

The cardiovascular changes during upper gastrointestinal endoscopy

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Abstract

Tracheal intubation under general anaesthesia causes a rise in heart rate and blood pressure. This can worsen myocardial ischaemia or precipitate infarction in patients with coronary artery disease. We monitored 50 sedated and non-sedated patients undergoing upper gastrointestinal endoscopy with continuous non-invasive blood pressure monitoring. We subsequently studied 15 patients undergoing endoscopy and oesophageal dilatation under general anaesthesia. We observed a significant ($P < 0.01$) rise in rate pressure product (RPP) occurring on oesophageal intubation in both sedated and non-sedated patients. There was no significant change in RPP on oesophageal intubation in patients under a general anaesthetic. The rise in RPP in response to tracheal intubation in those patients receiving a general anaesthetic was as great as the rise in RPP occurring in response to oesophageal intubation in the sedated and non-sedated patients. Oesophageal intubation in sedated and non-sedated patients produces a rise in RPP comparable to that associated with tracheal intubation in anaesthetised patients and may confer the same risk of worsening myocardial ischaemia in patients with coronary artery disease. © 1997 Elsevier Science B.V.

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1. Introduction

In the last 20 years, there have been enormous increases in the number of endoscopic procedures performed. A recent review from the Trent region suggests that since 1975 the incidence of upper gastrointestinal (GI) endoscopies has increased from <1 to 8.6 per thousand of the population [1]. The proportion of elderly and frail patients subjected to these procedures is also increasing [2].

A prospective audit of over 13 000 patients undergoing upper gastrointestinal endoscopy describes the mortality rate for diagnostic endoscopy to be at least 1 in 2000 procedures and the morbidity rate 1 in 200 [3]. Most of the morbidity and mortality was due to car-

diopulmonary complications such as myocardial infarction, stroke and pneumonia. A recent review suggests that since 1976, there has been a small increase in the mortality associated with this procedure and that the proportion of cardiopulmonary complications is increasing [2].

The changes in blood pressure and heart rate in response to tracheal intubation are well documented in the anaesthetic literature because of the association with myocardial ischaemia [4,5]. Multiple regimens designed to attenuate this response have been investigated. The haemodynamic responses to upper gastrointestinal endoscopy are not so widely appreciated and may be as great as those seen in anaesthesia and may cause a similar morbidity.

To find out the effect of sedation and anaesthesia on the haemodynamic side effects of upper GI endoscopy, we monitored 65 sedated, non-sedated and anaesthetised patients undergoing upper gastrointestinal en-

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doscopy, using continuous non-invasive blood pressure monitoring and pulse oximetry.

2. Method

Following ethics committee approval, 50 consecutive patients presenting for routine diagnostic upper GI endoscopy between the ages of 18 and 90 years were studied (ASA groups I–IV). The endoscopies were all performed by one experienced operator (GDB) using either a Pentax FG34X or FG29X forward viewer. Twenty patients received up to 100 mg of lignocaine topically to the pharynx approximately 5 min prior to endoscopy and no sedation. Thirty patients received midazolam sedation given over 30 s approximately 2 min prior to endoscopy. Patients aged 70 years and over received 2 mg midazolam and patients aged less than 70 years received 5 mg midazolam.

On arrival in the endoscopy suite, patients were monitored with the Finapres 2300e non-invasive blood pressure monitor together with pulse oximetry and received supplemental oxygen via nasal cannulae at 2 l/min throughout the procedure. Monitoring commenced at least 90 s prior to the endoscopy to record baseline levels of heart rate (HR) and blood pressure (BP). Heart rate, systolic and diastolic blood pressures were continuously recorded throughout the procedure and downloaded directly onto a computer for later analysis.

Fifteen patients between 18 and 90 years undergoing upper GI endoscopy and oesophageal dilatation under general anaesthesia were also studied. They received a standard anaesthetic technique. Premedication was with oral temazepam and atropine. They were pre-oxygenated for 3 min, induced with thiopentone at 3–5 mg/kg and given fentanyl 50–100 µg. Cricoid pressure was applied. Tracheal intubation was facilitated with suxamethonium at 1 mg/kg, the patients were ventilated and anaesthesia was maintained with oxygen in 66% nitrous oxide and enflurane 1–2%. Supplemental doses of suxamethonium were administered for continued muscle relaxation if clinically indicated or if the procedure was prolonged.

All patients were monitored continuously throughout the procedure with pulse oximetry, ECG, capnography and the Finapres 2300e non-invasive blood pressure monitor. Heart rate, systolic and diastolic blood pressure measurements were continuously recorded throughout the procedure and downloaded onto a computer for later analysis.

Statistical evaluation of the data was performed using analysis of variance, followed by Student's *t* test. Differences were considered to be significant if $P < 0.05$

Table 1
Gender and age characteristics of patient group

Patients	Male	Female	Mean age
Sedated	16	14	69
Non-sedated	10	9	72

3. Results

There were no statistically significant differences between the sedated and non-sedated patients in terms of age or sex (Table 1). One patient was excluded from the non-sedated group and one patient from the anaesthetised group due to technical problems with monitoring and failure of the information to download onto computer.

We compared the mean heart rate (HR), systolic blood pressure (SBP) and mean rate pressure product (RPP) over 30 s periods for each patient. Mean readings were taken over 10 s periods for 1 min following oesophageal intubation. Taking mean values over 10 or 30 s periods provided a more accurate reflection of the actual changes taking place as opposed to taking single values at intervals.

There were no significant differences in the baseline values of HR, SBP or RPP prior to oesophageal intubation in the patients sedated or not sedated. There was a significant rise in RPP in both sedated and non-sedated patients following oesophageal intubation ($P < 0.01$). There was no significant difference in the peak value of RPP in these two groups following oesophageal intubation (Table 2).

The rises in RPP that occur during upper GI endoscopy when patients are sedated (Fig. 1), not sedated (Fig. 2) or receive general anaesthesia (Fig. 3) are illustrated. In those patients receiving general anaesthesia, there were no significant differences in RPP prior to induction of anaesthesia, compared to the non-anaesthetised patients. There was no significant change in RPP on oesophageal intubation in the anaesthetised group compared to pre induction values (Fig. 3). There was a significant rise in RPP on tracheal intubation ($P < 0.01$) and the peak value of RPP attained was not significantly different to the peak value of RPP following oesophageal intubation in the sedated and non-sedated groups (Figs. 1–3).

Table 2
Rate pressure product

Patients	Baseline	(± SEM)	Peak	(± SEM)
Sedated	10 808	(± 551)	18 278	(± 1189)
Non-sedated	11 673	(± 594)	17 501	(± 1140)

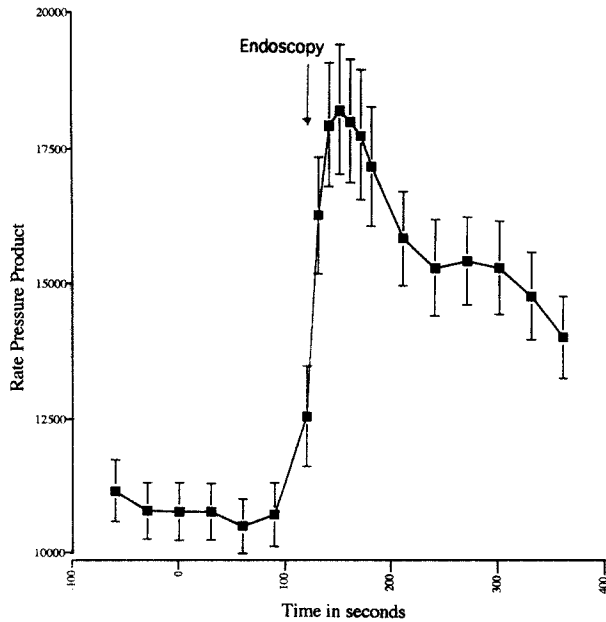


Fig. 1. Graph to show the changes in rate pressure product that occur in sedated patients undergoing upper gastrointestinal endoscopy.

The area under the RPP curve following oesophageal intubation ($t = 120$ s to $t = 300$ s) is 3.3 times greater in those patients receiving sedation, when compared to the same time interval after oesophageal intubation in patients receiving general anaesthesia ($t = 180$ s to $t = 360$ s) (Figs. 1-3). The area under the curve following oesophageal intubation in sedated patients ($t = 120$ s to $t = 360$ s) is 2.2 times greater, when compared to the same time interval after tracheal intubation (the first time of epipharyngeal stimulation) in those patients receiving general anaesthesia ($t = 60$ s to $t = 300$ s).

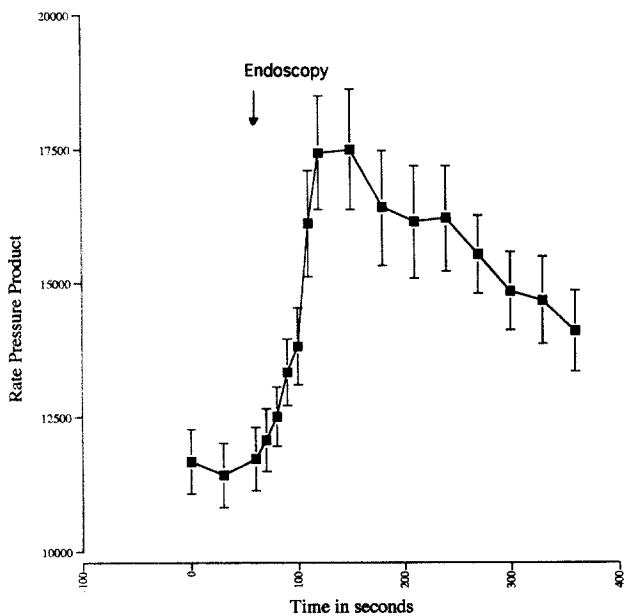


Fig. 2. Graph to show the changes in rate pressure product that occur in non-sedated patients undergoing upper gastrointestinal endoscopy.

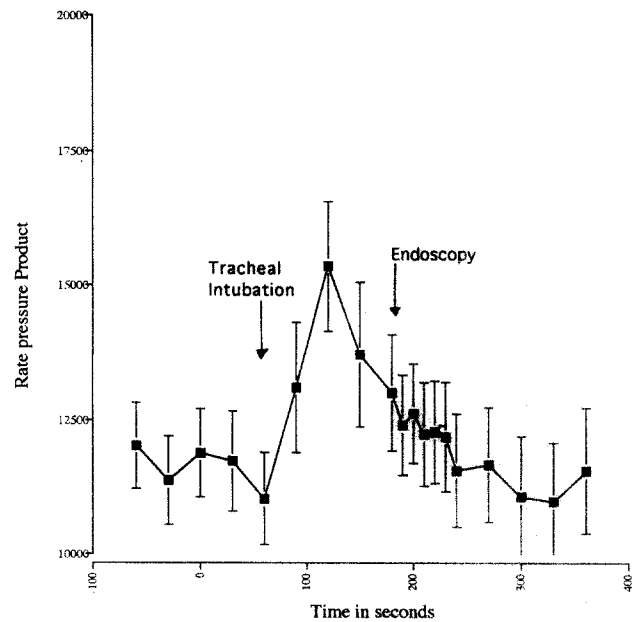


Fig. 3. Graph to show the changes in rate pressure product that occur in patients undergoing upper gastrointestinal endoscopy and oesophageal intubation under general anaesthesia.

4. Discussion

We have monitored continuously the changes in blood pressure and heart rate that occur during upper GI endoscopy and we have demonstrated a significant rise in HR, SBP and RPP in response to oesophageal intubation in both sedated and non-sedated patients. We demonstrated no difference in pressor response in those patients receiving midazolam sedation compared to those not sedated (topical local anaesthetic spray only). Patients who received a general anaesthetic did not demonstrate a significant pressor response to oesophageal intubation, however, the pressor response to tracheal intubation was of similar magnitude to the pressor response to oesophageal intubation in the other two groups. The greater area under the RPP curve in patients receiving sedation compared to patients receiving general anaesthesia suggests that their pressor response to oesophageal intubation is sustained for a longer period of time when compared to the pressor response to tracheal intubation. The myocardial oxygen demands may be greater in patients subjected to a more prolonged period of hypertension and tachycardia.

Although several other groups have documented a pressor response to oesophageal intubation by using intermittent monitoring they may have failed to document the most significant peaks of these changes [6-8]. The Finapres 2300e blood pressure monitor is an easy, non-invasive method of continuous blood pressure measurement and is more accurate than oscillometric methods of measurement [9]. It clearly demonstrates changes in blood pressure however brief, whereas inter-

mittent monitoring can miss transient changes and peaks of rises.

A recent audit of upper GI endoscopy carried out by the Royal College of Surgeons of England [3] has shown that the majority of endoscopists use a bolus injection method of sedation rather than the theoretically more correct safer slow titration method. The doses of midazolam used in our study had previously been found to produce a dysarthric and drowsy patient, who was still able to cooperate and in whom oesophageal intubation was easy and well tolerated. This was based on a study of 800 consecutive cases using bolus doses in this unit [10].

Perioperative cardiovascular changes have been extensively studied in patients undergoing coronary artery bypass surgery. Slogoff and Keats clearly demonstrated a relationship between post operative myocardial infarction and perioperative myocardial ischaemia [11]. Ischaemia was significantly associated with tachycardia and many ischaemic episodes occurred during intubation. Roy et al. also observed ischaemic episodes related most often to increase in heart rate and blood pressure following tracheal intubation in patients with known coronary artery disease during non-cardiac surgical procedures [4]. They concluded that intubation is one of the highest risk intervals in anaesthesia and surgery. It seems likely that patients with ischaemic heart disease undergoing upper GI endoscopy will be exposed to similar risks when subjected to hypertension and tachycardia particularly during oesophageal intubation.

Many asymptomatic patients have advanced coronary artery disease [12]. They are at risk of myocardial ischaemia during periods of increased oxygen demand, such as that which occurs during upper GI endoscopy and oesophageal intubation. There is a profound increase in the rate pressure product in the first few minutes of upper GI endoscopy associated with oesophageal intubation. A rise in rate pressure product increases myocardial oxygen consumption and may be associated with ST segment depression in some patients. RPP has been shown to be a reliable index of myocardial oxygen consumption [13]. A rise in RPP is known to correlate with angina [14].

Tomori and Widdicombe investigated reflex cardiovascular responses to mechanical stimulation of the upper respiratory tract in cats and found cardiovascular responses and sympathetic activity to be most pronounced during stimulation of the epipharynx [15]. This area is stimulated during upper GI endoscopy and likewise during laryngoscopy and tracheal intubation.

Our study clearly demonstrated the dramatic increase in rate pressure product that occurs on intubating the oesophagus in both sedated and non-sedated patients. In patients undergoing oesophagoscopy under general anaesthesia, the rise in RPP associated with tracheal intubation was well documented. The peak rise in RPP

was not significantly different to the rise in RPP occurring on oesophageal intubation in patients sedated or in those receiving topical local anaesthetic spray.

There has been an increase in the incidence of endoscopy related deaths in the last 20 years [2,3]. This is in comparison to anaesthetic related deaths which have fallen dramatically. One explanation for this is the increase in the use of monitoring equipment and better training standards for anaesthetists [16]. This may result in fewer critical events (hypoxaemia, arrhythmias, myocardial ischaemia) occurring during anaesthesia compared to those occurring with procedures performed under sedation. Methods to improve the safety of endoscopic procedures are urgently required.

Our patients routinely received supplemental oxygen via nasal canulae to avoid hypoxaemia and were continuously monitored using pulse oximetry. The greatest degree of oxygen desaturation occurs at the time of oesophageal intubation [17,18] i.e. at the time of increased myocardial oxygen demands, and this must therefore compound the risks of myocardial ischaemia. The Royal College of Surgeons' Working Party now recommends the administration of supplemental oxygen to all patients and we would endorse this in the face of our own results.

It is likely that the combination of tachycardia and hypertension at a time of oxygen desaturation is detrimental. This may contribute significantly to the cardiopulmonary morbidity and mortality associated with upper gastrointestinal endoscopy. The adverse myocardial effects are substantiated by ECG changes which occur at this time [7].

Increased use of monitoring with routine use of supplementary oxygen together with methods to prevent hypertension and tachycardia during endoscopy may confer greater myocardial protection. Active methods to protect the myocardium may reduce the incidence of myocardial complications and mortality associated with upper gastrointestinal endoscopy. This is clearly an area that needs further investigation.

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