Cardiovascular changes following insertion of oropharyngeal
and nasopharyngeal airways

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Background. The cardiovascular responses following the insertion of oropharyngeal airways in
anaesthetized patients have been found to be of little consequence. However, those following the
insertion of nasopharyngeal airways have not been studied. The aim of this investigation was to
compare the cardiovascular responses to the insertion of oropharyngeal and nasopharyngeal
airways in anaesthetized patients.

Methods. Twenty-four ASA I or II patients aged 16–65 yr, requiring nasotracheal intubation as part
of their anaesthetic management, received a standardized general anaesthetic and were randomly
allocated to receive either a nasopharyngeal or an oropharyngeal airway.

Results. The two groups were similar with respect to age, weight and gender. There was a significant
decrease in systolic pressure following the induction of anaesthesia in both groups. Following
nasopharyngeal airway insertion, there was a significant rise in systolic pressure above pre-insertion
levels (P<0.001), though not above pre-induction levels (P=0.808). There was no significant change
in the oropharyngeal airway insertion group (P=0.619). At 1 min post-insertion, the mean (SD)
systolic pressure in the nasopharyngeal airway insertion group, 122 (21.6) mm Hg, was significantly greater
than that in the oropharyngeal airway insertion group, 103 (15.3) mm Hg (P=0.017). Diastolic pressure
followed a similar pattern. In both groups, heart rate fell after induction and fell further after
insertion, and at 4 min post-insertion was significantly lower than pre-induction and pre-insertion
levels. There was no significant difference in heart rates between the two groups (P=0.372).

Conclusions. The pressor response following the insertion of nasopharyngeal airways in anaes-
thetized patients is significantly greater than that following the insertion of oropharyngeal airways.
However, the mean rise in arterial pressure does not exceed pre-induction level, and thus the
response is less severe than that likely to be associated with orotracheal or nasotracheal intubation.

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Pharyngeal airways, in conjunction with other airway inter-
ventions (head extension, chin lift and jaw thrust), are often
helpful, and sometimes essential, in the treatment of airway
obstruction in unconscious patients, whether the depressed
level of consciousness is due to anaesthesia, hypoxia, drugs,
head injury or metabolic abnormalities.1 Although orophar-
yngeal (Guedel) airways are usually used in routine
anaesthetic practice, there are some situations where naso-
pharyngeal airways may be more appropriate. For example,
they may be better tolerated in lightly anaesthetized patients.2
They are particularly useful in patients with clenched jaws,
trismus or maxillofacial injuries.3 They may be life-saving
rescue devices in any situation where treatment with oropha-
yngeal airways proves ineffective.4

The cardiovascular responses following the insertion of
oropharyngeal airways in anaesthetized patients has been
found to be of little clinical significance,5 but the pressor
responses to the insertion of nasopharyngeal airways have
not been studied. When investigating the pressor responses to
the three stages of nasotracheal intubation, Singh and Smith6
recently found that the nasal insertion of stiff plastic tubes,
derived from RAETM tracheal tubes, caused a significant rise
in arterial pressure compared with pre-induction values.
There is no information about whether soft pliable dedicated
nasopharyngeal airways used in normal clinical practice
would have similar effects.

Although transient increases in arterial pressures are of
little consequence in healthy patients, any increase in oxygen
demand in patients with ischaemic heart disease may lead to further ischaemia which could depress myocardial function and possibly cause infarction. Therefore this study aimed to compare the cardiovascular responses to the insertion of standard oropharyngeal and nasopharyngeal airways in anaesthetized patients.

Methods
Following local ethics committee approval, 24 ASA I or II patients, aged between 16 and 65 yr, undergoing elective maxillofacial surgery under general anaesthesia, requiring nasotracheal intubation as part of their anaesthetic management and giving informed written consent participated in the study. Patients with cardiovascular disease, morbid obesity, oesophageal reflux, bleeding diatheses, diabetes mellitus or a history of nasal obstruction, or who were taking vasoactive drugs, were excluded.

Thirty minutes before the induction of anaesthesia, the vasoconstrictor xylometazoline hydrochloride 0.1% was applied to the nasal mucosa. As the patient leaned forward slightly, the nozzle of the xylometazoline spray was inserted into a nostril and the patient was asked to sniff as the bottle was squeezed. Two sprays (0.09 mg per spray) were applied to each nostril. Electrocardiogram, indirect arterial pressure, arterial oxygen saturation and carbon dioxide concentrations were monitored in the anaesthetic room. All patients were investigated with the same calibrated and checked indirect arterial pressure machine (Agilent™ CMS2001) and an appropriately sized cuff was selected for each individual. After a stabilization period of at least 5 min, baseline recordings were made and the patient was given 100% oxygen. Anaesthesia was induced with fentanyl 1 μg kg⁻¹ and propofol 2.5 mg kg⁻¹ followed by atracurium 0.5 mg kg⁻¹, and the patient’s lungs were ventilated with oxygen 30%, nitrous oxide 70% and isoflurane by means of a face mask attached to a circle system. Ventilator settings were adjusted to maintain the end expired carbon dioxide concentration at 4.5–5% and the isoflurane vaporizer was adjusted to maintain the end tidal isoflurane concentration at 0.6%. Indirect arterial pressure measurements were taken at 1 min intervals.

After 4 min of ventilation, patients were allocated to receive either an oropharyngeal airway (oropharyngeal airway group) or a nasopharyngeal airway (nasopharyngeal airway group) using block randomisation stratified for gender. After group allocation, the oscillotonometer was switched to standby mode and the appropriate airways were inserted. Plastic Guedel airways (size 3 for males and size 2 for females) or nasopharyngeal (Portex™) airways (size 7 mm for males and 6 mm for females) were used. The nasopharyngeal airways were not softened by warming but were lubricated with a water-soluble gel. After completion of the insertion procedure, ventilation was resumed, the oscillotonometer was switched to automatic mode and four further arterial pressure determinations were made at 1 min intervals. If any difficulty was encountered in performing mask ventilation after the induction of anaesthesia or if significant epistaxis occurred after any insertion, the patient was withdrawn from the study and treated appropriately. After completing the cardiovascular recordings, the pharyngeal airways were removed and nasotracheal intubation was performed using a Macintosh laryngoscope.

Within-group data were analysed using repeated measures ANOVA with multiple comparisons versus control using Dunnett’s method. Between-group data were analysed using t-tests. Power analysis indicated that if the minimum clinically important difference in peak systolic arterial pressures was deemed to be 18 mm Hg, then a sample size of 12 patients in each group would be required when α=0.05 and β=0.2. Statistical analysis was performed using SigmaStat 2.03 (SPSS Inc., Chicago, IL).

Results
The groups were similar with respect to age, weight and gender (Table 1). There were no withdrawals from the study since satisfactory mask ventilation was maintained in all patients and no patient had significant epistaxis. Before the induction of anaesthesia (pre-induction) and immediately before the insertion of the airways (pre-insertion), there was no significant difference in mean systolic and diastolic arterial pressures between the two groups. After the induction of anaesthesia, arterial pressure decreased significantly in both groups. After the insertion of the nasopharyngeal airways (post-insertion), arterial pressure increased to a peak level at 1 min post-insertion and was significantly greater than pre-insertion levels for all 4 mind post-insertion (Table 2 and Fig. 1). It was not significantly greater than the pre-induction level. After the insertion of the oropharyngeal airways, there was no significant change in arterial pressure compared with pre-insertion levels. At 1 min post-insertion, arterial

<table>
<thead>
<tr>
<th></th>
<th>Oropharyngeal airway group</th>
<th>Nasopharyngeal airway group</th>
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</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>28.7 (18–42)</td>
<td>29.8 (17–41)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72 (15.3)</td>
<td>70.1 (15.2)</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>6/6</td>
<td>6/6</td>
</tr>
<tr>
<td>Smoking history (yes/no)</td>
<td>7/5</td>
<td>5/7</td>
</tr>
<tr>
<td>Pre-induction values</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic arterial pressure (mm Hg)</td>
<td>122 (16.6)</td>
<td>123 (10.2)</td>
</tr>
<tr>
<td>Diastolic arterial pressure (mm Hg)</td>
<td>69 (6.3)</td>
<td>73 (7.2)</td>
</tr>
<tr>
<td>Heart rate (beats min⁻¹)</td>
<td>74 (10.8)</td>
<td>79 (15.3)</td>
</tr>
<tr>
<td>Pre-insertion values</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic arterial pressure (mm Hg)</td>
<td>99 (19.8)</td>
<td>96 (13)</td>
</tr>
<tr>
<td>Diastolic arterial pressure (mm Hg)</td>
<td>51 (13.4)</td>
<td>49 (7.4)</td>
</tr>
<tr>
<td>Heart rate (beats min⁻¹)</td>
<td>70 (11.3)</td>
<td>72 (10)</td>
</tr>
</tbody>
</table>
pressure in the nasopharyngeal airway group was significantly greater than that in the oropharyngeal airway group (Table 2 and Fig. 1).

Before the induction of anaesthesia and immediately before the insertion of the airways, there was no significant difference in mean heart rate between the two groups. In both groups, heart rate fell after the induction of anaesthesia and fell further after insertion of the airways, and at 4 min post-insertion, heart rate was significantly lower than pre-induction and pre-insertion levels. There was no significant difference in heart rates between the two groups.

**Discussion**

In this paper we have shown that the insertion of standard nasopharyngeal airways in anaesthetized patients causes a significant rise in arterial pressure compared with pre-insertion levels, whereas the insertion of oropharyngeal airways has no significant effect. The significantly greater pressor response to nasopharyngeal airway insertion has not been reported previously and is presumably due to mechanical stimulation of the nose or the nasopharynx or both. However, the insertion of standard nasopharyngeal airways does not result in a rise in arterial pressure above pre-induction levels. Although the two publications cannot be compared directly, this finding contrasts with the results of Singh and Smith who found that nasopharyngeal airways derived from tracheal tubes, caused the arterial pressure to rise significantly above pre-induction levels. It is conceivable that the softer and more compliant material used in standard (Portex™) nasopharyngeal airways renders the device less stimulating to the nasal = nasopharyngeal mucosa than does the harder material used in the construction of tracheal tubes. The rise in arterial pressure only to pre-induction levels also contrasts sharply with the more severe responses seen after orotracheal and nasotracheal intubation.

It appears unlikely that the modest pressor response following the insertion of standard nasopharyngeal airways would result in deleterious effects in most anaesthetized patients. However, it should be borne in mind that, although indirect non-invasive arterial pressure monitors have been shown to be reasonably accurate and have been widely used in the assessment of the pressor response to intubation, they provide only intermittent reports and typically take 20–25 s to make a determination. It is thus possible that the peak arterial pressure was missed and that the true peak pressure could be higher than that observed in this investigation. Direct intra-arterial pressure measurement would have been more precise and continuous, but the use of this technique would not be ethically justifiable in patients presenting mainly for extraction of wisdom teeth.

It should also be borne in mind that in this work we studied only anaesthetized patients, in whom cardiovascular responses may have been partially attenuated by the anaesthetic agents used (particularly fentanyl, propofol and isoflurane). Therefore anaesthetists should be aware that the study does not exclude the possibility that the pressor response may be exaggerated, and thus potentially harmful, in other situations where nasopharyngeal airways may be used, for example in lightly anaesthetized patients, awake or comatose maxillofacial patients or patients with head injury or other intracranial pathology.

All the patients in this clinical trial received two sprays of xylometazoline to each nostril at least 30 min before the

### Table 2

Maximum values and maximum increases in arterial pressures and heart rates observed following insertion of oropharyngeal and nasopharyngeal airways. Data are presented as mean (SD). *P<0.05 compared with oropharyngeal airway group.

<table>
<thead>
<tr>
<th></th>
<th>Oropharyngeal airway group</th>
<th>Nasopharyngeal airway group</th>
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<tbody>
<tr>
<td><strong>Maximum observed values after airway insertion</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic arterial pressure (mm Hg)</td>
<td>103 (15.3)</td>
<td>122* (21.6)</td>
</tr>
<tr>
<td>Diastolic arterial pressure (mm Hg)</td>
<td>55 (12.6)</td>
<td>77* (17.7)</td>
</tr>
<tr>
<td>Heart rate (beats min⁻¹)</td>
<td>67 (9.6)</td>
<td>70 (10.1)</td>
</tr>
<tr>
<td><strong>Maximum observed increases (compared with pre-insertion values)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic arterial pressure (mm Hg)</td>
<td>6.1 (13.1)</td>
<td>26.2* (11.9)</td>
</tr>
<tr>
<td>Diastolic arterial pressure (mm Hg)</td>
<td>5.9 (10.3)</td>
<td>28.3* (13.1)</td>
</tr>
<tr>
<td>Heart rate (beats min⁻¹)</td>
<td>−2.4 (4.3)</td>
<td>−0.75 (6.2)</td>
</tr>
</tbody>
</table>

*Fig 1* Systolic and diastolic arterial pressure (mean, SEM) changes in oropharyngeal and nasopharyngeal airway insertion groups before induction immediately before insertion and 1, 2, 3 and 4 min post insertion. *P<0.05 between groups.
induction of anaesthesia. The local application of vasoconstrictors to reduce the incidence of epistaxis before nasotracheal or nasopharyngeal intubation is standard practice in anaesthesia for maxillofacial surgery. It is unlikely that this treatment had any affect on the results. It was applied to every patient before randomization. The mean arterial pressure of the 24 patients before the induction of anaesthesia was 122/71 mm Hg and the mean heart rate was 76 beats min\(^{-1}\). No patients had arrhythmias or any other ECG changes. These findings concur with those of previous studies\(^6\)\(^{11–13}\) involving nasal pre-treatment with vasoconstrictors.

The pressor response to the insertion of nasopharyngeal airways should be added to the other recognized complications of the device, which include failure of insertion, epistaxis (due to mucosal tears or avulsion of turbinates), laryngospasm (if the tube is too long), submucosal tunnelling and pressure sores. Contraindications include nasal airway occlusion, nasal fractures, coagulopathy (because of the risk of epistaxis), cerebrospinal fluid rhinorrhea (resulting from base of skull fracture) and adenoid hypertrophy (in paediatric patients).

References

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