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Double blind randomised controlled trial of effect of metoprolol on myocardial ischaemia during endoscopic cholangiopancreatography

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Abstract

***Objective*—To evaluate the effect of metoprolol, a β adrenergic blocking drug, on the occurrence of myocardial ischaemia during endoscopic cholangiopancreatography.**

***Design*—Double blind, randomised, controlled trial.**

***Setting*—University Hospital.**

***Subjects*—38 (two groups of 19) patients scheduled for endoscopic cholangiopancreatography.**

***Interventions*—Metoprolol 100 mg or placebo as premedication two hours before endoscopy.**

***Main outcome measures*—Heart rate, arterial oxygen saturation by continuous pulse oximetry, ST segment changes during endoscopic cholangiopancreatography (an ST segment deviation >1 mV was defined as myocardial ischaemia), electrocardiogram monitored continuously with a Holter tape recorder.**

***Results*—All patients had increased heart rate during endoscopy compared with rate before endoscopy, but heart rate during endoscopy was significantly lower in the metoprolol group compared with the placebo group (P = 0.0002). Twenty one patients (16 placebo, 5 metoprolol; P = 0.0008) developed tachycardia (heart rate > 100/min) during the procedure, and 11 patients (10 placebo, 1 metoprolol; P = 0.003) developed myocardial ischaemia. One patient in the placebo group had an acute inferolateral myocardial infarction. In the 10 other patients with signs of myocardial ischaemia during endoscopy the ST deviation disappeared when the endoscope was retracted. In all patients myocardial ischaemia was related to increases in heart rate, and 10 of the 11 patients had tachycardia coherent with myocardial ischaemia.**

***Conclusions*—Metoprolol prevented myocardial ischaemia during endoscopic cholangiopancreatography, probably through lowering the heart rate. Thus, tachycardia seems to be a key pathogenic factor in the development of myocardial ischaemia during endoscopy.**

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Introduction

Myocardial ischaemia (defined as ST segment deviation >1 mV on an electrocardiogram) may occur during upper gastrointestinal endoscopy.[1-4] Supplementary oxygen may increase arterial oxygen saturation during endoscopy[4-9] but does not decrease the incidence of myocardial ischaemia.[4 10 11] Previous studies have suggested tachycardia to be an important pathogenic factor in the development of myocardial ischaemia during endoscopy.[1 4] No controlled studies, however, have previously attempted to reduce heart rate during the procedure. We evaluated the effect of metoprolol, a cardiospecific β adrenoceptor blocking drug, on the occurrence of tachycardia and myocardial ischaemia during endoscopic cholangiopancreatography.

Subjects and methods

A total of 48 patients were included in the study. Two patients withdrew from the study because of anxiety (they had thrown away the pill before arriving for outpatient endoscopy), seven patients were excluded because of missing data as a result of equipment failure, and one patient was excluded because she had atrial fibrillation throughout the monitoring period which impeded the semiautomatic ST segment analysis. Thus, the study comprised 38 patients (16 men, mean (SD) age 56.9 (18.4) years). The patients were randomised (by a computerised randomisation code performed by the pharmaceutical company before the study) to receive a tablet of either metoprolol 100 mg (19 patients) or placebo (19 patients) as premedication. The tablet was given two hours before endoscopic cholangiopancreatography by a department nurse, and compliance was therefore 100% in the two groups. The study was double blinded, and the code was not broken until after final data analysis. Exclusion criteria were use of β adrenergic blocking drugs, calcium antagonists, or digitalis as well as signs of congestive heart failure or endocrine diseases.

Arterial oxygen saturation (Sao_2) was measured by continuous pulse oximetry (adhesive finger probe, Nellcor N-200, Pleasanton, California) sampling once a second into a bedside personal computer, and electrocardiogram was measured continuously with a Holter tape recorder (Spacelabs 90205, Spacelabs, Redmond, Washington). Continuous data were obtained from 20 minutes before endoscopy until 5-10 minutes after removal of the endoscope. The methods for Holter monitoring and analysis have been described elsewhere.[12] Myocardial ischaemia was defined as a change in ST level >0.1 mV

Table 1—*Characteristics of patients in two treatment groups. Figures are means (SD) unless stated otherwise. No significant differences between two groups*

Detail	Metoprolol (n = 19)	Placebo (n = 19)	Difference between group means (95% confidence intervals)	Odds ratios (95% confidence intervals)
Men/women	6/13	10/9		0.43 (0.12 to 1.54)
Age (years)	51.4 (18.9)	62.5 (16.6)	-11.1 (-22.8 to 0.6)	
Weight (kg)	70.1 (16.0)	69.6 (17.5)	0.5 (-10.6 to 11.5)	
Height (cm)	168.4 (7.8)	169.2 (9.8)	-0.8 (-6.7 to 5.0)	
Smokers	8	14		0.28 (0.08 to 1.01)
Systolic blood pressure (mm Hg)	139.2 (19.2)	146.8 (25.3)	-7.6 (-22.4 to 7.2)	
Diastolic blood pressure (mm Hg)	81.6 (10.9)	78.2 (10.7)	3.4 (-3.7 to 10.5)	
Mean blood pressure (mm Hg)(diastolic+1/3 (systolic-diastolic))	100.8 (11.8)	101.1 (12.5)	-0.3 (-8.3 to 7.8)	
Pulse rate/min	82.7 (16.2)	80.1 (12.4)	2.6 (-6.9 to 12.1)	
Duration of endoscopy (min)	31.4 (23.2)	37.4 (25.0)	-5.9 (-21.8 to 9.9)	
Diagnostic/therapeutic procedures	12/7	11/8		1.24 (0.34 to 4.47)
Lignocaine spray (mg):	77.2 (28.5)	80.0 (27.7)	-2.8 (-21.3 to 15.7)	
30	1	2		0.49 (0.05 to 5.06)
50	1	2		0.49 (0.05 to 5.06)
60	5	4		1.33 (0.30 to 5.81)
70	1	0		7.39 (0.15 to 99.99)
80	1	0		7.39 (0.15 to 99.99)
100	10	9		1.23 (0.35 to 4.31)
110	0	2		0.13 (0.01 to 2.13)
Diazepam (mg):	10.9 (4.2)	11.6 (4.9)	-0.7 (-3.7 to 2.4)	
5	1	1		1.00 (0.06 to 16.61)
7.5	5	5		1.00 (0.24 to 4.16)
10	8	6		1.55 (0.42 to 5.70)
12.5	0	1		0.14 (0.00 to 6.82)
15	3	4		0.71 (0.14 to 3.59)
20	2	1		2.02 (0.20 to 20.73)
25	0	1		0.14 (0.00 to 6.82)
Morphine (mg):	3.2 (4.6)	2.8 (4.8)	0.4 (-2.7 to 3.5)	
0	10	11		0.81 (0.23 to 2.87)
2.5	4	4		1.00 (0.21 to 4.66)
5	1	1		1.00 (0.06 to 16.61)
7.5	0	1		0.14 (0.00 to 6.82)
10	3	0		8.28 (0.81 to 84.88)
15	1	2		0.49 (0.05 to 5.06)
Hyoscine butylbromide (mg):	54.2 (35.2)	50.5 (26.1)	3.7 (-16.7 to 24.1)	
0	1	0		7.39 (0.15 to 99.99)
10	1	0		7.39 (0.15 to 99.99)
20	4	5		0.75 (0.17 to 3.30)
40	3	4		0.71 (0.14 to 3.59)
60	4	7		0.47 (0.12 to 1.89)
80	2	2		1.00 (0.13 to 7.73)
100	3	0		8.28 (0.81 to 84.88)
120	1	1		1.00 (0.06 to 16.61)

from baseline measured at 60 ms from the J point. Episodic hypoxaemia was defined as a sudden decrease in oxygen saturation of more than 4% from baseline,[13] and tachycardia was defined as a heart rate above 100/min. Data from the pulse oximeter and the Holter recording were compared manually. Synchronous time markers were placed at the start and end of each recording. Correlations between electrocardiographic abnormalities and oxygen desaturation were accepted if the electrocardiographic changes occurred within one minute on either side of the oxygen desaturation.

The endoscopic cholangiopancreatography was performed by the same endoscopist with an Olympus JF1T20 in all patients. We did not use premedication other than the study drug. A few minutes before endoscopy, intravenous diazepam was used for conscious sedation and lignocaine aerosol spray for pharyngeal topical anaesthesia (see table 1 for doses). Morphine and hyoscine butylbromide were used for pain and duodenal relaxation when needed (see table 1). The patients did not receive supplementary oxygen during the procedure. No data were available to anyone during the endoscopy session.

The study was in accordance with the Helsinki Declaration II and approved by the local ethics committee and the Danish Health Authorities. Patients were included after written informed consent. The Mann-Whitney test, the Fisher exact test, and Spearman correlations were used for statistical analyses. Level of significance was chosen at $P<0.05$. Group values are given as means (SD) if not otherwise stated.

A previous study from our group in patients undergoing endoscopic cholangiopancreatography found myocardial ischaemia in 10 of 16 patients (63%) studied.[1] Thus, with a type 1 error of 5%, a type 2 error of 20%, an expected effect rate in the control group of 63%, and a minimal difference between effect rates of 40%, a minimum of two groups of 17 patients was needed for the study.

Results

There was no difference between the two groups regarding patient characteristics (table 1). Table 2 summarises data from the Holter recordings and oximetry tracings according to treatment groups. All patients had increased heart rate during endoscopy compared with rates before endoscopy ($P<0.0001$), but heart rate during endoscopy was significantly lower in the metoprolol group compared with the placebo group ($P = 0.0002$; table 2). Twenty one patients (16 placebo, 5 metoprolol; $P = 0.0008$) developed tachycardia (heart rate >100/min) during the procedure. There were no significant differences in oxygen saturation results between the two groups ($P>0.5$; table 2). There was no significant correlation between the dose of hyoscine butylbromide given and the mean ($P = 0.6$) or maximum heart rate ($P = 0.4$) during endoscopy.

Table 2—*Results of pulse oximetry and electrocardiography according to allocated treatments. Figures are means (SD) unless stated otherwise*

Detail	Metoprolol (n = 19)	Placebo (n = 19)	Difference between group means (95% confidence interval)	Odds ratio (95% confidence interval)
Sao_2 before endoscopy (%)	97.4 (2.7)	97.4 (2.9)	0.0 (−1.9 to 1.9)	
Sao_2 during endoscopy (%)	96.2 (3.1)	95.8 (4.0)	0.3 (−2.0 to 2.7)	
Minimum Sao_2 during endoscopy (%)	89.5 (7.1)	90.6 (7.3)	−1.1 (−5.8 to 3.6)	
Heart rate/min before endoscopy	70.4 (9.9)	78.8 (14.9)	−8.4 (−16.7 to 0.0)	
Heart rate/min during endoscopy	87.4 (14.0)	108.5 (13.6)**	−21.1 (−30.2 to −12.0)	
Maximum heart rate/min during endoscopy	96.6 (15.5)	122.9 (17.7)**	−26.3 (−37.3 to −15.4)	
No with episodes of tachycardia (heart rate>100/min) during endoscopy	5	16**		0.10 (0.03 to 0.36)
No with episodes of hypoxaemia (>4% decrease) during endoscopy	10	8		1.51 (0.43 to 5.30)
No with ischaemia† (total) during endoscopy	1	10*		0.11 (0.03 to 0.42)
No with ischaemia† related to tachycardia during endoscopy	1	9*		0.12 (0.03 to 0.50)
No with ischaemia† without tachycardia during endoscopy	0	1		0.14 (0.00 to 6.82)
No with ischaemia† related to tachycardia and hypoxaemia during endoscopy	0	2		0.13 (0.01 to 2.13)
No with ischaemia† related to hypoxaemia without tachycardia during endoscopy	0	0		1.00 (1.00 to 1.00)
No developing acute myocardial infarction during endoscopy	0	1		0.14 (0.00 to 6.82)

*P<0.01 and **P<0.001 for comparison between groups, Mann-Whitney test or Fisher exact test.
Sao_2 = arterial oxygen saturation.
†Myocardial ischaemia was defined as ST deviation >0.1 mV from baseline.

Eleven patients (10 placebo, 1 metoprolol; P = 0.003) developed myocardial ischaemia during endoscopy. All instances of ischaemia were asymptomatic. In one patient (receiving placebo) the ST elevation persisted after endoscopy, and it was later verified that she had developed an inferolateral myocardial infarction during endoscopy. She recovered without effects. In the 10 other patients with signs of myocardial ischaemia during endoscopy, the ST deviation disappeared when the endoscope was retracted. In all patients myocardial ischaemia was related to increases in heart rate, and 10 (9 placebo, 1 metoprolol; P = 0.008) of the 11 patients with ischaemia had tachycardia consistent with myocardial ischaemia (table 2). There was no difference in age (P>0.2) or duration of endoscopy (P>0.2) in patients who developed ischaemia compared with those who did not. One patient receiving placebo had myocardial ischaemia without tachycardia, but the ST segment depression developed when heart rate increased from 60 to 95/min, and he did not have simultaneous hypoxaemia. One patient receiving metoprolol developed tachycardia and myocardial ischaemia during endoscopy (table 2). Of the 18 other patients receiving metoprolol, none developed myocardial ischaemia. Among the 11 patients with signs of myocardial ischaemia during endoscopy four had ST segment elevation (including the one with an acute myocardial infarction) and seven had ST segment depression. In both groups combined the mean and maximum heart rates during endoscopy were higher (P<0.01) in patients developing ischaemia (110 and 126/min, respectively) compared with patients without ischaemia (93 and 103/min, respectively). Five of the 15 patients undergoing a therapeutic procedure had myocardial ischaemia, and six of the 23 patients undergoing diagnostic cholangiopancreatography had signs of myocardial ischaemia during the procedure (P = 0.9).

No patient developed myocardial ischaemia related to episodes of hypoxaemia without concomitant tachycardia (table 2). Thus, in the two cases of ST deviation related to episodic hypoxaemia there was also simultaneous tachycardia (table 2).

One patient, a 63 year old man, experienced an episode of discomfort with a decrease in blood pressure from 130/90 mm Hg to 90/50 mm Hg, lasting 10-20 minutes, about 80 minutes after taking the active drug. He had an uneventful endoscopic procedure without hypoxaemia, tachycardia, or myocardial ischaemia. No other adverse events were encountered in the 37 remaining patients.

Discussion

Our principal finding was that metoprolol prevented the development of myocardial ischaemia during endoscopic cholangiopancreatography. Myocardial ischaemia was related to the development of tachycardia and not to hypoxaemia itself. Metoprolol lowered the heart rate during endoscopy.

We found an association between increased heart rate and the development of ST segment deviation during endoscopy, thereby confirming our previous results.[1,4] Furthermore, ST deviation was not related to hypoxaemia without concomitant tachycardia. Thus, increased cardiac oxygen demand (caused by tachycardia) rather than decreased oxygen supply (caused by hypoxaemia) is probably an important pathogenic factor in the development of myocardial ischaemia during endoscopy. Even in a risk group of cardiac patients supplementary oxygen did not reduce the overall incidence of myocardial ischaemia during endoscopy.[11] Also as most patients developing myocardial ischaemia did not have concomitant hypoxaemia their results do not support hypoxaemia per se as an important pathogenic factor in the development of myocardial ischaemia during endoscopy.[11] Thus, there are no data available in the literature to support a strong association between hypoxaemia and cardiac morbidity in gastrointestinal endoscopy.

We believe the present study to be the first controlled study evaluating the effect of β adrenergic blockade on the occurrence of myocardial ischaemia during endoscopy. Preliminary uncontrolled observations in nine patients showed that β adrenergic blockade with propranolol could prevent tachycardia during gastroscopy, and none of these nine patients developed myocardial ischaemia during the procedure.[2] During peripheral vascular surgery[14] and after cardiac operations[15] metoprolol has been reported to have a positive effect on the myocardial oxygen economy with reduced incidence of myocardial ischaemia. Further-

Key messages

- Metoprolol lowered heart rate during endoscopy
- Metoprolol prevented myocardial ischaemia during endoscopy
- Myocardial ischaemia was related to tachycardia more than to concomitant hypoxaemia
- Tachycardia seems to be a key pathogenic factor in the development of myocardial ischaemia during endoscopy

more, metoprolol and other β blockers have a strong anxiolytic effect comparable with the benzodiazepines but without concurrent sedation.[16-20] This may be another positive effect of metoprolol in gastrointestinal endoscopy, and it should therefore be included in future controlled trials with metoprolol. As we evaluated the effect of metoprolol only on myocardial ischaemia a larger randomised controlled trial with metoprolol should also include other variables of morbidity such as pancreatitis, haemorrhage, haemodynamic variables, and myocardial infarctions.

Guidelines for monitoring and oxygen therapy during endoscopy based on the assumption that hypoxaemia during endoscopy is dangerous have been issued in the United Kingdom and the United States,[21 22] although without supportive data.[23] The finding in our present and previous studies[1 4] showing hypoxaemia to be of minor or no importance in the development of myocardial ischaemia during endoscopy questions the rationale for these guidelines.

In conclusion, metoprolol prevented myocardial ischaemia during endoscopic cholangiopancreatography, probably through a lowering of the heart rate. Thus, the results support previous observations that tachycardia (and not hypoxaemia) is a key pathogenic factor in the development of myocardial ischaemia during endoscopy.

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A MEMORABLE PATIENT

Regaining my position

Born and raised in Oldham, in the heart of rugby league territory, it was only natural that I should often climb the hill from home to the Watersheddings ground to watch the Roughyeds play "the greatest game of all." Medical school in Liverpool, however, thrust me into a soccer obsessed city where the medical school rugby team offered only a game of the tedious and uninspiring plod for the unfit that is rugby union. With no easy transport, living on a student budget I drifted from the game I love for five years.

It was as a house physician at St Helens Hospital that I met the patient who changed that. As he recovered from his heart attack we would talk sometimes about rugby league and particularly "the Saints." "You should come and watch 'em, doc; you're one of us after all."

Without his urging I would probably never have taken my place one bitterly cold Sunday afternoon in the stands at Knowsley Road, smelt again those sweet smells of grass, chips, strong tea, and oil of wintergreen, or found again my passion. Neither would I have experienced the dazzling highs or wounding lows on the emotional roller coaster ridden by the supporter. Certainly without that man I would not have learnt a lesson which none of my medical tutors had considered important enough to teach.

As doctors, we all need an escape from our profession to be people in our own right; no longer shackled, however willingly, to our science and art but finding joy in the simplest pleasures. A chance to throw off professional standards and conduct and just do something else.

With the increasing workload I find myself under, the need for escape only grows. As a loyal home and away fan (on call permitting) I can now step out, decked in replica kit and scarf, into 80 minutes of pure self indulgence. I regularly thank that patient who gave me back myself and helped to keep me nearer sanity.—RICHARD BARNES *is a senior registrar in old age psychiatry in Merseyside*