JESSE PATT PRIZE IN SURGERY

1977 COMPETITION

Essay on the subject

"THE SURGEON AND SHOCK"

Submitted by:

Thomas P. Cripps
References in the text, indicated with a number thus: (1) are to be found on page...
I will not attempt in this essay to give a comprehensive review of shock; this has been done elsewhere for various aspects of the syndrome (1,2,3). Rather, I will try to give a survey of the subject with reference to surgery, and discuss various aspects of its management which seem to be of interest, pointing to some areas in which data is scarce or opinions diverse.

The first English language use of the word 'shock' was in 1743, in the translation of Henri François le Drian's second French edition of 'A Treatise of Reflections Drawn from Experience with Gunshot Wounds'; the translator used the word to convey the impression of a jolt or blow followed by progressive dizziness, loss of consciousness, and death. Since then the word has been used loosely and ambiguously in medical circles. For the purpose of this essay it will be defined as a syndrome associated with a state of inadequate tissue perfusion leading to generalized cellular hypoxia and vital organ damage. This definition does not preclude pressure on cardiovascular variables (e.g. hypotension, low cardiac output) and it precludes the hypotensive state of 'neurogenic shock' where tissue perfusion is adequate and causes the low blood pressure. Also excluded is the 'frightened surgeon syndrome', asphyxiation, O.v. de fibrillation and any other situation where the words 'shock' and 'surgeon' might be associated.

Shock may be of interest to the surgeon for several reasons. He may cause it, cure it, carry on despite it, treat it, prevent it - or even choose to ignore it. The simple question 'how frequent is shock in surgical practice?' has proved impossible to answer, at least for this writer. The closest approximation comes from the Glasgow Shock Team where one can guess that over the last 200 patients developed the shock syndrome (4) assuming the turnover of surgical cases 2,000 per annum at Glasgow Western Infirmary 1971-1972.) The complete absence of citations on this subject in Index Medicus is very revealing; the response of several surgeons to the same question is equally so! Whatever the incidence of shock, a mortality of 56% in the above study demands that it must be diagnosed and treated correctly and quickly when a patient develops the syndrome. Here one is not purely in the territory of the surgeon - but an understanding of the pathophysiology of shock as a rational basis for therapeutic measures (including surgery) is essential, and the subject is discussed below.
The fundamental inadequacy of tissue perfusion may be looked at with the aid of an elementary model of the vascular system (Figure):

**Figure: Model of Vascular System**

This comprises a two-chambered pump (the heart), an arterial system of high capacitance, with an arterial blood pressure, the tissues with variable input and output resistances and in parallel with a shunt, and finally the venous system of variable capacitance filling the heart with a central venous pressure; into this is put the blood volume. Shock could be due to failure of the heart to pump enough blood, or bypassing of the tissues by an otherwise sufficient cardiac output (shunting). The shunt could be anatomical or physiological—a subject which will be discussed later.

1. Failure to maintain cardiac output may be due to insufficient input with a low right atrial pressure—caused by absolute hypovolaemia or an increase in venous capacitance (relative hypovolaemia) or due to pump failure. The latter could be caused by left or right sided myocardial failure, infection, damage to the valves, septal defects, etc., or a pulmonary embolus. Alternatively, the problem might be an arrhythmia. Since all these affect either stroke volume or heart rate—the product of which is cardiac output—they can all potentially cause a low tissue perfusion state.

Irrespective of its cause, a low cardiac output state as above tends to have the same systemic consequences. Since:

\[
\text{BLOOD PRESSURE (B.P.)} = \text{CARDIAC OUTPUT (C.O.)} \times \text{PERIPHERAL RESISTANCE (P.R.)}
\]

a low c.o. tends to reduce B.P. and this induces vasocostriction via the baroreceptor reflex, increasing P.R. and thus tending to correct the blood pressure. There is at the same time sympathetic constriction of the low pressure capacitance vessels tending to increase venous return, stroke volume, and thus c.o. and B.P. Unfortunately this reflex vasocostriction (which ultimately involves
all organs except the brain and heart) reduces tissue perfusion, adding to the low flow initially causing it. Unless the pathology causing the latter is corrected or stabilised, decompensation occurs, a positive feedback loop is initiated, and the situation becomes progressively worse. In shock, the vasoconstriction and its sequelae are probably not entirely reflex in nature; current thinking in the field (3) is that a vasoactive 'shock factor' of unknown origin is also present.

2. Shunting may be present. A low C.O., but is clearest in high cardiac output states, of which septic shock is the most obvious. In the presence of a local focus, or systemic injection, a high or normal C.O. is associated with hypotension, tissue hypoxia and progressive metabolic acidosis - the latter being masked to some extent by an initially hyperventilation and possibly a respiratory alkalosis. In terms of the figure, this represents a shunt, probably mainly a physiological one with blood flowing through low resistance tissue beds without adequate gas exchange - presumably due to the sick state of the cells concerned. The presence of actual anatomical shunts is debatable and the proponents of this concept are, at present, in disfavour. High C.O. shock tends to suddenly convert to the low C.O. state described above, due to failure of the heart, venous return, or possibly the 'shock factor'; hypovolaemia in septic shock may be due to fluid leaking out of the vascular space into inflamed tissues.

At a cellular level, a lowered perfusion can initially be tolerated by increasing venous oxygen desaturation. Eventually. however, anaerobic metabolism will occur with consequent lactic acid production (see later). Initially preconditioning vasoconstriction causes the capillary hydrostatic pressure to decrease relative to that of the interstitial fluid, and (by using the Starling Principle) extracellular fluid phase invades - effectively augmenting the blood volume, and diluting it! On the other hand, in the latter and more severe stages of shock, the pre-capillary sphincter relaxes whilst the post-capillary sphincter remain in spasm; here, flow occurs in the opposite direction and blood volume may be reduced. In low flow states, red cells and sludge within the capillaries thus - according to some - further reducing tissue flow, and possibly triggering intravascular coagulation or irreversible damage. There seems scant evidence for this; Grammark, uraemia, shock in transparent chambers implanted in man found the sludging highly reversible once a normal flow returned.

Whilst the haemodynamic principles involved in shock are relatively well understood, metabolic events are poorly elucidated. Experimental shock, if prolonged, becomes 'irreversible' - even when normal haemodynamic factors are restored, and this is paralleled by the high mortality in shock patients. Vital organs may suffer ischaemic necrosis, the kidney and gut being two important examples, and death can result from failure of these organs. However, there also seems to be
a systemic metabolic lesion, involving failure of intracellular energy production. Cellular ATP levels fall, the mitochondria become leaky, and the cell swells; it seems unable to utilise glucose, insensitive to insulin, and continues to metabolise anaerobically producing further lactate even in the face of restored blood flow. The proponents of steroid therapy in shock believe that lysosomes are ruptured at this stage, and their proteolytic enzymes released, adding to the metabolic chaos. It is not known at present what initiates these biochemical changes; the presence of metabolic 'poisons' has never been proved in man, and the role of endotoxin (in the past by analogy to cause shock, given unstatistics promiscuous) is unclear. Similarly the status of a significant lactic acidosis in shock is unknown; what is known is that lactic acid concentration in shock patients is inversely related to prognosis (5, 6). At present, the incidence of lactic acidosis in surgical patients - and the possible significances of this in relation to the development of shock - is entirely unknown.

The final outcome of shock may be recovery to a pre-shock state. It may be immediate death, or death may occur following a set of associated sequelae; these comprise organ dysfunction of which intracranial death of the heart or kidney are simple examples. More rarely gut, adrenal, or central infarction can be observed. Two features of interest are the fatality of the liver and lungs. One of the first liver functions to be lost in shock seems to be the ability to convert lactic acid to lactic acid; this, of course, contributes to the general metabolic disability of the patient, no doubt aggravating any acidosis. In addition, many other systemic complications of shock, for example disseminated intravascular coagulation, may be associated directly - or by default - with liver dysfunction.

'Shock lung' is a fascinating syndrome which has only been recognized since the early 1960s, and has a legion of other names to describe it, which help to place it (e.g. 'Vanishing Lung') suggested etiologies ('post-traumatic', 'post transfusion') and many other aspects of the disease. Perhaps the best term to use is Adult Acute Respiratory Insufficiency (ARI) since it describes the syndrome. Allegedly (7) one third of all post-surgery or major trauma deaths are from ARI. The 'typical' pathological picture is of grossly inflated lungs with focal haemorrhage, interstitial and alveolar oedema and, if the syndrome has been present for some days there is hyperline, non-caseous formation making the pathological 'indistinguishable from well advanced interstitial (card) pneumonia' (8). This picture is consistent with an increased capillary permeability and distant from pulmonary oedema secondary to heart failure. Pulmonary function is impaired by non ventilation of (oedematous) alveoli - leading to physiological shunting, an increase in the work of breathing secondary to decreased compliance, and finally an actual diffusion block occurs across the alveolar membranes. At this stage being function compatible with life is ready impossible to attain and patients usually die (often of an overwhelming lung infection at this stage). Paradoxically, the lung tissue seems to be
requiring itself by this stage, with granular pneumocytic proliferation and hyperplasia; these (type II) cells appear to multiply as a response to alveolar damage, and may later de-differentiate to type I cells (8). Unfortunately, the type II cells are ten to fifteen times the thickness of a type I cell, and the patient rarely survives.

The aetiology of A.R.I. is not known, and many people feel that there are several independent causes (thus the plethora of names). However, the advent of A.R.I. in the Vietnam war in the early 1960’s suggests that there was an aetiologic factor present then for the first time. Massive blood transfusions were first carried out then, and evidence has been presented by several groups (e.g. (14)) that a 40% blood filter during transfusion reduces the incidence; this is allegedly due to the prevention of microthrombotic debris from the transfused blood impacting in the pulmonary capillary bed. However, others disagree with this, and dispute the evidence. Teplitz (15) points out that there is no evidence in man that microthrombi can do this sort of damage, nor is there evidence of them (pathologically) in A.R.I. He also observes that the advent of A.R.I. also coincided with the birth of aggressive mechanical ventilation therapy and pulmonary intensive care. In 1966 one in ninety fatal burn cases died with hypoxia readings in the lung; over a third had massive pulmonary oedema; by 1966 few died with simple massive pulmonary oedema whilst the new A.R.I. pathology was common — yet the population of patients had not changed on isos. He postulates that the difference is simply that patients survive long enough to develop A.R.I. Whatever its pathology — be it oxygen toxicity, microthrombi, toxacaral or due to shock in any event — one factor is certain: A.R.I. is an intraoperative disease, and would not exist if the patient was not strenuously resuscitated.

If surgeons could prevent shock, it would be unnecessary to diagnose or treat it. Therefore it is appropriate to consider this aspect of the subject at this stage. Hypovolaemic shock is the commonest encountered by the surgeon, and may be the result of trauma (therefore impossible for the surgeon to prevent) or of surgery. Since any surgical procedure is liable to involve fluid loss, an adequate pre-operative should be obtained, and all fluid losses during surgery should be replaced. Special risk groups like those who have poorly wounds, or have lost blood or fluid for any other reason should be identified and made none-volaemic in advance. Similarly, any patient likely to cause, or predispose to septicaemia, like diabetes, immunosuppression or lung disease should be corrected as far as possible in the preoperative stage — not forgetting the value of chest physiotherapy. The state of the cardiovascular system should be considered, and the possibility that it will not withstand a major insult;
shock due to pulmonary embolism may be avoided by preoperative heparinisation, and there is possibly a place for antibiotic prophylaxis. Assuming that the incidence of shock is inevitable, recognition of this at the earliest possible stage will improve survival. Very close monitoring of high risk groups should be undertaken, possibly including the routine use of a CVP line.

In the operation, a good surgical technique should be used - creating the minimum of blood loss and trauma, and giving a minimum opportunity for bacteria to colonise the wound. It is essential that adequate drainage is provided. 90% of patients dying in septic shock between 1968 and 1971 in the Intensive Therapy Unit at Glasgow Western Infirmary had evidence of continuing urinary focus of infection at the time of death (12). A significant and disturbing feature of the operative care of patients is that the 'surgeon' and the 'anaesthetist' usually have differing views of the amount of fluid lost and required: a feature found both in the literature and by asking the relevant parties. Lack of communication at this point may be the first step along a road to disaster.

Too often the diagnosis of shock brings forth an alarm reaction on the part of the physician. To this he responds by giving fluids, vasopressors, buffers, and any other recently tested routine. No significant measurements are made and, by virtue of natural homeostatic mechanisms, the patient survives, a triumph for some one or other drug, or is killed. On the other hand, if the patient dies, shock is pronounced irreversible! Thus (13).

When the syndrome is recognised, it is mandatory to diagnose its cause and put right the pathology which is creating it. With the diverse causes mentioned above, no one form of treatment is universally correct: classic oligaeamic shock might present with confusion, hyperpnoea, hypotension, oliguria, peripheral cyanosis and cold, clammy extremities - a picture likely in any shock state caused by inadequate cardiac output, whilst septic shock may present with hyperpnoea, pyrexia, confusion, warm, dry extremities and (at first) a respiratory alkalosis. Many patients, however, do not obey the textbooks, and 40% of initial diagnoses may later prove incorrect. Initially, of course, a thorough clinical examination should be undertaken, and the temperature, respiration, skin perfusion, heart rate and rhythm, blood pressure and state of peripheral veins be noted. Based upon this history, a provisional diagnosis may be made at this stage, and first line treatment initiated - for example setting up a drip if hypovolaemia is suspected, and ventilatory support if it is necessary. However this must be only provisional therapy, blood should be withdrawn and sent for cross-matching, and normal haematological studies, in addition to urea and electrolyte determination. If there is any suggestion of sepsis, sepsis.
culture must be carried out. An E.C.G. and chest X-ray should be performed in order to exclude infection, arrhythmias, pulmonary embolism and left ventricular failure, and a central venous pressure (C.V.P.) line must be set up.

If the C.V.P. is less than -3 cm H₂O, this demonstrates the need for increased venous return, and the infusion is stepped up as a matter of urgency; the type of fluid replacement will be discussed later. Changes of C.V.P. during infusion will reflect the adequacy of fluid replacement, and a rising pressure to beyond the normal height indicates that the infusion should be reduced. Since C.V.P. represents the right atrial filling pressure, reflecting venous return and right ventricular function, it does not represent left ventricular function, and consequently if there is any suspicion of cardiac disease, or if the diagnosis is in doubt, the C.V.P. should not be used as an index of left atrial pressure. Instead, a Swan-Ganz balloon catheter should be placed up the pulmonary artery and used to measure pulmonary wedge pressure; a value greater than 18 mmHg would suggest left ventricular dysfunction. A continuous record of arterial pressure may be achieved with an arterial transducer, and urine flow can be determined by catheterisation of the bladder.

In difficult cases, the cardiac output should be determined, and thus peripheral resistance (equation on page 2.) At this point it must be stressed that B.P. does not give a reliable index of C.O. Finally, an arterial line should be used to determine the blood PₐO₂, PₐCO₂ and pH. Thus we may tabulate the clinical data available:

**Table: Parameters for Diagnosis of Shock.**

<table>
<thead>
<tr>
<th>General Clinical Examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial Pressure</td>
</tr>
<tr>
<td>Central Venous Pressure</td>
</tr>
<tr>
<td>Pulmonary Wedge Pressure</td>
</tr>
<tr>
<td>Cardiac Output</td>
</tr>
<tr>
<td>Peripheral Resistance</td>
</tr>
<tr>
<td>Haematocrit, Blood Haemoglobin etc.</td>
</tr>
<tr>
<td>Blood Urea and Electrolytes</td>
</tr>
<tr>
<td>Blood Glucose, Lactate etc.</td>
</tr>
<tr>
<td>Arterial pH, PₐO₂, PₐCO₂</td>
</tr>
</tbody>
</table>

Chest X-ray
E.C.G.
Urine Flow
Temperature
With all these, I should be possible to characterise the type of shock, and initiate correct therapy. Unfortunately, this is an 'ideal' scheme in that availability of time, equipment and highly skilled personnel is required; this may be found on the far side of the Atlantic, but in Britain such intensive investigation -- even in a specialised intensive therapy unit - has been found unpracticable. For example, unless specialist technicians are available, cardiac output measurements are of little use; they will reflect an inexperienced, unpractised hand, and be unreliable if available at all (13). Consequently more must be based upon clinical acumen and guesswork. An understanding of the limitations of any one method of investigation may aid this guesswork. If the overworked and under-equipped British surgeon realises this, he may be able, with basic physiological principles to aid him, to come to a helpful diagnosis. The C.V.P. is a useful, indeed an essential measurement, but it does not necessarily give a clear diagnosis!

As soon as its cause is established, shock should be treated appropriately: once this has been done, the other physiological abnormalities are liable to return to normal spontaneously, whilst merely correcting the latter is of no lasting value. Therapy will not be described exhaustively, but a salient features are discussed below.

Fluid replacement is essentially easy; the patient is rehydrated until replete! However, the type of fluid used merits a brief discussion. Intuitively, one expects that the 'ideal' fluid replacement would equal that lost, both in volume and composition. This can be estimated from known losses of blood, from the gut, via burns and from changes in haematocrit; the 'ideal' amount is then replaced (monitoring C.V.P. as a guide against overhydration.) However, the normal body fluid distribution may be changed in shock: Shires et al. (9) discuss evidence that the extracellular fluid (E.C.F.) 'shunt space' is depleted in shock, even if fluid and total body fluid loss is made good. This is suggested, is due to sequestration of the E.C.F. by the intracellular compartment. Rehydration of this space can be achieved by a 1-2 litre infusion of E.C.F. composition (e.g. Ringer lactate) prior to, and whilst cross-matching for a blood transfusion. A second reason for haemodilution is that it decreases blood viscosity, thereby allegedly preventing or alleviating peripheral sludging in shocked tissues, without materially affecting oxygenation. An optimum haematocrit of 30-35% is suggested.

When a fluid is used, its metabolite effects should be considered in advance; if anaemia needs correction this is often attempted with lactate rather than bicarbonate, the rationale being that controlled conversion of lactate to bicarbonate will take place in the liver. However, since this is known to be one of the first hepatic functions lost in shock, the practice should be regarded with suspicion, for in this situation a lactate infusion may aggravate the metabolite acidosis.

Conventional therapy also includes administration of antibiotics chosen according to culture sensitivity, or, if absolutely necessary, the unit's own
policy for septic shock. In the case of heart failure, inotropic agents and diuretics (if the CVP is raised) may be used; pulmonary embolism may be treated with anticoagulation, antithrombin and streptokinase. However, if these fail, there are interesting, new, and incompletely evaluated ways of treating shock.

The use of vasodilator substances is always subject to debate. At one stage vasopressors were used in shock; some centres still do this. If the drug raises B.P. by increasing P.R. (which must do), this will aggravate the shocked state of tissues. Thus, using such a vasopressor in shock displays complete failure to understand the pathophysiology (cf. Section B). Inotropic agents like dopamine are justifiable in cases of refractory hypotension since they raise cardiac output. Peripheral vasodilators are, at least theoretically, a good idea, since they may increase tissue blood flow in low C.O. or high P.R. shock (obviously they are contra-indicated in high C.O. shock.) Foraster et al. (10) describe encouraging results both in increasing flow to the periphery and in alleviating left-sided heart failure. Dramatic increases in C.O. were achieved on infusing sodium nitroprusside or phentolamine into patients with heart failure, apparently without any decrease in myocardial oxygenation, for, at low infusion rates, any deep in blood pressure. They define parameters which must be monitored in this therapy: pulmonary wedge pressure must be less than 15-18 mm Hg, and an adequate systolic and diastolic coronary perfusion pressure must be present. However, the latter may be achieved by counterpulsation.

Evidently the exact role of inotropes is not certain. At present they must be regarded as of possible benefit in selected cases of shock. Even less certain are the indications for two 'biological' weapons now being used in shock—steroids and insulin. It is argued that if a cell cannot use glucose, and a state of relative diabetes exists—as in shocked tissues—then high infusion of insulin with dextrose and potassium could overcome this metabolic block. This seems to be the case in rabbits, whose condition in 'unresuscitable' shock may improve with such a regime, and one or two groups claim success in septica shock in man. Maybe such attempts are misguided, so it is possible that glucose is not the ideal caloric fuel in these conditions. An infusion of ATP with MgCl2 might supply 'raw' energy, whilst much work done recently points to the possibility that amino acids are an important energy source in post-traumatic catabolic states. One can hope that as the field is investigated the ideal 'meal' for a shocked cell will be found.

It is not unlikely that the biggest white elephant in the whole field of shock therapy is the use of steroids. Debate on the usefulness of huge, non-physiological doses has been heated, and entirely inconclusive, for years. Proponents of this therapy such as Lillehei, provide stunning evidence of their near magical curative properties (114). Apparently steroids stabilise lysosomes and cell membranes, protect against endotoxin shock, are positively cardio-inducine, reduce peripheral resistance, increase capillary perfusion, and prevent ARI yet have no adverse effect upon infection rates. Others are unable to repeat these results, and continue
Steroids either useless or dangerous - for example in ARF where the actual cause of death is usually a fulminating bronchopneumonia (B.) Frankyl (11a) has summarized the present position: 'by steroid administration we have saved 55 cats, 200 dogs, 100 guinea pigs, 20 baboons, and possibly 100 humans'.

In the field of ventilatory support, positive end-expiratory pressure (PEEP) is of interest since usually - but not invariably - it seems to have beneficial effects in hypoxic patients with pulmonary edema. PEEP is indicated if arterial PaO₂ falls below 60 mmHg when inspired PaO₂ is above 50%. Possibly secondarily to an increased functional residual capacity, as well as a raised alveolar PaO₂, PEEP tends to increase arterial oxygenation; however a fall in C.O., blood oxygenation, or both may be observed. This is unpredictable, but must be monitored. If present, it demonstrates that PEEP is not beneficial. For obvious reasons, PEEP will increase C.V.P. and this must be anticipated; it does not represent hypovolemic shock.

If all respiratory support fails, it is possible to oxygenate the blood by means of extracorporeal membrane oxygenation. This has been done successfully in the short term, and one could well conceive that shock patients who will ultimately recover, possibly including those regarded as terminal with ARF, would be eligible for such treatment. Yet, however, yet more impossible in Britain at the present time.

Another new form of therapy for shock is counterpulsation. This involves increasing the diastolic pressure either externally, by compressing the legs in this phase, or via an intra-aortic balloon. The point of this is to increase coronary blood flow, which occurs during diastole. Unfortunately, the external method, whilst being non-invasive, seems to be of little use. Intra-aortic balloon counterpulsation has, however, been of use in cases of cardiogenic shock secondary to heart surgery and seems helpful in maintaining coronary perfusion until a corrective operation can be performed. But it is of dubious value in non-cardiogenic shock, or as a long-term method for circulatory support.

Finally, what surgical procedures can be used to treat shock? These conditions are amenable to therapy. First, continued fluid loss in cardiogenic shock, this could be due to trauma, or continued loss secondly to gastro-intestinal pathology, incurable by other means, or it could be reactionary bleeding; and all of these must ultimately be arrested. Second, any area of continued infection in septic shock should be drained. In these two cases, the danger of continued fluid loss or sepsis must be weighed against the operative risk to the patient. With septic shock, the Glasgow study (12) and see section B. demonstrates that almost any risk is acceptable; the situation is less clear in hypovolemic states. The third indication for surgery is that of controllable cardiogenic shock; one example of this is papillary muscle rupture - where emergency operation (within six hours) and volume replacement will be curative in some cases. If necessary, volume replacement can be preceded by intraaortic balloon counterpulsation. Another rare case for corrective cardiac surgery is cardiac tamponade without ventricular rupture (often secondary to anticogulant therapy) where emergency pericardiosentesis cures the shock. In all these cases, prompt, accurate diagnosis and immediate treatment, are essential.
In situations where a patient recovers from shock, but has received irreparable organ damage as a result, the surgeon may be able to intervene again. Some people have suggested lung transplantation in A.R.S., whilst a less dramatic case is the excision of gut infected in a hypovolemic phase of shock. Perhaps the most ironic of all is the transplantation of a cadaver kidney to replace those which have suffered necrosis as a consequence of shock. This incurs long-term immunosuppression, and places the patient in a very high risk category - for the development of septicaemic shock...

Conclusions

This essay has not described shock in minute detail, for every aspect of it relevant to the surgeon. Instead, an attempt has been made to provoke an understanding of the few important and simple principles concerned with it, on which a scientific analysis of the subject may be built. It has tried to show that the surgical team should avoid shock by good technique and adequate mutual communication. In particular, there must be correct and prompt fluid replacement, the minimum of trauma in surgery, stringent antisepsis, and adequate drainage. But even the best team can make mistakes, and shock will always be an inevitable surgical complication; additionally, the patient may be shocked prior to emergency surgery.

Once shock is present, a diagnostic approach to the patient gives both a physiological understanding of the problem, and a basis for therapy. There are many well tried treatments, and several others which are new, and not properly evaluated, some of which have been discussed. Sometimes the surgeon can correct the lesion causing shock - be it his own fault or from some other cause. Sometimes he can put right damage wrought by the condition.

One can but hope that in the not too distant future, the therapy of shock will be so effective that the description of it by John Collins Warren (in 1815) as 'A momentary pause in the Act of Death' will be purely historical. However, until then we can only dream that shock will never be the result of error on the part of a surgeon.
(1) R.D. Bradway, Recent Adv. in Surgery (1976) p 375
(2) A.P. Thal, Shock: A Physiologic View (1971) Chicago: Year Book
(3) LeDouxham, Monograph on Shock (1976)
(6) Peirce et al., Can Med Assn J. 90 p 673 (1964)
(7) Rosch, Surgical Clinics of North America 55.3 p 613 (1975)
(8) Teplitz, Surgical Clinics of North America 56.5 p 1041 (1976)
(9) Shires et al., Surgical Clinics of North America 52.6 p 1341 (1973)
(10) Forrester et al., Surgical Clinics of North America 55.3 p 531 (1975)

B. Fennel
Summary up in " " " " "
(13) Dr. Pierre Fox, Intensive Therapeutic Unit, Oxford, Personal Communication (1977)
(14) Reul et al., Am Surg. 106:3 p 56 (1973)