Hypoxaemia during transoesophageal echocardiography

Anthony J I Scriven, Stuart M Cobbe

Abstract

***Objectives*—To establish the incidence and severity of arterial oxygen desaturation during transoesophageal echocardiography performed under light intravenous sedation; to determine which patients are at greatest risk; and to assess the effects of supplementary oxygen treatment.**

***Design*—Prospective study of 150 patients referred for transoesophageal echocardiography.**

***Setting*—Echocardiography laboratory in a tertiary cardiothoracic referral centre.**

***Main outcome measure*—Transcutaneous arterial oxygen saturation.**

***Results*—During transoesophageal echocardiography mean (SD) arterial oxygen saturation (SaO_2) fell in 144 of 150 patients (96%) from 95·4%(2·6%) to 90·7%(6·3%) ($p < 0·001$). Significant hypoxaemia, defined as $SaO_2 < 90\%$, was found in 27 of 150 patients (18%); in this group SaO_2 fell from 92·9%(3·5%) to 81·8%(9·6%) ($p < 0·001$), but rose rapidly on oxygen to 95·5%(2·4%) ($p < 0·001$). Two patients became profoundly hypoxaemic with SaO_2 values of 35% and 74%. The principal risk factors for hypoxaemia during transoesophageal echocardiography were mitral valve disease, severe mitral regurgitation, and New York Heart Association symptomatic class III or IV.**

***Conclusions*—Transcutaneous oximetry and supplementary oxygen should be available routinely during transoesophageal echocardiography.**

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Transoesophageal echocardiography is now a widely practised procedure with a well established clinical value.[1 2] Several reports have confirmed that the incidence of complications is low,[3] and these include specific studies of bacteraemia, endocarditis, ischaemia, and oesophageal trauma during or after transoesophageal echocardiography.[4–6]

The incidence of hypoxaemia during transoesophageal echocardiography has not been assessed, although technically similar procedures such as upper gastrointestinal endoscopy are known to cause hypoxaemia, and the risk of arrhythmia during periods of arterial oxygen desaturation has been documented.[7] Overall, the degree of desaturation during endoscopy is reportedly modest, about 4%-6%, but falls as low as 51% have been recorded.[8 9]

Many patients who undergo transoesophageal echocardiography are elderly, and may have valvar disease, coronary artery disease, or left ventricular dysfunction. These factors may increase the risk of hypoxaemia, and indeed some patients might already be hypoxaemic before the start of the procedure. The purpose of our study was to establish the incidence and severity of arterial oxygen desaturation during transoesophageal echocardiography, to identify the patients at greatest risk, and to assess the effects of supplementary oxygen.

Patients and methods

PATIENTS

One hundred and fifty consecutive patients undergoing transoesophageal echocardiography were studied. Mean (range) age was 54 (20–72) and 99 (66%) were women. Seventy (46%) were outpatients; the rest were hospital inpatients; patients in the intensive care unit, or those with a known requirement for oxygen treatment, or regular daytime sedation were excluded. Patients with known medical contraindications or intolerance to intravenous sedation were excluded. The indications for transoesophageal echocardiography were for assessment of prosthetic and native mitral valve dysfunction (44%), source of embolus (15%), infective endocarditis (12%), aortic dissection and aortic valve disease (9%), congenital heart disease (9%), multiple prosthetic valves (6%), and for miscellaneous reasons (5%). Forty one patients were in New York Heart Association (NYHA) class I, 73 were in class II, 31 were in class III, and five were in class IV.

PREMEDICATION AND SEDATION

All patients received routine premedication with lignocaine throat spray (Xylocaine, Astra) and intravenous midazolam (Hypnovel, Roche) (0·04–0·05 mg/kg). The mean (range) dose was 3·4 (1·5–10) mg. This is a low dose of midazolam: the manufacturer's package insert indicates the usual dose to be about 0·07 mg/kg. At the dose range used in our study, most patients experienced only mild and brief sedation that facilitated passage of the probe; most patients recovered sufficiently to complete the examination with little discomfort. Transoesophageal echocardiography was performed in the standard

Department of Medical Cardiology, Royal Infirmary, Glasgow
A J I Scriven
S M Cobbe

Correspondence to:
Dr A J I Scriven, Department of Cardiology, Leicester General Hospital, Gwendolen Road, Leicester LE5 4PW.

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manner with the patient in the left lateral decubitus position. The electrocardiogram was monitored continuously in all patients. Full resuscitation facilities were available in the echo laboratory, including flumazenil (Anexate, Roche), a specific benzodiazepine antagonist.

OXIMETRY AND OXYGEN TREATMENT

Arterial oxygen saturation (SaO_2) was measured transcutaneously with a Biox 3740 pulse oximeter (Ohmeda, Louisville, KY, USA) with continuous earlobe or digital monitoring. The SaO_2 was recorded before intravenous sedation (baseline) and at its lowest stable level during transoesophageal echocardiography. Experience showed that it was common for the SaO_2 to fall transiently below 90% immediately after passage of the transoesophageal probe; it usually recovered to above 90% within 10–20 seconds when the patient had settled. Patients whose SaO_2 remained persistently below 90% for the first one or two minutes of the procedure were unlikely to improve further. They were regarded as significantly hypoxaemic for the purpose of this study, and were given oxygen at 4 l/min through nasal cannulae. In these patients, the SaO_2 was again recorded during oxygen treatment.

STATISTICAL ANALYSIS

Results are expressed as mean (SD). Comparisons between groups were made with Student's t test for paired and unpaired data, and differences between proportions in the two groups by the χ^2 test. A two tailed p value $< 0{\cdot}05$ was taken as significant.

Results

ARTERIAL DESATURATION DURING TRANSOESOPHAGEAL ECHOCARDIOGRAPHY

Overall, SaO_2 fell in 144 of 150 patients (96%) and was unchanged in six patients. In the entire group SaO_2 fell from 95·4%(2·6%) to 90·7%(6·3%) ($p < 0{\cdot}001$). The mean (range) fall was 4·7% (1%-49%).

INCIDENCE OF SIGNIFICANT HYPOXAEMIA

Significant hypoxaemia was defined as a stable SaO_2 persistently below 90%. One hundred and twenty three patients (82%) maintained an SaO_2 at or above 90% during transoesophageal echocardiography (no hypoxaemia group); their baseline SaO_2 was 96·0%(1·9%), falling to 92·7%(2·6%) during transoesophageal echocardiography ($p < 0{\cdot}001$). Significant hypoxaemia was found in 27 patients (18%, hypoxaemia group). Baseline SaO_2 in this group was 92·9%(3·5%) and fell to 81·8%(9·6%) during transoesophageal echocardiography ($p < 0{\cdot}001$); after oxygen treatment, SaO_2 rose rapidly in all hypoxaemia group patients, to 95·5%(2·4%)($p < 0{\cdot}001$).

Two patients in the hypoxaemia group developed profound hypoxaemia (SaO_2 35% and 74%), despite small doses of midazolam (2·0 and 3·0 mg). The first patient developed transient apnoea and nodal bradycardia and the second patient became bradypnoeic. The transoesophageal echocardiography probe was withdrawn and both patients responded to flumazenil and oxygen. The more severe episode occurred in a patient aged 68 who had an atrial septal defect with a right to left shunt and unrecognised chronic obstructive airways disease. The other hypoxaemic episode was thought to be an idiosyncratic reaction to sedation in a patient aged 67 with a normally functioning mitral valve prosthesis and mild mitral regurgitation.

DOSE OF MIDAZOLAM

There was no difference in the dose of midazolam between the two groups. The mean (range) dose in patients without hypoxaemia was 3·7 (1·5–10·0) mg, and in the hypoxaemia group it was 3·2 (1·5–6·0) mg.

FACTORS ASSOCIATED WITH HYPOXAEMIA

The table shows that significant hypoxaemia during transoesphageal echocardiography was associated with NYHA classes III and IV; mitral valve disease including normal prosthetic mitral valves; and, especially, severe mitral regurgitation. Thus 23/27 (85%) of patients with hypoxaemia had mitral valve disease, of whom 15 had severe mitral regurgitation (3+ or 4+). The other eight patients had diagnoses including mitral stenosis or mixed mitral valve disease (three); or non-severe mitral valve disease with infective endocarditis (two), with severe left ventricular dysfunction (two), and with aortic stenosis (one). The four patients with hypoxaemia but no mitral valve disease all had NYHA class III or IV symptoms. In addition two had severe left ventricular dysfunction, one had aortic valve endocarditis, and one had an atrial septal defect with pulmonary disease.

Incidence of hypoxaemia in patient groups during transoesophageal echocardiography

Patients	*Total (n)*	*Normal (n)*	*Hypoxaemia* (n (%))*	χ^2	*p Value*
All patients	150	123	27 (18)	—	—
NYHA class I	41	41	0 (0)	12·3	0·001
NYHA class II	73	63	10 (14)	1·8	NS
NYHA class III or IV	36	19	17 (47)	27·4	0·001
MVD (including all MVR)	93	70	23 (25)	7·5	0·01
Severe MR (3 + or 4 +)	42	27	15 (36)	12·4	0·001
MVD in NYHA II	53	43	10 (19)	0·04	NS
MVD in NYHA III or IV	28	15	13 (46)	18·8	0·001

* Defined as arterial oxygen saturation below 90%. MVD, mitral valve disease; MVR, mitral valve replacement; MR, mitral regurgitation.

Discussion

Transoesophageal echocardiography is acknowledged to be a safe procedure with a very low complication rate. Reports concerning the morbidity and mortality of transoesophageal echocardiography have generally emphasised mechanical or anatomical problems.[3–6] Transcutaneous oximetry is not used routinely in many European centres. Thus the incidence and severity of hypoxaemia during transoesophageal echocardiography has not

been previously assessed, and the potential for morbidity is unknown.

In this study of transoesophageal echocardiography performed under light sedation, we have shown that a minor degree of desaturation is almost universal, and significant hypoxaemia may occur in a sizable minority. Studies of arterial oxygen saturation during upper gastrointestinal endoscopy have shown that about half of the total fall in Sa_{O_2} is caused by intravenous sedation alone, probably as a consequence of mild ventilatory depression. A further fall in Sa_{O_2} occurs during passage of the endoscope but this may be related to partial obstruction of the upper airway.[9]

Our findings indicate that the patients at risk of hypoxaemia are principally those with mitral valve disease or prosthetic mitral valve dysfunction, and who have symptoms of NYHA class III or more. This is not surprising in view of the frequent presence of pulmonary vascular congestion and pulmonary hypertension in this group. Conversely, patients with diagnoses other than mitral valve disease seem to be at low risk unless left ventricular function is severely impaired, or pulmonary hypertension is present. A few patients may be sensitive even to small doses of benzodiazepines.

In this series, the procedure was complicated by considerable respiratory depression in two out of 150 patients (1·3%). This suggests that although significant hypoxaemia may occur in a sizable minority of patients, the incidence of related clinical morbidity is low.

Although many operators perform transoesophageal echocardiography on unsedated patients,[3 10] light sedation may improve patient comfort and allow a longer, more detailed examination. The small additional risk associated with intravenous sedation is therefore acceptable provided arterial oxygen saturation is continuously monitored, and facilities for resuscitation, including oxygen, are available. As transoesophageal echocardiography is usually performed in darkened rooms, the early signs of hypoventilation and arterial desaturation would otherwise go unnoticed. Where necessary, oxygen rapidly reverses hypoxaemia, and routine oxygen treatment may largely prevent it.[8] Routine monitoring of arterial oxygen saturation is a useful additional safety measure and we recommend that it be used routinely during transoesophageal echocardiography.

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