Anaesthetic management of severe bradycardia during general anaesthesia using temporary cardiac pacing

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There are few reports of management of severe bradycardia with temporary cardiac pacing. We describe a 65-yr-old female patient who developed bradycardia and hypotension on two occasions during general anaesthesia for laryngoscopy. The first episode was treated with atropine, ephedrine, and colloid infusion and the second with a temporary pacemaker and ephedrine.

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Excessive vagal activity, which causes severe bradycardia and hypotension, can be life threatening. The trigger can be painful stimulation of the bronchial, pharyngeal, laryngeal, or oesophageal mucosa. Prompt treatment is needed with urgent restoration of venous return by leg elevation, head down tilt, and i.v. fluids, and the use of anticholinergic and sympathomimetic drugs. A pacemaker should be considered for patients with vasovagal syncope which is frequent and does not respond to medical treatment. The use of a temporary pacemaker for patients who develop bradycardia during general anaesthesia is controversial.

We describe two episodes of severe bradycardia in the same patient during general anaesthesia, the second of which was managed with a pacemaker.

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Case report

The patient was a 65-yr-old female who weighed 59 kg. She was admitted to hospital to have direct laryngoscopy and vocal cord biopsy, because of a history of hoarseness. This was to be carried out under general anaesthesia.

She gave a history of a myomectomy under general anaesthesia in 1970, which was uneventful, and asthma for the last 25 yr. Her medications were N-acetyl cysteine and an inhaler of fluticasone propionate.

She was given 5 mg diazepam by mouth before going to the operating theatre. She was monitored with non-invasive arterial pressure, electrocardiogram, and pulse oximetry. Her heart rate was 68 beats min\(^{-1}\) and arterial pressure 115/78 mm Hg. Anaesthesia was induced with propofol 150 mg, followed by vecuronium 6 mg and fentanyl 200 μg. After the trachea had been intubated with a microlaryngeal tube, her heart rate was 76 beats min\(^{-1}\) and arterial pressure was 136/82 mm Hg. Anaesthesia was maintained with 2% sevoflurane in nitrous oxide/oxygen, and the patient was positioned slightly head up to facilitate direct laryngoscopy. Sinus rhythm was present throughout the episode.

Immediately after direct laryngoscopy by the surgeon, the heart rate suddenly decreased to 28 beats min\(^{-1}\). Despite withdrawal of the laryngoscope, bradycardia persisted. Atropine 0.5 mg was given i.v., but the heart rate remained slow and the arterial pressure was 55/28 mm Hg. A further dose of atropine 0.5 mg i.v. had no effect but ephedrine 10 mg i.v. caused the heart rate to increase to 72 beats min\(^{-1}\). Hypotension persisted and was treated with infusion of 500 ml of hydroxyethyl starch. Arterial blood gas and serum electrolyte measurements were normal. After 12 min the patient became cardiovascularly stable. The operation was postponed, and the patient recovered without sequelae. Subsequent cardiac examination was normal.

Six months later the same patient returned for treatment of nasal polyps, by nasal endoscopy. Her hoarseness had resolved with medical therapy. On careful questioning the patient gave a history of attacks of syncope or pre-syncope resolved with medical therapy. On careful questioning the patient gave a history of attacks of syncope or pre-syncope resolved with medical therapy. On careful questioning the patient gave a history of attacks of syncope or pre-syncope resolved with medical therapy.

Subsequent cardiac examination was normal. Tilt-table testing was the only investigation that can precipitate a typical attack that can be directly observed, it is taken as the ‘gold standard’ for the diagnosis of vasovagal syncope.4

Clinically, syncope associated with autonomic dysfunction tends to be most frequent early in the day, and is more likely after a period of bed rest, after vigorous exercise, or during drug treatment that can reduce central circulatory volume. Unlike vasovagal faints, these episodes of syncope are not associated with bradycardia, sweating, or marked pallor.5 Our patient’s clinical presentation, and the absence of predisposing factors such diabetes mellitus led us to think she had a vasovagal response rather than autonomic dysfunction.

Treatment of vasovagal syncope with a pacemaker is controversial.6 Vasovagal syncope can be aborted by dual-chamber pacing, and if syncope does occur, pacing can prolong consciousness.7 However, in a report of 22 patients with neurocardiogenic syncope, pacing failed to prevent a decrease in arterial pressure during bradycardia caused by tilt testing.8

Temporary pacing is most commonly used to treat symptomatic bradycardia for short periods, either before a permanent pacemaker or when the bradycardias is not persistent.3,6 There are few reports of temporary pacing for general anaesthesia.9 We suggest that as well as drug treatment, temporary pacing is a useful form of treatment for this condition.

References


was withdrawn the day after surgery. The patient recovered completely and left hospital after 3 days.

Discussion

During anaesthesia, changes of heart rate may suggest changes in the depth of anaesthesia, changes in vagal activity, or the effects of drugs or possible hypoxia. Simple vagal reactions usually respond to when the stimulus is stopped. Here, the vagal response was excessive and severe hypotension persisted despite stopping the laryngoscopy.

Tilt-table testing may be useful in the diagnosis of vasovagal syncope, and can guide treatment. However the tilt-table test is positive in only 75% of patients with classic vasovagal syncope. The sensitivities of the various test methods vary widely and they are only 75–80% reproducible, suggesting that they may give an incorrect diagnosis.3 However, as tilt-table testing is the only investigation that can precipitate a typical attack that can be directly observed, it is taken as the ‘gold standard’ for the diagnosis of vasovagal syncope.4