A CASE OF POSTOPERATIVE RESPIRATORY INSUFFICIENCY AND PROLONGED UNCONSCIOUSNESS

BY

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In 1955, Rukes et al. described the dramatic response of several cases of pre- and postoperative shock to the administration of intravenous hydrocortisone where standard methods of treatment by blood transfusion and vasopressor agents had failed.

Our interest in the possible value of hydrocortisone in the treatment of some cases of postoperative respiratory insufficiency was roused by Hugín's (1957) analysis of Hunter's (1956) cases of "neostigmine-resistant curarization" especially by his observation that some of these patients presented a clinical picture suggestive of acute suprarenal cortical insufficiency. His own words on the subject are: "It seems to me possible that these patients could be saved by the rapid infusion of hydrocortisone, and there is probably no connection between this complication and the use of relaxant drugs or neostigmine."

As such cases fortunately occur infrequently in any one anaesthetist's practice, it is perhaps of interest to record a recent one of this type in which intravenous hydrocortisone formed part of the therapy.

CASE REPORT

A 78-year-old man was admitted to hospital at 2.15 p.m. on 27.5.57 with a history of severe lower abdominal pain of 24 hours duration. There was no history of vomiting, constipation or diarrhoea. Among the positive findings were bilateral reducible inguinal herniae and generalized abdominal tenderness, most marked in the right lower quadrant. Temperature 100.2° F. Pulse rate 110/minute. Respiratory rate 24/minute. B.P. 145/90 mm mercury. Urine: reaction acid with a trace of albumin.

He had previously had operations for repair of inguinal herniae in 1935 and 1945 and an operation for intestinal obstruction in 1947.

After examining the patient at 3 p.m., the surgeon decided that laparotomy should be performed and ordered morphine sulphate 10 mg subcutaneously. The patient was seen 2 hours later by the anaesthetist who considered him to be remarkably alert mentally for his age of 78. He was a big, robust man with a slightly toxic appearance, but with no apparent dehydration. Pre-operative medication of atropine sulphate 0.6 mg subcutaneously was ordered.

At 6 p.m. laparotomy was performed and the findings were:

1. Generalized purulent peritoneal exudate.
2. Paralytic distension of small and large intestines.
3. Gangrenous perforated appendix.
4. Incomplete obstruction of ileum by an omental band.
5. Loop of ileum in right inguinal hernial sac.

The operation consisted of appendicectomy and division of the omental band.

Anaesthetic Procedure.
Anaesthesia was induced with thiopentone sodium 300 mg given slowly intravenously, followed by d-tubocurarine chloride 20 mg. After rhythmic inflation of the lungs for two minutes, intubation was attempted unsuccessfully due to coughing and movement of the patient's head and arms. Following a supplementary intravenous injection of 200 mg thiopentone sodium, he settled and intubation with a no. 10 cuffed endotracheal tube was performed with ease.

Anaesthesia was maintained with nitrous oxide 3 litres and oxygen 1½ litres employing a Waters' to-and-fro rebreathing unit with controlled leak, and assisted respiration. Within 10 minutes of induction of anaesthesia the patient was breathing again with vigour, and in order to facilitate exploration of the abdomen a further 10 mg d-tubocurarine and 25 mg pethidine hydrochloride were given. Before closure of the peritoneum, 5 mg d-tubocurarine was administered.

Resuscitation.
At the conclusion of the operation, the administration of neostigmine methyl sulphate 2.5 mg, preceded by atropine sulphate 0.6 mg, resulted in regular respiration of adequate amplitude, but the patient remained unconscious. Throughout operation his condition had remained good, blood pressure and pulse maintaining their pre-operative values. Blood loss had been negligible.

On his return to the ward at 7.30 p.m. the hitherto adequate tidal exchange gave place to an inadequate diaphragmatic respiration with tracheal tug, whose response to intravenous injections of atropine and neostigmine in the previous dosage was short lived.
Re-intubation was performed with ease and with no reaction other than an occasional extrasystole over the next few minutes. There was no response either to inflation with 100 per cent oxygen or to inflation with 6 per cent carbon dioxide in oxygen. Intermittent positive pressure respiration with CO₂ absorption was then re-instituted. An intravenous infusion of 5 per cent dextrose in saline was set up. A short period of deep thoraco-abdominal respiration followed the administration of 4 ml 25 per cent solution of nikethamide but there was no alteration in depth of consciousness. Since 500 mg thiopentone is a very large dose in the aged, bemegride 25 mg was administered intravenously, but this failed to alter the amplitude of respiration or the level of consciousness. The patient remained deeply comatose with pupils small, but not pin point and unresponsive to direct light.

At 11.15 p.m. an intravenous solution of hydrocortisone 100 mg in 540 ml dextrose 5 per cent in water was run in rapidly and towards the end of this infusion the tone of the jaw muscles increased markedly and the tracheal tug disappeared. Respiration increased in amplitude to such a degree that assistance would no longer tolerate the endotracheal tube. Moreover for the first time respiration became mainly thoracic in type.

Despite the fact that the improvement in respiration was maintained the systolic blood pressure fell 1 hour later to 90 mm mercury. 15 mg methylamphetamine sulphate was given intravenously followed by 15 mg intramuscularly with no immediate response. Two hours later the systolic blood pressure was still 90 mm mercury but respiration had become more shallow. The administration of nikethamide in the previous dosage resulted in a similar increase in amplitude of respiration for about 2 minutes. From this point onwards, his tidal volume and blood pressure remained unchanged, but the level of consciousness gradually lightened until at 8.30 a.m. the patient would no longer tolerate the endotracheal tube. At 10 a.m. he was fully conscious and able to speak clearly and intelligently.

Blood Chemistry.

Records of serum electrolytes are not available for the pre-operative period but the results obtained on the first postoperative day are as follows:

- Serum urea: 91 mg per cent.
- Serum chlorides: 96 m.equiv./l.
- Serum alkali reserve: 24.8 m.equiv./l.
- Serum potassium: 6.2 m.equiv./l.

DISCUSSION

Over the past year several anaesthetists have reported the occurrence of postoperative respiratory insufficiency, generally in elderly and/or extremely ill patients. (Hunter, 1956; Foster, 1956; Kenny, 1956; Burchell, 1956). The dramatic improvement following intravenous hydrocortisone suggests that in this patient at least the response to anaesthesia and operation was due to a modification of suprarenal cortical activity. Foster (1956) administered 17-hydroxycorticosterone to two patients suffering from postoperative respiratory insufficiency, one of whom recovered consciousness temporarily. It would appear that the second patient, who also died, was already in a state of advanced circulatory failure when the drug was administered.

A diagnosis of adrenocortical insufficiency in such patients is not unreasonable. As a result of the probable atrophy of the suprarenal glands in old age a modification of the response to stress is not unlikely. In this case the stress was considerable with infection, haemoconcentration, electrolyte imbalance, narcosis and operation as contributory factors.

Foster (1956) after administration of potassium to several such patients with some success, concluded that the postoperative respiratory insufficiency was related to low intracellular potassium which allowed molecules of muscle relaxant to penetrate the blood/brain barrier and thus exert a central depressant action on brain cells. It has been shown however, that an increase in the potassium content of blood plasma may result in increased corticoid secretion (Vogt, 1951) and it is possible that this was partly the reason for the response of some patients in Foster's series to the intravenous administration of potassium solutions.

From the time of medication pre-operatively to that of prolonged unconsciousness post-operatively, the response of this patient to narcotics was abnormal. Two hours after the administration of 10 mg morphine subcutaneously this 78-year-old normotensive patient was very alert. A further abnormal reaction was the apparent resistance to the hypnotic action of thiopentone. In contrast, the duration and depth of unconsciousness post-operatively were not in keeping with this initial resistance. It is possible that the duration of unconsciousness may have been prolonged by the administration of dextrose solutions of which he had 1 litre of 5 per cent. Although the effect of administration of glucose on subjects under thiobarbiturate narcosis has been demonstrated only in experimental animals (Lamson, Greig and Robbins, 1949). An annotation (Lancet, 1950) warns of the possibility in humans.

Caughey and Garrod (1954) in a study of coma and disturbances of consciousness in hypopituitarism have noted that endocrine failure, particularly
of the suprarenal cortex, seemed to be an important factor in lowering the threshold of consciousness. It has also been noted (Dundee, 1957) that prolonged unconsciousness may occur in patients suffering from Addison’s Disease in whom replacement therapy had been inadequate, particularly when such patients are already under the influence of opiates and thiopentone. Dundee has also reported cases of prolonged unconsciousness and circulatory failure following anaesthesia in cases of drug-induced adrenocortical insufficiency.

The response of the brain to narcotics depends on the rate of cerebral metabolism and this latter may be altered by the blood level of adrenocorticooids. It seems reasonable to imagine, “without penetrating ever deeper into the maze of speculative pharmacology” (Hewer, 1957), that under a condition of altered cerebral metabolism due to modification of corticoid secretion an abnormal response to narcotics may be produced.

SUMMARY

A case of postoperative respiratory insufficiency and prolonged unconsciousness is presented.

The management is described and the possible aetiological factors discussed.

ACKNOWLEDGMENTS

We should like to thank Mr. F. Smith for his permission to publish the details of the case, and Dr. O. M. Watt for his encouragement and interest.

REFERENCES


