HALOTHANE AND BLOOD TRANSFUSION FOR PHAEOCHROMOCYTOMA
A Case Report

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SUMMARY

The management of a case of phaeochromocytoma is described, halothane being used as the main anaesthetic and blood transfusion given specifically to control the post-operative fall in blood pressure. Noradrenaline was not needed.

Operations for the deliberate removal of a phaeochromocytoma are fairly rare. This case is presented because the details of its management were stimulated by suggestions in a recent article (Rollason, 1964) that halothane may be an agent of choice, in spite of theoretical considerations, and that blood transfusion may avoid the need for noradrenaline after the vasoconstrictor tone is released. It is felt that our experience may be of value to others faced with a similar problem.

Halothane was used but, in spite of this, phentolamine was needed to control quite large swings of blood pressure. After removal of the tumour, however, control of hypotension was achieved by the use of blood alone and, although noradrenaline was kept in readiness, it was not used.

CASE REPORT

The patient admitted on May 27, 1964, was a married woman of 28 years with a 10-year history of intermittent attacks of palpitations accompanied by a throbbing sensation throughout the body. These became progressively more frequent and had been occurring almost daily for the past 2 years. She often vomited during attacks. There was no chest pain but a mild exertional dyspnoea had been present for 2 years. She had four children, three of them born within the last 10 years and the last, 9 months ago.

On examination, she was found to be an anxious woman of good build and nutrition though clinically anaemic, weight 104 lb., blood pressure 130/80 mm Hg and pulse 106 beats/min. The apex beat was half an inch lateral to the nipple line but there were no signs of cardiac failure. The liver edge was just palpable. There were no other abnormalities in the abdomen and no free fluid. There were no abnormal signs in the respiratory system. The haemoglobin was 11.5 gm/100 ml.

Two days after admission she had an attack of palpitations with some dyspnoea and was started on meprobamate. With an ambient ward temperature of 100°F, sweating, although present, was not a helpful clinical sign. On June 2, 1964, an electrocardiogram showed sinus tachycardia with evidence of anterolateral coronary ischaemia. The blood sugar was 144 mg per cent. An augmented histamine test done at this time produced headache, palpitations and vomiting but no rise in blood pressure.

On the following day, however, during a further attack, her blood pressure was found to be 250/150 mm Hg.

A phentolamine test during a subsequent attack produced a marked drop in blood pressure and a 24 hour specimen of urine contained 2000 mg catecholamines. A diagnosis of phaeochromocytoma was made. Intravenous pyelography, done on June 11, 1964, showed very poor excretion from both kidneys and a phenolsulphathalein test gave only 13 per cent excretion of the dye. In spite of this, the blood urea was 37 mg/100 ml, but the maximum concentration of urine was 1/10.

Radiography of the chest revealed no abnormality. Grade I and II hypertensive changes were present in the fundi. She continued to have daily hypertensive episodes which were controlled by phentolamine and on June 18, 1964, surgery was decided upon. The blood pressure rose to about 280/170 mm Hg in each attack and dropped to about 110/80 mm Hg after phentolamine.

Although it is said (Mathews, 1963) that the use of chlorpromazine makes the maintenance of arterial pressure troublesome and that this may be greater in a patient for whom hypotension may be anticipated, with the possibility of an inverted response to adrenaline, it was given for a week before operation in doses of 50 mg t.d.s. intramuscularly in an effort to control the attacks. They were largely controlled in this way and only one dose of phentolamine was required during this time for, although attacks occurred, the blood pressure did not rise to such high levels and the symptoms were less distressing.

Some difficulty in obtaining blood for transfusion meant postponement of the operation but it was finally performed on July 3, 1964.

Chlorpromazine medication was stopped 2 days before operation. Pre-operatively, she was sedated with oral butobarbitone 180 mg for the night and was given an intramuscular injection of morphine 10 mg prome-
thazine 50 mg and phentolamine 5 mg 1 hour before surgery.

On arrival in the theatre she was well sedated. The systolic blood pressure was 120 mm Hg and the pulse 100 beats/min.

Anaesthesia was induced with thiopentone 300 mg given slowly. This was followed with 3 per cent halothane, using the Macintosh induction inhaler with oxygen added to the air inlet by a T-piece system so that the final mixture inhaled by the patient contained 30 per cent oxygen. Once asleep, suxamethonium 50 mg was given. There were no fasciculations. The lungs were well inflated after reducing the concentration of halothane to 0.5 per cent and a No. 10 cuffed Magill orotracheal tube passed. Thereafter, ventilation was controlled using the Oxford Bellows and Mitchell inflating valve. The blood pressure did not rise. Bilateral exploration was intended and no further positioning was necessary. Slight movement, as the operation commenced, necessitated an increase in the concentration of halothane to 2 per cent but this caused the systolic pressure to drop to 80 mm Hg and it was felt unwise to continue in this way as it rose immediately the percentage was reduced, therefore tubocurarine 10 mg was added to enable 0.5–1 per cent halothane to be used. In spite of this the pressure again dropped but as exploration of the abdomen commenced, there was an abrupt rise to 250 mm Hg.

A large tumour was located above the right kidney and, although phentolamine was given as indicated in figure 1, the halothane concentration increased to 2 per cent for periods, and the surgery discontinued for short periods, wild swings in blood pressure became very difficult to control. Apart from a few extrasystoles at the first rise, the pulse remained regular and remarkably steady. A further 5 mm dose of tubocurarine was given during this phase.

As the last vein was clamped and the blood pressure started to fall, the halothane was reduced to 0.5 per cent and blood was run in rapidly, 800 ml being given during the succeeding half hour. The operative blood loss was very small. Noradrenaline was held in readiness but not needed as the drop was halted at 120 mm Hg.

There was no change whilst the abdomen was closed. The patient woke quietly on extubation and was taken to the recovery room. There, the systolic pressure dropped to 100 mm Hg; a further 400 ml of blood was quickly given which brought it back to 120 mm Hg and the pressure finally stabilized at about 110 mm Hg. Due to a misunderstanding, a noradrenaline drip containing 4 mg was started during the night following operation but this was later stopped and had no marked effect on the blood pressure. No other specific treatment was needed and the postoperative course was uneventful.

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**Fig. 1**

Record of blood pressure during operation.
DISCUSSION

This was a large and active tumour and the control of swings of blood pressure during the operation was not very successful. Rollason (1964) used 4 per cent halothane but our vaporizer delivers a maximum of 3 per cent and, moreover, early falls in arterial pressure with 2 per cent made us wary of risking a higher concentration. It was noteworthy, however, that there were no serious pulse irregularities as far as could be determined without electrocardiographic monitoring. The reason for this is not clear in view of the reports of undoubted ventricular fibrillation in the presence of halothane and adrenaline. Presumably, the cardiac muscle becomes relatively resistant to the effects of adrenaline after being subjected to high levels for a long period. Nevertheless a defibrillator was available in the theatre during the operation. In view of the response of the blood pressure to concentrations of halothane sufficient to permit control of ventilation and movement, tubocurarine was chosen instead of gallamine as the muscle relaxant so that the concentration of halothane could be reduced. The possibility of tachycardia occurring with gallamine was considered as the muscle relaxant so that the concentration of halothane could be reduced. The possibility of tachycardia occurring with gallamine was considered to outweigh the slight risk of hypotension with small doses of tubocurarine.

Phentolamine was given intermittently into the intravenous drip but it is probable that a continuous drip would have given smoother control of the blood pressure.

Following the suggestion of Rollason (1964) blood was given rapidly as soon as the tumour was removed. Although loss had been very small a total of 1200 ml was transfused within 1½ hours of its excision. Following this there was no evidence of raised venous pressure.

The theory that the blood volume is low due to vasoconstriction is difficult to understand since the constriction is intermittent. Unfortunately, blood volume studies were not done on this case, but it is doubtful if the only method available to us (Evans’s Blue dilution) would have given sufficiently reliable information. At first the theory would seem analogous to the situation in “shock” in which pharmacological release of vasoconstriction and immediate rapid transfusion of blood may restore the blood pressure effectively, yet, so far, there is no good evidence of permanent vasoconstriction in cases of phaeochromocytoma.

This would, however, not be surprising if we assume a continuing level of catecholamines in the blood insufficient except during a hypertensive attack to do more than produce a very mild elevation of blood pressure. If this were so it would seem rational to give blood when the level of catecholamines falls after removal of the tumour, giving time for restitution of the normal mechanisms for maintaining vasomotor tone. Certain it is that the use of noradrenaline is not an easy answer to the problem because large doses may be needed (Hardy, McPhail and Gallagher, 1962) possibly because of its storage in an inactive form at receptor sites (Burn and Rand, 1959). Also the response may be disappointing and then there are the difficulties involved in withdrawing noradrenaline treatment.

One patient cannot form a basis for conclusions but at least the result has been encouraging and will, we hope, spur others to investigate further with particular reference to pre- and postoperative blood volume studies in such cases.

REFERENCES


HALOTHANE ET TRANFUSION SANGUINE DANS UN PHAEOCHROMOCYTOME

DESCRIPTION DU TRAITEMENT D'UN CAS DE PHAEOCHROMOCYTOME OÙ L'HALOTHANE A ÉTÉ UTILISÉ COMME ANESTHÉSIQUE PRINCIPAL ET DES TRANSFUSIONS SANGUINES ONT ÉTÉ FAITES SPÉCIFIQUES POUR CONTRÔLER LA CHUTE TENSIONNELLE POST-OPÉRATOIRE. ON N'EN N'A PAS EU BESOIN DE NORA-DRÉNALINE.

HALOTHAN UND TRANSFUSION BEI PHAEOCHROMOZYTOM

ZUSAMMENFASSUNG

Es wird die Durchführung der Narkose bei einem Fall von Phäochromozytom beschrieben. Halothan wurde als das Hauptanästhetikum verwandt und eine Transfusion hauptsächlich zur Kontrolle des postoperativen Abfalles des Blutdruckes gegeben. Die Anwendung von Noradrenalin war nicht notwendig.