cardiomyopathy, by definition, is limited to the heart, occurs generally in young or full adulthood, is characterized by limited response to medical therapy, is not subject to palliative surgery, and carries a poor prognosis. Patients with this disorder would thus seem to be ideal candidates for cardiac transplantation. Indeed, many of the recipients of heart transplants have been patients with cardiomyopathy (Rider et al., 1975). It is quite likely that, when further advances in cardiac preservation and in immuno-suppressive therapy warrant expansion of cardiac transplantation centres, this will become an even more important therapeutic approach to the patient with cardiomyopathy.

14 The need for other therapeutic modalities

Clearly, therapy of congestive cardiomyopathy leaves much to be desired. Although this is in good part a function of the disease itself, future developments may increase therapeutic effectiveness. There is need for positive inotropic agents which are neither vasoconstricting nor arrhythmogenic, for antiarrhythmic agents without negative inotropic effects, and for diuretics which do not deplete tissues of potassium or magnesium. Agents favouring protein synthesis might be useful. Controlled studies of prophylactic pacing and antiarrhythmic agents are indicated, as is the evaluation of anti-inflammatory agents in the therapy of acute myocarditis.

Primary prevention

Primary prevention of cardiomyopathy warrants greater attention than it has been given. Hereditary myopathies with cardiac involvement and some familial cardiomyopathies might be prevented by genetic counselling. Early detection by means of amniocentesis may provide another avenue for prevention (Mahoney et al., 1977).

The introduction and wide application of vaccines against smallpox, poliomyelitis, rubella, measles, influenza and diphtheria undoubtedly have prevented many acute and perhaps some chronic cardiomyopathies. The recently developed vaccine against pneumococcal pneumonia should also be of value in protecting the heart. Vaccines against Trypanosoma cruzi are being developed. The danger of myocardial involvement with possible permanent late effects should be a major impetus to the future development of vaccines against Coxsackie viruses.

Finally, the recognition of cardiotoxins and their elimination or the control of their use are most important components of preventive cardiology. Examples are radiation and cobalt.

Secondary prevention

Earlier approaches to primary cardiomyopathy held that a patient so diagnosed was suffering from a heart muscle disorder of a specific cause as yet undiscovered. Increasingly, we have come to recognize that even an individual patient’s primary cardiomyopathy may be pluricausal, and that even...
in a secondary cardiomyopathy other aetiological factors may be playing a contributory role. These may be considered as risk factors. This concept of cardiomyopathy permits a therapeutic approach stressing the prevention of additional structural and functional damage to the myocardium. This approach to secondary prevention is presented in Table 2. Only a few items tabulated here warrant additional comment. The acute and chronic depressant effect of alcohol upon myocardial function, as well as its enhancing effect upon an already existing disease of heart muscle, have been so well demonstrated in both experimental animals and in man that its use should be severely restricted or proscribed altogether in patients with cardiomyopathy (Regan, 1973; Gould et al., 1971).

<table>
<thead>
<tr>
<th>Table 2. Secondary prevention of cardiomyopathy</th>
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| 1. Assure balanced nutrition  
Adequate proteins  
Adequate vitamins |
| 2. Treat  
Hypertension  
Anaemia  
Infection |
| 3. Prevent thrombo-embolic complication  
Anticoagulants |
| 4. Avoid  
Overweight  
Excessive activity  
Pregnancy  
Heat and humidity  
Potassium and magnesium loss  
Alcohol  
Cigarettes  
Drugs, esp. phenothiazines and tricyclic antidepressants  
Radiation |

Cigarette smoking should be considered a myocardial stress and avoided, in view of its chronotropic and positive inotropic effect, combined with an increased afterload and decreased arterial saturation for oxygen (Rabinowitz and Abelmann, 1977).

It is also evident that, in order to render this approach feasible and effective, early diagnosis of cardiomyopathy becomes most important.

Rehabilitation

Because of the generally guarded prognosis of congestive cardiomyopathy, it is easy for physicians, patients and their families to accept a defeatist attitude, if not with regard to therapy, certainly with regard to rehabilitation. The generally good response to therapy of early congestive failure must be kept in mind, and especially when heart failure has been precipitated by a treatable or preventable excessive cardiac load or intercurrent illness, a cautiously optimistic attitude is warranted. A plan for rehabilitation to self-care, normal family life and even gainful employment should be considered for all patients. Although specific information is not as yet available, it is not unreasonable to expect that in earlier stages of cardiomyopathy carefully designed physical exercise regimens may be of conditioning value, reducing exercise heart rate and blood pressure, as is the case in healthy subjects and in patients with ischaemic heart disease (Wilhemsen et al., 1975).

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References


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