Routine Arterial Oxygen Saturation Monitoring Is Not Necessary during Transesophageal Echocardiography

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Summary

Background: Transesophageal echocardiography (TEE) is now an established adjunct to routine echocardiography, its diagnostic impact making it an invaluable first-line diagnostic procedure in many cardiac conditions. However, there is no unanimity in the way the transesophageal procedure is carried out, especially with regard to the need for antibiotic prophylaxis, sedation, and the monitoring of oxygen saturation.

Hypothesis: This study was prospectively undertaken (1) to determine the presence and magnitude of oxygen desaturation and (2) the changes in heart rate and blood pressure following sedation for routine TEE in an unselected and consecutive group of patients to identify those at high risk.

Methods: Arterial oxygen saturation, heart rate, and systolic, diastolic, and mean blood pressure were monitored in 106 consecutive patients undergoing routine transesophageal echocardiography. Ninety-four (89%) patients received intravenous sedation with midazolam.

Results: Three min after midazolam administration there was a drop in oxygen saturation from 97 ± 2.5 to 95 ± 2.9 ($p < 0.001$), in systolic blood pressure from 139 ± 19.5 to 124.8 ± 22.2 mmHg ($p < 0.001$), in diastolic blood pressure from 86.6 ± 19.9 to 77.5 ± 17.7 mmHg ($p < 0.001$), and in mean blood pressure from 108.3 ± 18 to 95.6 ± 28.8 mmHg ($p < 0.001$). After introduction of the transesophageal probe and during the examination, there was a further drop in oxygen saturation with a maximum drop at the 15th min of the examination (93.7 ± 3.7 vs. 97 ± 2.5 , $p < 0.001$). The maximum blood pressure drop occurred at the 12th min into recovery: systolic blood pressure dropped from 139 ± 19.5 to

118 ± 20.8 mmHg ($p < 0.001$), diastolic blood pressure from 86.6 ± 16.9 to 75.8 ± 17.9 mmHg ($p < 0.005$), and mean blood pressure from 108.3 ± 18 to 92.5 ± 19.4 mmHg ($p < 0.01$). Patients with congestive heart failure had a greater drop in oxygen saturation compared with patients who were not in heart failure ($p < 0.01$). Twelve patients did not receive any sedation; however, they all showed a drop in oxygen saturation from 97.8 ± 2.3 to 94.6 ± 3.4 ($p < 0.001$), with a maximum drop at the 15th min during the transesophageal examination.

Conclusion: In patients with no chronic obstructive airway disease who are not in congestive heart failure, routine oxygen saturation monitoring is not deemed necessary during transesophageal examination. The cause of hypoxemia during the procedure is not only related to sedation but also to esophageal intubation.

Introduction

Transesophageal echocardiography (TEE) is now an established adjunct to routine echocardiography, and its diagnostic impact has made it an invaluable first-line diagnostic procedure in many cardiac conditions. There is, however, no unanimity in the way the transesophageal procedure is carried out, particularly with regard to the need for antibiotic prophylaxis, sedation, and the monitoring of oxygen saturation. Indeed, routine monitoring for oxygen saturation is not advocated by the recommendations of the British Society of Echocardiography.¹

The need for monitoring oxygen saturation and blood pressure during endoscopic procedures has been controversial.²⁻⁵ The frequency of cardiopulmonary complications during upper gastrointestinal endoscopy is rare, approximately six events per 10,000 patients.² Although there are several reports³⁻⁷ on the efficacy of monitoring during gastrointestinal endoscopy, only one study⁸ has been prospectively carried out for TEE. This procedure often involves patients who are critically ill, with their cardiovascular system severely compromised, and who should be closely monitored, but also in outpatients in clinically noncompromised patients in whom we hypothesize that monitoring may not be necessary.

This study was therefore prospectively undertaken in order to determine (1) the presence and magnitude of oxygen desaturation and (2) the changes in heart rate and blood

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pressure following sedation for routine TEE in an unselected and consecutive group of patients. We also sought to assess whether patients in heart failure are at a higher risk for oxygen desaturation.

Patients

In all, 106 consecutive patients (66 men and 40 women), with an average age of 56 ± 14.7 years (range 15–84 years) who were referred for TEE were included in the study. Of these, 29 patients were referred for infective endocarditis, 26 for prosthetic valve assessment, 17 for cardiac source of emboli, 11 for mitral valve disease, 10 for aortic dissection, and 13 for adult congenital heart disease.

Ninety-four patients received low-dose intravenous midazolam (0.04 mg/kg), 5–8 min prior to intubation, while in 12 others the procedure was performed without sedation. The nasopharynx of all patients was anesthetized with lignocaine 2% spray given 2–3 min before intubation. None of the patients received antibiotic cover of the procedure according to the routine practice in our laboratory. No patient in this study received routine oxygen unless saturations dropped below 85%. Patients who received sedation were divided into two groups according to the presence or absence of congestive heart failure. Thirty-one patients (33%) were in heart failure [20 in classes I–II and 11 in classes III–IV of the New York Heart Association (NYHA) classification]. The remaining 65 patients comprised the non-heart failure group.

Methods

A Toshiba SSH 160 A ultrasound system with a 5 MHz bi-plane transducer was used for TEE. The maximal diameter of the probe was 11 mm at the level of transducer tip.

Arterial oxygen saturation was measured by a transcutaneous Ohmeda 3700 pulse oximeter, with continuous digital monitoring. Heart rate and systolic, diastolic, and mean blood pressure were monitored using an electronic dynamap blood pressure cuff (Physio-Control, Life-State 200). Baseline recordings were obtained before and at 3-min intervals after midazolam administration, during the transesophageal examination and recovery.

Statistical Analysis

The paired and unpaired *t*-test and multiple regression analysis were used. All values are expressed as mean values $\pm$ 1 standard deviation.

Results

Patients with congestive heart failure were older, had lower baseline oxygen saturations and slightly lower diastolic blood pressure (Table I). The only difference between sedated and

TABLE I Sedated patients prior to transesophageal intubation

	Non-CHF group (n = 63)	CHF group (n = 31)	p Value
Age (years)	52.6 ± 13.9	65.6 ± 11.6	<0.0001
Midazolam (mg)	4.3 ± 2.3	3.6 ± 2.2	NS
SaO ₂ (%)	97.5 ± 2	95.1 ± 3.8	<0.001
HR (beats/min)	87.8 ± 11.8	82.1 ± 10	NS
SBP (mmHg)	140.8 ± 17.6	134 ± 24.6	NS
DBP (mmHg)	90.3 ± 15.4	81 ± 15.9	<0.05
MNP (mmHg)	11.9 ± 17.9	102 ± 18.2	NS

Abbreviations: CHF = congestive heart failure, DBP = diastolic blood pressure, HR = heart rate, MBP = mean blood pressure, SaO₂ = oxygen saturation, SBP = systolic blood pressure.

nonsedated patients was the resting heart rate (86.8 ± 16.8 vs. 105.7 ± 9.8 beats/min, $p < 0.05$).

Midazolam Administration

The total average dose of midazolam given was 3.9 ± 1.8 mg. There was a 2% drop of oxygen saturation from 97 ± 2.5 to 95 ± 2.9 ($p < 0.001$) with a concomitant increase in heart rate up until 3 min after the transesophageal probe introduction (Fig. 1).

Systolic blood pressure dropped from 139 ± 19.5 to 124.8 ± 22.2 mmHg ($p < 0.001$) (average drop 14.4 mmHg), diastolic blood pressure dropped from 86.6 ± 15.7 to 77.6 ± 17.7 mmHg ($p < 0.001$) (average drop 9.1 mmHg), and mean blood pressure dropped from 108.3 ± 18 to 95.6 ± 20.8 mmHg ($p < 0.001$) (average drop 12.9 mmHg) (Fig. 2).

Transesophageal Probe Introduction

This was uneventful in all patients and lasted for an average of 14.1 ± 4.1 min (range 3–18). Three minutes after probe introduction the heart rate increased from 82.5 ± 16.5 to $88.8 \pm$

FIG. 1 Arterial oxygen saturation and heart rate (mean values), before (O), after midazolam administration (M), after transesophageal probe introduction (T), and during recovery (R) in all patients. SaO₂(%) = oxygen saturation, HR = heart rate.

FIG. 2 Systolic (SBP), diastolic (DBP), and mean (MBP) blood pressure (mean $\pm$ 1 standard deviation) before (O), after midazolam administration (M), after transesophageal probe introduction (T), and during recovery (R) in all patients.

18.5 beats/min ($p < 0.01$) (Fig. 1) and the average systolic, diastolic, and mean blood pressure also slightly increased briefly ($p = \text{NS}$) (Fig. 2).

During Transesophageal Imaging

There was a drop in all hemodynamic parameters, with the largest drop being that of oxygen saturation from 97 ± 2.5 to 93.7 ± 3.7 ($p < 0.001$) recorded at the 15th min of transesophageal examination (Fig. 1). Systolic blood pressure dropped from 139 ± 19.5 to 118 ± 20.8 mmHg ($p < 0.001$) in all patients (Fig. 2). Diastolic blood pressure also dropped from 86.6 ± 16.9 to 75.8 ± 17.9 mmHg ($p < 0.005$) and mean blood pressure dropped from 108.3 ± 18 to 92.5 ± 19.4 mmHg ($p < 0.005$). The maximal blood pressure drop occurred at the 12th min into recovery. Following the early rise in heart rate during transesophageal probe introduction, this was gradually decreased throughout the examination with the maximum drop of heart rate seen at the 9th min into recovery. The changes in heart rate appear to follow those of oxygen saturation and systemic blood pressure (Fig. 3).

During Recovery

Oxygen saturation started returning to baseline exactly after probe extubation (Fig. 1), while the blood pressure drop was extended well into recovery until the 12th min (Fig. 2). Both oxygen saturation and blood pressure had fully recovered 18 min after probe extubation (Fig. 3).

Multiple regression analysis was undertaken with independent variables including the patient's age, the dose of midazolam used, the procedure duration, the systolic, diastolic, and mean blood pressures as well as heart rate at the 15th min of the examination. There was a positive correlation between heart rate and oxygen saturation at the time of maximal oxygen saturation drop (the higher the heart rate the smaller the oxygen saturation drop) ($n = 52$, $r: 0.29$, $p < 0.05$). The resting value of oxygen saturation also correlated positively with the maximal oxygen saturation drop (low resting values lead to greater saturation drop during the examination) ($n = 63$, $r: 0.5$, $p < 0.01$). Multiple regression analysis with independent variables, that is, patient's age, dose of midazolam, procedure duration, value of oxygen saturation, heart rate at the time of maximum blood pressure drop, and resting blood pressure levels revealed that the resting blood pressure level correlates positively with the maximal drop of blood pressure (the lower the resting blood pressure the greater the blood pressure drop) ($n = 53$, $r: 0.65$, $p < 0.001$ for systolic, $n = 52$, $r: 0.73$, $p < 0.001$ for diastolic, and $n = 51$, $r: 0.73$, $p < 0.001$ for mean blood pressure).

Patients with Congestive Heart Failure

These patients showed a greater drop in oxygen saturations compared with patients who were not in congestive heart failure ($p < 0.01$) throughout the transesophageal examination (Fig. 4). The change in blood pressure was similar in both groups.

In patients with heart failure, heart rate remained elevated after the early peak following probe introduction, without the gradual drop demonstrated in the non-heart failure group (Fig. 5).

FIG. 3 Arterial oxygen saturation and systolic blood pressure (average values) before (O), after midazolam administration (M), after transesophageal probe introduction (T), and during recovery (R) in all patients. $\text{SaO}_2(\%)$ = oxygen saturation, SBP = systolic blood pressure.

FIG. 4 Arterial oxygen saturation (mean value $\pm$ 1 standard deviation) before (O), after midazolam administration (M), after transesophageal probe introduction (T), and during recovery (R) in all patients (top) and in heart failure patients (bottom). $\text{SaO}_2(\%)$ = oxygen saturation, CHF = congestive heart failure.

FIG. 5 Heart rate (mean value $\pm$ 1 standard deviation) before (O), after midazolam administration (M), after transesophageal probe introduction (T), and during recovery (R) in all patients (top) and in patients with heart failure (bottom). HR = heart rate, CHF = congestive heart failure.

One of three patients with heart failure showed oxygen saturation $<90\%$. The oxygen saturation values at the 15th min of transesophageal examination between I–II and III–IV NYHA classes groups were 91.3 ± 5 and 89.5 ± 7.8 , respectively ($p = \text{NS}$). Only five patients demonstrated a minimum systolic blood pressure under 90 mmHg (Table II).

Nonsedated Patients

The 12 (13%) nonsedated patients also demonstrated an average of 2.6% drop in oxygen saturation, with the maximum drop recorded at the 15th min of transesophageal examination (Fig. 6); saturations dropped from $97.3 \pm 2.4\%$ to $94 \pm 5.5\%$

TABLE II Frequency distribution of individual values of oxygen saturations and systolic blood pressure in patients with (CHF) and without heart failure (No-CHF) at baseline, 3rd min after midazolam administration (Midazolam-3'), 15th min into the transesophageal examination (TEE-15'), and 12th min into recovery (Recovery-12')

$\text{SaO}_2\%$	Pre-procedure		Midazolam-3'		TEE-15'	
	No-CHF (n = 63)	CHF (n = 31)	No-CHF (n = 63)	CHF (n = 31)	No-CHF (n = 33)	CHF (n = 29)
100–95 (%)	54 (87)	24 (77)	40 (67)	12 (39)	17 (52)	5 (17)
95–90 (%)	9 (13)	5 (16)	21 (34)	13 (42)	16 (48)	14 (47)
90–85 (%)	0 (0)	0 (0)	2 (3)	5 (16)	0 (0)	8 (27)
85–80 (%)	0 (0)	2 (6)	0 (0)	1 (3)	0 (0)	2 (7)

SBP (mmHg)	Pre-procedure		Midazolam-3'		Recovery-12'	
	No-CHF (n = 63) (n = 54)	CHF (n = 31)	No-CHF (n = 31) (n = 31)	CHF (n = 31)	No-CHF (n = 63) (n = 63)	CHF (n = 31) (n = 31)
180–150 (%)	21 (33)	8 (26)	10 (15)	4 (12)	4 (7)	1 (3)
150–120 (%)	29 (47)	17 (55)	23 (37)	12 (39)	21 (39)	12 (39)
120–90 (%)	13 (20)	4 (13)	28 (45)	10 (32)	27 (50)	15 (48)
<90 (%)	0 (0)	2 (6)	2 (3)	5 (16)	2 (4)	3 (10)

Abbreviations: CHF = heart failure, $\text{SaO}_2\%$ = oxygen saturations, SBP = systolic blood pressure.

($p < 0.05$). Blood pressure did not change, but there was a non-significant rise in heart rate.

No patient suffered any complications or significant arrhythmias during the procedure.

Discussion

Transesophageal echocardiography is considered to be a safe procedure with low complication rate.⁹ However, practices differ at several institutions, particularly with regard to the need of antibiotic prophylaxis and oxygen saturation monitoring. In this study, oxygen saturation monitoring showed that all patients had mild arterial oxygen desaturation that was particularly prominent in patients with heart failure.

Midazolam decreases tidal volume,¹⁰ cardiac output,^{11, 12} and systemic vascular resistances.¹⁰ The reduction of peripheral resistance is generally more prominent in patients whose peripheral resistance is already high.¹⁰ The respiratory and cardiac depressant effects of midazolam probably lead to lung ventilation-perfusion mismatch and to oxygen saturation drop.¹³

The decrease in cardiac output^{11, 12} and peripheral resistance¹² induced by midazolam can also explain the reduction in systolic, diastolic, and mean blood pressure.

In concordance with Scriven and Cobbe,⁸ we found that patients in congestive heart failure show a greater degree of desaturation than those who are not in heart failure. It is possible that the existing impaired cardiac function, which may be related to the lower resting values of oxygen saturation together with the additional adverse effect of midazolam^{11, 12} may lead to a more prominent lung shunt so that patients with more severe heart failure tend to demonstrate a greater drop in oxygen saturation.

Nonsedated patients also desaturate during transesophageal examination. The additional decrease of oxygen saturation in sedated patients after probe introduction and the immediate improvement in oxygen saturation values immediately after extubation raises the possibility that esophageal intubation also contributes to arterial oxygen desaturation which may be due to a transient upper airways obstruction.¹⁴ This may be important to some patients irrespective of additional drug administration.

The extension of blood pressure drop into the recovery period probably indicates that this is not related to transesophageal probe introduction and is entirely midazolam-dependent. This is also supported by the fact that nonsedated patients showed no drop in their blood pressure. When the blood pressure drops, the drop lasts as long as midazolam remains active in the circulation.¹⁰ In our study, this period was approximately 40 min (from onset of blood pressure drop to blood pressure recovery).

It is believed that oxygen desaturation and mild hypotension can induce ischemic or arrhythmic episodes during TEE.¹⁵ None of our patients, however, had any evidence of ischemia or arrhythmia.

One study⁶ showed that monitoring oxygen saturation during gastroscopy is not necessary because the oxygen saturation

FIG. 6 Arterial oxygen saturation (mean value $\pm$ 1 standard deviation) before (O), after midazolam administration (M), after transesophageal probe introduction (T), and during recovery (R) in sedated patients (top) and in nonsedated patients (bottom). SaO₂(%) = oxygen saturation.

drop is similar to that occurring during normal sleep. However, the patients' status was not mentioned in that study. Another study,⁸ based on heart failure patients, recommends routine oxygen saturation monitoring during TEE.

Clinical Applications

Patients in congestive heart failure are more prone to hypoxemia, and in this category of patients we believe that oxygen saturation monitoring and oxygen support is crucial. In addition, patients with low (<85%) resting oxygen saturation values are at higher risk for severe hypoxemia and in these patients routine oxygen saturation monitoring is recommended. However, routine transesophageal oxygen saturation monitoring is not necessary in patients who are not in heart failure.

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