CARDIOGENIC SHOCK - A THEORIST'S VINEYARD

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Acute myocardial infarction is cursed with high immediate mortality, the main defiants being dysrhythmias heart failure and shock. The wider appreciation and awareness of the role of dysrhythmias in early sudden dissolution and their appropriate aggressive treatment both in the coronary care units and general medical wards has brought down the mortality rate from 40 percent to 10 per ent. The cardiogenic shock, however, is a great deterrant in forestalling further the still forbidding mortality rate from acute myocardial infarction. The main cause of desperate failure of the traditional treatment of cardiogenic shock has been the lack of haemodynamic data and sparse meters. The study of the haemodynamic profile of shock has been facilitated by the introduction of Swan-Gans balloon-tipped flow-directed catheter which without the aid of image intestifiers and flouoroscopy may be wedged in the pulmonary capillaries. This has permitted the monitoring of left ventricular events allowing the study of left ventricular function in more detail. Echocardiography by defining the movements of the interventicular septum and the posterior wall of the left ventricle has revealed much valuable information of immediate practical application in the study of the clinical profile of cardiogenic shock. Besides, the study of myocardial infarction by electron microscope has revealed many subcellular changes which may have bearing not only on the immediate treatment but as well on the long term prognosis. Biochemical investigations embracing the study of plasma and urinary catecholamines, lactic acid, vaso-active substances and enzymes have thrown considerable light on the nature of shock.

The main obstacle in the study of cardiogenic shock has been the lumping together of many conditions under the cardiogenic shock. Cardiogenic shock perhaps needs defining in terms of biochemical, haemodynamic and mechanicalderangements if more effective treatment is to be planned.

I would suggest that cardiogenic shock may be classified into *three man types.*

Type I including those cases of cardiogenic shock in whom there (is some percipitating factor such as tachyarrhythmia, bardyarrhythmia, myocardial depressant (drugs such as propranolol, quinidine or procaineamide, (dehydration due to sweating and use of diuretics. These patients have excellent prognosis provided the underlying cause is immediately dealt with. If unattended, the haemodynamic derangements may threaten the integrity of peri-infarction ischaemic myocardium rendering the condition irreversible.

Type II cardiogenic shock includes those cases of acute myocardialinfarction in whom there (is violent and complete depletin of the catecholamines as a result of sympathatic storm irrespective of the magnitude of myocardial

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injury. These patients initially, for the first 24 to 72 hours, suffer from a hyperdynamic circulatory state with tahycardia, raised, blood pressure,(increased peripheral vascular resistance and increased urinary catecholamines. This state of sympathetic overdrive is followed by symathetic depression representing exhaustion phase of stress reaction. These patients may have mild to moderately severe myocardial injury as revealed by electrocardiogram or enzyme profile. The main difficulty stems from catecholamine depletion so that there is no sympathetic support to the heart or blood vessels which fail their function: the heart slows and the peripheral arterioles and venules dilate with reduction in venous return and cardiac output. This is a very desperate situation, the limited cardiac output due to obliterated venous return from venous pooling in the capacitance vessels finding ineffective expression in the sea of dilated arterioiles with precipitious fall in blood pressure. In such a depleted catecholamine state, the only effective remedy lies in replenishing the catecholamines to provide sympathetic support to the heart and to constrict the artericles and venules. The rational treatment, therefore, lies in giving these patients 5 percent dextroes in water containing adequate amount of norepinephrine (4 to 12 mg per litre of fluid depending upon the response). Prophylactically, the hyperdynamic circulatory state in acute myocardial infarction should be contained by exhibition of propranolol thus forestalling its progression to shock. This group of patients, though constituting a precious minority, is worth retrieval by judicious use of beta-adrenergic blocking drugs in early stages and sympathomimetic drugs in later stages when pre-shock or shock occurs.

Type III shock embraces patients suffering from acute myocardial infarction who have suffered considerable loss of myocardium, usually 40 to 70 percent. In these patients, there are three main determinants of clinical course:—

1. Amount of muscle damage
2. Compliance of the infarcted area
3. Mitral incompetence

The *amount of muscle damage* has great bearing on the ejection fraction irrespective of whether the muscle damage was cumulative with previous infarcions or was suffered in the episode under review. The ejection fraction is about 60 percent with 10 percent muscle damage compared to 40 percent with 40 percent muscle damage. The extent of myocardial damage is a linear determinant of ejection fraction.

Compliance of the infarcted area is the major determining factor of the clinical course. The non-compliant infarcted area would be stiff and non-distensible withstanding the pressure build-up during systole by the uninjured myocardium which is probably utilising the Frank-Starting mechanism to its optimum. In such patients, raising the left ventricular filling pressure by 5 percent dextrose in water would initiate more froceful contraction of the healthy myocardium thus facilitating higher ejection fraction, increasing the cardiac output and ensuring better coronary perfusion. The latter would salvage the peri-infarction ischaemic myocardium so that the latter would contribute to still further enhanced ejection fraction. Better cardiac output would raise the blood pressure and, through baroceptors, decrease the peripheral vascular resistance. The latter would reduce the after-load and would obli-

terate the release of vascoactive substances and lactic acidosis by ensuring better tissue perfusion. These patients constitute a sub-group in type III shock which, if properly dealt with, may be retrieved from the vicious circle of shock insisting on self-perpetuation. These patients may respond to 5 percent dextrose in water. Norepinephrine or isoproterenol may be given to invoke inotropism, though the healthy myocardium may be already working, at the climax of the Frank-Starling's curve. In other words, the shift of the ventricular function curve of the healthy myocardium (further to the left may not be possible. Sympathomimetic drugs should, therefore, be exhibited only after maximum response to volume overload has been obtained. If the exhibition of sympathomimetic amines fail to improve the haemodynamic situation or if the clinical or heamodynamic profile deteriorates, they should be with held. This sub-group (*Sub-group A*) of patients usually has left ventricular filling pressure approaching to or about 15 mmHg. and cardiac index about 2 litre or more per minute per square meter of the surface area of the body.

The patients who have a *compliant infarction* suffer from gross mechanical disadvantages. They suffer from dyssynergy which is the bane of efficient mechanical performance by the heart. The dyssynergic myocardium lags behind the healthy myocardium in shortening since it needs further stretching by the transmission of systolic pressure build-up during the isovolumetric phase of cardiac cycle. It works at a Frank-Starling curve different from that of the healthy myocardium because of its shift to the right. Much of the useful work done by the normally contractile myocardium is disspated in stretching the infracted compliant myocardium. Pressure-volume work loop shows that much of the left ventricular blood meant for ejection into the aorta is accomodated in balooning the complaint infarcted area. A large size infarct may, therefore, accommodate a significant precentage of blood. Enhancing contractility of the myocardium by digitalis or catecholamines would cause greater build-up of pressure causing more balooning of the compliant infarct leaving still reduced amount of blood for ejection. The desperate attempts in this clinical profile to use inotropic drugs would aggravete the situation by reducing still further the already compromised ejection fraction.

Besides, the site of the compliant infarct is an important determinant of haemodynamic profile. It has been demonstrated by echocardiography that abnormal interventricular septal motion is associated with a significant obstructive lesion in the left anterior descending coronary artery (Jacob et al., 1973). In the absence of significant involvement of the anterior descending branch of the left coronary artery, the interventricular septal motion as demonstrated echocardiographically is invariably normal. Since cardiogenic shock is more commonly associated with anterior myocardial infarction and involvement of the anterior descending branch of the left coronary artery, it would follow that in all such cases, the interventricular septum would be involved. In a compliant infarction of the interventricular septum, one or several of the following mechanical difficulties may arise:

(*i*) *Impaired filling of the right ventricle* due to its reduced capacity from infarcted interventricular septum being pushed by the high pressure left ventricular chamber during diastole (Bernheim effect). This would cause rise

of central venous pressure (jugular venous pressure clinically) in the presence of normal or slightly raised left ventdiular filling pressure. This effect needs demonstration angiocardiographically.

(*ii*) *Prolapse of the septal leaflet* of the tricupids value during systole because of the poor support to its anchorage (valve ring and papillary muscle). This would cause tricuspid insufficiency further reducing the return of blood to the left ventricle.

(*iii*) The dyssynergic interventricular septum may obstrcut the right ventricular outflow tract of which it forms a wall during systole. This would further reduce blood flow to the left ventricle.

(*iv*) Perhaps, the compliant infarcted interventricular septum may prolapse into the left ventricular outflow tract causing its throttling and, hence, reduce the cardiac output.

(*v*) It has been demonstracted by Jacobs et al, (1973) that when the interventricular septum is spared as in posterior myocardial infarction, it would display markedly exaggerated movements into the left ventricular cavity. Such an enhanced movement of the interventricular septum may obstruct the left ventricular outflow tract, further aggravating cardiogenic shock.

These patients need separation from Type III, subgroup A who have non-compliant infarction. Clinically, these patients have both S3 and S4 (both the ventricular and atrial gallops) in contrast to the Type III, sub-group A who because of raised left ventricular end-diastolic pressure have S4 adtial gallop. These patients may be designated to belong to Type III, subgroup B. They do not respond to volume load or inotropic drugs. Infact, that latter would aggravate the situation by increased build-up of systolic pressure in the left ventricle causing greater mechanical disadvantage. These patients carry a mortality rate of 100 per cent. The only rational treatment would be infarctectomy and sphenous vein bypass graft of the obstructed coronary artery following emergency coronary arteriography.

There is another subgroup (Subgroup C) in Type III shock. This includes patients who suffer from *mitral incompetence*. If mitral incompetence is significant (R.F.—0.5 or more), the ejection fraction would be severely compromised. The inotropic drugs and volume load would considerably aggravate the regurgiatnt fraction making further inroads and encroachment on the ejection fraction. These patients carry 100 percent mortality, their treatment should be similar to that for type III subgroup B except that they would also need mitral valve replacement, a heroic measure indeed, though the only hope in this desperate situation with no prospects of survival.

References

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