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Complications Related to the Pressor Response to Endotracheal Intubation

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The pressor response to laryngoscopy and endotracheal intubation has been recognized since 1951.[1] It is a sympathetic reflex provoked by stimulation of the epipharynx and laryngopharynx.[2] The increases in blood pressure and pulse rate are usually transitory, variable, and unpredictable.[1,3,4] Hypertensive patients are more prone to have significant increases in blood pressure whether they have been treated beforehand or not.[5] Transitory hypertension and tachycardia are probably of no consequence in healthy individuals, but either or both may be hazardous to those with hypertension, myocardial insufficiency, or cerebrovascular disease. Several investigators have warned of this,[1,4–6] but so far there has been no report of complications in such a case.

We report two cases in which complications followed hypertensive episodes directly related to laryngoscopy and endotracheal intubation.

Report of Two Cases

Patient 1. A 28-year-old black multigravida was brought to the operating room for emergency cesarean section for chronic hypertension with superimposed pre-eclampsia, and possible abruptio placentae. Blood pressure had been poorly controlled with alpha-methyldopa, 250 mg, *t.i.d.*, and ranged from 200/130 to 240/140 torr. Before induction of anesthesia, blood pressure was 260/140 torr, with a pulse rate of 96/min. Following preoxygenation, induction of anesthesia was carried out with thiopental, 375 mg; endotracheal intubation was performed without difficulty after the administration of succinylcholine, 80 mg. Immediately after endotracheal intubation, the systolic blood pressure rose to more than 300 torr and the pulse rate increased to 160/min, at which point pulmonary edema promptly developed. Approximately 200 ml of fluid were aspirated from the endotracheal tube. Immediate treatment consisted of morphine sulfate, 10 mg, iv, furosemide 40 mg, iv, a head-up tilt, and positive end-expiratory pressure with oxygen.

After delivery of a living infant, the endotracheal tube was left in place, and the patient was ventilated with an Engström ventilator, using positive end-expiratory pressure. She was unresponsive initially, but gradually recovered consciousness. Blood pressures ranged from 200/100 to 100/70 torr, with pulse rates of 110–130/min. The patient was treated with furosemide, digoxin and procainamide. She remained in a state of borderline cardiac failure for two to three days, easily becoming decompensated without positive end-expiratory pressure. As the hypertension and cardiac failure subsided, she was gradually weaned from PEEP and the ventilator. She made a good recovery.

Patient 2. A 37-year-old white man with end-stage renal failure was brought to the operating room for emergency cadaver kidney transplant. He had a history of hypertension, and had been treated with large doses of alpha-methyldopa, which had been reduced some time previously following an episode of hepatitis. Since then, blood pressure had remained at a normotensive level with a small maintenance dose. Blood pressure before induction was 140/90 torr, with a pulse rate of 100/min. Following preoxygenation, induction was carried out with meperidine, 100 mg, in divided doses and thiopental, 250 mg. Endotracheal intubation was performed without difficulty following succinylcholine, 100 mg, iv. Approximately 5 minutes later, blood pressure was 240/160 torr, and a transient ventricular tachycardia developed; this reverted to normal sinus rhythm following a bolus injection of lidocaine, 100 mg, iv. Halothane was added to the N_2O–O_2 mixture the patient was receiving, and the blood pressure returned to 140/100 torr, and remained at this level. Halfway through the procedure, it was noticed that the left pupil was dilated and did not respond to light. The patient did not regain consciousness at the end of the surgical procedure and had a gradually downhill course, with evidence of cerebrovascular hemorrhage. At autopsy, a ruptured cerebral aneurysm was found.

Discussion

These two case reports illustrate complications that may be associated with the hypertensive and tachycardiac response to laryngoscopy and endotracheal intubation in susceptible patients. In view of the frequency of occurrence of some hypertension during laryngoscopy even in normotensive patients, and the numbers of patients who come to operation with pre-existing cardiovascular disease, it is perhaps rather surprising that complications have not been reported. One reason for this may be the transient nature of the hypertension, which usually lasts less than ten minutes.[3,4,7]

It is possible, however, that some of the complications that occur during induction, or even later in the course of anesthesia, may in fact be precipitated by episodes of hypertension and tachycardia following endotracheal intubation. This may not be appreciated

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with intermittent blood pressure measurements because of the transient nature of the response. It is important, therefore, to be aware of the potential problem, and to be particularly cautious with patients who are already hypertensive, or who have other cardiovascular or cerebrovascular disease. When endotracheal intubation is essential, blood pressure should be monitored very closely during induction, continuously by direct intra-arterial measurement if applicable, but certainly at intervals of less than 5 minutes.

Techniques that have been shown to modify the pressor response should be considered. In principle, the response may be diminished or modified locally, centrally or peripherally, and attempts have been made to accomplish this using all of these approaches, with varying success. Regional and topical anesthesia have been used to block afferent impulses.[8,9] Intravenous lidocaine[10] and "deeper" inhalational anesthesia[1] have been used to modify the response at the central nervous system level. Trimethaphan[4] and phentolamine[11] have been used for their peripheral effect. Although prophylactic use of beta-adrenergic receptor blockers has been proposed by Prys-Roberts *et al.*,[5] Siedlecki[4] found that practolol, a cardioselective beta-blocking agent, was ineffective. None of these pharmacologic approaches has proved entirely satisfactory, because the reflex is not completely blocked, because the method itself carries some additional risk, or because the agents used may be too long-acting or have undesirable side-effects.

A different approach would be to minimize stretching the tissues of the epipharynx and laryngopharynx, which presumably trigger the reflex response.[1,2] In theory, blind nasal intubation, by avoiding laryngoscopy, might achieve this. Prys-Roberts[5] reported four such cases in which 10 per cent CO_2 was used as an aid to blind nasal intubation, and in which there was no pressor response, but the series is too small to evaluate the technique.

In conclusion, one must appreciate that a "crash" induction such as was utilized in both of the patients in this report provides little protection against the pressor or chronotropic effects of endotracheal intubation. In management of all such potentially susceptible patients, there should be an awareness of the hazard, close monitoring, adequate "depth" of anesthesia, and all appropriate additional measures taken to modify, if not entirely to suppress, the response.

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