Precipitous bradycardia induced by laryngoscopy in cardiac surgical patients

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We present three patients with severe ischaemic heart disease who developed profound bradycardia (heart rates of 15 beats·min⁻¹, 10 beats·min⁻¹ and asystole) at the time of laryngoscopy and spraying of the trachea with topical lidocaine. All patients had received fentanyl 20–30 µg·kg⁻¹ and pancuronium 0.1–0.13 mg·kg⁻¹ and in each case laryngoscopy was performed three minutes after the administration of these agents. We believe that in these cases the dosage of fentanyl was inadequate and since increasing the dosage of fentanyl to at least 50 µg·kg⁻¹ and eliminating the manoeuvre of laryngeal spraying with topical anaesthesia, we have not encountered this problem.

For over 40 years it has been appreciated by anaesthetists that the performance of laryngoscopy and intubation can be accompanied by reflex circulatory responses. With the introduction of the non-depolarizing muscle relaxants in the 1940's and 1950's it was realised that the shift in technique from intubation under deep planes of volatile anaesthesia to the lighter planes made possible by the use of relaxants could predispose the patient to the development of intubation-related arrhythmias, including bradyarrhythmias.¹

Consequent upon the increase in understanding of the pathophysiology of ischaemic heart disease, there has been a shift in emphasis towards the importance of hypertension and tachycardia associated with laryngoscopy and intubation and the subsequent development of myocardial ischaemia.² This emphasis has led to the underestimation of the degree and severity of bradycardia which can occur with laryngoscopy and intubation.

Key words

ANAESTHESIA: cardiac; COMPLICATIONS: bradycardia.

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We therefore would like to present three patients with severe cardiac disease who developed significant bradycardia at the time of laryngoscopy and spraying of the trachea and vocal cords with topical anaesthetic solution.

Case reports

Case 1

Fifty-four-year-old 64 kg female, with a long past history of hypertension and ischaemic heart disease, was admitted to the coronary care unit with unstable angina. Her medication consisted of propranolol 320 mg daily, hydrochlorothiazide 25 mg daily, amiloride 5 mg daily and isordil 80 mg orally daily. She was commenced on intravenous nitroglycerine for chest pain which continued despite this therapy and was complicated by one episode of sinus arrest lasting six seconds. Coronary angiography revealed triple vessel disease and the patient was scheduled for urgent coronary artery bypass surgery.

She was premedicated with morphine 7.5 mg IM, scopolamine 0.3 mg IM and received 7 mg diazepam IV during insertion of arterial, venous and pulmonary artery catheters. At induction blood pressure (BP) was 150/80 mmHg, heart rate (HR) 48 bpm, mean pulmonary artery pressure (MPAP) 17 mmHg and pulmonary capillary wedge pressure (PCWP) 10 mmHg. Induction of anaesthesia was with fentanyl 1600 µg (25 µg·kg⁻¹) pancuronium 8 mg and nitrous oxide with 50 per cent oxygen. Following induction her heart rate increased to 65 bpm and BP was 125/70 mmHg. After three minutes of ventilation, laryngoscopy and spraying of the cords with 1 mg·kg⁻¹ of ten per cent lidocaine was followed by progressive bradycardia to a minimum rate of 10 bpm. The BP fell to 75 mmHg systolic (Figure 1). A precordial thump was given and heart rate recovered to 75 bpm over the following 12 seconds. Subsequent laryngoscopy and intubation performed two minutes after recovery was uneventful. An arterial blood gas specimen at this time showed pHa 7.47, PaCO₂ 36 mmHg, PaO₂ 148 mmHg. Serum electrolytes were normal.

Case 2

Sixty-four-year-old 73 kg male presented with unstable

FIGURE 1 Patient 1. Effect of laryngoscopy and laryngotracheal spraying with topical lidocaine. Arterial line tracing. L1 = Laryngoscopy and spraying.

angina following a long history of ischaemic heart disease including three previous myocardial infarctions. Continuous ECG monitoring revealed occasional sinus bradycardia to a rate of 45 bpm. Medication consisted of verapamil 360 mg daily, isordil 30 mg daily and prazosin 1 mg daily. Intravenous nitroglycerine and morphine were required for pain relief. His coronary angiogram revealed triple vessel disease and he was scheduled for urgent coronary artery bypass grafts.

Premedication was with morphine 10 mg and scopolamine 0.4 mg. Diazepam 10 mg IV was administered during vascular cannulation.

At induction of anaesthesia BP was 125/55 mmHg, HR 54 bpm, MPAP 20 mmHg and PCWP 9 mmHg. The patient was induced with fentanyl 2200 μ g (30 μ g·kg⁻¹)

and the heart rate fell to 43 bpm. Pancuronium 8 mg was given and the heart rate returned to around 65 bpm. After three minutes of ventilation with 100 per cent oxygen, laryngoscopy and vocal cord spraying with 1 mg·kg⁻¹ of lidocaine were performed. Heart rate rapidly slowed to less than 15 bpm and the systolic pressure fell to 50 mmHg (Figure 2). The laryngoscope was removed and both pulse rate and BP reverted to normal over the following 20 seconds. Uneventful intubation was performed two minutes later. An arterial blood gas specimen taken at this point revealed pHa 7.38, PaCO₂ 45 mmHg, PaO₂ 251 mmHg. Serum electrolytes were normal.

Case 3

Sixty-two-year-old 98.5 kg male presented with unstable angina after a 12-month history of ischaemic chest pain. At the time of surgery medication consisted of isordil 40 mg daily, prazosin 3 mg daily, metoprolol 200 mg daily, nifedipine 60 mg daily, allopurinol 300 mg daily and intravenous nitroglycerine infusion. His ECG showed sinus rhythm with left ventricular hypertrophy. Coronary angiography demonstrated severe triple vessel disease with left inferior wall hypokinesia and a dilated left ventricle. Because of continuing chest pain he was scheduled for urgent coronary artery bypass surgery. He was premedicated with morphine 10 mg IM and scopolamine 0.4 mg IM. Vascular cannulation was facilitated with 10 mg of diazepam IV. Immediately prior to induction BP was 125/80 mmHg, HR 50 bpm, MPAP 16 mmHg and PCWP 8 mmHg.

Anaesthesia was induced with fentanyl 2000 μ g (20 μ g·kg⁻¹), pancuronium 10 mg and he was ventilated with nitrous oxide 50 per cent in oxygen. Three minutes later,

FIGURE 2 Patient 2: (a) Bradycardia with fentanyl induction and vagolytic effect of pancuronium. (b) Effect of laryngoscopy and laryngotracheal spraying with lidocaine and subsequent intubation. Arterial line traces. F = Fentanyl; P = Pancuronium; L₁ = Laryngoscopy and spraying; L₂ = Laryngoscopy and tracheal intubation.

at the time of laryngoscopy and tracheal spraying with $1 \text{ mg} \cdot \text{kg}^{-1}$ of topical lidocaine, asystole occurred (Figure 3). External cardiac massage was commenced and sinus rhythm returned at a rate of 55 bpm after 20 seconds, with satisfactory BP.

Approximately one minute after the initial laryngoscopy the patient was intubated and again demonstrated a reduction in heart rate to 45 bpm for a period of 12 seconds. An arterial blood gas analysis at this time showed pH 7.42, PaCO_2 31 mmHg, PaO_2 189 mmHg. Serum electrolytes were normal.

Discussion

The initiation of reflex changes in heart rate and rhythm has been well known for many years and as early as 1949 Burstein³ described cardiac arrest as a result of surgical interference with the larynx under inadequate anaesthesia.

The incidence of arrhythmias during tracheal intubation can be as high as 90 per cent.⁴ A relative sympathetic predominance with tachycardia and hypertension is the usual course. The use of potent narcotics in cardiac anaesthesia, in particular fentanyl, has become widely accepted as a safe reliable and reproducible technique with which to obtain "cardiac stable" anaesthesia. Kautto⁵ demonstrated that $6 \mu\text{g} \cdot \text{kg}^{-1}$ of fentanyl will attenuate the circulatory effects of laryngoscopy and doses of 25–75 $\mu\text{g} \cdot \text{kg}^{-1}$ are regularly used for induction with usually little change in heart rate or cardiac output.⁶ However, fentanyl, like morphine, can produce bradycardia. This effect is probably due to a reduction in the outflow of sympathetic activity from the central nervous system⁷ rather than peripheral interference with catecholamine output from nerve endings. This bradycardia is usually limited in degree and easily reversed by anticholinergic drugs including pancuronium, as occurred in patient 2.

The most outstanding feature of all three cases which we are reporting is that the occurrence of a precipitous bradycardia immediately followed the manoeuvres of laryngoscopy and spraying of the trachea and vocal cords with room temperature topical anaesthetic solution. Although Savarese *et al.*⁸ refer in passing to the production of precipitous bradycardia during vagotonic manoeuvres such as laryngoscopy, tracheal intubation, chest incision or sternal splitting in cardiac surgery performed under potent narcotic anaesthesia, we have been unable to find any documented cases occurring in patients receiving this combination of fentanyl and pancuronium.

We believe fentanyl administered in the dose range of 20–30 $\mu\text{g} \cdot \text{kg}^{-1}$, although adequate in producing a level of anaesthesia to prevent tachycardia and hypertension, proved inadequate in the prevention of reflex bradycardia at the time of laryngoscopy and upper airway spraying in our patients. Many cardiac anaesthetists using high-dose

FIGURE 3 Patient 3. Asystole following laryngoscopy and laryngotracheal spraying with topical lidocaine. Return of sinus rhythm after 20 seconds of external cardiac massage. Effect of subsequent laryngoscopy and intubation. Arterial line tracing. L1 = Laryngoscopy and spraying; ECM = External cardiac massage; SR = Sinus rhythm; L2 = Laryngoscopy and tracheal intubation.

narcotic techniques routinely employ tracheal spraying prior to the introduction of the endotracheal tube, with a view to attenuating the heart rate and blood pressure responses of laryngoscopy and intubation and to reduce the chance of ventricular arrhythmias by decreasing ventricular irritability.⁹ We believe this practice to be unnecessary. Stoelting¹⁰ has certainly demonstrated that when tracheal intubation can be reliably accomplished with a short period of direct laryngoscopy, pharmacological attempts (such as topical tracheal or intravenous lidocaine) to minimise the pressure responses are not helpful and further, sudden reflex cardiac arrest occurring due to impingement of water on the upper respiratory tract is a cause of death well recognised by forensic experts.¹¹ Anecdotally (at our institution) some degree of bradycardia associated with laryngoscopy and laryngeal spraying seems to occur with an incidence of up to 1:200 cases. Since recognising the limitation of the dose range of narcotic which was employed in the reported cases (20–30 $\mu\text{g} \cdot \text{kg}^{-1}$) we have increased our induction dose of fentanyl to at least 50 $\mu\text{g} \cdot \text{kg}^{-1}$ pre-laryngoscopy and have eliminated the manoeuvre of laryngeal spraying. Following this modification in technique we have not seen the reflex bradycardia described in over 450 successive cases.

Two of the three patients (patients 1 and 2) are of particular interest as both had exhibited either sinus arrest or sinus bradycardia in the preoperative period. We feel that those patients who have demonstrated bradyarrhythmias preoperatively are potentially more at risk for the development of reflex related bradyarrhythmias at the time of laryngoscopy. All patients were receiving either beta adrenergic blockers or calcium channel blocking agents. Patient 3, who exhibited the most dramatic response, was receiving both nifedipine and metoprolol.

Whereas these agents do not always provide protection against heart rate acceleration⁴ many beta blocking agents in clinical use today have the potential of producing substantial sinus bradycardia and A-V block. Verapamil, when given to patients on beta adrenergic blocking drugs, can produce asystole¹² but this is not the case with nifedipine which tends to promote an increase in heart rate.¹³ The combination of metoprolol and nifedipine was probably non-contributory to the development of bradycardia in patient 3.

In conclusion we have documented three patients with severe cardiac disease in whom the development of precipitous bradycardia occurred at the time of laryngoscopy and spraying of the trachea and vocal cords. A history of pre-existing bradyarrhythmias and bradycardia at the time of induction should alert the anaesthetist to the potential for development of severe reflex bradyarrhythmias. We believe this bradycardia to be a manifestation of reflex vagal activity in the presence of narcotic anaesthesia of insufficient depth to ablate this reflex.

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Résumé

Nous présentons trois patients souffrant d'ischémie cardiaque grave, chez lesquels une bradycardie profonde s'est manifestée (rythme cardiaque de 15 par minute, 10 par minute et asystolie) au moment de la laryngoscopie et de l'application de lidocaïne en aérosol dans la trachée. Tous les patients avaient reçu 20-30 $\mu\text{g}\cdot\text{kg}^{-1}$ de fentanyl et 0.1-0.13 $\text{mg}\cdot\text{kg}^{-1}$ de pancuronium et dans chaque cas la laryngoscopie était pratiquée trois minutes après l'administration de ces agents. Nous croyons que dans ces cas le dosage de fentanyl était inadéquat et, depuis que nous avons augmenté le dosage de fentanyl à au moins 50 $\mu\text{g}\cdot\text{kg}^{-1}$ et éliminé la manoeuvre d'application laryngienne avec un anesthésique local en aérosol, ce problème ne s'est pas répété.