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Update on Management of Congestive Heart Failure

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Syndrome of congestive heart failure (C.H.F.)is such a common and so well recognized clinical entity that an average physician is lulled into a feeling of self security that he understands this entity very well. Quite the contrary, however as modern techniques become available to assess various parameters of cardiac function and performance one realizes that this syndrome defies simple understanding or definition. The simplest definition is that, "this is a condition of impaired cardiac pumping."

If we accept this simple definition there are still a vast number of unresolved questions relating to the etiology pathophysiology, clinical recognition, treatment and prognosis of this syndrome. A proper understanding of these parameters in the light of our present knowledge is essential to gain insight into proper preventive and therapeutic measures. I shall like to review some of the data in the light of our present knowledge with the hope that the recognition and management of this syndrome will be easier for a general physician.

Etiology:

Any condition that damages the myocardium or produces a state of increased pressure or volume overload over a certain period of time can produce this syndrome. These commonly include;

Pressure Overload:

1. Hypertension.
2. Aortic stenosis.
3. Hypertrophic obstructive cardiomyopathy.

Volume Overload:

1. Mitral regurgitation.
2. Aortic regurgitation.
3. A.V. fistula.
4. L-R shunts.

Myocardial Damage:

1. Myocardial ischemia.
2. Infections of the myocardium e.g. viral myocarditis.
3. Inflammations of the myocardium e.g. rheumatic myocarditis.
4. Infiltrative diseases of the myocardium e.g. sarcoidosis, amyloidosis etc.
5. Drugs adriamycin, emetine, phenothiazine, tricyclates etc.
6. Toxic e.g. diptheria.
7. Miscellaneous; D.C. shock etc.

It is interesting to note that hypertension was the leading cause of heart failure in Framingham Study, published in 1971. This was true even if concurrent rheumatic or coronary artery disease was present. This finding emphasizes the importance of controlling hypertension as an important prophylactic measure in the management of hypertension. However, since the publication of this study treatment of hypertension has improved remarkably and at least in the developed countries this condition is relatively less pronounced.

A very recent study published by Franciosa et al in 1983 in the United States, shows that ischemic heart disease (I.H.D.) was the cause of C.H.F. in 40-75% of the cases. Although hypertension might have been present in the earlier years in at least some of these patients, hypertensive readings were not recorded in these patients at the time of their evaluation. Myocardial ischemia can cause heart failure by any one of the following mechanisms;

1. Damage to a large area of heart muscle.
2. Global hypokinesia of the left ventricle with relatively normal looking coronaries i.e. ischemic cardiomyopathy.

In the remaining 25-60% of the cases in this series no evidence of myocardial ischemia was evident at the time of evaluation. Thus other causes of heart failure enumerated previously can be instrumental in such cases. In our society rheumatic heart disease remains a very important cause of congestive heart failure. This disease can effect the myocardium by two mechanisms;

1. Direct Muscle Damage - - - rheumatic myocarditis
2. Secondary Muscle Damage - - - as a result of

    a. Volume overload as in mitral regurgitation or aortic regurgitation.
    b. Pressure overload as in aortic stenosis or mitral stenosis.

Clinical Diagnosis:

Diagnosis is not difficult when a patient with advanced heart failure presents to a physician. Traditional signs of heart failure such as:

1. Low volume arterial pulse.
2. Raised central venous pressure (C.V.P.)
3. Peripheral edema.
4. Enlarged tender liver.
5. Bilateral basal end inspiratory rales.
6. Cardiac enlargement.
7. (S_3) Gallop.

However, in early cases of congestive heart failure these sign may not be over and yet it is in these cases that a proper diagnosis is essential and necessary. In such cases following additional measures may be helpful.

Hepatojugular Reflux.

This simple bed side maneuver is quite helpful to pick up early cases of cardiac decompensation. A sustained pressure over the right hypochondrium in a normal person will produce a transient or no change in the C.V.P. However in patients with cardiac decompensation a significant increase in C.V.P. is noted and persists as long as the pressure is maintained. This reflux is produced by the following mechanisms;

1. Increase blood flow to the already congested right atrium due to mobilization of the visceral blood.
2. Raised diaphragm which will produce a tamponade like effect.

It is evident that this sign is an indicator of right atrial hypertension and as such suggests right heart decompen sation. However, most common cause of right heart failure (R.H.F.) is left heart failure (L.H.F.) and thus it is present in some cases of even early L.H.F. Nevertheless, its interpretation must be in the light of this knowledge.

Non-Invasive Measurement of Cardiac Size & Function.

Various non invasive methods like;

1. Chest X-ray,
2. Chest X-ray flouroscopy,
3. Echocardiography,
4. Nuclear pool studies.

can give us very useful indications regarding the size and pumping reserve of the heart. These procedures are now readily available in most of the large cities of Pakistan. Evidence of cardiac enlargement or weakness of cardiac pumping by these techniques even in asymptomatic patients must be taken seriously and the patient managed accordingly.

Exercise Testing.

In the early stages of heart failure some sort of physical stress may be necessary to bring out the cardiac decompensation. This can be assessed by using the standard criteria of the New York Heart Assocaition, which is entirely dependant on the subjective account of the patient This method is not without its limitations since individual patients differ in their capacity to elaborate a certain symptom. Furthermore, the assessment may be further limited by the individual habits of a patient. A sedentary person may hardly be aware of his limitations since he never exerts, yet an active person may complain of severe limitation of his physical performance and yet may be able to do very well when assessed objectively on a treadmill or a bicycle ergometer.

In view of this limitation, other more objective methods have been devised to assess cardiac decompensation during physical stress.

These include:–

1. Measurement of O_2 consumption by collection of expired gas during progressive exercise on a treadmill or a bicycle ergometer. This allows calculation of maximum O_2 consumption that patient can achieve. When the test is limited by dyspnea or fatigue then a peak O_2 consumption of less than 25 ml/kg/min. will suggest impaired O_2 consumption. In patients with cardiac dysfunciton it will be accompanied by a rising respiratory quotient, high level of expiratory O_2 suggesting anaerobic metabolism and a peak cardiac out put response.

2. Heart failure can also be assessed by a rising filling L.V. pressure and an attenuated cardiac out put response during stress. These measurements can only be carried out during invasive cardiac catheterization and thus have their limitations'

3. Newer noninvasive techniques like CO_2 rebreathing and impedance cardiography are being devised and may help the future clinician.

Pathophysiology:

Experimental models of heart failure by traumatizing an animal heart are difficult to produce. Thus this defies

the common notion that heart failure is due to damage to the heart muscle. Similarly, in patients with extensive myocardial infarction, signs of acute heart failure may completely resolve initially only to reappear in the months to come without any apparent evidence of further damage to the myocardium.

In a recent study this syndrome has been produced in a canine heart by damaging a large area of the myocardium after repetitive D.C. shocks. This model confirms that the syndrome so well known to us all is the result of a number of positive feed back mechanism involving the heart, peripheral circulation, and a number of vital organs. This has made the issue more complex yet it has given us an understanding that in patients with congestive heart failure treating the damaged muscle alone may not be enough unless these other factors are also dealt with accordingly. Thus understanding of these mechanisms is essential to treat such patients properly.

These mechanisms are;

1. Aortic impedance or ventricular after load.
2. Ventricular preload.
3. Myocardial hypertrophy.
4. Sodium retention.
5. Peripheral edema.
6. Myocardial ischemia.
7. Neurohumoral factors.
   a. Renin angiotensin-aldosterone.
   b. Sympathetic system.
   c. Vasopressin-ADH.

Aortic Impedance or Ventricular Afterload:

This is the resistance that L.V. faces at the time of contraction. Thus its emptying is inversely proportional to this obstruction which is' largely dependant on the vasomotor tone of the peripheral arteries and arterioles. Whereas a normal ventricle shows no significant drop in its emptying even with marked increase in this obstruction, a decompensated heart is very sensitive and even a small increase in the impedance can produce a significant drop in the cardiac performance Fig. 1. In patients with heart failure there is enhanced activity of various neurohumoral vasoconstrictive systems explained below. This generates a positive feed back loop as shown in Fig. 2. This triggers a vicious cycle and impairs the cardiac function further.

Ventricular Preload:

This plays an important role in the symptomatology of heart failure. Elevated L.V. end diastolic pressure produces pulmonary venous congestion which is the cause of dyspnea. Furthermore it leads to pulmonary hypertension and secondary R.H.F. with elevation of C.V.P. Factors responsible for this are;

1. Reduced L.V. emptying.
2. Sodium retention.
3. Reduced compliance of the venous bed. This increases the C.V.P. even with normal venous volume.
4. Reduced compliance of the ventricle, that is its inability to distend to a desired level during diastole without raising its pressure.

Myocardial Hypertrophy:

Pressure and volume overloaded ventricles react to these loads by devloping hypertrophy of their muscle mass. This is helpful in the initial stages to maintain a near normal cardiac out put. However, with passage of time the hypertrophied muscle fails due to; 1. Reduced compliance, 2. Reduced stroke volume. as a result of diminishing functional muscle fibres due to replacement by the collagen fibres.

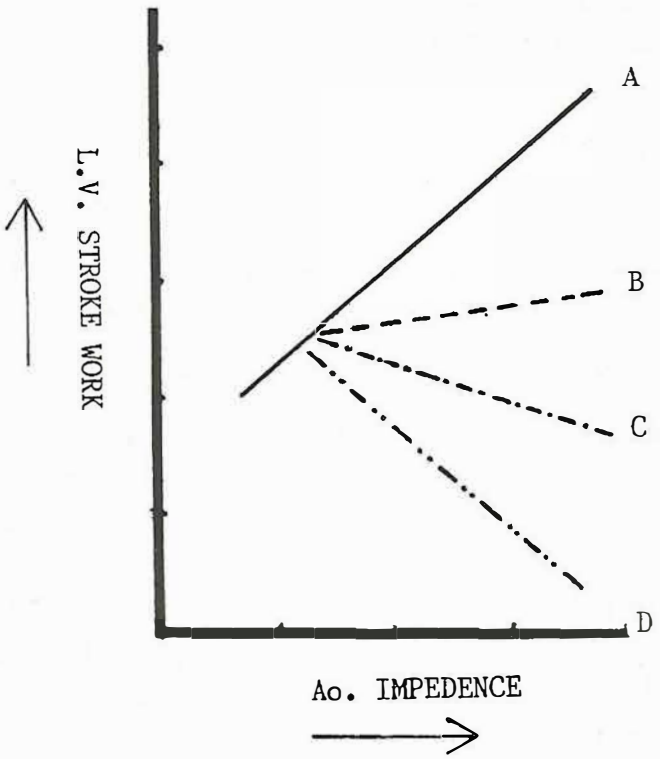

Fig 1 Relationship of Left ventricular stroke volume with aortic impedence.
A. Normal
B. Minimal left vent. dysfunction.
C. Moderate left vent. dysfunction.
D. Severe left vent. dysfunction.

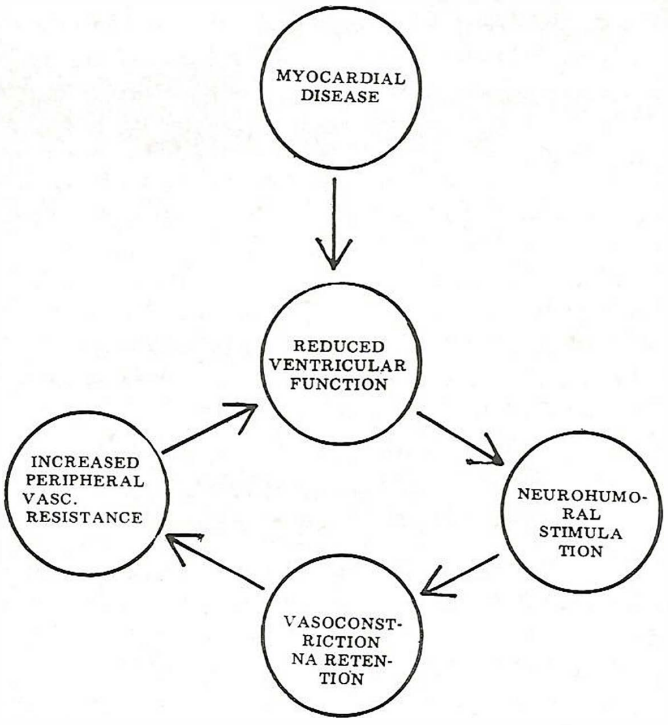

Fig-1.

It is thus imperative that hypertrophy must be kept to the minimum by adequate control of pre and afterloads.

Sodium Retention:

This is brought about by;

1. **Hemodynamic factor:**

   The low cardiac out put produces low renal perfusion, low glomerular filtration and as a result low over all excretion of sodium.

2. **Hormonals Factor:**

   a. Increased sympathetic tone, through the stimulation of baroreceptors.
   b. Renin-angiotensin - aldosterone, brought about by the hypoperfusion of the juxta glomerular apparatus of the kidney.

All these factors impair the functional capacity of the kidneys to excrete sodium at the desired level thus resulting in overall sodium retention.

Peripheral Edema:

Following factors play an important role in the formation of peripheral edema;

1. Raised CVP.
2. Sodium and water retention with reduction in plasma oncotic pressure.
3. Reduction in the lymphatic drainage.

Peripheral edema interferes with peripheral compliance characteristics and thus have an indirect effect on the venous pressure itself. Thus once formed it tends to get worse.

Myocardial Ischemia:

Myocardial perfusion of the subendocardial areas is usually maintained during diastole. This is facilitated by low ventricular diastolic pressure (V.D.P.) However, as heart failure sets in and left V.D.P. rises, subendocardial ischemia takes place irrespective of the patency of the coronaries and further impairs left ventricular function thus setting up a positive feed back cycle. This is further augmented in patients with ischemic heart disease who have proximal coronary artery obstruction and thus low pressure in the subendocardial circulation. It is thus obvious that once congestive heart failure sets in myocardial ischemia is inevitable irrespective of the cause.

Neurohumoral Response:

Three such factors seem to be operative in patients with C.H.F. These are;

1. Increased sympathetic activity and high norepinephrine levels, leading to tachycardia and systemic vasoconstriction.
2. Increased plasma renin activity by RENIN-ANGIOTENSIN-ALDOSTERONE mechanism. However, in some cases renin level may be normal and even low. In such cases peripheral vasodilatorss are ineffective or even harmful, since the low renin does not trigger angiotension synthesis and as a result there is no reactive vasoconstriction.
3. Increased vasopressin levels which produce;

   a. Vasoconstriction.
   b. Antidiuretic effect.

The sum effect of these factors is to support the blood pressure in a failing heart. However, this increase in the aortic impedance is inappropriate under the circumstances and in fact may further deteriorate the ventricular function as stated earlier.

Prognosis:

In the recent years it has become clear that mortality of most of the cardiac diseases is related to the L.V. reserve This is true in patients with acute myocardial infraction as well as those with chronic I.H.D. irrespective of the extent of coronary artery disease. Similarly sudden death is much more common in patients with L.H.F. and risk of ventri-

cular premature beats is also related directly to the underlying L.H.F. Thus presence of L.H.F. is a major risk factor contributing to the mortality in cardiac patients irrespective of the etiology.

TREATMENT OF CHF

Following observations have been made recently in this regards:

- Even the most severely failing heart is capable of improving its performance with the help of various vasodilators and ionotropic agents when used intelligently.
- The quality of life, exercise tolerance and perhaps the life expectancy is altered with appropriate pharmacological intervention at the appropriate time.

But before embarking on any therapeutic regimen it is always rewarding for the physician to set the goals & objectives of treatment. Furthermore, these objectives must be achieved at the minimum cost to the patient both bodily and financially. The prime objective of any therapeutic regimen is to improve the quality of patients life so that he can make a comfortable living. It is hoped that by improving the quality of life and by keeping patient free of the symptom of heart failure, may be one can prolong his life as well.

It is thus important that a detailed history highlighting patients symptoms, activities, nature of job and his living condition is essential. This must be combined with a thorough physical examination and noninvasive assessment of his cardiac function as out lined earlier. The treatment can then be instituted on the following lines;

General Measures:

Patients who have early symptoms and minimal limitations of their activities can benefit a lot from such measures. These includes;

- Weight loss if needed.
- Restriction of salt in-take.
- Avoidance of undue physical stress which may require adjustment and changes at his job or at home. Patient living in multistorey houses who have to climb stairs many times a day can simply benefit by moving to the ground floor. This if possible must always be advised & proves very effective.

Concurrent control of other condition which may aggravate CHF like hypertension, various supraventricular tachycardias, respiratory insufficiency, anemia, thyrotoxicosis, and renal failure is also very essential and may stabilize the cardiovascular status. Similarly, drugs that have a negative effect on myocardial contractility like, beta blockers, calcium antagonist etc. must always be discontinued or reduced appropriately.

Drug Therapy:

Once it has been decided to put the patient on treatment it is always helpful that the physician explain the importance of taking the drugs regularly and the expected side effects if any. He or she must also be warned regarding the use of certain drugs that may have an undesirable effect on the weakened myocardium and may produce serious complications like arrhythmias, e.g. ephedrine related compounds & tricyclate antidepressants which are commonly purchased over the counter without a doctor's prescription.

Diuretics:

These compounds play an important role in the management of those patients who have evidence of volume overloading as manifested by raised central venous pressure, enlarged liver, edema and pulmonary congestion. Diuresis removes the extra volume circulating thus unloading the ventricle and reducing the preload which in turn will improve the myocardial perfusion as explained earlier.

The choice between the potent loop diuretic like furosemide and a more milder and long acting one like a thiazide is made according to the underlying situation. In milder form of congestion where the patient is not in any significant distress thiazide may be preferrable. However, in moderate to severe CHF loop diureties are necessary. Dose & frequency of these drugs can be tailored to the need of the patient. Since a majority of such compounds are short acting, some patients may prefer to have it in the early morning so that they have the diuresis early in the day & can have a more relaxing afternoon & evening. Others may prefer it in divided doses twice or thrice a day, so that they have a more uniform diuresis. Patients with symptom of paroxysmal nocturnal dyspnea usually benefit by an extra dose of a short acting loop diuretic like furesamide given in the early part of the after noon. Similarly by keeping a close watch on peripheral signs of vascular congestion like, dependant edema, raised JVP, pulmonary basal rales, and maintaining a daily record of the patient's weight, one can determine the optimum dose of the diuretics. The same can be taught to an intelligent & cooperative patient that he or she may adjust the dose depending on the fluctuation of their daily weight as well presence or absence of pedal edema.

However, one very imporant factor to be kept in mind in patients with long standing CHF is that aggressive diuresis may reduce the optimum filling of the compromised ventricle which is essential to produce a certain amount of contractility based on Frank-Starling curve. This is

explained further in Fig 3.

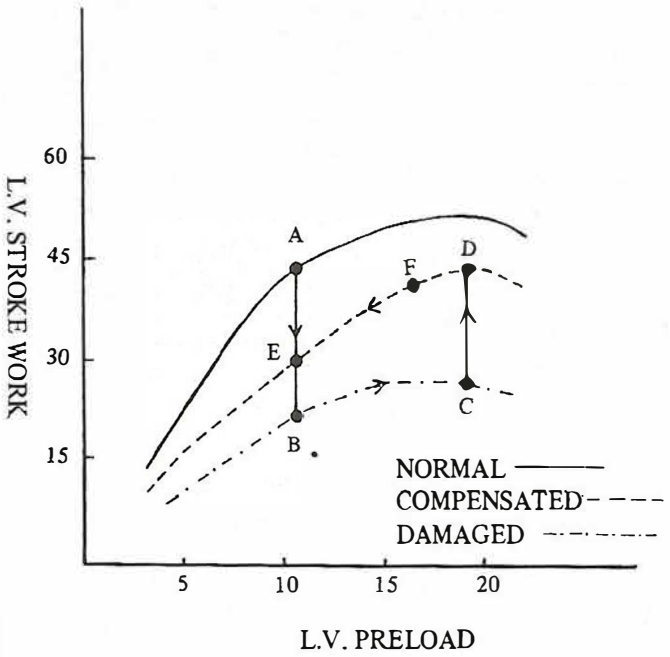

Fig 3.

An L.V. working at point 'A' of the normal Frank Starling Curve is generating an optimal stroke work. When damaged its contractility drops and it moves to a new point B with significant drop in the cardiac out put. However, as a result of the drop in the stroke volume there is a progressive increase in the residual L.V. diastolic volume which subsequently increases the preload & improves the contractility some what to a new point 'C' on a new curve for this damaged L.V. Later secondary increase in the contractility due to release of catecholamine or use of ionotropic agent like digitalis, the contractility increases further to a new point D. This improved state of contractility will unload the L.V. of its preload some what thus moving it to a point 'E' on this new "compensated" curve, thus bringing the stroke work to almost near normal level. However, if the preload is reduced further to the initial level by the use of excessive diuretics it will shift the patient to a new position 'F' where the contractility of the L.V. will be markedly reduced and the patient may develop symptom and sign of significant low cardiac out put state.

Unloading Agents:

These agents have been used over the last two decades or so in the management of CHF. But recently studies have proved that used intelligently these agents not only improve the quality of life irrespective of the underlying cause but also reduce the mortality over a three year follow up.

Most commonly used agents available here are hydralazine, iso sorbide nitrate, prazosin & recently angiotensin I converting enzyme inhibitor like captopril' These act by reducing the venous or arteriolar tone, as in case of the nitrates & thus reducing the preload & aortic impedance simultaneously. Thus nitrates are ideal in patients with congested circulation and low forward out put. The remaining act by reducing the after load or aortic impedance due to their selective action on arteriolar bed and are more beneficial in those circumstances where the forward flow is less as in case of hypertensive heart failure, mitral regurgitation. The recommended dosage for such compounds is as follows;

DRUGS	DOSE	SIDE EFFECTS
1. HYDRALA ZINE	50-100 mg BID Max.800mg/day	Reflex tachycardia Lupus like syndrome
2. ISOSORBIDE NITRATE	10-20 mg QID Max.320 mg/day	Headache, flushing, Postural Hypotension
3. CAPTOPRIL	6.25 mg TID Max. 50 mg TID	Proteinurea, Azotemia, Blood Dyscrasia, Hypotension.
4. PRAZOSIN	1-2 mg initial dose, 3-5 mg TID subsequently.	– First dose effect of severe postural hyptosion, dryness of mouth, drug tolerance.

In patients with low blood pressure the initial dosage of these drugs have to be monitored very closely since a precipitous drop can some time prove catastrophic. However if the patient has tolerated first few doses well, subsequent drop in pressure is unusual and indeed it may improve as the hemodynamic status is stabilized.

Inotropic Agents

Digitalis is a time tested drug & has been popular with physicians around the world for the treatment of C.H.F. However, there has been some recent skepticism regarding its use in the United Kingdom particularly in those patients who are in sinus rhythm. There is no question that digoxin produces a mild to moderate improvement in all parameters of cardiac function. It is also established that low dosage is more beneficial than the larger dose used previous-

ly. Thus it is important that its efficacy must be judged against its hazards particularly in patients with compromised kidney function or serious sinus or A.V. nodal disease.

Inspite of a vigorous interest in other oral inotropic agent none as yet been found safe enough to be used orally. These agents can be categorised under two headings;

a. **Beta-adrenergic agonist.**

In this category are included; persuterol, prenalterol, terbutaline, salbutamol & some other research compounds not yet marketed. Some of the above drugs act mostly through beta 2 stimulation – and have their predominant effect on bronchiolar and arteriolar dilatation. It is likely that the hemodynamic improvement with these compounds is due to the sum effect of these compounds on inotropicity as well as vaso dilatation. It is said that their prolong use may down regulate the beta receptor & thus may render these compunds ineffective.

b. **Non-adrenergic drugs;**

These drugs consist of a heterogenous group with variable mechanisms of action. Some are inhibitor of phosphodiesterase & thus raise the level of cardiac cyclic AMP. Other act by increasing the permeability of the myocardial cell to calcium or may make the contractile apparatus more sensitive to calcium. The only drug in this group that is marketed in the U.S.A. is amrinone. This too is available for I.V. infusion only since oral preparation when given over a long period produces serious blood dyscrasia.

It is thus obvious that the drug therapy of a patient with C.H.F. is to be individualized and only those drugs are used which improve the quality of his life without producing the serious side effects. An intelligent approach in this regard will definitely alleviate the miseries of many such patients.

Surgical Therapy:

– Various mechanical supports have surfaced in the last decade or so.

These include;

1. **Surgical Correction**

Modern cardiovascular surgery has changed the out look of many patients with various valvular and congenital malformations. They if left alone will develop progressive, and in certain situations like aortic stenosis, a very rapidly debilitating C.H.F. It is thus important that a physician must scrutinize such patients and evaluate them properly for an appropriate surgical intervention. Recently, more invasive therapeutic modalities like balloon valvuloplastly for pulmonary stenosis in infants & aortic stenosis in the elderly offers a new hope to such patients with minimal morbidity at a very low cost.

2. **Heart Transplant**

It has been shown that the quality of life in such patients with end stage heart disease who do not respond to the medical treatment is better with this procedure. However, the cost, availability of donors and limited numbers of centres doing this procedure limits its popularity.

3. **Mechanical Heart**

Survival of Dr. Barney Clark, after receiving the mecha nical heart at Univ. of Utah does raise the hope that future may have some prospect to offer in this field. However, at present this approach is not very practical and remains more of an experimental modality for various obvious reasons.

In summary, it can be said that the problem of C.H.F. remains a challenge to all of us in the field of medicine and it is thus necessary that a multidisciplinary, well coordinated effort must be made to effectively control such patients: by the epidemiologists & the public health authorities to prevent various conditions that lead to CHF like rheumatic fever and hypertension, by the clinicians to recognize the entity early and to treat it effectively, by the pathophysiologists to understand and resolve various aspects of the disease which are not yet clearly understood, by the pharmacologists, who must continue to come forward with newer and more effective agents in the light of our growing knowledge, and by the bioengineers and surgeons to devise more practical ways of supporting or replacing a failing or a failed heart.

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