General discussion

IRVING. There is an undoubted case for antibiotics in septicaemic shock. In cases of cardiogenic shock and traumatic haemorrhagic shock, where there is no obvious bacterial involvement initially, does Dr Pentecost consider that wide spectrum coverage is necessary, because of the undoubted inhibition of the reticulo-endothelial system which has been shown to occur in experimental animals?

PENTECOST. I have no idea, but since the mortality is 85-90% in cardiogenic shock, perhaps we ought to use antibiotics in massive doses.

BAU. Patients with haemorrhagic shock who have been given large amounts of bank blood, sometimes deteriorate rather than improve. There are various reasons for this. For example, large loads of citrate and potassium, low levels of platelets and Factor VIII may have adverse effects (Basu, 1968). The lysis of aged red cells will release thromboplastins and may give rise to a form of defibrinogenation syndrome. It is better to give fresh blood to these patients.

It is possible that low tissue perfusion in shocked patients causes stasis and intravascular clotting; this then stimulates a secondary fibrinolytic response. Study of a small number of pregnant women with non-haemorrhagic shock showed that all had low levels of fibrinogen, platelets and Factor V. This hypocoagulability prevents further intravascular clotting, and does not require treatment with fibrinogen. The level of fibrinogen-degradation products in these patients was also high suggesting adequate fibrinolytic response. There is little to be gained by giving streptokinase. Administration of a fibrinolytic inhibitor such as Trasylol may inhibit a protective response and be harmful. The main emphasis should be on prevention of the development of coagulation disturbances by improving tissue perfusion.

LITTON. If you are going to treat coagulation defects and fibrinolysis you must know what you are doing, because it is easy to do more harm than good. You must have very careful laboratory monitoring.

WALTERS. Mr Pearson yesterday with peritonitis and E. coli septicaemia who responded well to digoxin after transfusion to a normal CVP had failed.

PIGOTT. Levene of New York told me recently that in low perfusion states the hepatic vein potassium concentration reaches high levels. Does Dr Schröder consider this to be true, and if so, would Dr Pentecost be worried by hepatic vein concentrations of 19-20 mEq/l, and would he advocate the use of digitalis and insulin as Levene does?

SCHRÖDER. We measured glucose and free fatty acids but not potassium unfortunately.

PENTECOST. There is no evidence of a rise in potassium. There was a letter from Black in The Lancet about a year ago (Black & Ralston, 1967) in which he was intrigued by the suggestion that glucose, potassium and insulin might be of value in the management of myocardial infarction, but was unable to find evidence of a generalized hypokalaemia. We have not been able to find increased loss of potassium from the body in myocardial infarction. We did a controlled trial of intravenous glucose, potassium and insulin in the management of infarction and could detect no benefit.

McGowan. In many centres patients are carefully monitored by ECG and if they were suffering from potassium intoxication it would soon be apparent.

SCHRÖDER. In the early stages of myocardial infarction urinary excretion of potassium is increased. The mean arterial potassium concentration in the majority of patients is decreased. Later there is a lowered potassium output, perhaps due to an aldosterone effect.

PENTECOST. We measure 24-hr collections of urine and might have missed this transient phase.

PLEDGER. The problems in managing oligoaemic shock have been underplayed. How much Dextran 70 are people prepared to give? No one has mentioned Shire's work on giving large quantities of Ringer-lactate solution. Does warming blood or adding sodium bicarbonate have a place in management?

GRUBER. It is now generally agreed that 1.5 g/kg body weight of Dextran 70 can be given in 24 hr. This
means for a 70-kg man 1-1½ litres to begin with. If the patient continues to bleed, however, Dextran will also be lost, and more will have to be given during the next treatment period. Much larger amounts have been given, especially in burns, with apparently no difficulties from bleeding.

With regard to Shire's work and the use of large amounts of electrolyte solution I believe his theory is based on erroneous assumptions and false measurements (see references in my paper). The question is where are these large quantities of electrolytes lost? Why should there be 8 litres of fluid deficit? It is still in the body but cannot be measured by conventional techniques involving equilibration time.

HOPKIN. If more than 2 units of blood are given it should be warmed and a little calcium added. The most dangerous thing is the likelihood of hypothermic cardiac arrest.

STEWART. Dr Pentecost, how do you administer metaraminol?

PENTECOST. Both doses of 2 mg are given as a bolus injection. Subsequently the blood pressure is kept at about 100.

STEWART. If you give a slow infusion—this is also true of adrenaline—arrhythmias reported after bolus injection are not seen.

Although it is agreed that tissue anoxia is the basic problem, there has been no comment about hyperbaric oxygenation. My limited experience of two patients is that it did no good. The haemodynamic situation was not studied, and we know for instance that increased oxygen can lead to a decrease in cerebral blood flow and the net result is very little difference in oxygen reaching the cerebral tissue. While not condemning it, I think it is another possibility to be looked at from the point of view of research.

MATHESON. Little attention has been paid to the particulate content of stored blood. If you try to force stored blood through a microfilter, enormous pressures are needed. This depends on storage at low temperature and is due to platelet aggregates. During transfusion this particulate matter is presumably filtered in the lungs. There is also the question of the function of the red cell. There has been recent interest in the relationship between red cell content of organic phosphates, particularly 2,3-diphosphoglycerate and ATP, and the ability of the red cell to transport oxygen. The red cell content of organic phosphates falls off very rapidly after storage. Little is known about the ability of stored red cells to carry oxygen when transfused.

These points, together with the risk of hepatitis, make it important to avoid blood transfusion if possible and to reserve it for major blood loss.

On the question of the transfusion of large quantities of added salt solution, as Dr Gruber said, a lot of work now disclaims Shire's earlier postulates on the basis of sulphate space measurements. But there is no doubt that Shire's animals did survive, and the reason may depend on the fact that saline transfusion does sustain the plasma volume for a longer time after haemorrhage than in the normal person. There is also the question of degree of haemodilution; and with extreme haemodilution the animals don't survive.

References

