HEART BLOCK FOLLOWING PROPOFOL: A CASE REPORT

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Propofol is a new anaesthetic agent with a smooth and rapid onset of action similar to that of thiopentone [1], but with a very fast clearance from blood [2] and, consequently, a short duration of action. It also appears to have a low potential for histamine release [3] and does not cause suppression of steroid synthesis [4]. However, bradycardia [5] and hypotension [6] have been described as complications following use of the agent, but heart block has not been documented. We report two cases of ventricular asystole apparently with total heart block; in one patient propofol was used as an induction agent and in the other, continuous i.v. anaesthesia with propofol was used.

CASE REPORTS

Patient 1

The patient was an obese 54-yr-old female who weighed 98 kg, presenting for total abdominal hysterectomy and bilateral salpingo-oophorectomy. Significant previous medical history included hypertension for which she was receiving sotalol 80 mg day\(^{-1}\) and hydrochlorothiazide 12.5 mg day\(^{-1}\). She also had a hiatus hernia. Preoperative cardiovascular data included arterial pressure of 150/90 mm Hg, a regular heart rate of 72 beat min\(^{-1}\) and an ECG which showed a left ventricular strain pattern, but no conduction defects. The remainder of the clinical examination was normal. Preoperative investigations included a chest x-ray, and haemoglobin, urea and electrolyte estimations, which were all normal. For the calculation of drug doses a lean body mass of 80 kg was assumed. Anti-hypertensive medication was continued on the morning of surgery, and premedication consisted of papaveretum 10 mg and promethazine 25 mg i.m. 1 h before operation.

In the operating theatre the heart rate was 83 beat min\(^{-1}\) and arterial pressure 140/80 mm Hg. ECG leads were attached and arterial pressure was monitored every 1 min during the induction sequence using a non-invasive automatic device. In view of her hiatus hernia, a rapid sequence induction was performed. Following pre-oxygenation, anaesthesia was induced with propofol 200 mg given over 30 s, followed by suxamethonium 80 mg. Cricoid pressure was applied and, after fasciculations had ceased but before intubation, it was noticed that although there were regular P-waves with a rate of 70 min\(^{-1}\) and an ECG which showed a left ventricular strain pattern, but no conduction defects. The remainder of the clinical examination was normal. Preoperative investigations included a chest x-ray, and haemoglobin, urea and electrolyte estimations, which were all normal. For the calculation of drug doses a lean body mass of 80 kg was assumed. Anti-hypertensive medication was continued on the morning of surgery, and premedication consisted of papaveretum 10 mg and promethazine 25 mg i.m. 1 h before operation.

In the operating theatre the heart rate was 83 beat min\(^{-1}\) and arterial pressure 140/80 mm Hg. ECG leads were attached and arterial pressure was monitored every 1 min during the induction sequence using a non-invasive automatic device. In view of her hiatus hernia, a rapid sequence induction was performed. Following pre-oxygenation, anaesthesia was induced with propofol 200 mg given over 30 s, followed by suxamethonium 80 mg. Cricoid pressure was applied and, after fasciculations had ceased but before intubation, it was noticed that although there were regular P-waves with a rate of 70 min\(^{-1}\), there were no QRS complexes, and no pulse could be felt. Tracheal intubation was performed, and QRS complexes reappeared spontaneously with normal configuration and persisted for several normal beats. Manual ventilation of the lungs was instituted with 100 % oxygen and 3 % enflurane introduced. Within a few seconds, QRS complexes again disappeared, although P-waves persisted. Following 10 P-waves, normal QRS complexes returned, with a good output. A third similar episode occurred after an interval of a few seconds of normal sinus rhythm. Atropine 0.6 mg was administered i.v., and no further episodes occurred. This sequence of events took place over the

SUMMARY

We report two cases of ventricular arrest with persisting atrial activity in association with propofol anaesthesia. In both cases, anticholinergic agents corrected the arrhythmia. It is recommended that anticholinergic drugs be given routinely when propofol is used in association with vagal stimulants.
space of 1 min, and no arterial pressure recordings were made at this time. The next arterial pressure recording taken immediately after the atropine was given was 120/70 mm Hg, with a heart rate of 70 beat min⁻¹. Surgery proceeded uneventfully, and the patient recovered from the anaesthetic normally. A post operative ECG was identical to the preoperative tracing.

Patient 2

This patient was a 46-yr-old male with proven malignant hyperpyrexia susceptibility scheduled to undergo elective cholecystectomy. Preoperative evaluation included a full blood count, chest x-ray, ECG and urea and electrolyte measurements, all of which were normal. Preoperative arterial pressure was 130/90 mm Hg and heart rate 80 beat min⁻¹. Before induction, a radial arterial and a central venous catheter were inserted, and ECG leads attached. A halothane-free machine was used, and a continuous propofol anaesthetic technique adopted, with an induction dose of 180 mg and boluses of propofol 25–40 mg given as indicated by increases in heart rate or arterial pressure. Neuromuscular blockade was achieved with alcuronium 20 mg and controlled ventilation was maintained with 50% oxygen in nitrous oxide. The procedure was uneventful, and at the end of surgery residual neuromuscular blockade was antagonized with neostigmine 2.5 mg and glycopyrrolate 0.4 mg. Within a few seconds, QRS complexes disappeared, although P-waves persisted at an unchanged rate; arterial pressure was unrecordable and the patient began to sweat profusely. A further glycopyrrolate 0.4 mg was administered, and QRS complexes returned immediately with restoration of a normal arterial pressure. Intermittent episodes of heart block occurred with ventricular standstill of brief duration, for the next 30 min, but thereafter no further episodes were seen. The patient was monitored for the next 24 h and remained stable. Subsequently, further surgery was required for a wound dehiscence, and this was managed with regional blockade and was uneventful.

DISCUSSION

The cardiovascular effects ascribed to propofol include hypotension resulting from a combination of decreased systemic vascular resistance and a reduced cardiac output, and minimal changes in heart rate, although occasional bradycardia, responsive to atropine, has been described [4, 6]. Continuous propofol anaesthesia has been administered in combination with intermittent doses of suxamethonium, without serious bradycardia being reported, although the patients were premedicated with atropine [7]. Arrhythmias have not been described previously, but there has been a report of cardiac arrest following the use of propofol. In this patient, the arrest responded rapidly to the administration of atropine and a brief period of cardiac massage [8]. Another patient in this report had a severe bradycardia which responded to atropine.

Both of the patients described here received cholinergic stimuli before the onset of arrhythmia. In the first, suxamethonium was administered without an anticholinergic before intubation, and in the second neostigmine was given accompanied by a relatively small dose of glycopyrrolate. In both patients the ventricular arrest responded to administration of an anticholinergic agent, suggesting that vagal stimulation was responsible at least partly for the events. However, if a vagally mediated cause for the ventricular arrests is to be postulated, it is surprising that P-waves continued unchanged in both cases.

Both patients had some cardiovascular disturbance. The first patient was hypertensive, receiving beta-blockers, with some evidence of ventricular strain. The second patient suffered from malignant hyperthermia susceptibility, and may therefore have had underlying myocardial abnormality, although there was no clinical or ECG evidence for this. The safe use of propofol in MH has been described recently [9].

There is some evidence that propofol may depress the SA node directly (K. J. Hopkins, personal communication), but no evidence at present that the drug affects atrioventricular conduction. The explanation for the events observed therefore remains obscure, but it would seem advisable at present to recommend that an anticholinergic drug be administered whenever propofol is given in combination with potential cholinergic stimulant agents. This is advisable particularly if the patient is also receiving a beta-blocker.

REFERENCES

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