FACTORS IN THE ASSESSMENT OF CLINICAL SHOCK

Circulatory patterns in clinical shock

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Summary

The term 'shock' is used to denote a pathological process, but in clinical practice it must be recognized by clinical methods.

Successful treatment requires both the haemodynamic abnormality and also the causal disease to be defined.

Because of the dynamic nature of the shock process and the multiplicity of the precipitating causes the clinical signs vary with time and also from case to case. Nevertheless, certain broad patterns tend to occur and their relationship to diagnosis and treatment are discussed.

As the title of this paper implies, I shall not use the word 'shock' to describe any particular syndrome, but merely as a conveniently brief term for referring collectively to a variety of circulatory disorders which might progress to fatal circulatory failure. The syndrome of hypotension, pallor, sweating, rapid weak pulse, etc., which I shall call the 'classical shock syndrome', is only one of a number of patterns, although most cases will exhibit this syndrome at some stage if untreated. Some authors appear to regard these patterns as quite distinct phenomena specifically associated with Gram-negative or Gram-positive infections, but they are, in fact, interrelated, and patients often exhibit transitions from one pattern to another, either spontaneously or in response to treatment. I shall describe these patterns in clinical terms, indicating whenever possible, the underlying pathological changes.

The causes of shock are many and the clinical signs vary in detail with the cause. A large number of clinical syndromes occurs, but whilst, in the present state of knowledge, it would be unrewarding to discuss all of these, certain broad patterns are discernible which are worthy of our attention. I shall concentrate on states of hypotension, although it is well known that in some circumstances the fall in blood pressure correlates poorly with the pathological process, as in the early stages of haemorrhage (Grant & Reeve, 1951). Nevertheless, when the blood

pressure *does* fall, most people take notice, and I think it is still an important sign though often there are earlier warning signs.

These hypotensive patients can be divided into two broad groups according to the pulse volume, assessed by feeling the radial artery. This is usually a good clinical index of the stroke output, though it may be modified by other factors. There are patients with a poor pulse volume indicating a low output, and those with a good pulse volume indicating normal or high output.

The poor-pulse-volume states are the most common and will be dealt with first. There are many causes, but most of the early studies were made in haemorrhage. It was established that the fall in blood volume causes first a fall in central venous pressure and then a low cardiac output; the blood pressure falls when vasoconstriction is inadequate to compensate for the fall in cardiac output. At each stage compensatory activity in the sympathetic nervous system gives rise to signs of the classical shock syndrome such as sweating, venous constriction, pallor and tachycardia.

An acute fall in cardiac output may occur from many other causes, and the mechanisms of its production may differ (Fig. 1). Myocardial infarction, for example, affects the myocardium directly while bacterial infection may act at three points, any one of which may dominate the picture at a given time, though this dominance may change as I hope to show. It follows that for rational treatment it is necessary to identify the haemodynamic fault and to determine and treat the underlying cause.

In the poor-pulse-volume group the main haemodynamic fault may be determined by measuring the central venous pressure (CVP).^{*} If this is high it indicates a primary cardiopulmonary cause, or myocardial failure secondary to some other cause such as bacterial infection. The differential diagnosis

^{*}In the following examples all CVP measurements were made by observation of the external jugular vein, and are expressed in centimetres above (+) or below (-) the sternal angle. The upper limit of normal is - 3 cm (see page 506).

FIG. 1. Mechanisms by which different causes of shock produce a low cardiac output.

within this group is usually resolved on other clinical evidence.

If the CVP is low, then whatever the cause, and irrespective of other factors which may be present, transfusion will be the first treatment to correct this haemodynamic fault. Diagnosis of the cause in this group may be easy from the history of, say, a rigor or drug overdose, but is not always so, particularly in post-operative patients. Here it is important not to miss infection and especially peritonitis. A pulse rate of 140/min usually indicates infection or pancreatitis, and transient arrhythmias are not uncommon especially in the elderly. Non-circulatory signs may also be very useful. Cyanosis is much more common in infective cases and petechiae, if present, are almost specific in this context. Confusion and jaundice may indicate infection some time before shock occurs, and a sudden rise in respiratory rate is often a valuable indication even of non-respiratory infection (Fig. 2); like the previous two signs it may appear before the blood pressure falls, and is often

FIG. 2. Respiratory rate in forty-five consecutive patients with acute hypotension. The rate is often increased in patients with infection located outside the respiratory system. Stippled columns, infection (non-respiratory); open columns, no infection.

present when the temperature is still normal or even subnormal.

The change from a normal circulation to a low blood pressure and poor pulse volume occurs at variable rates. On the whole I regard these as differences of detail, but there is one subgroup I want to stress because this syndrome is commonly misdiagnosed as massive pulmonary embolism, a mistake that has often had disastrous consequences for the patient.

I am referring of course to the patient who collapses dramatically with a fall in blood pressure and a barely palpable pulse of 140/min, perhaps with some irregularity. There may be pallor, or intense cyanosis, and the respiratory rate often shoots up to 40/min or more. Not infrequently the patient experiences a sense of constriction in the chest and some cases I have looked into in retrospect are said to have complained even of central chest pain. The similarity of this picture to massive pulmonary embolism is obvious, but I have now seen so many such cases where pulmonary embolism was excluded at autopsy that I am quite unable to accept a diagnosis of pulmonary embolism as the cause of such collapse without autopsy confirmation, or at least CVP measurements in life. By far the most common unexpected finding at autopsy is infection, though not long ago my colleague, Dr C. H. L. Howells, found intraperitoneal bleeding from a slipped ligature; occasionally no cause is identified. The infections which may cause this syndrome are many (Table 1).

TABLE 1. Infections causing pseudo-pulmonary embolism syndrome

1. Peritonitis	< Gastro-intestinal operation Gynaecological operation
2. Septicaemia	
3. Pneumonia	< Staphylococcal Others
4. Enteritis	< Staphylococcal Idiopathic

Post-operative peritonitis from a leaking anastomosis is probably the most important, the usual signs

of peritonitis being absent at the time of collapse. Dr McGowan and I have stressed previously that in a patient who collapses in this manner after an abdominal operation, a pleural rub over the attachment of the diaphragm is strong evidence in favour of peritonitis and is against massive pulmonary embolism (Walters & McGowan, 1963).

The mechanism of this syndrome is not understood. It has been shown experimentally in a number of species that intravenous bacterial toxins may cause pulmonary vasoconstriction and also closure of the terminal airways (Leading Article, *Lancet*, 1966), but I know of no comparable studies in this clinical syndrome. In my view the CVP should always be measured in such circumstances. In massive pulmonary embolism it is raised and has, so far, always been low in cases of the pseudo-embolism syndrome. But I do not know what happens to it within 10–15 min of the onset because I never see these cases at such an early stage. Diagnosis may not be easy, but it obviously helps to be aware of the pseudo-embolism syndrome. If there is the slightest doubt, I believe that antibiotics should be used.

The other major group consists of patients who become hypotensive with a good pulse volume. The cases we saw originally were in the aftermath of some acute episode which we had not witnessed. They had normal blood volumes and were obviously coming to no harm from their hypotension. We presumed that hypotension was due to vasodilatation, and we called it 'benign hypotension'. However, we gradually came to realize that a good pulse volume did not always indicate a benign state and some patients were very ill and continued to deteriorate. The combination of hypotension and a good pulse volume does, in fact, include a wide range of syndromes varying once again in detail, and it is among these that we see many transitions from one pattern to another. There are a number of causes, the most important being bacterial infection. Other causes I have seen from time to time are listed in Table 2.

TABLE 2. Causes of hypotension with good pulse volume

1.	Bacterial infection
2.	Acute pancreatitis
3.	Drugs
4.	Haemorrhage
5.	Myocardial infarction
6.	Phaeochromocytoma

Patients may pass straight into this state from normal, but frequently it appears during recovery from a poor-pulse-volume state. This is illustrated by Case 1 (Fig. 3). This patient (Case 8 of McGowan & Walters, 1963) first exhibited the classical shock syndrome after a long operation for an impacted

FIG. 3. Case 1 (post-operative shock ? cause). Transition from a poor pulse volume to a good pulse volume state produced by transfusion with plasma, but with persistent 'benign' hypotension. Each arrow represents the infusion of 450 ml plasma.

gallstone. The cause of shock was not obvious but he was transfused rapidly with plasma to raise a low CVP to normal. The pulse volume improved greatly after rapid infusion of a large volume of plasma and the blood pressure became easily measurable, but the systolic never rose above 85 mmHg, and indeed settled at 70 mmHg overnight. The following morning he was very well, sitting up reading the paper. Renal function remained good and the blood pressure rose slowly to normal over the next 4 days.

A variant of this response is shown by Case 2 (Fig. 4)—a case of acute pancreatitis. Here transfusion

FIG. 4. Case 2 (acute pancreatitis). Similar response to transfusion with plasma as in Fig. 3, but with a transient rise in blood pressure.

ultimately brought about a rise of blood pressure which, however, soon fell again to 80 mmHg when the transfusion was slowed. The pulse volume had

improved and continued to do so while the blood pressure remained low. This patient remained ill for a time, but the blood pressure slowly rose to normal without further active measures, the continuing hypotension apparently causing no harm.

In such patients, the fact that the blood pressure remains low is of no clinical consequence *if the pulse volume improves and the underlying cause is controlled*. Renal function is unimpaired, unless damage occurs during the low output phase, even though the time taken for spontaneous recovery of the blood pressure is very variable (Fig. 5). That it is justifiable to

FIG. 5. Time for spontaneous recovery of blood pressure in patients who remained hypotensive with a good pulse volume after transfusion.

interpret the good pulse volume as indicating a good cardiac output is supported by data from other similar patients shown in Table 3.

TABLE 3. Cardiac output in hypotensive patients with good pulse volume

Case	Blood pressure (mmHg)	Cardiac output (l/min)
1. Staphylococcal septicaemia	85/50	9.4
2. Peritonitis	100/40	6.5
3. Peritonitis	80/60	7.0
4. Peritonitis	100/50	7.4
5. Pseudomonas septicaemia		
5. Carcinoma of pancreas	70/40	5.1

The good-pulse-volume hypotensive state may also be entered from the side of the normal circulation, and as in the case of poor-pulse-volume states, the rate of transition is variable. Some cases decline rapidly as illustrated by Case 3 (Fig. 6). Very soon after delivery this patient's blood pressure was 90/60 mmHg, temperature 102°F, and pulse and respiratory rates 140 and 40/min, respectively. The liquor was obviously infected and treatment with antibiotics was begun immediately. The central venous pressure was normal and the pulse volume was good, so no fluid was given. Recovery of normal blood pressure was quite rapid in this case, but other similar cases may remain hypotensive for longer periods, recovery being indicated not only by the fall of temperature

FIG. 6. Case 3 (liquor amnii infected with *Esch. coli*). Sudden onset of hypotension with a good pulse volume. Early diagnosis and treatment led to rapid recovery without transfusion.

and pulse rate, but also by the return of the respiratory rate to normal, which may be an early sign that the infection is being controlled.

Other cases show a much slower decline. Case 4 (Fig. 7) became febrile after vulvectomy, due to a streptococcal wound infection, and was treated with tetracycline and chloramphenicol without much success. The blood pressure appeared to be falling slowly and on the 8th day it was 105/70 mmHg. The CVP was normal and the pulse volume good. It looked as though the patient was slowly declining due to a septicaemia, and when the blood culture was positive the following morning, treatment with erythromycin was begun, as she was sensitive to penicillin. There was rapid improvement shown by a fall in temperature and respiratory rate. But during the night the blood pressure fell further to 95/60 mmHg. As the pulse volume was still good, with a falling respiratory rate, fluid was not given rapidly, and the blood pressure was normal again in 2 days.

Although there may be no need for a rapid intravenous infusion in such a case, the cause must be sought assiduously, because if the signs of such a decline in blood pressure are ignored, deterioration

Temperature (°F)	104	104	102	97	
		Diarrhoea Confused Petechiae			
Days post-operation	(2)	(7)	(8)	(9)	(10) (11)
Blood pressure	140 80	110 70	105 70	105→95 60	105 60 130 80
Respiration		38	35	35→	18
Pulse rate (volume)		120 (G)	120 (G)	116 (G)	100 (G)
CVP			-3		
		Tetracycline Chloramphenicol →		←Erythromycin	

FIG. 7. Case 4 (vulvectomy; *Strep. pyogenes* from blood culture). Slow decline of blood pressure with continuing good pulse volume(G). Recovery without transfusion.

		800 ml blood		Penicillin Streptomycin	
Blood pressure	125/80	80/55	100/60	90/50	—
Pulse rate	136	132	116	126	160
Pulse volume	Good	Weak	Good	Good	Very Weak
CVP	-3.5	-6	-3	-1	0
Hours after admission	0	10	12	20	29

FIG. 8. Case 5 (acute pyelonephritis; *Esch. coli* from blood culture). Progressive decline first with hypotension and a poor pulse volume due to low CVP. Subsequently, low blood pressure with a good pulse volume and normal CVP, and finally poor pulse volume with myocardial failure.

might become too rapid to be reversed, and might progress into the classical shock syndrome with a low cardiac output, as illustrated by Case 5 (Fig. 8) (Case 3 of McGowan & Walters, 1963). This woman, aged 44 years, was admitted with anuria and pyrexia following a gynaecological operation in another hospital. She was slightly pyrexial but the circulation was satisfactory. Ten hours after admission the blood pressure had fallen to 80/55 mmHg with a poor pulse volume, and this was primarily due to a fall in CVP. Transfusion restored the pulse volume over about 2 hr, but the blood pressure did not rise to normal, i.e. this is the same response as seen in Figs. 3 and 4. She remained in this state for a time, the blood pressure falling to 90/50 mmHg. This is not, in itself, an ominous sign if the pulse volume is good, but the CVP had now risen to -1 cm, an abnormally high level for the method of measurement employed, so that now we had both vasodilatation and some myocardial impairment due to infection. Nine hours later the CVP had risen further to zero and the pulse volume was very weak, so that now cardiac failure dominated the picture.

This patient with a coliform septicaemia therefore showed three distinct circulatory patterns, dominated first by a low CVP and then by vasodilatation and myocardial failure respectively, and manifest clinically by a poor pulse volume, good pulse

volume, and then a poor pulse volume again from a different haemodynamic fault. Antibiotics were not begun until after a bacteriological report had been received on the urine, the blood culture result not being available until after death. This case, therefore, also illustrates the folly of waiting for such results—I might add by way of excuse that this occurred in 1956.

I think most bacterial cases declining in this manner will ultimately pass through the classical shock syndrome. Some cases, however, will maintain a hyperdynamic state until death. Case 6 (Fig. 9) was a woman aged 40 years with peritonitis and a *Pyocyaneus* septicaemia. An episode of hypotension with a poor pulse volume responded very quickly to rapid intravenous fluid, and the blood pressure rose to normal. But, just like Case 2, the blood pressure fell again, though more slowly, with a bounding pulse. The cardiac output fell from 13 to 7.5 l/min, so that the alterations in blood pressure were probably due to the changes in cardiac output. She remained in this high output state but the respiratory rate remained high and she became more confused, ultimately lapsing into coma. Jaundice appeared and she was obviously getting much worse despite the maintained cardiac output. This was sustained until very shortly before death about 16 hr after the onset.

FIG. 9. Case 6 (peritonitis; *Ps. pyocyanea* from blood culture). General deterioration and progressively falling blood pressure with a bounding pulse sustained until shortly before death.

Hepatic cirrhosis is said to modify the pattern in this way (Udhoji & Weil, 1965), but in my experience most cases of this sort are due to infection and do not have cirrhosis. Alterations in oxygen consumption have led to the suggestion that tissue perfusion is reduced in the hyperdynamic patient, despite the high cardiac output and low peripheral vascular resistance, because of arterio-venous shunts in the micro-circulation (Siegel, Greenspan & del Guercio, 1967).

Finally, I want to comment on the view that Gram-negative and Gram-positive organisms produce characteristic patterns. Last year, *Antibiotic News* described a report from Weil's laboratory (Kwaan, Bradley & Weil, 1966) that Gram-negative bacteria produce low cardiac output states with low tissue perfusion, and Gram-positive bacteria produce high output states which correspond to our good-pulse volume states. I have demonstrated that Gram-negative infections may also produce high output states, as in the last case, and that the pattern may change progressively as the patient declines (Case 5).

Gram-positive infections also produce both high and low output states. For example, Case 4 had streptococcal septicaemia with a good pulse volume and required no infusion, but we have recently seen

another whose cardiac output failed to rise above 2.8 l/min despite an adequate CVP and correction of acidosis. Many cases of staphylococcal infection also have a low output pattern, with or without a preceding high output state, and may even cause the pseudo-pulmonary embolism syndrome I have described.

Attempts to identify the type of organism from the haemodynamic pattern are to be welcomed, but must not, at present, be used as a basis for treatment. This was brought home very forcibly by a recent case of bacteraemic shock 3 days after prostatectomy. The picture was one of poor pulse volume and marked red and blue mottling of the skin. This, we thought, was certainly a Gram-negative septicaemia and instituted treatment accordingly. Fortunately, the urine was taken to the laboratory and the deposit Gram-stained immediately, as it always should be in these cases. It was a mass of staphylococci which were subsequently grown from the blood culture, by which time, of course, the patient was better. If the type of organism cannot be predicted with certainty in these circumstances, then I doubt if it ever can be. The essential procedure in all these cases is to assess the haemodynamic pattern with a view to correcting the fault, bearing in mind that a change of pattern may call for a revision of treatment, and to make every effort to determine the cause so that specific treatment may also be directed at this.

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