Hospital Practice

PREVENTION OF HYPOXAEMIA DURING UPPER-GASTROINTESTINAL ENDOSCOPY BY MEANS OF OXYGEN VIA NASAL CANNULAE

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Summary Hypoxaemia during oesophagogastroduodenoscopy is well documented and contributes to cardiac arrhythmias and occasional deaths from endoscopy. In 50 patients sedated with intravenous midazolam and examined with a large-diameter endoscope, oxygen desaturation was abolished by giving oxygen (2 litres/min) by way of nasal cannulae throughout the procedure.

INTRODUCTION

UPPER-GASTROINTESTINAL endoscopy with modern fibreoptic instruments is generally a safe procedure in experienced hands, with a mortality of less than one in five thousand.[1] Reviews of the major complications of oesophagogastroduodenoscopy (OGD) suggest that cardiopulmonary difficulties account for 50% of the morbidity and 60% of deaths from the procedure.[2-6] The cardiac arrhythmias often observed during both OGD[7-9] and fibreoptic bronchoscopy[10-12] are most commonly associated with periods of maximum oxygen desaturation.[12]

TABLE I—CLINICAL DETAILS OF PATIENTS

	This series (n = 50)	Previous series* (n = 100)
Sex (M/F)	29/21	52/48
Mean age (yr)	58·0 (15·2)	62·4 (15·0)
Dose of midazolam (mg)	6·7 (3·0)*	6·3 (2·8)†
Time from injection to intubation (min)	4·1 (1·1)	3·8 (1·2)
FEV_1	86 (22)	90 (26)
FVC	89 (20)	93 (23)
FEV_1/FVC	77 (12)	77 (11)
Baseline oxygen saturation (%)	95·7 (1·8)‡	95·4 (2·2)§

SD given in parentheses.
*Range 2–15 mg. †Range 1–15 mg. ‡Range 90–99%. § Range 86–99%.

Arterial hypoxaemia during OGD is well documented.[7,13-15] We have reported that significant hypoxaemia occurred in 15–20% of patients and was due to a combination of the depressant action of the premedication causing hypoventilation and the obstructive effect of the endoscope on the patient's upper airway.[20] In many endoscopy units midazolam is replacing diazepam as the intravenous sedative of first choice.[21] We have shown that, as with diazepam, many elderly patients require extremely small doses of midazolam to achieve adequate levels of sedation.[22]

We now report the effect of supplementary oxygen on oxygen saturation in 50 patients undergoing endoscopy and compare the findings with those previously reported in 100 patients in whom no supplementary oxygen was given.[20]

PATIENTS AND METHODS

50 consecutive patients attending for endoscopy on 1 day of the week were studied in the same way as previously described[20] except that oxygen was given by way of nasal cannulae. Informed consent was obtained from all patients. For 48 of the 50 patients ventilatory function tests (forced expiratory volume in 1 s [FEV_1], forced vital capacity [FVC], and the ratio of these two, all expressed as percentage predicted) were carried out. Each patient was fitted with

A. C. DE GROOT AND OTHERS: REFERENCES—*continued*

2. Javier R, Dumler F, Park JH. Long-term treatment with minoxidil in patients with severe renal failure. *J Cardiovasc Pharmacol* 1980; 2 (suppl 2): 149–55.
3. Zappacosta AR. Reversal of baldness in a patient receiving minoxidil for hypertension. *N Engl J Med* 1980; 303: 1480–81.
4. Seidman M, Westfried M, Maxey R. Reversal of male pattern baldness by minoxidil: a case report. *Cutis* 1981; 28: 551–53.
5. Vestey JP, Savin JA. Atrial of 1% minoxidil used topically for severe alopecia areata. *Acta Dermatol Venereol* 1986; 66: 179–80.
6. Fenton DA, Wilkinson JD. Topical minoxidil in the treatment of alopecia areata. *Br Med J* 1983; 287: 1015–17.
7. Vanderveen EE, Ellis CN, Kang S, et al. Topical minoxidil for hair regrowth. *J Am Acad Dermatol* 1984; 11: 416–21.
8. Fiedler-Weiss VC, West DP, Buys CM, Rumsfield JA. Topical minoxidil dose-response effect in alopecia areata. *Arch Dermatol* 1986; 122: 180–82.
9. Weiss VC, West DP, Fu TS, Robinson LA, et al. Alopecia areata treated with topical minoxidil. *Arch Dermatol* 1984; 120: 457–63.
10. Tosti A. Topical minoxidil useful in 18% of patients with androgenetic alopecia: a study of 430 cases. *Dermatologica* 1986; 173: 136–38.
11. De Villez RL. Topical minoxidil therapy in hereditary androgenetic alopecia. *Arch Dermatol* 1985; 121: 197–202.
12. Headington JT, Novak E. Clinical and histologic studies of male pattern baldness treated with topical minoxidil. *Curr Ther Res* 1984; 36: 1098–106.
13. Olsen EA, Weiner MS, Delong ER, Pinnell SR. Topical minoxidil in early male pattern baldness. *J Am Acad Dermatol* 1985; 13: 185–92.
14. Olsen EA, Delong ER, Weiner MS. Dose-response study of topical minoxidil in male pattern baldness. *J Am Acad Dermatol* 1986; 15: 30–37.
15. Novak E, Franz TJ, Headington JT, Wester RC. Topically applied minoxidil in baldness. *Int J Dermatol* 1985; 24: 82–87.
16. Stern RS. Topical minoxidil. A survey of use and complications. *Arch Dermatol* 1987; 123: 62–65.
17. Editorial. Unapproved use of minoxidil. *J Am Acad Dermatol* 1986; 14: 828–29.
18. Anon. Rogaine—US approval recommended despite efficacy doubts. *Scrip* March 25, 1987 (no 1190): 26–27.
19. Earhart RN, Ball J, Nuss DD, Aeling JL. Minoxidil-induced hypertrichosis: treatment with calcium thioglycolate depilatory. *South Med J* 1977; 70: 442–43.
20. Nuck BA, Fogelson SL, Lucky AW. Topical minoxidil does not act as an anti-androgen in the flank organ of the golden Syrian hamster. *Arch Dermatol* 1987; 123: 59–61.
21. Parker LN, Lifrak ET, Odell WD. Lack of a gonadal or adrenal androgenic mechanism for the hypertrichosis produced by diazoxide, phenytoin and minoxidil. *Biochem Pharmacol* 1982; 31: 1948–50.
22. Wester RC, Maibach HI, Guy RH, Novak E. Minoxidil stimulates cutaneous blood flow in human balding scalps: pharmacodynamics measured by laser doppler velocimetry and photopulse plethysmography. *J Invest Dermatol* 1984; 82: 515–17.
23. Cohen RL, Alves MEAF, Weiss VC. Direct effects of minoxidil on epidermal cells in culture. *J Invest Dermatol* 1984; 82: 90–93.
24. Baden HP, Kubilus J. Effect of minoxidil on cultured keratinocytes. *J Invest Dermatol* 1983; 81: 558–60.
25. Franz TJ. Percutaneous absorption of minoxidil in man. *Arch Dermatol* 1985; 121: 203–06.
26. DeGreef H, Hendrickx I, Dooms-Goossens A. Allergic contact dermatitis to minoxidil. *Contact Dermatitis* 1985; 13: 194–95.
27. Tosti A, Bardazzi F, De Padova MP, Caponeri GM, Melino M, Veronesi S. Contact dermatitis to minoxidil. *Contact Dermatitis* 1985; 13: 275–76.
28. Baral J. Scalp comedones after topical minoxidil. *J Am Acad Dermatol* 1985; 13: 1051.
29. Olsen EA. Scalp comedones after topical minoxidil. *J Am Acad Dermatol* 1985; 13: 1051–52.
30. Ranchoff RE, Bergfeld WF. Topical minoxidil reduces blood pressure. *J Am Acad Dermatol* 1985; 12: 586–87.
31. Feinstein RP. The effect of topical minoxidil on blood pressure. *J Am Acad Dermatol* 1985; 13: 673–74.
32. Vanderveen EE, Ellis CN. Topical minoxidil reduces blood pressure. *J Am Acad Dermatol* 1985; 12: 587.
33. Yates VM, King CM, Harrop B. Topical minoxidil in the treatment of alopecia areata. *Br Med J* 1984; 288: 1087.
34. Rook A, Dawber R. Diseases of the hair and scalp. Oxford: Blackwell, 1982: 12.
35. Price VH, ed. Rogaine (topical minoxidil, 2%) in the management of male pattern baldness and alopecia areata (proceedings of a sponsored symposium). *J Am Acad Dermatol* 1987; 16: 647–750.
36. Olsen EA, Delong ER, Weiner MS. Long-term follow up of men with male pattern baldness treated with topical minoxidil. *J Am Acad Dermatol* 1987; 16: 688–95.
37. Price VH. Summary (of symposium). *J Am Acad Dermatol* 1987; 16: 749–50.
38. Hamilton JB. Patterned loss of hair in man: types and incidence. *Ann NY Acad Sci* 1951; 53: 708–28.

TABLE II—DISTRIBUTION OF OXYGEN SATURATIONS

Oxygen saturation (%)	% of patients						
	Baseline		On oxygen	After midazolam		During endoscopy	
	With O_2	Without		With O_2	Without	With O_2	Without
95–100	79	77	98	92	28	84	13
90–94	21	21	2	8	52	10	52
85–89	0	2	0	0	16	4	20
80–84	0	0	0	0	2	0	8
70–79	0	0	0	0	2	2	3
60–69	0	0	0	0	0	0	2
<60	0	0	0	0	0	0	2

TABLE III—CHANGES IN MEAN (SD) OXYGEN SATURATION

	Mean (SD) oxygen saturation (%)		
	Men (n = 29)	Women (n = 21)	Total (n = 50)
Baseline	95·6 (1·7)	95·9 (1·8)	95·7 (1·8)
On oxygen	98·1 (1·2)	97·6 (1·7)	97·9 (1·4)
After midazolam	97·5 (1·5)	97·3 (2·2)	97·4 (1·8)
During endoscopy	97·0 (1·9)	95·2 (5·4)	96·3 (3·8)

an induction plethysmograph vest (P. K. Morgan Ltd, Chatham, Kent) to record rate and depth of respiration throughout the procedure.

All patients underwent endoscopy lying on the left side, after receiving a spray (4% lignocaine) to anaesthetise the back of the throat. An ear oximeter (Hewlett Packard, Co 4720 1A Santago, California) was attached to the right ear to record continuously the patient's oxygen saturation throughout the procedure. After baseline oxygen saturation had been recorded, 2 litres of oxygen were delivered per minute by way of nasal cannulae for the rest of the procedure. Once a new baseline oxygen saturation had been reached and a baseline respiratory rate and excursion established, an intravenous injection of midazolam ('Hypnoval', Roche) was given as previously described.[20] The intubation was not started until oxygen saturation had stopped changing and a new baseline had been reached.

The endoscopy was carried out by G. D. B. in all patients; in 48, the Olympus GIFT forward-viewing instrument was used and in 2 the smaller Pentax 28 FGA instrument.

Changes in oxygen saturation were assessed by means of the paired Student's *t* test and p values cited are two-sided.

RESULTS

21 women and 29 men were studied. All features were similar to those of the previously reported patients[20] (table I). After the administration of 2 litres/min oxygen, the patients' mean (SD) oxygen saturation rose significantly from 95·7% (1·8%) to 97·9% (1·4%) (range 92–100%; $p < 0.001$). After the injection of midazolam, the respiratory excursion was reduced in 40 of the 50 patients (80%) but no effect was noticed in the remaining 10. Despite the sometimes pronounced hypoventilation, the lowest recorded oxygen saturation between the start of the injection of midazolam and intubation did not change (97·4% [1·8%]; range 90–100%). In our previous series[20] oxygen saturation fell to 92·1% (4·2%; range 73–98%) when respiratory excursion was similarly suppressed. The distribution of oxygen saturations is shown in table II.

During the endoscopic procedure, the lowest recorded oxygen saturation fell only very slightly (not significantly) in this series to 96·3% (3·8%; range 79–100%; table III). In contrast, in the previous series[20] the mean lowest oxygen saturation recorded during endoscopy fell significantly to 89·0% (7·0%; range 51–98·5%).

The mean (SD) change from baseline in oxygen saturation after midazolam was +1·7% (1·3%), in patients who received oxygen and −3·3% (3·4%) in those who did not ($p < 0.0001$ in each group). The mean changes during endoscopy were +0·48% (3·6%) and −6·4% (7·3%), respectively ($p < 0.0001$ in each group).

DISCUSSION

Thus, administration of oxygen (2 litres/min) by way of nasal cannulae largely prevents hypoxaemia during OGD. A similar effect of supplementary oxygen during bronchoscopy is well documented,[23–26] and it is surprising that this method has, to our knowledge, not previously been used during OGD.

Discussion of methods of preventing hypoxaemia and thus, by implication, dangerous cardiac arrhythmias[7] during OGD has centred on reducing the dose of sedation used, when possible avoiding simultaneous sedation with narcotics such as pethidine or 'Fentanyl',[17,18] and using an endoscope of smaller diameter (8·5 mm) to minimise mechanical obstruction of the upper airways.[7,18]

Although endoscopy can be carried out without intravenous sedation, the procedure is less well tolerated[7] and patients are reluctant to undergo it again. Small endoscopes (8·5 mm) are better tolerated but they are less durable, and the image quality is poorer, and biopsy samples smaller.[1] Furthermore, larger instruments (12–15 mm diameter) are increasingly being used for therapeutic procedures, such as laser photocoagulation, electrodiathermy, and thermal cautery. It is easy to see how profound hypoxaemia followed by serious cardiac arrhythmias might occur if such procedures were carried out by means of large-diameter instruments in sick, bleeding, elderly patients, many of whom may have a history of cardiac disease.[7]

In our present series, we have largely prevented hypoxaemia during OGD despite giving adequate doses of intravenous sedation and using one of the largest endoscopes available. On the basis of our study, we strongly suggest that supplementary oxygen should be used in elderly patients, particularly those with a history of cardiovascular disease.

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REFERENCES

1. Cotton PB, Williams CB. Practical gastrointestinal endoscopy. Oxford: Blackwell, 1982.
2. Schiller KFR, Cotton PB, Salmon PR. The hazards of digestive fibre-endoscopy: a survey of British experience. *Gut* 1972; 13: 1027.
3. Taylor PA, Cotton PB, Towey RM. Pulmonary complications after oesophagogastroscopy using diazepam. *Br Med J* 1972; i: 666.
4. Silva SE, Nebel O, Rogers G, Sugawa C, Mandelstam P. Endoscopic complications. *JAMA* 1976; 235: 928–30.
5. Davis RE, Graham DY. Endoscopic complications: the Texas experience. *Gastrointest Endoscopy* 1979; 25: 146–49.
6. Katon RM. Complications of upper gastrointestinal endoscopy in the gastrointestinal bleeder. *Dig Dis Sci* 1981; 26 (suppl 7): 47S–54S.
7. Lieberman DA, Wuerker CK, Katon RM. Cardiopulmonary risk of esophagogastroduodenoscopy: role of endoscope diameter and systemic sedation. *Gastroenterology* 1985; 88: 468–72.
8. Levy N, Abinader E. Continuous electrocardiographic monitoring with Holter Electrocardiocorder throughout all stages of gastroscopy. *Am J Dig Dis* 1977; 22: 1091–96.
9. Mathew PK, Ona FV, Damevski K, Wallace WA. Arrhythmias during upper gastrointestinal endoscopy. *Angiology* 1979; 30: 834–40.
10. Shrader DL, Lakshminarayan S. The effect of fiberoptic bronchoscopy on cardiac rhythm. *Chest* 1978; 73: 821–24.
11. Luck JC, Nesseder OH, Rubenstein MJ, Morrissey WL, Engel TR. Arrhythmias from fiberoptic bronchoscopy. *Chest* 1978; 74: 139–43.

References continued at foot of next page