REGURGITATION AND ASPIRATION

Sir,—Dr Scott (1978) postulates that, where there is regurgitation of gastro-oesophageal contents into the pharynx, there exists in spontaneously breathing anaesthetized patients a mechanism for preventing irritant fluid reaching the smaller bronchi and that this mechanism is absent in paralysed patients. However, there is also a mechanism (present in spontaneously breathing anaesthetized patients and absent in paralysed patients) which prevents reflux into the pharynx of any material present in the oesophagus. This latter mechanism depends upon the muscle tone of the cricopharyngeal sphincter. This tone is present in the majority of patients even at relatively deep levels of anaesthesia when the patient is not paralysed.

Some years ago I studied the pattern of relaxation of the cricopharyngeal sphincter in response to suxamethonium (Davies, 1963). The loss of tone in the sphincter following the injection of a large bolus of suxamethonium was examined in 50 adult patients using a 1.5-cm diameter lax, water-filled balloon attached to a low inertia mercury manometer. The patients were anaesthetized with thiopentone 250 mg followed by nitrous oxide, oxygen and halothane. Of the 50 patients, 26 were known to have been at a level of anaesthesia at least as deep as plane II (Stage III). In these 26, decrease in balloon pressure following paralysis ranged from zero to 48 cm H\textsubscript{2}O. The median value was 18 cm H\textsubscript{2}O. In 21 of the 26 the decrease was greater than 10 cm H\textsubscript{2}O.

A lax balloon 1.5 cm in diameter does not give an accurate measure of the tone present in the closed sphincter. However, these results suggest that, in the majority of instances, a patient undergoing inhalation anaesthesia will have a sphincter tone which is considerably greater than during full neuromuscular blockade and this would tend to prevent reflux of oesophageal contents into the pharynx.

Whether or not the trachea should be intubated in a patient with a full stomach depends on the type of operative procedure. Where a patient is to undergo a procedure requiring only a short, light anaesthetic, a non-intubation technique may be favoured. However, for major abdominal surgery, one must balance the dangers of intubating the trachea against those of maintaining prolonged anaesthesia without a safeguarded airway. If endotracheal intubation is to be undertaken, it may well be that this could be most safely performed using a purely inhalation technique and without the aid of muscle relaxants.

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CAROTID ARTERY PALPATION DURING INTERNAL JUGULAR VEIN CANNULATION AND SUBSEQUENT VENTRICULAR FIBRILLATION

Sir,—One of the hazards of cannulation of the internal jugular vein is puncture of the carotid artery. To avoid this, and to help in locating the vein, the artery may be sought beforehand (English et al., 1969; Mostert, Kenny and Murphy, 1970; McConnell and Fox, 1972). We describe a case where palpation of the artery near the carotid sinus was followed by ventricular fibrillation.

A 61-year-old-man was admitted to hospital for a coronary artery bypass graft for ischaemic heart disease. He had been taking propranolol 80 mg three times daily until the day of operation.

Premedication comprised papaveretum 15 mg, hyoscine 0.3 mg and droperidol 5 mg. The heart rate was 40 beat min\textsuperscript{-1}, in sinus rhythm (SR), and the arterial pressure was 190/100 mm Hg. Atropine 0.3 mg i.v. was given, and the heart rate increased to 50 beat min\textsuperscript{-1} SR. Induction of anaesthesia with papaveretum 20 mg and thiopentone 250 mg was uneventful; endotracheal intubation was performed after the administration of pancuronium 8 mg. Heart rate and arterial pressure remained unchanged.

The patient was tipped 20° head down and the surface markings of the right internal jugular vein were mapped out. The right carotid artery was not palpable at the root of the neck, but it was found opposite the thyroid cartilage at the level of proposed venepuncture. At the moment of palpation tachycardia developed (e.c.g. showed 200 beat min\textsuperscript{-1}) which changed rapidly to ventricular fibrillation. The heart was defibrillated successfully with 200 J. An antecubital fossa vein was used for insertion of a central venous line. The operation of coronary artery bypass grafting was successful. After operation, the patient denied symptoms of previous carotid sinus syndrome.

Carotid sinus massage may have a variable effect on the rhythm of the heart. Scherf and Schott (1973) described factors including: (1) the site of stimulation (on the carotid sinus or below it), (2) if stimulation causes vagal excitation or sympathetic inhibition and (3) the condition of the heart, particularly its rhythm at the time of stimulation. They described a case of ventricular tachycardia following carotid sinus massage for paroxysmal supraventricular tachycardia, and cases of ventricular fibrillation following carotid sinus massage in digitalized patients.

It is possible that in our patient palpation of the carotid artery stimulated the carotid sinus. The patient was already receiving propranolol, and had been given hyoscine with the premedication. These drugs could have caused bradycardia before induction of anaesthesia. Further vagal stimulation from the carotid sinus may have allowed ectopic ventricular foci to cause escape beats.

We have no e.c.g. tracing to support this explanation and the recent injection of atropine also casts doubt on our hypothesis, but until the carotid artery was palpated, the patient was in sinus rhythm and after palpation he developed ventricular tachycardia.

In future, we will identify the carotid artery with care.

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