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CARDIOVASCULAR AND CATECHOLAMINE RESPONSES TO LARYNGOSCOPY WITH AND WITHOUT TRACHEAL INTUBATION

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SUMMARY

The catecholamine and cardiovascular responses to laryngoscopy alone have been compared with those following laryngoscopy and intubation in 24 patients allocated randomly to each group. Following induction with fentanyl and thiopentone, atracurium was administered and artificial ventilation undertaken via a face mask for 2 min with 67% nitrous oxide in oxygen. Following laryngoscopy, the vocal cords were visualized for 10 s. In one group of patients, ventilation was then re-instituted via a face mask, while in the second group the trachea was intubated during the 10-s period and ventilation of the lungs maintained. Arterial pressure, heart rate and plasma noradrenaline and adrenaline concentrations were measured before and after induction and at 1, 3 and 5 min after laryngoscopy. There were significant and similar increases in arterial pressure and circulating catecholamine concentrations following laryngoscopy with or without intubation. Intubation, however, was associated with significant increases in heart rate which did not occur in the laryngoscopy-only group.

Since King and colleagues (1951) first described the reflex circulatory responses to direct laryngoscopy and tracheal intubation, there have been numerous publications concerning both the response and the manoeuvres by which it may be attenuated (Prys-Roberts et al., 1971; Derbyshire and Smith 1984). The majority of studies treat laryngoscopy and intubation as a single stimulus, which manifestly they are not. Although many anaesthetists believe that laryngoscopy is the major stimulus, the authors are unaware of any study that has specifically examined this question.

Recent work has correlated the cardiovascular and sympathoadrenal responses to laryngoscopy and intubation (Russell et al., 1981; Derbyshire et al., 1983). The present study was, therefore, designed to compare the cardiovascular and sympathoadrenal responses either to laryngoscopy alone or to laryngoscopy with intubation.

PATIENTS AND METHODS

Twenty-four adult patients (ASA grades I or II) undergoing elective surgery under anaesthesia requiring tracheal intubation gave their informed consent for the study, which was approved by the District Ethical Committee. Patients with a history of acid reflux or those whose body weight exceeded 15% of predicted values were excluded from the trial. The patients were allocated randomly to two groups comprising laryngoscopy with, or without, intubation of the trachea.

Premedication comprised diazepam 10 mg orally 1–2 h before operation. In the anaesthetic room a 16-gauge cannula was placed in a vein in the antecubital fossa after local intradermal injection of 0.5% plain lignocaine. A Dinamap cuff was placed on the contralateral arm, and ECG electrodes attached to record standard limb lead II. After a stabilization period of 5 min, control readings of arterial pressure (Dinamap) and heart rate (ECG) were noted, and 10 ml of blood withdrawn from the cannula for the measurement

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of plasma adrenaline and noradrenaline concentrations.

Induction of anaesthesia was achieved with fentanyl 0.1 mg followed, after 2 min, by a dose of thiopentone (3–4 mg kg^{-1}) sufficient to induce sleep, given over 60 s, and followed by atracurium 0.5 mg kg^{-1}. A Guedel type pharyngeal airway was inserted and the patient's lungs were ventilated with 67% nitrous oxide in oxygen via a face mask attached to a Bain breathing system receiving a fresh gas flow of approximately 90 ml kg^{-1}.

Ventilation and fresh gas flows were adjusted to maintain the end-expired carbon dioxide at a concentration of 5–5.5%, as measured by a Gould Capnograph using a sampling catheter placed under the mask.

Two minutes after the administration of atracurium, the arterial pressure and heart rate were recorded and a further venous sample obtained.

Immediately following sampling, the airway was removed and laryngoscopy performed using a Macintosh blade, enabling a clear view of the vocal cords for a 10-s period. For patients in the laryngoscopy-only group, the laryngoscope was then removed, the airway replaced and ventilation of the lungs recommenced via the face mask. For patients in the laryngoscopy and intubation group, the trachea was intubated during the 10-s period after which the airway was replaced and ventilation continued. Male patients received a 9.0-mm and female patients an 8.0-mm cuffed disposable tracheal tube.

Subsequently, in both groups the lungs were ventilated for a further 5 min and the end-tidal carbon dioxide concentration maintained at 5–5.5%. Venous blood samples were obtained and measurements of arterial pressure and heart rate were recorded at 1, 3 and 5 min after laryngoscopy. At the end of the study period in the laryngoscopy-only group, the patient's trachea was intubated and anaesthesia continued in the usual manner.

The 10-ml blood samples were collected into heparinized tubes and centrifuged as soon as possible. The separated plasma was analysed for noradrenaline and adrenaline concentrations using a high pressure liquid chromatographic technique, originally described by Hallman and colleagues (1978), which has been adapted in our laboratory (Fell, Achola and Smith, 1982; Derbyshire et al., 1983).

Data were analysed using paired and unpaired Student's t tests as appropriate.

RESULTS

The two groups were similar in respect of age, weight and sex (table I).

There were no significant differences between the groups in respect of arterial pressure at any time during the study (fig. 1). Following induction, systolic arterial pressure decreased significantly in both groups ($P < 0.01$) and following laryngoscopy with or without intubation, the systolic pressure increased significantly ($P < 0.001$) to values similar to those obtained before induction.

The diastolic arterial pressure also decreased in both groups after induction, but the changes were not significant. After laryngoscopy, diastolic arterial pressures increased significantly by 24% (laryngoscopy-only) and 36% (laryngoscopy and intubation) ($P < 0.001$, both groups) to values significantly greater than the pre-induction values (laryngoscopy, $P < 0.05$; laryngoscopy and intubation, $P < 0.001$). In both groups, diastolic arterial pressure decreased gradually over the next 5 min to values similar to those noted before induction.

Mean heart rates were similar before induction (fig. 2) and although there was a slight increase after induction, this was not significant. Laryngoscopy alone caused a small non-significant increase in mean heart rate, but in the laryngoscopy and intubation group there was a significant increase ($P < 0.001$) of 19%. Heart rates in both groups decreased gradually over the next 5 min, but they remained significantly different from each other for the whole period ($P < 0.01$). At 5 min after laryngoscopy alone, heart rate was significantly lower than that before induction ($P < 0.05$).

There were similar and significant increases in both plasma noradrenaline and adrenaline concentrations at 1 min following laryngoscopy with or without intubation ($P < 0.001$) (figs 3, 4). Noradrenaline concentrations remained significantly increased at 3 min after laryngoscopy in both groups.

TABLE I. *Details of patients (mean (SEM))*

Group	Age (yr)	Wt (kg)	Sex
Laryngoscopy-only	42.3 (4.5)	68.7 (3.9)	9M:3F
Laryngoscopy and intubation	45.3 (3.8)	68.1 (3.5)	8M:4F

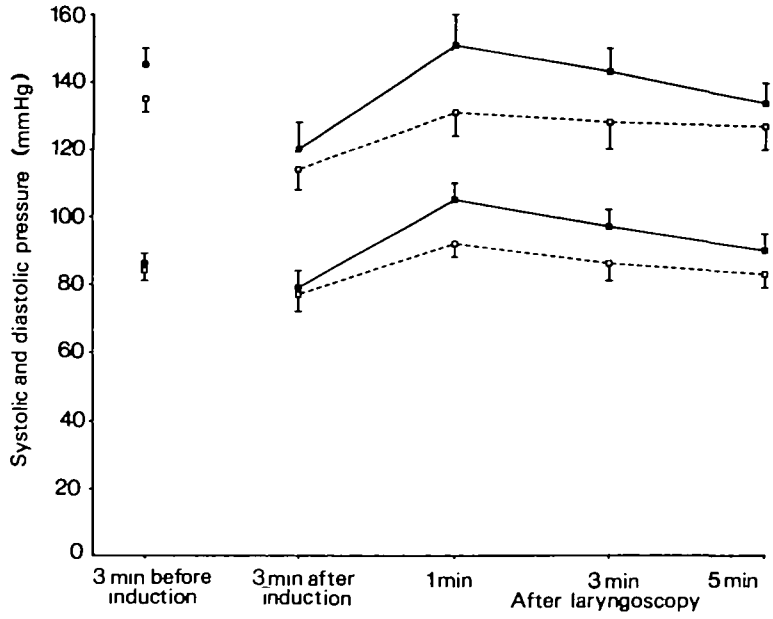

FIG. 1. Systolic and diastolic arterial pressures (mean ± SEM) in both groups before induction, and for the 5-min period after laryngoscopy. □- -□ = Laryngoscopy only (n = 12); ■——■ = laryngoscopy and intubation (n = 12).

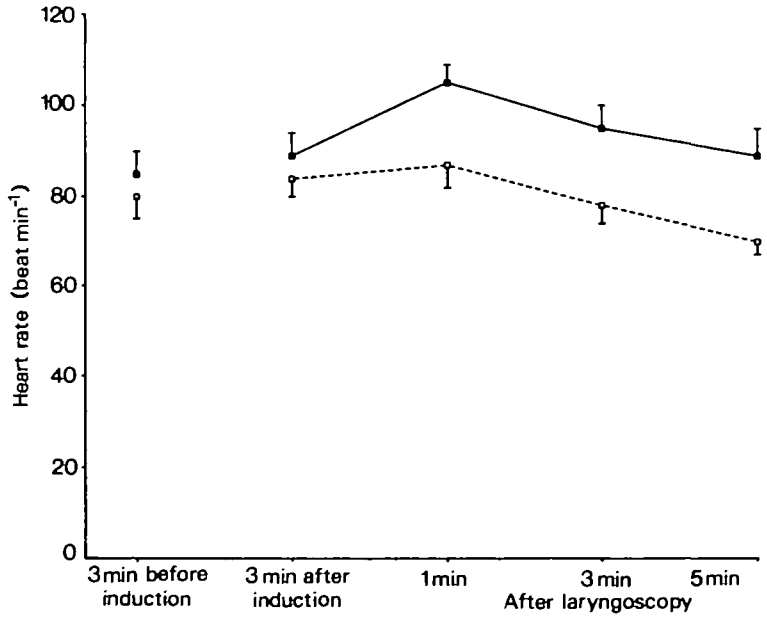

FIG. 2. Heart rates (mean ± SEM) in both groups before induction and for the 5-min period after laryngoscopy. □- -□ = Laryngoscopy only (n = 12); ■——■ = laryngoscopy and intubation (n = 12).

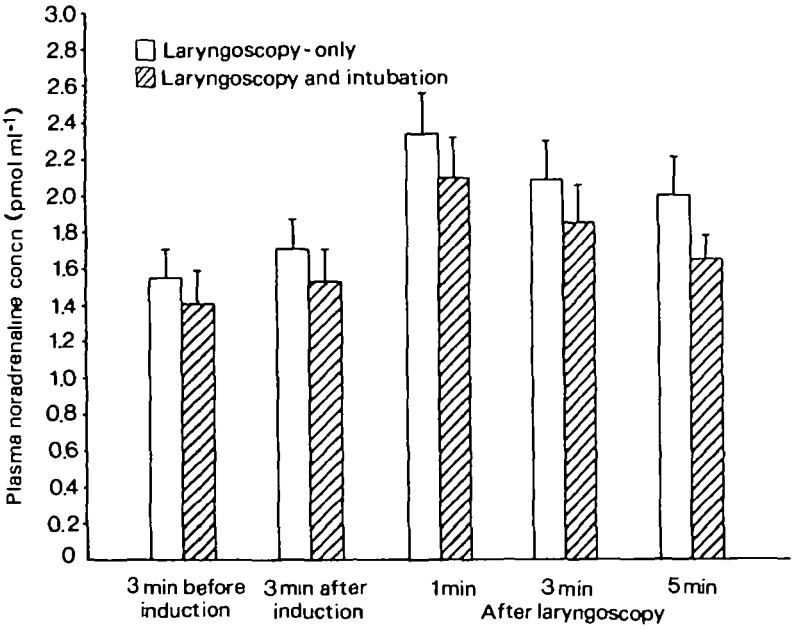

FIG. 3. Plasma noradrenaline concentrations (mean ± SEM) in both groups at each stage of the study. $n = 12$ (both groups).

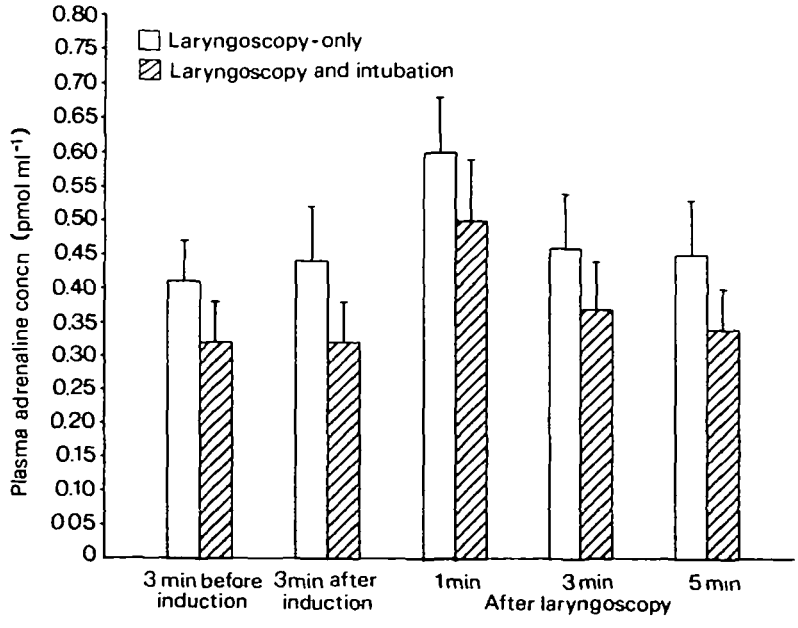

FIG. 4. Plasma adrenaline concentrations (mean ± SEM) in both groups at each stage of the study. $n = 12$ (both groups).

DISCUSSION

This study has shown that laryngoscopy alone generates the same pressor response and sympathoadrenal responses (in terms of circulating catecholamine concentrations) as laryngoscopy followed by intubation. This suggests that the major cause of the sympathoadrenal response to tracheal intubation arises from stimulation of the supraglottic region by tissue tension induced by laryngoscopy and that the placing of a tube through the cords and inflating a cuff in the infraglottic region contributes very little additional stimulation.

Laryngoscopy with intubation, however, was associated with significant increases in heart rate which were not apparent in the laryngoscopy-only group. This is difficult to explain, but implies that laryngoscopy produced a balanced stimulation of vagal and cardiac accelerator fibres, whereas intubation produced less vagal stimulation. The lack of vagolytic properties of the induction agents used in the study may have exaggerated any vagal stimulatory effect of the laryngoscopy.

Tomori and Widdicombe (1969) have shown that, in the paralysed cat, nervous activity in cervical sympathetic efferent fibres was significantly increased by stimulation of the epipharynx and laryngopharynx only, which also caused the greatest increase in mean arterial pressure. Stimulation of the tracheobronchial tree did not produce a significant increase in the activity of these fibres and caused only a small increase in mean arterial pressure.

Although Prys-Roberts and colleagues (1971) noted that tachycardia and hypertension occurred well before the act of intubation in treated and untreated hypertensives, data from only a single patient were presented to support this observation and, surprisingly perhaps, this observation does not seem to have initiated further studies to differentiate the contribution to sympathoadrenal activity of laryngoscopy from that of tracheal intubation. Stoelting (1977), however, did demonstrate changes in cardiovascular variables after laryngoscopy before intubation. Instead, subsequent studies have concentrated on the investigation of different methods of attenuating the sympathoadrenal responses induced by the combined act of laryngoscopy and tracheal intubation. This aspect of clinical investigation has been summarized elsewhere (Derbyshire and Smith, 1984).

The association between increased plasma catecholamine concentrations and pressor responses to tracheal intubation has been discussed by Derbyshire and Smith (1984). The present study has confirmed the work of Russell and colleagues (1981) and Derbyshire and colleagues (1983) showing that changes in plasma noradrenaline concentrations are linked with changes in mean arterial pressure. The association between changes in plasma adrenaline concentration and heart rate are less certain; in the present study there was a significant increase in plasma adrenaline concentration in both groups, but an increase in heart rate in only the laryngoscopy plus intubation group. In neither the study of Russell and colleagues (1981) nor that of Derbyshire and co-workers (1983) was there a significant correlation between plasma adrenaline concentration and heart rate or pulse pressure.

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