PREOPERATIVE EVALUATION OF THE PATIENT WITH MYOCARDIAL DISEASE: CLINICAL REVIEW*

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A PERCEPTIVE, ACCURATE ASSESSMENT of the cardiac patient by the anaesthetist is necessary for proper estimation of the anaesthetic risk and formulation of a safe anaesthetic course. In this review basic cardiac and circulatory physiology will be reviewed and related to the pathological changes that develop in various types of heart disease.

CARDIOVASCULAR RESERVE

In heart disease, the reserve capacity of the cardiovascular system is utilized to make up the deficit imposed by the disease, even during routine activity.[1]

Factors that contribute to cardiac reserve include:

(*a*) *Venous oxygen reserve*. With increased tissue oxygen consumption, the arterio-venous difference of oxygen content of haemoglobin can widen; that is, more oxygen is extracted by the tissues.

(*b*) *Maximum effective heart rate*. Cardiac output increases with increasing rate up to 170–180 beats per minute, beyond which there is inadequate filling time.

(*c*) *Stroke volume reserve*. Reserves of both systolic volume (in increased contractility) and of diastolic volume (due to Starling's Law) can contribute to increasing cardiac output.

(*d*) *Work of the heart*. Conversion of energy to useful work can increase in the healthy heart, but the ability to do this decreases as the myocardium fails.

(*e*) *Coronary vascular reserve*. Coronary flow can increase to meet rising myocardial oxygen requirements through a decrease in arteriolar resistance.

(*f*) *Cardiac enlargement*. The heart hypertrophies in response to increased pressure load and dilates because of long-term volume load.

All these variables respond to the need for greater cardiac output and work in circumstances such as exercise. In fact, the reserve capacity of the healthy heart is large, and particularly so in people in good physical condition.

RESPONSES TO CHRONIC LOADS

Chronic volume load

Abnormal conditions such as valvular insufficiency, severe anaemia and hyper-thyroidism, cause a sustained increase in cardiac output, leading to a chronic volume load. Dilatation of the left ventricle is one response to extra work. The increased work allows normal oxygen delivery, but cardiovascular efficiency is reduced. Myocardial oxygen requirements are increased at rest and greatly increased with exercise. Hence coronary oxygen delivery must rise with increased coronary flow even at rest and this is achieved by arteriolar dilatation. That is, coronary reserve capacity is reduced and, in total, exercise tolerance is diminished.

The heart can compensate for a chronic volume load and venous oxygen reserve is not utilized. The cardiac output is increased by a larger stroke volume and at rest the rate is usually still normal. However, ventricular diastolic and residual volumes are larger than normal and cardiac reserve at rest is diminished. With exertion the heart must increase its rate to raise cardiac output, using up remaining reserve.

Chronic pressure load

The heart is chronically subjected to greater pressure than usual in arterial hypertension and aortic stenosis. At rest, cardiac output and arterio-venous oxygen difference are not disturbed. However, the myocardium must develop more tension during systole to eject a normal volume into the aorta. The useful work required is much greater, even at rest, as is the total energy expended by the heart during exercise. In response, the muscle mass becomes larger, necessitating delivery of more oxygen. This requires a higher coronary flow, using up coronary reserve at rest. Later in the course of hypertension, the

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left ventricle dilates. Decompensation ultimately occurs when the total reserve of coronary flow is inadequate to maintain adequate oxygen supply to the myocardium.

Congestive Heart Failure

Cardiac function remains compensated as long as the cardiovascular reserve capacity suffices for the amount of physical activity undertaken.[2] More strenuous work or exercise brings on shortness of breath, fatigue, and a pounding heart beat.

When the cardiovascular reserve capacity is depleted, the maximum sustainable cardiac output becomes limited. A larger proportion of the available oxygen is extracted by tissues and the arterio-venous oxygen difference increases. The most striking finding is severe limitation of exercise tolerance.

The first evidence of congestive failure is the decompensating point for the gradual reduction in muscle contractility. There is also a loss of mechanical efficiency of contraction. The heart in failure does not use the energy from its substrates as efficiently as the normal heart.

Failure of both sides of the heart can occur separately or in sequence, each having different symptoms and signs.

Left ventricular failure

Cardiac output below normal at rest indicates chronic failure of the left ventricle. Reduced blood flow through organs and tissues facilitates greater than normal oxygen extraction at rest. The increased oxygen consumption of even mild exertion further reduces venous oxygen content, with discernible symptoms.

Reduction of blood flow through muscle leads to weakness and fatigue and reduction of flow through the splanchnic bed gives rise to gastrointestinal symptoms.

By far the most significant effects of left ventricular failure are pulmonary hypertension with pulmonary congestion. The increased volume of blood in pulmonary vessels with the increased pressure distends capillaries and veins. Interstitial fluid collects between capillary and alveolar walls and impairs oxygen transfer. Overt pulmonary oedema is due to fluid collection in the alveoli themselves. Bronchial congestion stimulates mucus production and this leads to cough. Oedema of the bronchial mucosa increases resistance to air flow, resulting in "cardiac" asthma.

Dyspnoea on exertion with rapid shallow breathing is the most common symptom. This has been ascribed to reflexes from vascular distension, along with increased rigidity of the lungs and the impairment of gas exchange by interstitial fluid. Orthopnoea develops later, due to fluid from dependent parts of the body entering the vascular compartment on lying down.

Right ventricular failure

Failure of the right ventricle may result from chronic pressure load, usually after left-sided failure with pulmonary congestion and hypertension. Other causes leading to right heart failure alone include pulmonary valvular stenosis, mitral stenosis or pulmonary disease. Since the right ventricle is an efficient pump it seldom fails from a volume load alone.

Right ventricular decompensation produces signs rather than symptoms. Such signs are peripheral oedema and systemic venous engorgement. Tissues with the greatest venous distensibility are enlarged. These are central and peripheral venous channels, the splanchnic vascular bed, liver, spleen and kidneys. The enlarged, palpable, tender liver is typical and is due to passive venous congestion. The congested small peripheral veins and increased venous pressure are easily detected, even in the jugular vein.

Peripheral oedema, particularly in dependent parts, increases in the erect position and disappears on lying down. The patient confined to bed develops sacral oedema.

Effusions into serous cavities occur in the later stages of right-sided failure with ascites, hydrothorax, and hydropericardium. No correlation has been found between the degrees of oedema and of ascites, possibly because interstitial fluid has a low protein content (0.5 per cent) while ascitic fluid contains 5 to 6 per cent of protein.

Another aspect of congestive failure and oedema is that the body retains more fluid than normal and extracellular fluid accumulates. The retention of salt and water by the kidneys is likely a response to multiple effects of decreased cardiac output.

Diagnosis of Incipient Failure

Frank cardiac failure is easily recognized. A patient booked for an elective operation is not likely to be untreated and in overt failure. How-

ever, patients with incipient failure[3] and reduced cardiovascular reserve may slip by the referring doctor and be booked for operation.

Symptoms

The anaesthetist may identify the patient bordering on failure by these symptoms: (*a*) cough, particularly at night and when recumbent; (*b*) restlessness at night and insomnia; (*c*) restlessness and vague irritability during the day; (*d*) fatigue and general weakness without obvious cause; (*e*) abdominal discomfort and pain, indigestion, due to collection of fluid; (*f*) perspiration at rest from the increased sympathetic activity; (*g*) rapid and shallow breathing at rest.

Signs

On examination these signs may be present: (*a*) sinus tachycardia without other evident cause; (*b*) wheezing, due to the pulmonary congestion; (*c*) pulsus alternans: every other systolic peak is reduced, indicating poor left ventricular function; (*d*) distended neck veins: the internal jugular vein may be palpable; (*e*) gallop rhythms: these are diastolic events, with S3 heard during rapid ventricular filling and S4 related to atrial systole.

Other aids in diagnosis of incipient failure

The electrocardiogram may reveal left ventricular hypertrophy, intraventricular conduction disturbances, previous infarction, presence of ischaemia, or rhythm and rate disturbances.

The chest roentgenogram may show the butterfly pattern of overt failure, which is really intra-alveolar oedema. An earlier sign is the presence of Kerley-B lines which are fine horizontal lines in the costophrenic angles due to increased pulmonary venous pressure.

Treatment of Cardiac Failure

The local medical man-power situation determines whether the anaesthetist is involved in treating cardiac failure preoperatively. Certainly anaesthetists must know how to manage these patients before and during operation.

All but the most dire emergencies must have operation deferred for several days, to allow effective reversal of the decompensated cardiac function.

Digitalis[4] is the mainstay of management, to improve myocardial performance. Digoxin may be given intravenously in urgent situations to produce its full effect in two to three hours. One guideline is to administer 0.9 mg per m^2 of body surface intravenously, in divided doses.

Diuretics[5] are the other group of drugs indicated, to reduce the accumulation of body water and return blood volume to normal. Massive diuresis causes a major loss of potassium, which itself may induce cardiac arrhythmias. One must also be careful not to produce dehydration and a contracted blood volume, which results in unstable blood pressure during anaesthesia. In other words, it is better to tend to underdigitalize patients and to limit diuresis immediately before operation.

Perioperative Problems in Cardiac Patients

The patient with a weakened myocardium, even though compensated, has these added risks: (*a*) with an enlarged blood volume there is more likelihood of fluid and electrolyte abnormalities such as hyponatraemia, hypokalaemia and decreased serum osmolality; (*b*) there is an increased incidence of thromboemboli, and particularly pulmonary emboli, from the reduced blood flow; (*c*) ventilatory problems occur more often with associated respiratory infections and pneumonia; (*d*) impaired liver and renal function alter the rate of inactivation and elimination of anaesthetic drugs.

The risk is that with the added stress of anaesthesia and operation the compensated heart is pushed into congestive failure. Cardiac reserve is partially used up at rest. With decreased cardiac performance adrenergic activity is increased, possibly associated with peripheral vasoconstriction. Over a long period the heart compensates both by hypertrophy and by dilatation of atrial and ventricular cavities. As a later attempt to remain compensated the heart rate is chronically rapid.

Patients in this stage of their disease may go into acute failure from the operative stress.

Factors Precipitating Acute Failure

The whole gamut of events from before operation through the post-operative period can contribute to acute decompensation. The important factors are: (*a*) fear, apprehension, and preoperative pain, all increasing myocardial output

and oxygen requirements; (*b*) anaesthetic agents, as myocardial depressants and peripheral vasodilators;[6] (*c*) controlled ventilation, decreasing venous return, and cardiac output;[7] (*d*) fluid overload during operation: due to reduced ability to handle it an average amount may be excessive; (*e*) fluid retention post-operatively because of inadequate kidney function; (*f*) reduced body activity after operation with diminished ventilation and general circulation of blood.

The real problem in these patients is their relative inability to increase cardiac output to meet the increased metabolic demand imposed by all aspects of the operation.

The risk of precipitating acute failure[3] is particularly high in patients with rheumatic disease and mitral stenosis, atherocalcific disease such as aortic stenosis and chronic obstructive lung disease.

The Patient Who Has Had an Infarction

Several large retrospective studies have clearly documented the significant risk of anaesthesia and operation in patients with a previous infarction.[8-10] Both Tarhan[8] and Plumlee[9] found a 0.13 per cent incidence of postoperative infarction in two series totalling over 50,000 cases. Tarhan's group[8] found the incidence of post-operative infarction in patients with previous infarction to be 6.6 per cent. This is 50 times greater than the infarction rate for patients who had not had an identified previous infarction. Steen, Tinker and Tarhan[10] subsequently reported on over 73,000 patients.[11] Again, the reinfarction rate post-operatively was 6.1 per cent, with the rate related to site of operation (higher for thorax and upper abdomen), and to length of operation. They showed also that post-operative infarction carried a mortality that averaged 69 per cent.

The other pertinent information[8] is that post-operative infarction occurred in 37 per cent of patients operated upon within three months of a previous infarction. This decreased to 16 per cent in the second three months and remained constant at about 5 per cent from then onward.

The implications are clear. No one should have an elective operation in the six months after an infarction. After that time the risk of reinfarction remains at 5 to 6 per cent. Patients with previous infarction need to be managed even more carefully during operation than patients with coronary artery disease who have not frankly infarcted.

Pre-operative Preparation of the Cardiac-Patient

The principles of proper preparation for anaesthesia and operation of the patient with reduced cardiac reserve include:

(*a*) take the time to obtain an adequate history relative to the cardiac status;

(*b*) determine the physical findings and E.C.G. report;

(*c*) make an assessment of cardiac reserve capacity and the extent of myocardial damage;

(*d*) ensure that the appropriate treatment for failure and arrhythmias has been given;

(*e*) give digitalis and diuretics if overt or incipient failure is present;

(*f*) get pulmonary function studies and start breathing exercises (e.g. the incentive spirometer);

(*g*) stop the patient from smoking; even several days of abstinence will improve the lungs;

(*h*) correct electrolyte abnormalities, anaemia and clotting problems;

(*i*) sedate effectively with heavy doses. Morphine is an excellent premedicant[11] to reduce apprehension and to allow more of the blood volume to be pooled peripherally, to reduce pulmonary blood volume.

Summary

The patient with chronic disease of the myocardium must be evaluated perceptively as to the extent of abnormal function. The degree of reduction of cardiac reserve must be estimated and all efforts must be made to improve cardiac performance before operation; then a carefully managed anaesthetic is needed to assure a smooth operative and post-operative course.

Résumé

L'auteur présente un rappel de la physiologie fondamentale circulatoire et cardiaque et sa relation avec les modifications pathologiques qui surviennent dans différentes maladies du cœur.

La capacité de réserve cardiaque comprend la réserve en oxygène du sang veineux, la capacité d'augmentation de la fréquence, la réserve de volume systolique, la capacité d'augmentation du travail myocardique, la réserve coronaire vasculaire et l'hypertrophie du muscle cardiaque.

Le cœur peut subir une surcharge chronique soit en volume, soit en pression. Surviennent

alors les changements compensateurs qui diminuent la réserve cardiaque et la tolérance à l'exercice.

Il y a insuffisance cardiaque congestive lorsque la capacité de réserve cardiaque ne rencontre plus les besoins de l'activité physique. La dyspnée et la tachycardie peuvent survenir même au repos. L'insuffisance ventriculaire peut être gauche ou droite, ou globale. L'insuffisance gauche se caractérise surtout par la congestion pulmonaire et la droite par l'oedème périphérique.

A son début, l'insuffisance se manifeste par la toux, la nervosité (diurne et nocturne), l'insomnie, la fatigue, les malaises abdominaux, la sudation et la respiration rapide et superficielle.

L'insuffisance franche se manifeste par la tachypnée, le wheezing, le pouls alternant et la distension des vaisseaux du cou.

A l'exception des cas urgents, en présence d'insuffisance cardiaque congestive, toute opération doit être différée jusqu'à l'amélioration du fonctionnement cardiaque par la digitale et les diurétiques.

A la période opératoire, les facteurs suivants peuvent précipiter la défaillance: l'appréhension, la douleur, les agents anesthésiques, la ventilation contrôlée, la surcharge et la rétention liquidienne.

Le vrai problème réside dans l'incapacité relative du cœur à augmenter son débit pour suffire à une demande métabolique accrue pendant et après l'opération.

Les malades ne devraient jamais subir d'interventions non urgentes dans les six mois qui suivent un infarctus du myocarde. Après six mois, le risque de réinfarctus post-opératoire tombe à environ six pour cent.

Les principes d'une préparation préanesthésique adéquate sont les suivantes:

(*a*) l'obtention d'une anamnèse complète, la recherche des signes physiques en rapport avec l'état cardiaque et l'électrocardiogramme;

(*b*) l'évaluation de la réserve cardiaque;

(*c*) le traitement de l'insuffisance et des troubles du rythme;

(*d*) l'évaluation de la fonction respiratoire, l'arrêt du tabac et l'initiation aux exercices respiratoires;

(*e*) la correction des débalancements électrolytiques et de l'hémoglobine;

(*f*) une sédation lourde.

Si on parvient à évaluer correctement la réserve cardiaque et améliorer la performance du cœur avant l'opération, une anesthésie signée par la prudence devrait assurer une évolution calme et sans problèmes.

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