THE INFLUENCE OF PYREXIA ON THE SIGNS OF SHOCK

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The way in which co-existent pyrexia may alter the classical picture of shock is not specifically described in the literature on the subject although the Medical Research Council Memorandum on the Treatment of Wound Shock (1957) does describe a variation of the shock syndrome seen in hot climates. The following case illustrates such an atypical picture as seen in a patient suffering from perforation due to diverticulitis of the colon with accompanying pyrexia.

Case History.

A man of 60, whose bowels had not moved for three days, complained of the sudden onset of generalized abdominal pain at 11.30 a.m. At the same time he felt cold and had a series of shivering attacks. He was seen at 3 p.m. by his family doctor whose findings were as follows:

Temperature 103°F; pulse rate 84/min; blood pressure 160/80 mm Hg.

Heart sounds were of good quality with no added sounds.

Respiratory system: nothing abnormal discovered.

Abdomen: tumid with generalized tenderness and guarding; no loss of liver dullness detected.

The patient gave a history of having had three severe attacks of coronary thrombosis during the previous ten years. However, these attacks had all the typical features of that disease and differed completely from the present illness.

The patient was seen again at 7 p.m. when the blood pressure was 120/80 mm Hg. There was loss of liver dullness. A diagnosis of a perforated viscus was made and the patient admitted to hospital where a straight X-ray of the abdomen revealed the presence of gas under the diaphragm. At 9 p.m. the patient was admitted to the ward where the blood pressure was 70/55 mm Hg. This sudden and severe drop was thought to have been brought about by the ambulance journey and the manipulations in the X-ray department. An intravenous drip of 10 per cent dextrose in saline was started; 100 mg of pethidine were given intramuscularly and the decision was made to perform a laparotomy as soon as the patient's condition permitted.

I first saw the patient at 11 p.m. His temperature was still 103.2°F. I found him to be a well-built man of ruddy complexion, mentally alert with a pulse clearly palpable at a rate of 84/min. The capillary refill time was rapid. There was no suggestion of cyanosis of the skin which was warm and dry. The blood pressure was 60/50 mm Hg but the patient's general appearance gave a completely false impression belying the seriousness of his condition.

The intravenous drip was changed to dextran and blood was taken for grouping and cross-matching. By midnight the blood pressure had risen to 100/60 mm Hg, and it was thought that his condition would improve little more.

After the patient had been premedicated with atropine sulphate 0.6 mg, anaesthesia was induced with halothane and oxygen. A cuffed oral endotracheal tube was passed and the anaesthesia continued with the same mixture. A closed circle circuit with carbon dioxide absorption was used, the halothane being vaporised by the basal oxygen flow from the trichloroethylene bottle on the top circuit. The peritoneal cavity was full of faeculent fluid together with large quantities of solid faeces. No perforation in an area of diverticulitis of the colon could be found and the abdomen was closed with a transverse colostomy through the incision. The blood pressure had been maintained at 100/60 mm Hg during the operation and the patient was conscious on leaving the theatre. On return to the ward his blood pressure again started to fall and did not respond to further intravenous therapy. The patient died at 3 a.m.

Discussion

If a human subject is strapped to a tilting table and changed from the horizontal to the vertical position, pooling of the blood occurs in the lower limbs. The faint which can occur may be delayed by reflex vasoconstriction of the vessels of the lower limbs. If the same experiment is repeated with an increased external temperature fainting occurs sooner, because the compensatory vasoconstriction in the lower limbs is prevented by the vascular mechanisms of temperature regulation (Wright, 1952).

The Medical Research Council Memorandum on the Treatment of Wound Shock (1957) describes how the classical picture of shock—pale, cold, sweating and probably slightly cyanosed skin, together with low blood pressure and rapid pulse—may be modified in hot climates to one of "warm hypotension"—warm extremities, pale or well coloured face, low blood pressure and rapid pulse.
Similarly it seems reasonable to expect that a co-existent pyrexia might modify the signs of shock and the above case confirms this.

Patients suffering from both severe shock and pyrexia are not commonly seen, but accurate assessment of their state is essential for the proper choice of the method of anaesthesia. Measurement of the blood pressure at frequent intervals is necessary as the external appearance and pulse may be quite misleading and the degree of shock may increase considerably without obvious change in the external appearance. In choosing a method of anaesthesia it must be remembered that the pyrexia deprives the patient of a varying proportion of the reflex vasoconstrictor response to fall in blood pressure and any anaesthetic drug or technique which may cause such a fall in blood pressure should be avoided.

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REFERENCES


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