

ANAESTHETIC MANAGEMENT FOR THE EXCISION OF CAROTID BODY TUMOURS — A REPORT OF TWO CASES

W K Van

SYNOPSIS

Carotid body tumours are extremely rare tumours arising from the chromaffin tissue of the carotid sinus. Very little has been written on the anaesthetic management during surgery to excise such tumours. Yet, for the unwary anaesthetist, the anaesthetic course is fraught with dangers.

This paper presents two patients with such tumours. Between the two cases, most of all the possible complications that may occur are illustrated. These include difficult intubation, hypertensive crises during the surgical manipulation of the tumour, sudden torrential haemorrhage, reflex bradycardia and sudden hypotension after excision of the tumour.

INTRODUCTION

Carotid body tumours are extremely rare tumours. Marchand first described this fleshy, highly vascular and usually well circumscribed tumour in 1891 (1). Up till 1979 only 600 such tumours had been described in English literature (2). Dent, Thompson and Fry in America have collected only 16 cases in 30 years (3). It comes as no surprise, therefore, that there is a paucity of anaesthetic literature concerning the anaesthetic management of such tumours. Yet anaesthesia for the removal of such tumours are fraught with problems for the anaesthetist who is not wary of them.

Department of Anaesthesia
Tan Tock Seng Hospital
Moulmein Road
Singapore 1130

W K Van, MBBS, FFARACS, AM
Senior Registrar

CASE HISTORIES

Case No 1

M.S.A., a 46 year old male, weighing 62 kg presented with a history of a lump at the left angle of the jaw for 6 months. The mass was also clearly visible in the oropharynx and extended past the midline. There was no evidence of Horner's Syndrome or of any cranial nerve palsies.

The patient was hypertensive with blood pressure readings ranging from 150/100 mm. Hg. to 160/110 mm. Hg. at rest. He was being treated with methyl-dopa, 125 mg., twice a day. His EKG however showed a marked degree of left ventricular hypertrophy. His blood chemistry was within normal limits. An assay for vanillyl-mandelic acid was not done.

A pre-operative angiogram showed a very vascular tumour. Compressing the left carotid artery demonstrated a good cerebral cross-flow from the right carotid artery.

The patient was pre-medicated with atropine 0.3 mg., morphine 15 mg., and promethazine 25 mg. After establishing an intra-venous line, anaesthesia was induced with 375 mg. of thiopentone. After a bolus dose of 100 mg. of succinyl di-choline, the patient was intubated with a #8.5 nasotracheal tube. Nasotracheal intubation was chosen as the surgeon had indicated that there was a possibility that intra-oral work may be carried out. No difficulty was encountered during intubation despite a huge mass in the oro-pharynx.

He was then paralysed with d-Tubocurarine and artificially ventilated with a 30% N₂O:70% O₂ mixture. PaCO₂ was maintained at around 30-35 torr.

The heart rate and rhythm were continuously monitored using lead II of a 3 lead EKG. The blood pressure was monitored via an intra-arterial line.

During the early phases of neck dissection, the blood pressure rose from 170/100 mm. Hg. to 200/130 mm. Hg. This surge in blood pressure was brought under control by an infusion of 0.01% sodium nitro-prusside. By titrating the rate of infusion of sodium nitro-prusside, the blood pressure was maintained at 150/90 mm. Hg. At one stage of the dissection, torrential haemorrhage caused the blood pressure to plunge to 110/50 mm. Hg. This was rapidly corrected by blood transfusions.

In the final stages of surgical dissection, traction on the tumour caused the heart rate to fall to 30/min. with a concomitant fall in blood pressure. This bradycardia, however, responded to a bolus injection of 0.6 mg. of atropine.

After complete excision of the tumour, the blood pressure settled at 150/70 mm. Hg. without the need for sodium nitro-prusside infusion. Closure of the surgical incision proceeded without further changes in heart rate or blood pressure.

The patient was reversed with the usual atropine/neostigmine mixture and extubated. Post-operative recovery was uneventful.

Case No 2

L.H.K., a 29 year old female weighing 36.6 kg. presented with a history of having had a lump at the side of the neck for 9 years. She only sought medical attention because she felt a numbness over the lump.

Except for a lump measuring 4.5 cm x 5.0 cm x 2.5 cm on the left side of the neck near the angle of the jaw, there were no other pathological findings. She had a blood pressure of 12/70 mm. Hg. and a resting pulse of 90/min. There was no oro-pharyngeal exten-

sion of the mass.

Her blood chemistry was essentially normal. No assay for vanillyl-mandelic acid was done. A carotid angiogram showed a vascular tumour. Cross compression studies showed good blood flow from the contralateral cerebral vessels.

The patient was pre-medicated with atropine 0.6 mg., morphine 10 mg., and promethazine 25 mg. Anaesthesia was induced with thiopentone 250 mg. After muscle paralysis was achieved by a bolus dose of 60 mg. succinyl di-choline, the patient was intubated with a #8.0 naso-tracheal tube. As expected, no difficulty was encountered during intubation. The patient was artificially ventilated with a 30% N₂O:70% O₂ mixture d-Tubocurarine was used for continued muscle relaxation.

The patient's heart rate and blood pressures were continuously monitored. The blood pressure remained stable throughout the whole operation, except for two occasions when surgical manipulation of the tumour caused the blood pressure to climb to 160/100 mm. Hg. This surge of blood pressure was brought under control by an infusion of 0.01% sodium nitro-prusside. There were no significant changes in the cardiac rhythm.

Dissection of the tumour away from the internal carotid proved to be difficult and it was decided that it would be safer to remove the tumour together with the internal carotid artery. Despite radiological evidence of good cerebral cross-flow, a stump pressure reading was taken. A stump pressure of 75 mm. Hg. was recorded while the patient's blood pressure was 120/70 mm. Hg.

After the completion of surgery, the patient was reversed with atropine and neostigmine. The patient remained drowsy although she was breathing adequately. She was not extubated and was discharged to the post-operative ward on a T-piece system. 2 hours later, the patient became fully conscious and was extubated. Subsequent post-operative course was uneventful.

DISCUSSION

The carotid bodies, situated at the bifurcation of the common carotid artery serve a very important function of detecting changes in arterial oxygen tension. The cells from which carotid body tumours arise from are thought to have their origin from the neuro-ectoderm (4) and are generally considered by some pathologists as one of the sub-types of APUDomas. There are others who feel that these rare tumours be classified under a group of their own (4, 5).

These tumours can on occasion secrete catecholamines (6) and are also known to be commonly associated with pheochromocytomas (7). Pre-operative catecholamine studies are of great value in cases like these (8). It was regrettable that no such studies were done on these 2 patients especially since the patient in Case No 1 was hypertensive.

Manipulation of these tumours may release catecholamines and lead to dangerous surges of blood pressure (4). These rises in blood pressure were seen in both the patients reported here. These abrupt increases of blood pressure can be readily controlled by vaso-dilators like sodium nitro-prusside or by alpha-adrenergic blockers like phentolamine (6). Pre-operative treatment with long acting alpha adrenergic blockers like phenoxy-benzamine should also be considered; especially when pre-operative catecholamine levels are elevated (4).

Hypotension is another problem that might be

troublesome after the carotid body tumour has been excised. The sudden decrease of catecholamines may necessitate the use of a nor-adrenaline infusion to correct the profound hypotension (2). While no such event occurred in the two patients reported here, the patient in Case No 1 did settle at a lower post-operative blood pressure than that pre-operatively.

A careful discussion with the surgeon as to his plan of action and a careful clinical examination of the patient is vital. A decision to perform intra-oral surgery might make naso-tracheal intubation necessary. On the other hand, a huge oro-pharyngeal extension of the tumour may make even oro-tracheal intubation a hazardous procedure.

Carotid angiography is an important pre-operative study (2). Not only does it determine the integrity of the cerebral cross circulation should resection of the carotid vessels be necessary, it also may show up an undiagnosed contra-lateral carotid body tumour. Even then, when a decision to resect the carotid artery is taken, a stump pressure reading should be done as an intra-operative check. A level of at least 60 mm. Hg is regarded to be indicative of adequate cerebral cross circulation.

Massive torrential haemorrhage may yet be another problem the anaesthetist has to face during this procedure (3). While some tumours may be easily dissected from the carotid bifurcation, there have been reported occasions when dissection of particularly adherent tumours have resulted in average blood losses of 2000 ml. It would seem prudent to have at least 4 units of blood readily available all the time.

Bradycardia, severe enough to cause falls in blood pressures, may also occur during dissection of the tumour. Unlike carotid endarterectomies where it would be relatively simple to infiltrate lignocaine 1.0% around the carotid bulb to block the afferent impulses, this manouvre is not possible in the presence of a huge, vascular, fusiform tumour occupying the carotid bifurcation. However, as demonstrated in Case No 2, atropine seemed to circumvent the problem effectively.

Neurological complications may be noticed post-operatively especially if dissection has been particularly difficult. These include hemiplegia, recurrent laryngeal nerve palsy, Horner's syndrome and hypoglossal nerve palsy (10). A diminished response to

hypoxia (11) and disturbances in baroreceptor function (12) may also occur if bilateral removal of carotid bodies has been carried out.

ACKNOWLEDGEMENTS

The author would like to thank Mr G Baratham, Senior Neurosurgeon, Tan Tock Seng Hospital; Dr K H Ng, former Consultant, Department of Anaesthesia, Tan Tock Seng Hospital and Dr Alex S C Lee for their help in these two case studies.

REFERENCES

1. Gratiot J H: Carotid body tumours: Collective review. *International Abstracts of Surgery* 1943; 77:177-86.
2. Wright D J, Pandya A, Noel F: Anaesthesia for carotid body resection; a case report and review of literature. *Anaesthesia* 1979; 34:806-8.
3. Dent T L, Thompson N W, Fry W J: Carotid body tumours. *Surgery* 1976; 80:365-72.
4. Levit S A, Sheps S G, Episona R E, Remine W H, Harrison E G: Catecholamine secreting paraganglioma of the glomus jugulare region resembling pheochromocytoma. *New England Journal of Medicine* 1969; 281:805-11.
5. Carotid Body Tumour In: Bloodworth J M B, *Endocrine Pathology*, 2nd. Edition, Publishers: William & Wilkins. 497-8.
6. Clarke A D, Matheson H, Boddies H G, Removal of a catecholamine secreting chemodectoma. *Anaesthesia* 1976; 31:1225-30.
7. Sato T, Saito H, Yoshinaga K, Shibota Y, Sasano N: Concurrence of carotid body tumour and pheochromocytoma. *Cancer* 1974, 34, 1787-95.
8. Lee C D, Levine H L, Bevin E G, Tucker H M: Tumours of the carotid body: Experience with 41 operative cases. *American Journal of Surgery* 1981; 142:362-5.
9. McKay R D, Sundt T M, Michenfelder J D, Gronert G A, Messick J M, Sharbrough F W, Piepgras D G: Internal carotid artery stump pressure and cerebral blood flow during carotid endarterectomy. *Anesthesiology* 1976; 45:390-9.
10. MacComb W S: Carotid Body Tumours. *Annals of Surgery* 1948; 127:269-77.
11. Nakayama K: The surgical significance of the carotid body in relation to bronchial asthma. *Journal of the International College of Surgeons* 1963; 39:374-89.
12. Holton P, Wood J B: The effects of bilateral removal of the carotid body and denervation of the carotid sinuses in two human subjects. *Journal of Physiology (Lond.)* 1965; 181:365-78.