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Arterial Oxygen Desaturation During Esophagogastroduodenoscopy

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This prospective study evaluated the incidence and severity of arterial oxygen desaturation during esophagogastroduodenoscopy (EGD) and assessed clinical factors in relation to arterial oxygen desaturation. Following pulmonary function testing, 192 patients underwent elective EGD with continuous recording of arterial oxygen saturation (SaO_2). 62 patients (32.3%) showed arterial oxygen desaturation (SaO_2 decrease $>$ 4% from baseline SaO_2); severe arterial oxygen desaturation ($SaO_2 \leq 85\%$) reflecting hypoxemia (arterial oxygen tension $\leq$ 50 mmHg) was found in 17 patients (8.9%). These changes were most frequent at the esophageal stage of EGD but most marked at the gastroduodenal stage of EGD. The incidence of arterial oxygen desaturation was not related to age, sex, preendoscopic pulmonary function tests, smoking, or duration of endoscopy. These data support that continuous monitoring of SaO_2 should be standard procedure during endoscopy because there was no identifiable preendoscopic risk factor for arterial oxygen desaturation during EGD. But there was no persistent drop in SaO_2 longer than 2 minute. We suppose that oxygen supplement may not be required during EGD even in patients with modestly impaired pulmonary function tests.

Key Words: *Esophagogastroduodenoscopy, Complication, Pulmonary function test, Hypoxemia*

INTRODUCTION

Esophagogastroduodenoscopy (EGD) is associated with low morbidity and mortality and is considered safe procedure even in critically ill patients. Although this procedure is considered safe, there was confusion surrounding what is appropriate monitoring for these patients during endoscopy of the gastrointestinal tract (Fleischer, 1989). Recent reviews of the major complications during EGD reveal that cardioplumonary events, although uncommon, account for 50% of the morbidity and 60% of the deaths (Schiller et al., 1972; Silvis et al., 1976; Davis et al., 1979). Electrocardiographic changes during EGD have been reported both in patients with and those without a history of cardiovascular disease (Lieberman et al., 1985). Although arrhythmias such as sinus tachycardia are common in patients undergoing EGD (34%), more serious arrhythmias are seen less commonly (14%), and serious complications such as ventricular arrhythmia, angina, myocardial infarctions, and cardiac arrests are unusual.

One potential pulmonary complication during endoscopy is arterial oxygen desaturation. Arterial oxygen desaturation has been reported to occur during EGD and, in fact, the cardiac arrhythmias previously reported during EGD were most common associated with periods of maximum arterial oxygen desaturation (Levy et al., 1977; Mathew et al., 1979; Rostykus et al., 1980). In previous studies, arterial oxygen desaturation during EGD has been related to endoscopic size (Lieberman et al., 1985), premedication (Rozen et al., 1982), patient age (Rozen et al., 1982), and severity of underlying chronic obstructive lung disease (Levy et al., 1977; Mathew et al., 1979; Rostykus et al., 1980; Ona et al., 1981; Rozen et al., 1982). There are con-

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flicting data concerning the precise etiology of arterial oxygen desaturation, and no study has determined a means of identifying patients at high risk for desaturation. Continuous monitoring of arterial oxygen saturation during EGD is not routine in many institutions and because of this, the exact incidence and severity of arterial oxygen desaturation are unknown. If undetected, arterial oxygen desaturation may lead to one of the serious complications described above. In addition, few previous studies evaluating arterial oxygen desaturation during EGD have carefully evaulated lung function prior to assessing the effect of endoscope size and or premedication on the development of arterial oxygen desaturation.

The purpose of this prospective study was to determine the incidence, etiology, endoscopic stages of arterial oxygen desaturation, and its influence on the heart rate changes during EGD.

MATERIALS AND METHODS

Patient population

Patients undergoing elective EGD were recruited for study. Exclusion criteria were: unstable hemodynamic status, acute hemorrhage, the need for continuous oxygen supplementation, or inability to give informed consent. A detailed history was obtained from each patient, including informations about previous lung disease, routine examinations, and smoking history.

Protocol

All patients were studied in a standard endoscopy suite; all patient undergoing in either the intensive care unit or operating room were excluded.

Spirometric tests of lung function, considered standard measurement for airway obstruction (Gaensler et al., 1966; Higgins et al., 1973; Jenkinson, 1984), were performed in triplicate prior to administration of any medication, and the best attempt was recorded. The tests used an electronic spirometry Microspiro HI-298 (Chest corporation, Tokyo, Japan), and included forced expiratory volume in the first second (FEV1), and forced vital capacity (FVC). FEV1/FVC was then calculated. The endoscopist was blind to these results. All patients had continuous monitoring and recording of arterial oxygen saturation (SaO_2) and the heart rate by pulse oximetry with Transend Oxy Shuttle (Sensor Medics Co., Anaheim, California, USA). The 'arterial oxygen desaturation' was defined as SaO_2 decrease greater than 4% from baseline SaO_2 and severe arterial oxygen desaturation reflecting 'hypoxemia' as SaO_2 lower than 85% equivalent to arterial oxygen tension (PaO_2) lower than 50 mmHg in usual hemoglobin-oxygen dissociation curve (Prout et al., 1972; Dark et al.,1990). The endoscopic stage of maximal aretrial oxygen desaturation was remarked as the oropharyngeal stage for introduction of the endoscope from teeth to epiglottis, as the esophageal stage for from epiglottis to gastroesophageal junction and as the gastroduodenal stage for from gastric cardia to the duodenum. Each patient was continuously observed and if noted to desaturate below 85% of SaO_2, was stimulated and encouraged to breathe deeper and more frequently. Oxygen was administrated if oxygenation did not improve.

Esophagogastroduodenoscopy

After NPO from midnight, premedication consisted of oral ingestion of 1 ml of 3% simethicon solution with 10 ml of water 15 min prior to EGD and a gargle of 5 ml of 0.3% benoxinate hydrochloride solution for 5 min, followed by intramuscular injection of 20 mg of scopolamine butylbromide 20 minutes prior to EGD. All patients were examined in the left lateral positions with an Olympus GIF P20 (9 mm in outer diameter) gastroscope (Olympus Corporation, Lake Success, NY) utilizing standard endoscopic procedure.

Statistics

Correlations between SaO_2 changes and each of the following were evaluated: age, smoking history, severity of obastructive lung disease, procedure duration and endoscopic stages. Statistical analysis included: chi-square test and Student's paired t-test. Results were considered statistically significant when an obtained p value was less than 0.05.

RESULTS

Patient population

One hundred ninty-two patients were entered into

Table 1. Characteristics of the population

No. of patients	192
Age (yr)	40.9±12.8
Sex (female:male)	104 : 88
FEV1 (% predicted)	97.6±16.8
FVC (% predicted)	100.7±16.3
Smoking (pk yr)	6.4±12.2

Results are expressed as mean±SD except number of patients and sex.

the study. Population characteristics are summarized in Table 1. The patients had a mean age of 41 year, FEV1 97.6%, and FVC 100.7% of predicted value by age matching.

Arterial oxygen desaturation

Arterial oxygen saturation results are summarized in Table 2. The nadir mean SaO_2 significantly decreased from inital mean SaO_2 during EGD. The arterial oxygen desaturation occurred frequently. About one third of the patients desaturated (62/192, 32.3%), whereas severe arterial oxygen desaturation reflecting hypoxemia, occurred in 8.9% (17/192). The arterial oxygen saturation decreased most frequently at the esophageal stage of EGD (126/192, 65.6%) and the arterial oxygen desaturation occurred most frequently at the esophageal stage of EGD (46/192, 25.5%). But severe arterial oxygen desaturation, hypoxemia, occurred more frequently at the gastroduodenal stage than the esophageal stage of EGD. The heart rate also increased of 30 beats per minute from initial heart rate during EGD. Also, the heart rate increased most frequently at the esophageal stage of EGD (126/192,

Table 2. Summary of arterial oxygen saturation (SaO_2)

Initial SaO_2 (%)	96.7±12.2
Nadir SaO_2 (%)	92.0±6.7*
Desaturation	62/192 (32.3%)
Endoscopic stage of desaturation	
Oropharyngeal stage	1 (1.6%)
Esophageal stage	46 (74.2%)
Gastroduodenal stage	15 (24.2%)
Hypoxemia	17/192 (8.9%)
Initial heart rate (/min)	98.2±18.8
Maximal heart rate (/min)	129.0±20.6**
Procedure duration (min)	3.9±1.6

'Desaturation' was defined as SaO_2 decrease greater than 4% from baseline SaO_2 and 'hypoxemia' as nadir SaO_2 lower than 85%.
Results are expressed as mean±SD except desaturation and hypoxemia.
* $p<0.001$ by paired t-test (initial SaO_2 to nadir SaO_2)
** $p<0.001$ by paired t-test (initial heart rate to maximal heart rate)

Table 3. The changes of the arterial oxygen saturation (SaO_2) and the heart rate (HR) according to the endoscopic stages.

Endoscopic stages	Oropharyngeal	Esophageal	Gastroduodenal
SaO_2 decresement (%)*	0.1±0.9 (1/44)	4.8±5.4 (46/126)	12.7±10.1 (15/22)
HR increasement (/min)**	4.7±9.4 (10)	28.7±17.5 (126)	40.0±19.6 (56)

Results are expressed as mean±SD.
* The number of patients showing the arterial oxygen desaturation are depicted in the parenthesis with total number of patient at stage of maximal arterial oxygen saturation decreasement from baseline during endoscopy. The p value less than 0.001 by ANOVA between each stages.
** The number of patient showing increasement of heart rate maximally are depected in the parenthesis at each stage during endoscopy. The p value less than 0.001 by ANOVA between each stages.

Table 4. Arterial oxygen desaturation and hypoxemia during endoscopy

	Desaturation		Hypoxemia	
	not developed	developed	not developed	developed
No of patients	130	62	175	17
Age (yr)	41.0±12.3	40.7±13.9	41.2±12.7	37.9±13.9
Sex (female:male)	68 : 62	36 : 26	94 : 81	10 : 7
FEV1 (% predicted)	97.0±15.9	98.9±18.5	97.3±16.8	101.1±15.6
Smoking (pk yr)	5.8±11.7	7.8±13.3	6.3±12.1	7.8±14.2
Procedure duration (min)	3.9± 1.7	3.8± 1.3	3.8± 1.6	4.1± 1.6
Heart rate change (/min)	27.7±19.0*	37.2±19.0*	30.3±19.5	35.0±18.5

'Desaturation' was defined as SaO_2 decrease greater than 4% from baseline SaO_2 and 'hypoxemia' as nadir SaO_2 lower than 85%.
Results are expressed as mean±SD except number of patients and sex.
* $p<0.005$ by Student's t-test

65.6%), but the heart rate increased more markedly at the gastroduodenal stage than the esophageal stage of EGD.

Statistical analyses were listed in Table 4. During EGD, there was no correlation between patient age, sex, FEV1, smoking amount, and procedure duration and both arterial oxygen desturation and hypoxemia except heart rate change (=maximal heart rate - initial heart rate).

Each patient was continuously observed and if noted to desaturate below 85% of SaO_2, was stimulated and encouraged to breathe deeper and more frequently. But, oxygen was not administrated in any patients because there was no clinical indication.

Although continuous cardiac monitoring was not performed in all patients, no symptomatic cardiac arrhythmias or hypotensive episodes occurred. Blood pressure was recorded prior to and several times during procedure, and no hypotensive episodes occurred.

DISCUSSION

Gastrointestinal endoscopic procedures are reported as safe procedures with little morbidity and mortality. Experienced endoscopists rarely experience serious complications (Schiller et al., 1972; Silvis et al., 1976; Davis et al., 1979).

The present study of both inpatients and outpatients demonstrates that arterial oxygen desaturation occurs commonly during routine endoscopic procedure of stable patients. Not only was the overall incidence of arterial oxygen desaturation high (62/192, 32.3%), but also a substantial portion of those patients who desaturated experienced severe desaturation, that was hypoxemia ($SaO_2 \leq 85\%$, $PaO_2 \leq 50$ mmHg) (17/62, 27.4%). We expected to find a strong correlation between the severity of obstructive lung disease and low arterial oxygen saturation; however, this was not the case. Predisposing factors to arterial oxygen desaturation were difficult to determine. Hypoxemia and cardiac arrhythmias during endoscopy have been reported and variably related to endoscope size, patient age, and sedative used (Levy et al., 1977; Mathew et al., 1979; Rostykus et al., 1980; Ona et al., 1981; Rozen et al., 1982; Lieberman et al., 1985). The longer procedure, the greater the risk for significant arterial oxygen desaturation. This may relate most to the amount of sedatives and possibly, the type of sedative used. In this study, no sedative was used. A prior study evaluated the contribution of endoscope size to hypoxemia during EGD (Lieberman et al., 1988), and found the larger endoscope to be associated with greater arterial oxygen desaturation. In this study, a relaively small-diameter endoscope was used for all EGD's, so this factor was not a variable. However, the endoscope utilized in this study is approximately the same size as the smaller endoscope in the previously mentioned study, which makes it unlikely as a contributing factor.

The pathogensis of the hypoxemia during EGD was suggested that: (1) hypoventilation due to drugs or physical presence of the endoscope, (2) V/Q mismatching or shunting due to aspiration, drugs, or supine position of the patients, and (3) combination of these factors, or vagally mediated bronchospasm have been suggested for the reduction in arterial oxygen tension (Rostykus et al., 1980; Rozen et al., 1981; Rozen et al., 1982; Pecora et al., 1984). Arterial oxygen saturation levels decreased in patients sedated with a combination of diazepam, meperidine, and promethazine, suggesting that a combination of the drugs may have interacted to depress the oxygen level (Atluri et al., 1978). Hypoventilation has been postulated as a possible reason for the significant hypoxemia that is seen to occur in other studies. However, alveolar hypoventilation, by definition, must include a rise in arterial carbon dioxide tension along with the hypoxemia, but this event was not seen to occur in any studies (Pecora et al., 1984). Therefore, hypoventilation cannot be implicated as the etiology for the changes which did occur. Some patients aspirated small quantities of material during endoscopy. These patients were sedated and had local anesthesia in the form of lozenges and throat spray (Prout et al., 1972). The mechanical effect of the endooscope in the oropharynx may result in impaired oxygenation or contribute to pulmonary aspiration (Prout et al., 1972; Rozen et al., 1981). Due to supine and sedated position of the patient, this may occur; however, the short duration of the most EGDs makes this an unlikely explanation.

The most likely reason for the changes in arterial oxygen tension is a V/Q mismatch (Pecora et al., 1984). This may be caused by the introduction of the endoscope into the oropharynx which may induce vagal reflexes causing vasospasm and/or bronchospasm. These reflex spastic responses could induce the V/Q mismatch and, therfore, account for the hypoxemia (Widdicombe et al., 1970; Gold et al., 1972; Rostykus et al., 1980; Ona et al., 1981). The heart rate was decreased of 2-40 per minute (mean $\pm$ SD, 10.4 ± 7.6) at early stage of EGD in some patients (30/192, 15.6%), probably due to increased vagal tone. Esophageal stimuli, even swallowing alone, have been suggested as having a causal association with myocardial mal-

perfusion, thus inciting either tachy- or brady-arrhythmias. Such reflexes may lead to decreased cardiac output and subsequent pulmonary perfusion deficits (Weiss et al., 1934; Branwood et al., 1949; Bajoj et al., 1972). These finding supports that the arterial oxygen decrease and the heart rate increase occurred frequently in the esophageal stage of the endoscopy in this study. But severe arterial oxygen desaturation, hypoxemia, occurred more frequently at the gastroduodenal stage than the esophageal stage of EGD and the heart rate also increased more markedly at the gastroduodenal stage than the esophageal stage of EGD. These may be related to more severe V/Q mismatching. Gastric distensions causes a predictable vagal effect on the coronary artery flow. It is possible that reduced coronary flow resulted in a reduction in cardiac output sufficient to reduce pulmonary flow, thus inducing a perfusion deficit (Palmer, 1976). Then, the arterial oxygen desaturation became more severe at the gastroduodenal stage than at the esophageal stage as shown in this study. Minimal inflation of the esophagus and the stomach may be helpful to decrease the vagal tones of these stages of EGD. But, the fact that oxygen tension drops as early as at the esophageal stage prior to significant gastric distension and oxygen tension rises at the gastroduodenal stage during the procedure suggests that as the patient becomes more accustomed to the presence of the endoscope, the reflexes may diminish and the mismatching would become less evident, thus improving ventilation and perfusion and, therefore, oxygenation.

Unlike bronchoscopy, the period of reduced oxygen saturation is generally short, with the average duration of desaturation under 5 min during endoscopy (Fine, 1990). But, because the risk of oxygen desaturation is not predictable, all patients undergoing endoscopy should have monitoring of oxygen saturation (Dark et al., 1990). There is a disagreement among the authers as to whether the monitoring should be for all patients or for high - risk patients. Finally, there is no study which addresses the effect of monitoring oxygen saturation on clinical outcome. The use of extracorporeal equipment to monitor patients may be useful adjunct to patients surveillance, but is never a substitue for conscientious clinical assessment. However, in those situations where the individualized need of the patients indicates that measurement of cardiac rhythm or oxygen saturation will complement the clinical assessment, the use of electrocardiographic monitoring or pulse oximetry may be beneficial.

Several studies advocated the use if continuous low-flow oxygen for patients undergoing EGD (Tirlapur et al., 1982; Bell et al., 1987; Barkin et al., 1989). Some authors have advocated the use of "low-flow" oxygen during gastrointestinal endoscopic procedure as standard practice to avoid these episodes. However, the use of low-flow oxygen does not necessarily prevent desaturation. Each patient was continuously observed and if noted to desaturate below 85% of SaO_2, was stimulated and encouraged to breathe deeper and more frequently. There was no persistent drop in SaO_2 longer than 2 minute in this study. So oxygen was not administrated in any patients because there was no clinical indication. Although no patient in the current study sustained an adverse event related to hypoxia, arterial oxygen desaturation was noted frequently. Continuous monitoring of arterial oxygen saturation should be considered during all gastrointestinal endoscopic procedures, so that, in the event of severe desaturation, corrective measures can be taken to circumvent potentially serious complications.

CONCLUSION

In conclusion, this study demonstrates that arterial oxygen desaturation occurs frequently during gastrointestinal endoscopic procedures. Despite utilizing a largely outpatient population, arterial oxygen desaturation occurred in 32% of patients, and severe desaturation occurred in 27% of those. In addition, it is apparent that predisposing factors to arterial oxygen desaturation and arterial hypoxemia during EGD are difficult to determine. The use of preoperative spirometry was not reliable in predicting the severity of arterial oxygen desaturation. These data support that continuous monitoring of SaO_2 should be standard procedure during endoscopy. But, there was no persistent drop in SaO_2 longer than 2 minute. We suppose that specific therapy, such as oxygen supplement, may not be required during endoscopy even in patients with modestly impaired pulmonary function tests.

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