THE CARDIAC RISK IN ANAESTHESIA AND SURGERY

By GRAHAM W. HAYWARD, M.D., F.R.C.P.

Assistant Physician, St. Bartholomew's Hospital, Physician, National Heart Hospital

The cardiac risk of anaesthesia and surgery is in most cases small and the operative mortality is very little higher in patients with heart disease than in patients without heart disease. The experience gained in recent years of operative treatment of mitral stenosis in patients with congestive failure and operations for relief of coronary insufficiency has shown that even direct operation on a damaged heart can be carried out safely. The strain on the heart of the operation itself is less than that imposed by even moderate physical exertion and if the cardiac patient has been free of symptoms suggesting myocardial insufficiency the operation is usually well tolerated, provided there is no sudden haemorrhage, prolonged hypotension or shock. The chief risks in the cardiac patient are hypoxia occurring either during induction of anaesthesia or at any stage of the operation and in the early post-operative period when chest infections, or excessive administration of fluids intravenously may precipitate heart failure and prolonged immobilization may cause thrombo-embolic complications.

The Anaesthetic Risk

The patient with heart disease is particularly susceptible to hypoxia and even a slight impairment of oxygen supply to the myocardium is badly tolerated, causing heart failure, increasing the risk of cardiac arrhythmias and sometimes causing sudden death from ventricular fibrillation. The risk is greatest in patients with coronary artery disease and in certain types of heart disease where sudden death is common.

In coronary disease the oxygen supply to the myocardium may be adequate at rest but if hypoxia should occur during anaesthesia acute coronary insufficiency with cardiac infarction or sudden death due to ventricular fibrillation may be caused. The danger of hypoxia is generally recognized in patients giving a history of angina pectoris or previous cardiac infarction but it is not always remembered that there may be extensive coronary narrowing in patients who are free from symptoms and in whom the electrocardiogram is normal. An extensive system of intercoronary anastomoses has developed which enables the coronary circulation to be maintained under ordinary conditions but this collateral circulation is particularly vulnerable to hypotension and hypoxia.[1] This occult coronary disease is impossible to detect clinically or with the electrocardiograph and the state of the peripheral vessels gives no indication as to the health of the coronary arteries so that the need for avoiding hypoxia in all patients is again emphasised.

Sudden death may occur in patients with aortic valvular disease, particularly aortic stenosis, syphilitic aortitis or aortic incompetence with involvement of the mouths of the coronary vessels and in heart block of all degrees. The cause of death is usually ventricular fibrillation and hypoxia should be particularly avoided in this group.

Cardiac Arrhythmias. Minor cardiac arrhythmias, usually ectopic beats, are common during anaesthesia but they are rarely serious. The more serious arrhythmias are cardiac arrest, due to reflex cardiac standstill during intubation in the induction period, or ventricular fibrillation caused by hypoxia and hypercapnia. These two types of cardiac arrest are clinically indistinguishable as no heart sounds can be heard and the blood pressure has fallen to zero. There will usually be no time for electrocardiographic differentiation and immediate priority must be given to restoring the oxygen supply to the brain and heart within two to three minutes by means of positive pressure breathing with 100 per cent. oxygen and cardiac massage which if efficiently carried out can maintain a blood pressure of 50 to 70 mm. Hg. Massage alone may cause return of normal rhythm but if it does not, intracardiac adrenalin (0.1 to 0.3 ml. of 1:1000 solution) should be given in cardiac standstill. If ventricular fibrillation is recognized by inspection or palpation of the ventricle the electric defibrillator is the most effective way of restoring normal rhythm or if this is not available procaine amide (100 to 200 mg.) should be injected into the auricle and cardiac massage continued.[3]

Frequently recurring ectopic beats in patients

with heart disease may be a precursor of ventricular tachycardia or fibrillation and in these cases, in addition to correcting any hypoxia which may be present it is advisable to give procaine amide hydrochloride intravenously (200 mg. initially followed by 100 mg. a minute up to a total of 1 g.)

These anaesthetic risks are in nearly all cases preventable and with a competent anaesthetist and modern anaesthetic techniques should rarely occur except in direct cardiac surgery when they may be unavoidable. It is particularly important in the cardiac patient to give adequate premedication as anxiety and a nervous tachycardia may precipitate heart failure especially in the patient with valvular disease. The orthopnoeic patient should be anaesthetized propped up but when fully anaesthetized may be placed in the recumbent position. The breathing of the orthopnoeic patient usually improves under anaesthesia, the signs of pulmonary congestion decreasing as a result of decreased return of blood to the right heart with muscular relaxation, improved oxygen supply to the heart muscle and a damping down of the reflexes which may contribute to the production of pulmonary oedema.[4]

The Surgical Risks

The chief risk to the heart from the operation itself is hypotension, whether caused by shock, haemorrhage or hypotensive drugs as the amount of coronary blood flow is determined largely by the level of blood pressure in the aorta.[5] Prolonged hypotension which lowers the coronary blood flow will decrease the oxygen supply to the heart muscle and may produce all of the early effects of hypoxia which have been discussed previously and in the patient with heart disease may cause cardiac infarction which will become apparent in the early post-operative period or cause heart failure.

In assessing the risks of drug-induced hypotension it should be remembered that the patient under general anaesthesia will tolerate a degree of hypotension which would cause severe effects in the conscious patient. The metabolic needs of the body are lowered under anaesthesia and there is decreased peripheral resistance due to widespread vasodilatation so that the work of the heart is decreased. A much lower level of coronary blood flow may under these circumstances be adequate for the needs of the heart muscle and provided there is full oxygenation of the blood no signs of coronary insufficiency will necessarily appear.

In spite of these circulatory adjustments caused by anaesthesia the patient with heart disease should not be exposed to the risk of hypotension and every effort should be made to keep the systolic pressure over 100 mm. Hg. particularly in patients with known coronary disease. Although the incidence of complications due to controlled hypotension has not been as high as was thought probable a few years ago the potential risk of this method of treatment remains and it should be used in patients with heart disease only if the added safety of the surgical procedure under hypotension makes the risk worth while.

The use of hypothermia to reduce cellular metabolism and to enable the circulation to be stopped for a short period is still in the experimental stage and this method is at present used chiefly in the surgery of the heart itself. It seems probable that when the technique of inducing hypothermia has been perfected this method may have a wider application in surgery of the brain and elsewhere where a bloodless field would be an advantage. The cardiac risk of this method is ventricular fibrillation which may occur when the body temperature falls to 26°C. and the method of preventing or treating this arrhythmia under these circumstances required much further study.[6]

Post-operative Complications

The patient with heart disease who has given rise to no anxiety from the anaesthetic or surgical points of view during the actual operation may get into difficulties during the early post-operative period and post-operative complications which are not serious in the patient with a normal heart may precipitate congestive failure in the cardiac patient. The recognition of early heart failure is not always easy in the post-operative period and examination of the heart itself may not produce the clue although the appearance of a triple rhythm or the persistence of an arrhythmia such as auricular fibrillation may be helpful in this respect. Laboured breathing which is relieved when the patient is propped up is often the earliest manifestation of left ventricular failure and at this stage there may be no clinical signs of pulmonary congestion although later râles will appear at the lung bases. Failure of the right heart can be recognized by an increase in the venous pressure, judged by inspection of the neck veins, as it is usually impossible to detect slight enlargement of the liver after an abdominal operation and oedema of the legs or over the sacrum may be due to causes other than heart failure.

If heart failure appears for the first time after operation, some definite cause for the failure should be sought as more rational therapy may then be possible. The precipitating cause is usually to be found outside the heart itself, although anaemia may be responsible for weakness of the myocardium, prolonged hypotension may have caused cardiac infarction or acute subendocardial ischaemia which only manifests itself when the

patient recovers from the anaesthetic, or a rapid ventricular rate due to uncontrolled auricular fibrillation may have induced failure. The commonest precipitating causes of heart failure during this period are:

1. *Chest complications*, either collapse of a lobe or chest infection. These embarrass the circulation because of hypoxia, tachycardia and the increased respiratory effort due to frequent cough and bronchospasm.

2. *Excessive Administration of Intravenous fluids.* Particular care should be taken to avoid giving too large amounts of sodium containing fluids intravenously in patients with heart disease as salt retention may easily precipitate heart failure. If the fluid is given too rapidly, acute overloading of the circulation may result and may be recognized by the occurrence of a frequent cough due to pulmonary congestion and an increase in the jugular venous pressure. The correction of anaemia by transfusion may cause the same overloading of the circulation but even in the presence of heart failure the anaemia may safely be corrected by repeated small transfusions of packed cells and the beneficial effect on the heart muscle will outweight the risks of transfusion. Fresh blood is probably better than ' bank ' blood for transfusion in cardiac patients as stored blood may contain an excessive amount of potassium in the plasma.

3. *Postural Changes.* The cardiac patient is best nursed in the propped up position when the work of the heart is lessened, chest movements are better and there is less hypostatic congestion of the lungs. Prolonged recumbency and particularly elevation of the foot of the bed which may be necessary in pelvic operations are positions which are badly tolerated by the patient with incipient heart failure, breathing being impeded by the headward displacement of the diaphragm and by pooling of blood in the dependent thorax.

4. *Cardiac Arrhythmias.* The commonest arrhythmia which appears during operation and persists afterwards is auricular fibrillation. The ventricular rate under these circumstances is often rapid as the patient is not under the influence of digitalis and the rapid heart rate may precipitate failure. This usually responds rapidly to control of the ventricular rate with digitalis and if the arrhythmia persists the decision as to the use of quinidine to restore normal rhythm can be made later when the patient's general condition has improved.

5. *Thrombo-embolic Complications.* The patient with heart disease not infrequently develops phlebothrombosis or thrombophlebitis in the leg or pelvic veins if immobilized in bed for any reason, and if in addition the factors of anaemia and infection are added as a result of operation the risk of this complication and pulmonary embolism is increased. Active leg exercises and early mobilization of the patient will minimize the risk of thromboembolism, but if it should occur femoral vein ligation to prevent pulmonary embolism should be considered if there are localizing signs in the legs or alternatively the use of anticoagulant drugs if these are not contraindicated surgically.

6. *Subacute Bacterial Endocarditis.* This complication of surgical treatment occurs in patients with valvular heart disease, commonly rheumatic in aetiology but sometimes syphilitic or arteriosclerotic or acyanotic congenital heart disease (ventricular septal defect, patent ductus arteriosus, coarctation of the aorta or bicuspid aortic valve). The precipitating cause is most commonly dental extraction, the streptococcus viridans being liberated into the blood stream at the time of extraction and lodging on the heart valves to set up the typical infective vegetations. Although bacterial endocarditis may follow any operation which is associated with infection, operations on the prostate or rectum seem particularly prone to cause this complication often in patients whose only sign of heart disease is a mitral systolic murmur of variable intensity and quality. The organism in these cases is commonly the streptococcus faecalis which is insensitive to penicillin alone but sensitive to large doses of penicillin (10 mega daily) together with streptomycin (2 g. daily). The obvious clinical manifestations of bacterial endocarditis may be delayed for three to six weeks after operation but any unexplained pyrexia should lead to this diagnosis being considered in a patient with signs of valvular disease and multiple blood cultures should be taken. Any operation on a patient with valvular or congenital heart disease which is likely to cause a bacteraemia should be carried out under penicillin cover to prevent bacterial endocarditis occurring and steps should be taken to ensure an adequate penicillin blood level during the period of surgical intervention.

Practical Management

With adequate premedication, a smooth induction of anaesthesia and the avoidance of hypoxia or hypotension during operation most cardiac patients will tolerate surgery well. There is no need for the routine administration of digitalis to all patients with heart disease and the use of this drug should be restricted to patients with auricular fibrillation or congestive failure. If there are signs of congestive failure before operation, anaesthesia and surgery should if possible be postponed until the failure has been treated, as in this way the risk to the patient will be considerably reduced. A period of medical treatment with rest,

low sodium diet, digitalis and injections of mersalyl will improve cardiac efficiency and make the operative and post-operative course smoother. If emergency surgery is essential and there is no time for adequate pre-operative treatment of heart failure the operative risk is increased although even then it is often surprising how well these patients do. Under these circumstances the patient should be digitalized rapidly, using digoxin 1.5 to 2 mg. in a single oral dose which will produce an effect in three to four hours. If greater speed of action is needed, 1 mg. of digoxin should be given slowly intravenously, but the intravenous route must be avoided if the patient has been receiving digitalis by mouth previously. These patients should be anaesthetized propped up, but may be placed flat when the laboured breathing of heart failure has been relieved by anaesthesia.

In the post-operative period they should be nursed propped up, digitalis should be continued and mercurial diuretics should be started early. Particular care should be taken to prevent chest infection and leg exercises should be started early to minimise the risk of thromboembolism.

The aetiology of the heart disease is in general less important than the pre-operative functional status of the patients in assessing the risk of anaesthesia and surgery. However, in certain types of heart disease, particularly aortic valvular disease or heart block, the risk of difficulties under anaesthesia is increased even though the patient has no symptoms. Patients with known coronary disease, with a history of angina pectoris or previous cardiac infarction are especially susceptible to hypotension or hypoxia but if these two factors can be avoided operation is usually well tolerated. A history of recent cardiac infarction within the previous three months, or of increasing severity of anginal attacks, should cause all except emergency operations to be postponed. The particular risk in these patients is the occurrence of serious ventricular arrhythmias but if operation is essential the risk can be decreased by giving quinidine sulphate 5 gr. three times a day pre-operatively and continuing the drug during the early post-operative period.

Prostatectomy in the elderly patient with hypertension and arteriosclerotic heart disease carries a mortality nearly three times as high as in the patient with a normal heart[7] and the increased risk should be taken into consideration when deciding the nature of the surgical treatment Partial thyroidectomy for hyperthyroidism can be safely carried out in patients with heart disease as with adequate pre-operative treatment with thiouracil drugs the operation can be done when the patient is in the euthyroid state. If however, the heart disease is serious, treatment with radio-active iodine is preferable.

The use of local instead of general anaesthesia for cardiac patients has little to recommend it except for minor procedures such as dental extraction and a properly administered general anaesthetic is both safer and pleasanter for the patient.

REFERENCES :

1. BLUMGART, H. L., SCHLESINGER, M. J. and DAVIS, D. (1940), *Amer. Heart J.*, **19**, 1.
2. DEUCHAR, D. C. and VENNER, A. (1953), *Brit. med. J.*, **ii**, 134.
3. McMILLAN, M. K. R. (1955), *Brit. med. Bull.*, **11**, 229.
4. HAYWARD, G. W. (1955), *Brit. med. J.*, **i**, 1361.
5. ECKENHOFF, J. E., HAFKENSCHIEL, J. H. and LANDMESSEN, C. M. (1947), *Amer. J. Physiol.*, **148**, 582.
6. DELORME, E. J. (1955), *Brit. med. Bull.*, **11**, 221.
7. MORRISON, D. R. (1948), *Surgery*, **23**, 561.

Bibliography continued from page 91—*E. G. McQueen, M.B., M.R.C.P., and F. H. Smirk, M.D., F.R.C.P.*

GRIMSON, K. S., TARAZI, A. K. and FRAZER, J. W. (1955), *Circulation*, **11**, 733.

HAMILTON, M. DOYLE, A. E., McQUEEN, E. G. and SMIRK, F. H. To be published.

HOOBLER, S. W. (1954), *Amer. J. Med.*, **17**, 259.

HUGHES, W. M., MOYER, J. H. and DAESCHNER, W. C., Jr. (1955), *Arch. intern. Med.*, **95**, 563.

KEITH, N. M., WAGENER, H. P. and BARKER, N. W. (1939), *Amer. J. Med. Sci.*, **197**, 332.

KLOHS, M. W., DRAPER, M. D. and KELLER, F. (1954), *J. Amer. chem. Soc.*, **76**, 2843.

McQUEEN, E. G. and BLACKMAN, J. G. (1955), *Proc. Univ. Otago med. Sch.*, **33**, 5.

McQUEEN, E. G., DOYLE, A. E. and SMIRK, F. H. (1955), *Circulation*, **11**, 161.

MORRISSEY, D. M., BROOKES, V. S. and COOKE, W. T. (1953), *Lancet*, **i**, 403.

MULLER, J. M., SCHLITTLER, E. and BEIN, H. J. (1952), *Experientia*, **8**, 338.

RESTALL, P. A. and SMIRK, F. H. (1950), *N.Z. med. J.*, **49**, 206

SCHLITTLER, E., ULSHAFER, P. R., PANDOW, M. L., HUNT, R. M. and DORFMAN, L. (1955), *Experientia*, **11**, 64.

SCHOTTSTAEDT, M. F. and SOKOLOW, M. (1953), *Amer. Heart J.*, **45**, 331.

SCHROEDER, H. A. (1952), *Circulation*, **5**, 28.

SMIRK, F. H. (1952a), *Proc. Univ. Otago med. Sch.*, **30**, 13.

SMIRK, F. H. (1952b), *Lancet*, **ii**, 1002.

SMIRK, F. H. and CHAPMAN, O. W. (1952), *Amer. Heart J.*, **43**, 586.

SMIRK, F. H., DOYLE, A. E. and McQUEEN, E. G. (1954), *Lancet*, **ii**, 159.

SMIRK, F. H. and HAMILTON, M. (1955), *Proc. Univ. Otago med. Sch.*, **33**, 11.

SMIRK, F. H. and McQUEEN, E. G. (1955), *Lancet*, **ii**, 115.

SMITHWICK, R. H. (1955), *J. Chronic Dis.*, **1**, 477.

TAYLOR, R. D., CORCORAN, A. C., DUSTAN, H. P. and PAGE, I. H. (1954), *Arch. intern. Med.*, **93**, 705.

TRAPOLD, J. H., PLUMMER, A. J. and YONKMAN, F. F. (1954), *J. Pharmacol.*, **110**, 205.

VAKIL, R. J. (1949), *Brit. Heart J.*, **11**, 350.