

Arterial blood gas tensions during upper gastrointestinal endoscopy

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SUMMARY Arterial blood gas tensions were measured before and during upper gastrointestinal endoscopy, with (group 1) and without (group 2) sedation with intravenous diazepam. There was a highly significant fall in the PaO_2 , which occurred in both groups and was therefore not attributable to diazepam. Measurement of FEV_1 and FVC before endoscopy had no predictive value for those patients whose PaO_2 fell the most.

Upper gastrointestinal endoscopy is a safe procedure with a low morbidity and mortality. With its increasing use, previously described complications (Schiller and Prout, 1976), of which those of a respiratory nature are most prominent, could become more apparent. Cyanosis is not infrequently noted during the examination, respiratory depression or apnoea may result from the sedatives administered (Dundee and Haslett, 1970), and pulmonary aspiration can occur in up to 29% of procedures (Prout and Metreweli, 1972). Electrocardiographic abnormalities have been demonstrated in a variable proportion of examinations (De Masi and Akdamar, 1969; Sturges and Krone, 1973; Pyörälä *et al.*, 1973; Fujita and Kumura, 1975), particularly in the presence of ischaemic heart disease, and it has been suggested that hypoxia may be a contributory factor.

Although blood gas tensions have been shown to change during fiberoptic bronchoscopy (Salisbury *et al.*, 1975), to our knowledge such changes have not been studied during upper gastrointestinal endoscopy.

The purpose of this study was to measure the change, if any, in arterial blood gas tensions before and during endoscopy and to relate any changes observed to the administration of sedative and occurrence of cardiac arrhythmias. Forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) were measured beforehand to assess their predictive value for patients at risk.

Methods

Sixty-five consecutive patients who presented for

upper gastrointestinal endoscopy and gave fully informed consent for the study were investigated. One hour before the procedure, the FEV_1 and FVC were measured, using a wedge bellows spirometer (Vitalograph Ltd.). Symptoms and past history of cardiovascular disease, respiratory disease, and smoking habit were recorded. The results of the previous chest radiograph and ECG were noted.

Fifty patients (group 1) received 15 puffs of metered lignocaine throat spray (Xylocaine spray, Astra Chemicals Ltd.: one puff equals 10 mg lignocaine), followed by intravenous sedation with diazepam. A further 15 patients (group 2) were subjected to endoscopy, having only 15 puffs of throat spray and no premedication of intravenous sedation. A radial arterial puncture was made for a baseline arterial blood gas tension analysis, after which the patient's throat was sprayed with lignocaine and in group 1 intravenous diazepam administered until drowsiness was achieved, the amount given being noted. Endoscopy was performed with an Olympus GIFK endoscope and a second arterial blood sample was taken when the stomach and duodenum had been examined, but with the endoscope still remaining *in situ*. This occurred at a mean time of 12 minutes. Blood gases were measured using a Radiometer ABL 1 automatic blood gas analyser.

The patients were monitored electrocardiographically throughout the procedure and any arrhythmias noted. In addition, the efficacy of sedation and the occurrence of cyanosis were noted.

Results

Table 1 gives the details of the patients in the study and Table 2 shows the results for PaO_2 and PaCO_2 .

Table 1 Details of patients

Group	Male		Female		Age (yr)		Mean Hb (g/dl)	Diagnosis													
	no.	%	no.	%	Mean (yr)	Range		Ulcer				Gastritis		Duodenitis		Carcinoma		Normal		Other	
								Gast. no.	%	Duod. no.	%	no.	%	no.	%	no.	%	no.	%	no.	%
I	35	70	15	30	55.6	18-82	12.8	7	14	4	8	8	16	7	14	4	8	17	34	3	6
II	11	73	4	27	55.3	26-76	13.4	3	20	1	7	3	20	3	20	0		4	26	1	7

Table 2 Arterial PaO₂ and PaCO₂ before and during endoscopy with and without diazepam

Group	No.	Initial PaO ₂ (KPa)		PaO ₂ during endoscopy (KPa)		Percentage difference	
		Mean ± SE		Mean ± SE		Mean ± SE	
I (Diazepam)	50	12.32 ± 0.24		10.62 ± 0.23		13.39 ± 1.42*	
II (No diazepam)	15	12.34 ± 0.53		10.36 ± 0.42		14.78 ± 3.69*	
		Initial PaCO ₂		PaCO ₂ during endoscopy		Percentage difference	
I (Diazepam)	49	4.87 ± 0.08		5.20 ± 0.09		7.08 ± 1.40*	
II (No diazepam)	15	4.63 ± 0.11		4.49 ± 0.13		3.63 ± 2.28	

*P < 0.001.

obtained before and during the examination in both groups of subjects. In group 1 the mean PaO₂ of 10.6 ± 0.23 KPa found during the examination is significantly lower than the mean initial PaO₂ of 12.3 ± 0.24 KPa (P < 0.001). The mean percentage difference in PaO₂ between the initial and subsequent readings of 13.4% is highly significant (P < 0.001). Similar results for PaO₂ were obtained in group 2, where no diazepam was used. The number of patients examined in this group was limited to 15 as the study was continued only until it had been confirmed statistically that there was no difference, in respect of the fall of PaO₂, between the two groups. In group 1 there was a significant increase in the PaCO₂ during endoscopy, contrasting with a marginal decrease in PaCO₂ in group 2 where no diazepam was administered. The Figure shows the least squares regression line for both groups for the correlation between the percentage change in PaO₂ and the starting value. There was no significant difference in the separate regression lines for the two groups, and thus the combined regression line, which is highly significant (P < 0.01), is shown. It is clear that in all but two subjects there was a fall from the initial PaO₂, regardless of the starting value, although there was a tendency for those with the highest values to fall by a greater extent. Although the FEV₁ correlated with the initial PaO₂ (P < 0.05),

neither the FEV₁ nor the FVC were of predictive value for the percentage fall in PaO₂.

After the exclusion of those patients with pre-existing arrhythmias, in group 1 arrhythmias occurred in seven patients (14%), of whom two gave a history of ischaemic heart disease. In group 2, only one patient, who had ischaemic heart disease, had an arrhythmia. All arrhythmias except one were multiple ventricular ectopics, the exception being atrial fibrillation. All reverted spontaneously after the procedure. The occurrence of arrhythmias was not related to the degree of fall in the PaO₂.

There was no correlation between the dose of diazepam administered or depth of sedation produced and the percentage fall in oxygen tension.

Discussion

This study clearly shows that there is a marked fall in the arterial PaO₂ during uncomplicated upper gastrointestinal endoscopy. Although the mean PaO₂ during endoscopy was only just below the lower limit of normal, in many subjects it fell significantly further, the lowest recorded value being 5.9 KPa. The observation that there was an equally notable fall in PaO₂ when no diazepam was administered indicates that this effect is not due to the diazepam. It therefore seems probable that it is either the presence

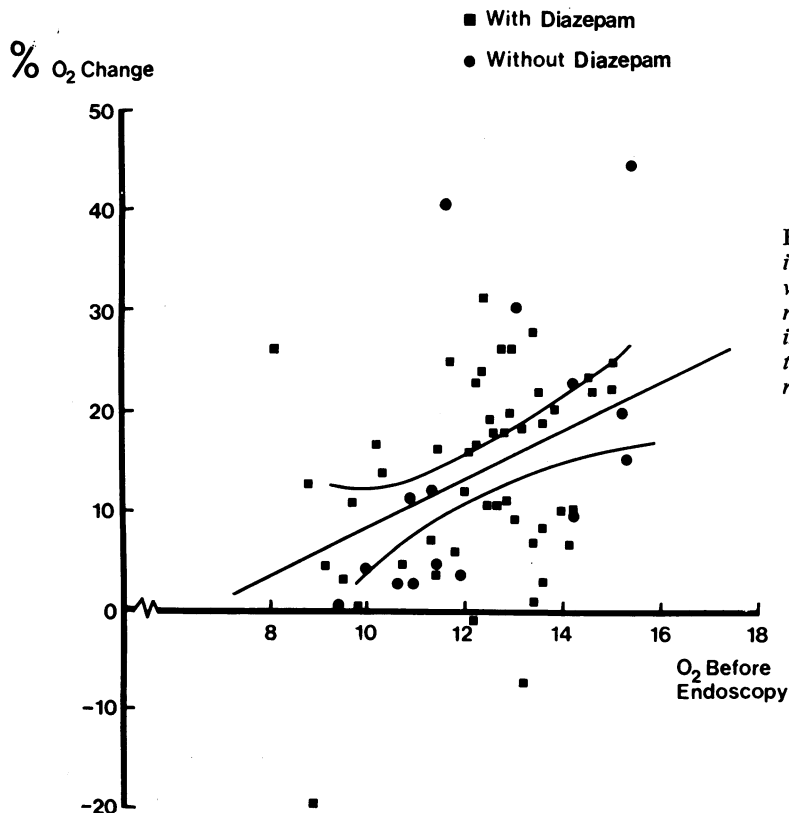


Figure The percentage change in PaO₂ plotted against the starting value, with the least squares regression line for the combined data, is shown. The curved lines show the 95% confidence limits for the regression line.

of the endoscope or the lignocaine throat spray, or a combination of the two, which is responsible for this finding. It has been previously reported that lignocaine throat spray may affect blood gas tensions (Salisbury *et al.*, 1975). However, diazepam may contribute to some extent in that the mean PaCO₂ rose only when diazepam was administered.

Although hypoxia may contribute to the generation of cardiac arrhythmias, in this study no clear relationship between hypoxia and cardiac rhythm abnormalities could be shown in our patients.

Although upper gastrointestinal endoscopy is a safe and diagnostically invaluable procedure, the present study indicates that circumspection should be used in submitting to endoscopy those patients with respiratory disease, and the previously advocated use of potential respiratory depressants as pre-medication (Dunn *et al.*, 1970; Mayes *et al.*, 1970; Prout and Schiller, 1976) is to be discouraged.

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