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ASPECTS OF TREATMENT*

Hypovolaemia and phaeochromocytoma

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Summary

The state of shock which frequently follows removal of a phaeochromocytoma is due to the hypovolaemia resulting from prolonged excessive secretion of vasoconstrictor substances and should be treated with intravenous fluids. The gradient between central and peripheral temperatures provides a reliable guide to the hypovolaemic state. Two illustrative cases are described.

The relationship between hypovolaemic states and the occurrence of a gradient between the central and the peripheral temperature in the body is now recognized, although not so widely as desirable. Ibsen[1] was one of the first to draw attention to this and subsequent authors have also presented it[2,3]. Observations of central and peripheral temperature give valuable information about the clinical state and should be made use of in all cases of illness or anticipated illness associated with circulatory instability, whether medical or surgical in origin. Without the information they can give great opportunities are missed in the understanding and management of patients who are either very ill or in danger of becoming so. Phaeochromocytoma provides an excellent example of this, and the grave illness which can arise from it is often largely dependent on hypovolaemia. The recognition of hypovolaemia can be readily made from the demonstration of a gradient between the central and peripheral temperatures. Additional information is provided by the central venous pressure (which will be low), a low urinary output, and perhaps a low arterial pressure. It is rarely necessary clinically to make actual blood volume estimations. Temperature records form a simple and valuable guide to the presence of and to the treatment of hypovolaemia.

The central temperature can be recorded from the rectum, from the nasopharynx, from the lower one-third of the oesophagus, or from the tympanic membrane. The peripheral temperature is best recorded from the ball or the medial side of the big toe. The temperature at these sites is conveniently and efficiently recorded by an electrothermometer, many examples of which are now available.

It has long been known that removal of a phaeochromocytoma may be followed by a shock-like collapse, and it was thought that this was due to the sudden removal of the vasoconstrictor substances that were being produced by the tumour and that the cir-

*Fellows interested in submitting papers for consideration with a view to publication in this series should first write to the Editor.

culatory collapse should be treated or prevented by administering vasoconstrictor drugs such as noradrenaline, and this had become a routine procedure. It is now recognized that the essential cause of the postoperative circulatory collapse in these cases is related to hypovolaemia that may be revealed or worsened by the operation. I am indebted to Professor R B Welbourn for his advice and guidance in this matter and also to some of my associates at Guy's Hospital.

As long ago as 1942 Kaltreider *et al.*[4] found that prolonged infusion of adrenaline caused a reduction in the circulating plasma volume and this was again demonstrated in 1958 by Finnerty and his colleagues[5] in the case of noradrenaline. Finnerty *et al.* noted that the acute reduction of arterial pressure in some hypertensive patients is followed immediately by an increase in plasma volume and a decrease in the haematocrit and plasma protein. They decided to perform the reverse experiment by producing an acute elevation of arterial pressure, and for this they selected noradrenaline as the pressor agent. Their observations on 10 subjects (5 men and 5 women) revealed that an average increase of 15% in the maximum arterial pressure was associated with an average decrease of 15% in plasma volume, no change in the red cell count, an average increase of 8% in the haematocrit, and an average rise of 5% in plasma protein.

The first report on the estimation of blood volume in patients with phaeochromocytoma came from Brunje *et al.*[6] who reported on 3 patients. They recalled that although the shock-like collapse following removal of a tumour was regarded as due to a vasopressor deficiency, little attention had been paid to the actual mechanism producing the shock. They postulated that the prolonged high level of circulating vasopressors from a phaeochromocytoma might cause a chronic decrease in the total blood volume and if this were so the removal of the source of the vasopressors should result in the relaxation of the vascular bed to normal, in turn resulting in hypovolaemic shock unless the reduced blood volume was restored to normal.

The first of their 3 patients began to develop shock as the tumour vessels were ligated. Vasopressors were given and the systolic blood pressure rose to 200 mm Hg (26.6 kPa), but the pulse was so weak that the diastolic pressure could not be recorded. One litre of blood was given and to this the patient responded well. In their next 2 patients they estimated the plasma volume and whole blood volume before and after operation. The first of these two patients had a whole blood volume of 2.978 l before operation, with a predicted normal volume of 3.720 l. He was given 1 l of blood before operation to correct the reduced blood volume and during operation another litre of blood to replace blood loss. The blood pressure was reasonably well controlled during and after operation and the blood volume on the second day had expanded to 3.6 l. The second patient had a whole blood volume of 5.274 l before operation. During the operation the estimated blood loss was 1.5 l and 3 l was given. No vasopressors were needed during or after operation. The blood volume after operation was 5.72 l, the predicted volume being 5.65 l.

Several authors have recorded blood volume measurements in their cases; Leather *et al.*[7], for example, recorded 6 cases, in 2 of which before operation they found a diminution in total blood volume. After operation the blood volume and haematocrit ratio returned to normal. Most authors now state that hypovolaemia may be present as a result of phaeochromocytoma and suggest that this should be treated by intravenous plasma or blood, but they do not give many measure-

ments. It is felt that the circulating blood volume is only infrequently decreased but that more fluid than is lost at operation must be provided because of dilatation of the vascular bed following the removal of the tumour.

Professor Welbourn has shown me details of 3 of his own patients before and after removal of a phaeochromocytoma and comments that there is evidence that the blood volume is reduced in some patients but not in all. He advises that more careful measurements of blood volume are required at all stages of therapy in more patients.

Two patients operated on at Guy's Hospital, both by Mr Noel Glover, to whom I am indebted for permission to refer to them, illustrate the important part that management of hypovolaemia may play. One was a man, 62 years old, with several years of illness who was admitted as an emergency and had three attacks in which the arterial pressure rose to very high figures and was then followed by hypotension (110/80 mm Hg (14.6/10.6 kPa)), which caused him to collapse. After three attacks in quick succession the correct diagnosis was appreciated and he was given intravenous phenoxybenzamine; this was followed by a fall of arterial pressure to 70/50 mm Hg (9.3/6.7 kPa). The central venous pressure was zero and hypovolaemia was recognized to be present. Over a period of 2 h 4.5 l of normal saline was given and the blood pressure rose to 100/70 mm Hg (13.3/9.3 kPa) and the venous pressure rose to 5 cm H_2O (0.49 kPa). Unfortunately an electric thermometer was not available, but the extremities were noted to be cold. No blood volume estimations were made. Fluids were given by mouth and the intravenous drip was maintained. Although no precise observations are available, he presumably continued to be reasonably normovolaemic because the clinical course continued stable. Radiographic studies had shown a mass in the right renal region and this was removed by Mr Glover. The measured blood loss was 155 ml and 1 l of blood was given. His clinical course was completely stable and the central and peripheral temperatures were acceptable (rectal 36°C, and toe 32°C) on return to the intensive care unit and then rapidly stabilized at 37°C and 35°C. The central venous pressure was 5 cm H_2O (0.49 kPa).

A second patient, a man 46 years old, was also admitted as an emergency. He had cold extremities and was drenched in sweat, and no peripheral pulses could be felt. His blood pressure was not recordable. The venous pressure was low. On the next day he had improved, his limbs were warm, and a vanillyl mandelic acid test was positive. A left arteriogram showed a tumour and this was removed by Mr Glover at 7 p.m. During the night the patient's condition was grave; the arterial pressure was 60/50 mm Hg (8/6.7 kPa) and the central venous pressure, which had been as high as 11 cm H_2O (1.08 kPa), fell sharply to 5 cm H_2O (0.49 kPa), 4 cm H_2O (0.39 kPa), and 3 cm H_2O (0.29 kPa) and at 2 a.m. was -4 cm H_2O (-0.39 kPa). Blood transfusion of 1 l was given and the total fluid administered was 2.89 l; the urinary output was 617 ml. The giving of this fluid caused the arterial pressure to rise slowly to 100/70 mm Hg (13.3/9.3 kPa) and the central venous pressure rose to 3 cm H_2O (0.29 kPa). At this time central and peripheral temperatures were recorded, the rectal being 39°C and the toe temperature 35°C, and thereafter they continued at about this level. But during the preceding 14 h, when no temperature records were being made, it is doubtful whether they had been at this level, seeing that the patient had received nearly 3 l of fluid. From all other experiences there would have been a

large temperature gradient. From 12 noon until 12 midnight on this day his fluid intake, by mouth and intravenously, totalled 4.93 l—that is, since operation he had received virtually 5 l. By then the rectal and toe temperatures were steady, the central venous pressure was 11 cm H_2O (1.08 kPa), and the arterial pressure was 90/70 mm Hg (12/9.3 kPa). Thereafter his condition continued stable. The conclusion is irresistible that he had been grossly hypovolaemic and the recognition of this led to abandonment of the use of noradrenaline that had been used until midnight under the time-honoured assumption that it was what was needed. The vigorous treatment of the hypovolaemia led to his recovery as soon as the true need was recognized.

The existence of hypovolaemia should be recognized from the general features of the rapid, poor-volume pulse, the low arterial and low central venous pressure, and low urinary output. The recording of a significant gradient between the central and the peripheral temperatures (rectal and toe) will confirm this, and indeed the appearance of a temperature gradient should serve as a much earlier warning of impending circulatory decline. The recording of these temperatures should be a routine in any patient in whom circulatory deterioration is likely to occur. The observations are simple to make and provide reliable information about the volaemic state without the need to call upon actual blood volume estimations. Moreover, the temperature records form a sensitive, accurate, and invaluable guide to the treatment of the hypovolaemia and its progress to clinical control.

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