The incidence of the shock syndrome in a general hospital

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Summary
During a 1-year period (May 1971–May 1972), patients developing the shock syndrome in a large general hospital were referred to a multi-disciplinary shock team. Sepsis, haemorrhage and severe hypoxaemia were the principal precipitating factors. Mortality exceeded 70% in the septic and cardiogenic shock series and was lowest in shock attributable to fluid depletion and drug overdose. Whereas improvement in mortality in the haemorrhagic shock group is likely to result from more rapid and aggressive primary surgery, septic shock, particularly arising from intestinal complications, will require a more complex solution. A shock team seems an important addition to the services of a modern general hospital.

Introduction
The high mortality amongst patients with the shock syndrome together with an increase in the incidence of shock due to sepsis, trauma and drug overdose (Weil and Shubin, 1967) has led to the establishment of several shock units in different parts of the world, predominantly in North America. Data on the nature and magnitude of the shock problem in the United Kingdom are relatively sparse and for this reason a 3-year prospective study was initiated at the Western Infirmary, Glasgow. The present data, obtained during the first year of the study, are concerned principally with the incidence of shock. The information serves to define more precisely which groups of patients are at special risk and should be of value both to clinicians involved in the centralized care of such critically ill patients and to those who are confronted with the problem only occasionally.

Materials and methods
The study was performed in the Western Infirmary, Glasgow, a general teaching hospital of 667 beds. There is a fully equipped 5-beded Intensive Therapy Unit (ITU) staffed on a continuous basis by junior and senior medical personnel in addition to specially trained nursing personnel. Most of the prolonged shock resuscitations were performed in the ITU although the initial treatment of some of the patients was performed in the ward or, in the case of those patients with trauma, in an Accident Care Area situated near the entrance to the hospital.

On May 1 1971, a Shock Team became available, comprising an Intensive Therapist and three Clinical Research Fellows whose specialties of origin were Anaesthesia, General Surgery and Orthopaedic Surgery. The clinical duties of the three Research Fellows were exclusively concerned with the care of shock patients, and when not thus engaged, they were involved in experimental projects related to various aspects of shock. Four technicians assisted the clinical team particularly in setting-up and calibrating apparatus during the early stages of resuscitation when avoidance of delay was important. The total group of eight personnel was divided into two teams which were available to answer shock calls on alternate weeks. The members of the Shock Team were supernumerary to the staffs of the ITU and of the Accident Service but the activities of all three groups were carefully integrated.

The patients were referred from all parts of the hospital; none had a terminal illness. Individual clinicians were found to vary in their interpretation of the severity of shock and to avoid delays in referral, a simple shock scoring system was devised. The system was based on the 20 clinical features most commonly noted at the time of referral and permitted allocation of the patients into categories of mild, moderate, severe and profound shock. Comparison of the severity of shock within and between groups of patients was thus facilitated. In most instances monitoring was instituted as soon as the patient was seen. In addition to the usual respiratory and cardiovascular measurements, some of the more recent monitoring developments were appraised including core/peripheral temperature gradient (Joly and Weil, 1969), cardiac output using
thermal dilution (Branthwaite and Bradley, 1968), pulmonary artery pressure and mixed venous blood gas analysis.

The detailed treatment of these patients is not an important component of this presentation but the following principles were followed. Initially, any deficit in fluid balance was made good until the central venous pressure exceeded 10 cm of water. Inotropic agents were used when elevation of the central venous pressure was not accompanied by a concomitant increase in blood pressure (or cardiac output) and improvement in peripheral perfusion. Alpha-receptor blocking agents were used in the presence of sustained vasoconstriction. Steroids were not used consistently. Oxygen administration and intermittent positive pressure ventilation were instituted when indicated on conventional grounds.

**Results**

Within the first year 110 patients were referred to the Shock Team. Of these, sixty-six were referred from surgical units (or operating theatres), twenty-six from the Accident Service and eighteen from medical units. The overall mortality in the series was 56%.

The patients were sub-divided into categories based on the principal factor apparently causing shock (Table 1). The majority of the patients presented as a consequence of sepsis, haemorrhage or severe hypoxaemia. There was a further miscellaneous group in which the aetiological factors were hypovolaemia, myocardial infarction, pulmonary embolism and drug ingestion. Mortality exceeded 70% in the septic and cardiogenic shock series and was lowest in those attributable to fluid depletion and drug overdose. Although some form of classification of clinical shock is important, many patients presented with several contributing factors, e.g. significant hypovolaemia and hypoxaemia were found frequently in association with sepsis.

The subsequent data are presented separately for each category of shock.

**Table 1. Western Infirmary Shock Project (first year)**

<table>
<thead>
<tr>
<th>Clinical category of shock</th>
<th>No. presenting</th>
<th>No. died</th>
<th>(% mortality)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sepsis</td>
<td>35</td>
<td>25</td>
<td>(71)</td>
</tr>
<tr>
<td>Haemorrhage (trauma)</td>
<td>23</td>
<td>8</td>
<td>(35)</td>
</tr>
<tr>
<td>Haemorrhage (non-trauma)</td>
<td>18</td>
<td>11</td>
<td>(61)</td>
</tr>
<tr>
<td>Hypoxaemia</td>
<td>14</td>
<td>8</td>
<td>(57)</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>8</td>
<td>6</td>
<td>(75)</td>
</tr>
<tr>
<td>Hypovolaemia</td>
<td>7</td>
<td>2</td>
<td>(29)</td>
</tr>
<tr>
<td>Drug</td>
<td>5</td>
<td>1</td>
<td>(20)</td>
</tr>
</tbody>
</table>

**Septic shock** (Table 2)

The thirty-five patients in this category had a mean age of 57 years; twenty-five died (a mortality of 71%). These patients were all at least moderately shocked when first seen; other common features included dehydration, hypoxaemia (ten had an arterial oxygen tension of less than 60 mmHg; nine of eleven receiving oxygen on referral had an arterial oxygen tension of less than 100 mmHg), hyperventilation (all but four had an arterial carbon dioxide tension of less than 35 mmHg; fourteen of less than 30 mmHg) and metabolic acidosis (eighteen had an arterial base deficit exceeding 6 mEq/l; ten exceeding 10 mEq/l). The evidence for sepsis took the form of positive blood cultures (fifteen of twenty-eight patients) or widespread tissue infection, e.g. generalized peritonitis. The commonest organisms were *Escherichia coli*, proteus, bacteroides and *Streptococcus pyogenes*, with occasional isolates of *Staphylococcus aureus*, Klebsiella, Clostridium welchii and *Enterococcus*. Gentamicin was the antibiotic most frequently used with kanamycin, cephaloridine, ampicillin and cloxacinil as occasional alternatives; lincomycin was used only once. In seven instances the organism isolated in culture proved in retrospect to be resistant to the antibiotic in use for treatment. Bacteroides organisms were the resistant organisms in three of the patients. Peritonitis secondary to gastrointestinal disease was the principal precipitating factor with genitourinary sepsis accounting for most of the remainder.

The effect of treatment was to relieve shock in nineteen patients. In sixteen patients, shock was either not relieved or recurred. Arrhythmias, particularly atrial fibrillation (eight patients), were a common occurrence and nine patients died in acute cardiac failure. Seven patients had a more slowly progressive combination of cardiac and respiratory failure. Renal failure was not considered to have contributed significantly to the death of any of these patients. The duration of shock exceeded 12 hr in twenty-three patients, but only four of the patients
who subsequently died survived for more than 3 days.

There was no significant difference between survivors and non-survivors in respect of age. The source of infection, however, appeared to be of importance. No patient with peritonitis arising from large bowel disease and only one with small bowel disease survived while, by contrast, the patients with gall bladder disease or gynaecological sepsis did well. Arterial oxygen and carbon dioxide tensions were not significantly different in survivors and non-survivors but the latter were significantly more acidic at the time of referral. Two other features of note were the duration of shock and failure to eliminate sepsis. In general, the longer the period of shock prior to inception of treatment, the less likely was survival. In five of the patients at a second laparotomy and an additional nine at autopsy, a continuing significant focus of infection was present.

**Haemorrhagic shock**

**Trauma**

Twenty-three patients were treated in the Accident Care Area for shock consequent on trauma; the mean age of the series was 38 years. Eight patients died (a mortality of 35%). Most presented with multiple injuries; three resulted from stab wounds. Two-thirds of these patients were severely shocked on admission. Arterial blood gas analysis revealed that hypoxaemia developed frequently (of seventeen patients, seven had an arterial oxygen tension of less than 60 mmHg) and was not always corrected by oxygen administration and positive pressure ventilation; changes in arterial carbon dioxide tension were masked by the frequent use of assisted ventilation but metabolic acidosis was a marked feature (in all but two patients, the arterial base deficit exceeded 5 mEq/l and in five exceeded 15 mEq/l). A marked fall in haemoglobin and packed cell volume occurred in those patients in whom blood transfusion was delayed and hypothermia (a core temperature of less than 35° C.) was observed in eight patients. Unlike the septic shock series the overall duration of this form of shock rarely exceeded 6 hr.

Of the eight patients who died, six were under 32 years of age and the average duration of stay in hospital prior to death was 3 hr. No patient died within 1 hr of admission. Continuing blood loss was the only or a major contributing factor in the death of seven of these patients; other contributing causes included severe hypoxaemia (three patients) and sudden cardiac failure (three patients). Intrabdominal bleeding occurred in eight patients and four died from causes directly attributable to continuing intra-abdominal blood loss. In none was there an associated lesion (i.e. head injury) of such severity as to render recovery unlikely and, in all, the intra-abdominal lesions appeared to be amenable to surgical treatment. In a fifth patient death occurred as a result of intractable blood loss from a fracture of the maxilla which was the only severe injury. On average, non-survivors received twice as much blood replacement as survivors (13·5 units and 5·6 units respectively; \(P<0.05\)). Only one of five patients with a metabolic acidosis exceeding 15 mEq/l survived.

**Non-trauma**

Eighteen patients presented with shock attributable to bleeding from peptic ulcer (eight patients), aortic aneurysm (three patients), oesophageal varices (two patients), the prostatic bed (two patients), torn inferior vena cava (one case) and ruptured uterus (one case). All but two of the patients were in the fifth or seventh decades; eleven patients died (a mortality of 61%). Of the patients whose shock score was calculable two were severely shocked, the remainder moderately. Hypoxaemia and hypocapnia were not marked features during the early stages of this form of shock and a metabolic acidosis exceeding 10 mEq/l was noted on only three occasions. The fall in packed cell volume and the duration of shock were comparable to the trauma group but hypothermia was unusual. No patient was in hospital for less than 2 hr prior to death.

Of the eleven patients who died, only two were under 60 years of age. The patients with bleeding from aortic aneurysms, oesophageal varices and the prostatic bed all died as did four of the eight patients with peptic ulcer. Continuing or recurrent blood loss was the sole or a major contributing factor in the death of eight of these patients; other contributing causes included severe hypoxaemia (two patients) and sudden cardiac failure (six patients). The average blood replacement was not significantly different between survivors and non-survivors (11 and 12 units respectively) but no patient receiving more than 12 units of blood survived.

**Hypoxaemia**

There were fourteen patients in whom the main precipitating factor of shock appeared to be severe hypoxaemia although normally other complicating factors were present including trauma, either surgical or accidental, acute or chronic blood loss and dehydration. Six of the patients presented immediately after general surgical operations and two after crushed chest injuries; two had long-standing respiratory disease and four were associated with other miscellaneous conditions. The patients were all in the seventh and eighth decades and were either moderately or severely shocked on referral; eight died (a mortality of 57%).

The average arterial oxygen tension in ten patients of this group prior to oxygen administration was
Incidence of the shock syndrome

46±11 mmHg (mean ± standard deviation). Unlike the previous groups, hypoventilation was a significant finding with the arterial carbon dioxide tension exceeding 45 mmHg in seven patients and falling within the normal range in the remainder; only five patients had a base deficit exceeding 5 mEq/l. The duration of shock was rather longer than in the haemorrhagic shock group although rarely exceeding 8 hr.

Of the eight patients who died five were in the post-operative category. Both the patients with long-standing respiratory disease died as did one patient with overwhelming bronchopneumonia. Death was attributed to a number of factors including sudden cardiac arrest (five patients), respiratory arrest (two patients) and one delayed hepatic failure. Neither age nor severity of the initial blood gas disturbance were factors distinguishing survivors from non-survivors.

Cardiogenic shock

Eight patients presented with shock due to primary cardiac failure. Six had a history of recent myocardial infarction and two followed acute pulmonary embolism. All but one were in the seventh decade and all were moderately or severely shocked on referral. One of the patients with pulmonary embolism survived as did one of the patients with myocardial infarction although the duration of shock in the latter was only 1 hr. Correction of severe hypoxaemia and moderate dehydration may have been a significant factor in the outcome of this patient. The remaining patients died 3–26 hr after the onset of shock from cardiac failure.

Hypovolaemic shock

Seven patients presented with shock in which the main precipitating factor appeared to be dehydration. Like those in the hypoxaemic group, other complications were usually present including hypoxaemia (five patients), infection (three patients) and myocardial ischaemia (three patients). All were over 55 years of age (four over 70 years of age). One was severely shocked on referral and the remainder moderately shocked. Five of these patients presented in the early post-operative period and three were associated with chronic medical conditions. One patient with acute pancreatitis died as did one of the post-operative group. Sudden cardiac failure was the terminal event in both cases.

Drug shock

Five patients presented with drug overdose associated with severe hypotension of at least 5 hr duration. One patient was in the third decade and the remainder were in early middle age. One patient was severely shocked on referral and the remainder moderately shocked. Three patients had ingested a mixture of barbiturates, tricyclic antidepressants and tranquillizers, one carbitral and one salicylates. The patient with salicylate intoxication died 18 hr after admission with sudden cardiac arrest.

Discussion

The study has established that in this hospital, two patients per week, on the average, present with the shock syndrome. Unfortunately, from the administrative point of view, the patients tend to arrive in groups of two or three at a time. It is probable that this figure represents the bulk of shock arising in the hospital during the period of study although it is reasonable to assume that a number of patients with some of the prodromata of the shock syndrome responded to early vigorous treatment and did not require to be referred. This would be true particularly of the non-traumatic haemorrhagic shock group. The majority of patients referred to the Shock Team were already moderately or severely shocked.

The groups of patients in which treatment was especially complex were those with shock secondary to sepsis and trauma, and to haemorrhage of non-traumatic origin in the elderly. The high mortality in cardiogenic shock is common to many series (Adgey, 1972) and treatment is strikingly unsuccessful. Other causes of shock were more amenable to early treatment, e.g. hypovolaemia.

In the septic shock series continuing abdominal sepsis was present in a surprising number of patients. The importance of the fundamental surgical precept of adequate drainage is emphasized. The presence of organisms resistant to the antibiotic in use for treatment was a disturbing observation and clearly there is a need for improved collaboration with the bacteriological service. The bacteroides group of organisms may prove to be of increasing significance in septic shock (Lancet, 1973; Okubadejo, Green and Payne, 1973). The frequency of sudden death of cardiac origin in nine of these patients underlines the problem of myocardial irritability and depression in established septic shock. These features may be attributable to myocardial ischaemia, to the direct effect of endotoxin on the myocardial cell (Hinshaw et al., 1972) or to the indirect effect of circulating myocardial depressant substances (Fisher et al., 1973). The equally common problem of sub-acute progressive cardiorespiratory failure when taken in conjunction with hepatic and renal dysfunction suggests a widespread disturbance, possibly disseminated intravascular coagulation (Milligan et al., 1973).

In the trauma group one of the more disturbing features was the apparent difficulty in adequately controlling haemorrhage, particularly of intra-abdominal or pelvic origin. Undoubtedly delay in
laparotomy was a contributing factor and this observation has led to a more aggressive approach to the investigation of such patients where injuries to the abdomen and pelvis do not prove immediately responsive to conservative resuscitative measures. The other difficulties in trauma patients relate to diagnosis and therapy in a rapidly changing clinical situation.

Perhaps one of the more encouraging aspects of this first year of the 3 year shock project has been the speed with which the Shock Team has become integrated within the framework of the acute medical and surgical services. Clearly a gap in the existing services of the hospital has been filled. In the case of patients suffering from traumatic shock the value of the Shock Team appears to lie in the fact that a small number of medical personnel becomes increasingly experienced in handling problems which have to be tackled immediately and rapidly, if significant improvement in survival is to be obtained. In the case of shock secondary to sepsis the role of the Team is in providing intensive and sustained care of acutely ill patients, frequently over prolonged periods of time. Medical staffing in most Intensive Therapy Units does not permit such sustained effort without detracting from the care of other patients in the Unit.

In terms of actual treatment of the shock syndrome little advance has been made during the year of study. The data obtained during this initial phase had to be assimilated before appropriate changes in the treatment regimen could be instituted on a logical basis. Information already emerging from the second year of the project indicates the value of these background data.

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References


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