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The Use of Magnesium Sulfate in the Anesthetic Management of Pheochromocytoma

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The perioperative management of a patient with a pheochromocytoma requires adequate adrenergic receptor blockade and minimizing stimuli that may provoke catecholamine release. Preoperative preparation usually entails the use of alpha-adrenergic blockers such as phenoxybenzamine, careful iv fluid replacement, and beta-adrenergic blockers if required. Intraoperatively, control of the effects of excessive catecholamine release usually is maintained by deep anesthesia, coupled with the use of adrenergic receptor blocking drugs such as phentolamine and propranolol and/or sodium nitroprusside. Despite all of these therapeutic maneuvers, the total elimination of cardiovascular disturbances seldom is achieved and some fluctuation in arterial blood pressure and heart rate often occurs. Magnesium sulfate has been shown to inhibit the release of catecholamines from both the adrenal medulla[1,2] and peripheral adrenergic nerve terminals,[3] as well as blocking catecholamine receptors directly, in addition to a direct dilator effect on vessel walls.[4] It, therefore, appeared worthy of a trial in this condition as an adjunct to conventional therapy.

REPORT OF A CASE

A 16-year-old girl presented with peripheral gangrene and labile hypertension. A diagnosis of pheochromocytoma was confirmed by angiography and by finding elevated urinary vanilmandelic acid levels. An initial dose of phenoxybenzamine 50 mg iv was infused over 2 h, followed by 20 mg orally every 8 h for 5 days with careful iv fluid replacement monitored by central venous pressure monitoring. The first doses of phenoxybenzamine produced hypotension with tachycardia, which responded well to iv fluid therapy. Over the next few days, good cardiovascular stability was achieved without the need for beta-adrenergic receptor blockers. During this time, the supine arterial blood pressure remained within the range 110/60–130/80 mmHg, and on standing this decreased to between 100/60 and 90/40 mmHg. There were no hypertensive episodes. The heart rate remained below 100 bpm at all times, and there were no dysrhythmias detected or ECG abnormalities noted. After 3 days the patient then was judged to be ready for surgery in accordance with the criteria recommended by Roizen.[5]

She was premedicated with chlorpromazine 50 mg, papaveretum 15 mg and scopolamine 0.4 mg im as well as being given 10 mg phenoxybenzamine orally. On arrival in the anesthetic room, arterial blood pressure was 190/120 mmHg and her heart rate was 170 bpm. She was sedated further with 0.1 mg fentanyl and 10 mg droperidol iv without benefit to her cardiovascular status. Ventilation was assisted gently and 50% nitrous oxide in oxygen inhaled. Although the patient was now well sedated, the severe tachycardia and hypertension persisted, although the patient was peripherally warm and vasodilated. Because tachycardia appeared to be the major problem, and in view of the fact that several alpha-adrenergic blockers already had been given, propranolol was administered in 2-mg increments with continuous ECG monitoring and blood pressure recording using an automatic doppler device. After 10 mg of propranolol, little improvement had been achieved and arterial blood pressure was 180/120 mmHg and heart rate 149 bpm. At this point, abandonment of the procedure was contemplated, but in view of her excellent preoperative status, it was felt that this was an acute response to the stress of coming to the operating theatre. Therefore, it was decided to make one further attempt to control the cardiovascular disturbances at this time, and a trial of magnesium therapy was initiated.

The patient was transferred to the operating room, an arterial line inserted into the left radial artery under local anesthesia, and a magnesium sulfate infusion commenced at a rate of 2.0 g/min. After 90 s, the arterial blood pressure had decreased to 130/90 mmHg and the heart rate to 122 bpm. As the central venous line had become displaced prior to the patient coming to theatre, a right subclavian catheter was inserted under local anesthesia prior to induction of anesthesia. This produced a mild increase in arterial blood pressure, which responded well to infusion of an additional 2.5 g magnesium sulfate. As eyelash and pharyngeal reflexes were still present, anesthesia then was induced with 150 mg thiopental iv, paralysis with 15 mg alcuronium iv, and the trachea then intubated. There was no cardiovascular response to endotracheal intubation and no change in heart rate (fig. 1).

Surgery proceeded uneventfully under halothane, nitrous oxide, and oxygen with controlled ventilation until mobilization of the tumor was attempted. The tumor was tightly adherent to the inferior vena cava, and ligating the venous drainage proved to be impossible. Extensive tumor handling would be necessary to remove the mass, and severe disturbances of cardiovascular function were anticipated. As expected, arterial blood pressure and heart rate began to increase, and before resorting to phentolamine and sodium nitroprusside, as had been planned, a further infusion of magnesium sulfate 2.0 g terminated the upward trend, and a further 2 g restored the arterial pressure and heart rate to normal levels, despite the fact that tumor handling was continuing throughout. Despite further manipulation of the mass, there were no further cardiovascular disturbances. Following tumor removal, mild hypotension and bradycardia occurred (arterial blood pressure 80/60 mmHg and heart rate 64 bpm), but this responded adequately to iv fluid therapy alone, and inotropic support was not required, although 1 g calcium gluconate was given to counteract any remaining magnesium effects. This produced no significant change in cardiovascular status. Neuromuscular blockade

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was reversed with 2.5 mg neostigmine and 1.2 mg atropine, following which good muscle tone readily was restored and satisfactory spontaneous ventilation established. The postoperative course was uneventful.

Discussion

The anesthetic management of pheochromocytoma has been well established, and the basic scheme of treatment followed here was in accordance with that recommended by Black and Montgomery.[6] Although good control of the cardiovascular system appeared to have been established in the days preceding the operation, presumably the stress of impending surgery triggered the release of catecholamines, despite heavy premedication. The rationale for using magnesium sulfate to improve control after phenoxybenzamine preoperatively, and chlorpromazine, 10 mg of droperidol, and 10 mg of propranolol intraoperatively, had failed is based on the observations that magnesium can inhibit the release of catecholamines, as well as exert a direct action on adrenergic receptors and blood vessels.[1–4] Little use has been made of these effects in clinical practice, although we have been using the agent with success to improve the control of the autonomic disturbances of tetanus,[7] which poses a similar problem in regard to the release of catecholamines.[8,9] Magnesium sulfate has a mild but transient effect on the arterial blood pressure and maintains cardiac output, often at increased levels.[10] This may be associated with a tachycardia resulting from the inhibition of the release of acetylcholine at the vagal terminals.[11] The sustained reduction in pulse rate and blood pressure produced in this case is, therefore, most likely to have been due to antiadrenergic effects, although whether this was through inhibition of release or via a peripheral action is impossible to state with certainty. Biochemically, magnesium offers some protection to the myocardium by preventing the excessive destruction of ATP and by preserving glycogen stores. It also limits the production of lactate by heart muscle during catecholamine stimulation but does not block the adrenergically mediated increase in contractility.[12,13] The ability of magnesium to reduce cardiovascular disturbances by inhibiting catecholamine drive without depressing myocardial contractility appears to be useful in the management of patients with tetanus and may well prove to be of value in pheochromocytoma.

In summary, a case of pheochromocytoma is presented in which alpha- and beta-adrenergic blockers failed to provide adequate cardiovascular control. A preinduction infusion of 2.5 g of magnesium sulfate restored cardiovascular parameters to acceptable values. Further disturbances of cardiovascular function during tumor excision were well controlled with the use of magnesium sulfate and halothane as the only intraoperative antiadrenergic drugs.

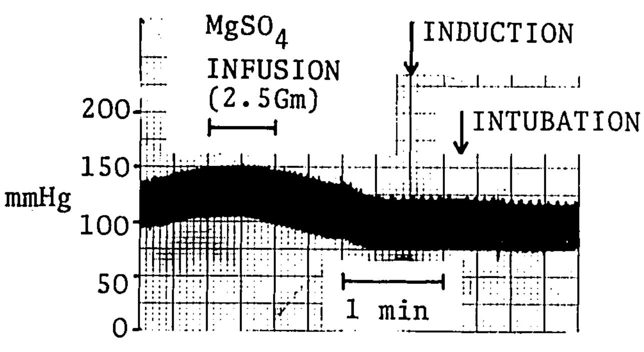

FIG. 1. The change in arterial blood pressure on insertion of the subclavian line is shown, together with the response to a further infusion of magnesium sulfate. The absence of cardiovascular disturbances during induction of anesthesia and intubation of the trachea is demonstrated.

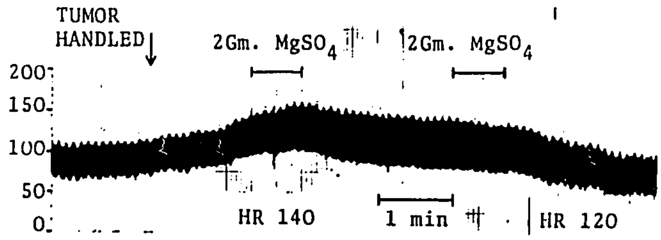

FIG. 2. The increase in arterial blood pressure during the period of tumor handling is illustrated, and the termination of this increase by further infusions of magnesium sulfate also shown. The mass was being manipulated throughout the period of the trace. HR = Heart rate, in bpm.

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Airway Management in a Patient with Sleep Apnea Using a Permanent Silicone Tracheal Cannula

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The sleep apnea syndrome, if not detected and appropriately treated, can have life-threatening implications.[1,2] Apnea, defined as complete airflow cessation for greater than 10 s,[2] has been divided into three types by Gastaut *et al.*[3]: central, mixed, and obstructive. Obstructive sleep apnea is by far the most prevalent type,[4] and a tracheostomy that can be closed during the day and opened at night has become the treatment of choice for this condition.[5]

During the last 5 years, more than 100 tracheostomies have been performed at our institution for obstructive sleep apnea, and the airway most frequently inserted is the silicone tracheal cannula¶ (STC) (fig. 1*A*). This permanent tracheostomy tube is easily inserted, is inconspicuous, maintains normal speech, and hence has been well accepted by patients.[6,7] This STC has an internal diameter of 8 mm, with a wall thickness of 1 mm. An inner flange secures the cannula to the anterior tracheal wall to prevent anterior displacement. Immediately following insertion, a wing-shaped faceplate secures the cannula to the skin of the neck to prevent posterior displacement. After healing of the tracheostomy, a ring washer replaces the faceplate. (fig. 1*B*). During waking hours, the patient obstructs the lumen with a plug. During sleep the plug is removed to provide an alternative airway and thus prevents upper airway obstruction. Because of the more frequent and anticipated widespread use of this STC in the treatment of sleep apnea, we report the anesthetic management of a patient with such an airway undergoing peripheral surgery.

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¶ Manufactured by Boston Medical Products, Inc., 180 Cambridge Street, Boston, Massachusetts 02114.

REPORT OF CASE

A 60-year-old man, weighing 67 kg, was admitted for arthroplasties of his right hand. His history was significant for severe steroid-dependent arthritis, recently diagnosed sleep apnea, and associated multifocal premature ventricular contractions. Since his apnea was related to upper airway obstruction, an STC had been inserted under local anesthesia 1 month prior to the current admission. Subsequently, Holter monitoring revealed a marked decrease in the frequency of his ventricular dysrhythmias. Posttracheostomy evaluation in our Sleep Laboratory during the current admission showed no airway obstruction during sleep, but residual central sleep apneic activity persisted (*i.e.*, less than 30 events during an 8-h period). Examination of the tracheostomy and STC was unremarkable. Prior to tracheostomy he had undergone multiple general anesthetics with endotracheal tubes for joint surgery without complication.

There are no reports available concerning the management of the airway containing a STC during general anesthesia. Therefore, we questioned whether 1) this thin-walled tube would accept and support