Electrocardiographic changes after alcohol septal ablation in hypertrophic obstructive cardiomyopathy

J Kazmierczak, Z Kornacewicz-Jach, M Kisly, R Gil, A Wojtarowicz

Abstract

***Objective*—To report acute and mid-term electrocardiographic changes in patients with hypertrophic obstructive cardiomyopathy (HOCM) after alcohol ablation of the first large septal branch of the left anterior descending coronary artery; and to relate electrocardiographic data with the left ventricular outflow tract pressure gradients.**

***Patients*—Nine consecutive symptomatic patients with HOCM (mean (SD) age 45 (12) years).**

***Methods*—Analysis of baseline and post-procedure ECGs and 24 hour ambulatory monitoring (up to six months). ECG data were related to left ventricular outflow tract pressure gradients.**

***Results*—One patient developed complete atrioventricular block requiring permanent pacing. The PR interval was significantly prolonged up to third month after ablation. Immediately after the procedure all patients developed right bundle branch block. At the sixth month of follow up, right bundle branch block was present in four patients. New anterior ST elevation developed immediately after ablation in five of the nine patients, and new Q waves in four. The QRS duration was significantly prolonged immediately after ablation and during follow up. There was significant but transient prolongation of QT-mean and QTc-mean intervals. QT dispersion, QTc dispersion, and JTc-mean interval were not affected. JT and JTc dispersions were transiently prolonged. No serious ventricular arrhythmias were recorded during Holter monitoring, either before or after the procedure. There were no significant correlations between the left ventricular outflow tract pressure gradient and QTc, QT-d, QTc-d, JTc, JT-d, JTc-d, or QRS duration before and after ablation.**

***Conclusions*—Alcohol septal ablation for HOCM induces significant changes in the resting ECG in most patients, despite the occlusion of a relatively small artery. The changes include new Q waves, new bundle branch block, transient anterior ST segment elevation, atrioventricular block, and transient prolongation of QT interval.**

(*Heart* 1998;**80**:257–262)

Keywords: hypertrophic obstructive cardiomyopathy; alcohol septal ablation; electrocardiography; QT interval

Department of Cardiology, Pomeranian Medical School, Powstanców Wlkp.Str. 72, 70-111 Szczecin, Poland
J Kazmierczak,
Z Kornacewicz-Jach,
M Kisly
R Gil
A Wojtarowicz

Correspondence to:
Dr Kazmierczak.

Accepted for publication
24 April 1998

Alcohol ablation of the first large septal branch of the left anterior descending coronary artery is a new therapeutic method of reducing outflow tract obstruction and improving left ventricular function in patients with hypertrophic obstructive cardiomyopathy (HOCM).[1–6] Alcohol septal ablation induces myocardial infarction of the subaortic part of the interventricular septum, where the ventricular conduction system begins. One would therefore expect there to be significant changes in the electrocardiogram. We present the electrocardiographic changes in a group of nine patients who underwent the procedure.

Methods

PATIENTS

We report nine consecutive symptomatic patients with subaortic HOCM , in whom alcohol septal ablation was performed. Eight had dyspnoea, five had chest pain; seven were in New York Heart Association class II and two in class III. There were eight men and one woman, mean (SD) age 45 (12) years (range 26 to 63 years). All patients had normal coronary arteries.

In all patients the first septal branch of the left anterior descending artery was ablated. In six, 3 ml of alcohol was used; in the remaining three, 4 ml (3.3 (0.5) ml per patient).

A reduction in the left ventricular outflow tract pressure gradients immediately after the procedure, measured haemodynamically, was achieved in all patients. The left ventricular outflow tract pressure gradient at rest was reduced from 56 (58) mm Hg (range 18 to 108) before treatment to 12 (13) mm Hg (range 0 to 40, $p < 0.01$). The postextrasystolic gradient could be reduced from 139 (45) mm Hg (range 69 to 208) to 37 (34) mm Hg (range 0 to 81, $p < 0.01$). During the Valsalva manoeuvre, the gradient decreased from 132 (31) mm Hg (range 89 to 180) to 25 (21) mm Hg (range 0 to 60, $p < 0.02$). Doppler echocardiographic measurements of the pressure gradients before and after the procedure are given in table 1.

During occlusion of the septal artery and alcohol injection, all patients complained of mild to moderate chest pain and a feeling of pressure in the thorax. Administration of 3–5 mg of morphine was necessary in all patients. During first 24 hours after the procedure three of nine patients complained of chest pain or discomfort in the thorax, requiring administration of up to 5 mg of morphine.

On discharge, eight of the nine patients had improved subjectively while one (with an

Table 1 Maximum left ventricular outflow tract pressure gradients (mm Hg) before (baseline) and after alcohol septal ablation measured by Doppler echocardiography at rest

Patient	*Baseline*	*Immediately after*	*After 3 days*	*After 9 days*	*After 3 months*	*After 6 months*
1	29	10	10	10	11	11
2	13	8	10	10	13	5
3	90	40	30	45	8	10
4	108	12	30	10	18	26
5	65	5	45	45	49	32
6	117	13	25	15	6	8
7	69	10	35	20	11	13
8	55	35	51	76	–	–
9	33	25	15	10	–	–
Mean (SD)	64 (36)	18 (13)*	28 (15)*	27 (23)†	17 (15)*	15 (10)*

*$p < 0.01$ *v* baseline; †$p < 0.02$ *v* baseline.

Table 2 Morphological features of the electrocardiograms before (baseline) and after alcohol septal ablation

		After ablation					
	Baseline	*1 h*	*3 d*	*7 d*	*9 d*	*3 m*	*6 m*
Number	9	9	9	9	9	7	7
Sinus rhythm (n/total)	9/9	8/9*	8/9*	8/9*	7/9†	6/7‡	6/7‡
New ST elevation (n/total)		5/9	5/9	5/9	1/9	1/7	0/7
New Q waves (n/total)		4/9	4/9	4/9	4/9	2/7	2/7
RBBB (n/total)	1/9	2/9	2/9	1/9	2/9	1/7	1/7
RBBB + LAH (n/total)	0/9	6/9	4/9	5/9	4/9	3/7	3/7
RBBB + LPH (n/total)	0/9	1/9	0/9	0/9	0/9	0/7	0/7
Inc RBBB + LAH (n/total)	1/9	0/9	0/9	0/9	0/9	0/7	0/7
LBBB (n/total)	1/9	0/9	1/9§	1/9§	1/9§	1/7§	1/7§

*One patient with complete heart block; †one patient paced and one patient with junctional rhythm, 75/min; ‡one patient paced; §a different patient from at baseline.
h, hours; d, days; m, months; inc RBBB, incomplete right bundle branch block; LAH, left anterior hemiblock; LBBB, left bundle branch block; LPH, left posterior hemiblock; RBBB, complete right bundle branch block.

implanted pacemaker) reported no change in his subjective symptoms. After six months, subjective improvement was still present in all seven patients followed at that time.

Serial creatine kinase activity (mean (SD)) peaked at 1529 (340) IU (range 1051 to 2076) at 14.0 (7.3) hours after the procedure, and aspartate transaminase at 218 (71) IU (range 107 to 320) at 24.8 (3.3) hours.

Drug dosage was decreased twofold to fourfold on the day before ablation and continued at that level after the procedure in all patients: verapamil (120 mg/day) in seven, propranolol (90 mg/day) in one, and acebutolol (200 mg/day) in one.

Seven patients were followed for six months and the remaining two for nine days. Precise description of the haemodynamic and echocardiographic results and the clinical outcome were presented in an earlier paper.[3]

ALCOHOL ABLATION PROCEDURE

The protocol for the alcohol ablation has been described previously.[1–4] Briefly, before the procedure the left ventricular outflow tract pressure gradient was measured by two catheters placed in the left ventricle and the ascending aorta. Measurements were made at rest, after induction of premature ventricular contraction, and during the Valsalva manoeuvre. The ostium of the left coronary artery was intubated by a 7F Judkins (or VL) coronary angioplasty guiding catheter (Cordis Corporation, Miami, Florida, USA). A small diameter over-the-wire angioplasty balloon (Cobra 2.0 mm, balloon length 10 mm (Cordis)) was then advanced into a septal branch of the left anterior descending coronary artery, previously chosen at diagnostic angiography, and the branch was occluded by balloon inflation. The measurement of the left ventricular outflow tract pressure gradient was then repeated. Echocardiographic contrast injections (1–2 ml of Levovist, Schering, Germany) through the balloon catheter, with simultaneous transthoracic echocardiographic recording, were used to assess the area of the septum supplied by this artery and to relate it to the location of the myocardial hypertrophy. After this, 1 ml of 98% ethanol was slowly injected through the inflated balloon into the vessel. If the reduction of the left ventricular outflow tract pressure gradient was insufficient, further 1 ml doses of ethanol were injected at five minute intervals, up to maximum of 4 ml. Ten minutes after the last injection the balloon was deflated and the catheter removed. Pressures were then measured again.

ECG ANALYSIS

Resting 12 lead electrocardiograms were recorded at a paper speed of 50 mm/s before ablation and then after one hour, after three, seven, and nine days, and after three and six months. The following features were noted: basic rhythm, any new ST segment elevation, any new Q waves, atrioventricular block, and the presence of bundle branch block. The heart rate was calculated from the first four complexes. QRS axis in the frontal plane was calculated according to general rules (values from the range −90° to ±180° were also included). QRS duration was measured in all leads and the arithmetical mean was calculated. The longest PR interval was taken for analysis. For QT measurements an average of three consecutive intervals was taken from all leads. The mean QT (QT-mean) was the arithmetical mean from all 12 leads. QTc was calculated with the formula of Bazett for each lead separately, and a mean value was taken for further analysis (QTc-mean). For QT and QTc dispersions, the difference between the maximum and the minimum QT and QTc intervals was used (QT-d, QTc-d). To avoid the possible influence of bundle branch block on QT interval, the JT interval—defined as QT−QRS—and its derivatives (corrected JT (Jtc), JT dispersion (JT-d), and JTc-d)) were also analysed. Correlations between QTc-mean, QT-d, QTc-d, JTc-mean, JT-d, JTc-d, QRS, and the left ventricular outflow tract pressure gradient before and after ablation were assessed.

AM AMBULATORY MONITORING

All patients underwent 24 hours of ambulatory ECG monitoring at the following times: (1) before the procedure, (2) on the third and seventh day after the procedure, and (3) after three months of follow up. Recordings were obtained on magnetic tape by a dual channel recorder (Excel; Oxford Medical, Oxford, UK). Analysis was focused on ventricular arrhythmias and atrioventricular conduction disturbances. Ventricular tachycardia (VT) was defined as more than six consecutive wide complexes at a rate of > 130 beats/min. Three

Table 3 The localisation of a new ST segment elevation and new Q waves

Patient	*New ST segment elevation*	*New Q waves*
3	V1 V2	I V3 V4
4	V1 V2 V3	I II F V5 V6
5	–	I II L V5 V6
6	V1 V2 V3 V4	–
7	–	V1
8	V1 V2	–
9	V1 V2	–

to six consecutive wide beats at a rate of > 130 beats/min were considered to be non-sustained VT.

STATISTICAL ANALYSIS

Statistical calculations were performed with the Excel 4.0 program using the paired *t* test. Significance was assumed when the p value was less than 0.05. Values are expressed as mean (SD).

Results

MORPHOLOGICAL FEATURES OF THE ECG

Alcohol ablation of the first septal branch of the left anterior descending coronary artery caused distinctive changes in the resting ECG in all patients.

After the procedure all but one patient were in sinus rhythm until the eighth day when one patient developed a junctional rhythm (rate 75/min). This was associated with a change in the QRS axis from −15° to +42° and in QRS morphology (in lead V1 from RsR to rSR). By the third month after the procedure this patient had returned to normal sinus rhythm.

New anterior ST segment elevation in at least two consecutive leads developed immediately after the procedure in five of the nine patients; however, the classic evolution typical of myocardial infarction was not seen. On discharge from hospital this elevation persisted in one of the nine patients and in this patient it was still present by the third month. By the sixth month of follow up ST elevation was not recorded in any patient (table 2).

New Q waves developed one hour after ablation in four of the nine patients. At the third and sixth month follow up, new Q waves were still present in two (table 2).

The localisation of the new ST segment elevation and the new Q waves is shown in table 3. There was a tendency for the QRS axis to rotate leftward, from −17(38)° before the procedure to −28(89)° by the ninth day after the procedure, and to −45(72)° by the sixth month.

The mean heart rate did not change significantly immediately after the procedure and during follow up. Typical acute ECG changes caused by alcohol septal ablation are shown in fig 1.

ATRIOVENTRICULAR CONDUCTION

At baseline all but one patient had a normal PR interval. Two patients developed complete atrioventricular block in the catheter laboratory; one required temporary and the other permanent DDD pacing. The PR interval was

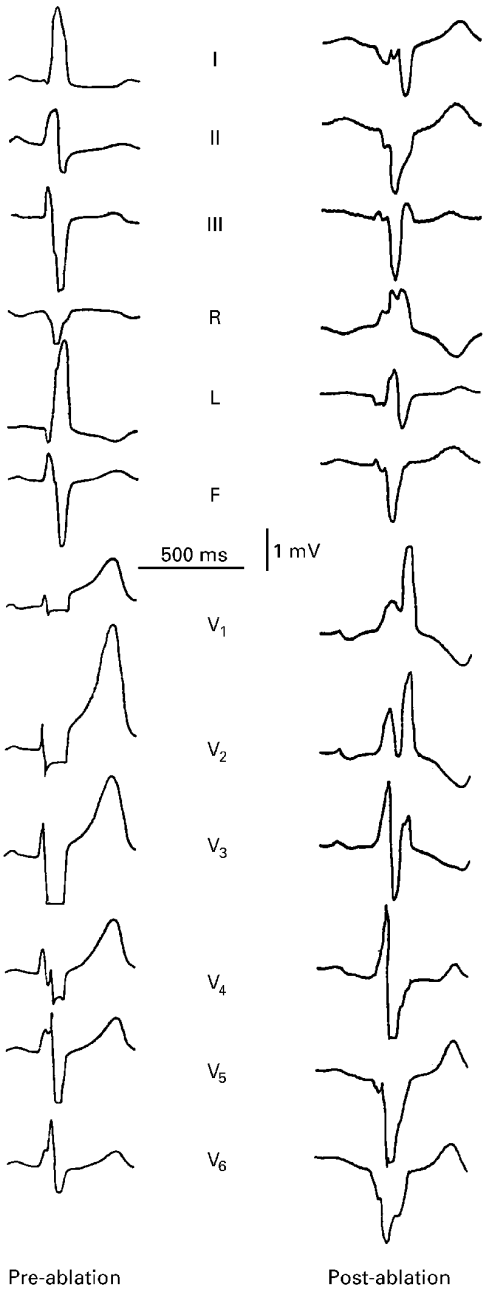

Figure 1 ECG of patient 5 before and after alcohol septal ablation. A new right bundle branch block developed. QRS duration increased from 140 ms to 260 ms. Paper speed of 50 mm/s.

prolonged from 160 (26) ms to 199 (43) ms ($p < 0.01$). This prolongation persisted during follow up, but from the third month was not large enough to be significant (table 4).

QRS MEASUREMENTS AND INTRAVENTRICULAR CONDUCTION

The average QRS duration was prolonged from 95 (19) ms at baseline to 138 (18) ms ($p < 0.01$) immediately after ablation. The significant prolongation persisted during follow up, though in three patients the QRS duration returned to baseline values (table 4). Intraventricular conduction disturbances were present in three patients before ablation—in one there was incomplete right bundle branch block, in

Table 4 Quantitative ECG parameters before (baseline) and after alcohol septal ablation

		After ablation					
	Baseline	*1 hour*	*3 days*	*7 days*	*9 days*	*3 months*	*6 months*
Number	9	9	9	9	9	7	7
HR (beats/min)	62 (8)	57 (4)	67 (13)	68 (15)	62 (13)	61 (7)	69 (9)
QRS axis (°)	−17 (33)	−35 (92)	−27 (70)	−47 (79)	−28 (89)	−33 (71)	−45 (72)
PR (ms)	160 (26)	199 (43)**	174 (41)	188 (42)**	183 (24)**	177 (27)	183 (30)
QRS (ms)	95 (19)	138 (18)**	120 (27)**	125 (31)**	123 (32)**	113 (28)*	115 (26)*
QT-mean (ms)	433 (24)	492 (24)**	447 (29)*	438 (29)	456 (33)**	450 (25)	422 (31)
QTc-mean (ms)	439 (32)	480 (23)**	470 (36)**	465 (47)*	460 (38)	453 (24)	452 (19)
QT-d (ms)	71 (34)	76 (19)	94 (30)	80 (35)	66 (18)	64 (13)	69 (29)
QTc-d (ms)	73 (36)	74 (19)	99 (32)*	86 (43)	68 (25)	65 (13)	74 (33)
JT-mean (ms)	341 (22)	352 (30)	341 (37)	316 (28)**	331 (36)	342 (17)	309 (20)**
JTc-mean (ms)	342 (22)	346 (20)	342 (23)	332 (23)	335 (24)	340 (21)	330 (22)
JT-d (ms)	71 (29)	79 (24)	102 (32)*	78 (40)	64 (13)	70 (19)	77 (24)
JTc-d (ms)	73 (31)	76 (24)	109 (39)*	83 (48)	65 (18)	71 (19)	83 (28)

Values are mean (SD).
*$p < 0.05$ *v* baseline; **$p < 0.01$ *v* baseline.
HR, heart rate; JT-d, JT dispersion; JTc, JT correction with square root; JTc-d, JTc dispersion; JTc-mean, mean JTc from 12 leads; JT-mean, mean JT from 12 leads; PR, PR interval; QT-d, QT dispersion; QTc, QT correction with square root; QTc-d, QTc dispersion; QTc-mean, mean QTc from 12 leads; QT-mean, mean QT from 12 leads.

another there was complete right bundle branch block, and in the third there was complete left bundle branch block. All patients developed complete right bundle branch block immediately after the procedure. In two patients intraventricular conduction was normalised by the second day, and another developed incomplete left bundle branch block by the seventh day instead of complete right bundle branch block. At three and six months' follow up, complete right bundle branch block was present in four of seven patients (including the patient with DDD pacing, who showed dominant sinus rhythm during ambulatory monitoring when the pacing frequency was temporarily programmed to 50 beats/min), and incomplete left bundle branch block in one patient (table 2).

QT AND JT MEASUREMENT

QT-mean was significantly prolonged at one hour and on the third and ninth day after ablation, but returned to baseline by the third month. QTc-mean before the procedure was 439 (32) ms, and exceeded 440 ms in three of the nine patients. Immediately after the procedure it was prolonged to 480 (23) ms ($p < 0.01$) and exceeded 440 ms in all nine patients. On discharge and at the three and six month follow up QTc-mean interval was 460 (38) ms, 453 (24) ms, and 452 (19) ms, respectively (all NS *v* baseline) and exceeded 440 ms in six, five, and four patients, respectively. JT-mean interval was significantly different from baseline only on the seventh day and at the six month follow up. These differences were caused by a slightly higher heart rate (table 4). JTc-mean interval did not change significantly during follow up.

No significant changes were found in QT dispersion and QTc dispersion except for QTc-d on the third day. JT-d and JTc-d increased from 71 (29) ms at baseline to 102 (32) ms ($p < 0.05$) by the third day, and from 73 (31) ms to 109 (39) ms ($p < 0.05$), respectively. By the ninth day JT-d and JTc-d had returned to preablation values (table 4).

AMBULATORY MONITORING

Holter monitoring at baseline revealed neither serious ventricular arrhythmias nor atrioventricular conduction disturbances in any patient. With the exception of single ventricular extrasystoles, ventricular arrhythmias were not seen during the procedure even at the time of alcohol injection. The only abnormalities during follow up were ventricular extrasystoles (10–20/hour) in two patients and complete heart block followed by dual chamber pacing in one patient. Holter data are summarised in table 5.

Table 5 Holter monitoring data before (baseline) and after alcohol septal ablation

		After ablation		
	Baseline	*3 days*	*7 days*	*3 months*
VES 10–20/h (n/total)	2/9	0/9	2/9	2/7
VT (n/total)	0/9	0/9	0/9	0/7
nsVT (n/total)	1/9	0/9	0/9	0/7
High grade AV block (n/total)	0/9	1/9	1/9	1/7*
Pacing (n/total)	0/9	1/9	1/9	1/7

*The patient with implanted pacemaker showed periods of sinus rhythm or DDD pacing.
AV, atrioventricular; nsVT, non-sustained ventricular tachycardia (3–6 complexes); VES, ventricular extrasystoles; VT, ventricular tachycardia.

CORRELATION OF THE LEFT VENTRICULAR OUTFLOW TRACT PRESSURE GRADIENT WITH ECG DATA

There was no significant correlation between the left ventricular outflow tract pressure gradient and QTc, QT-d, QTc-d, JTc, JT-d, JTc-d, and QRS duration before and after the ablation.

Discussion

Alcohol septal ablation leads to myocardial damage of subaortic part of the interventricular septum. The necrosis, ischaemia, or perinfarction oedema could affect branches of the His bundle.

Complete right bundle branch block developed immediately after ablation all nine patients. This is not surprising, however, because the right bundle branch is usually supplied by the septal branches of the left anterior descending coronary artery.

There was a total reversal of the new intraventricular conduction disturbances at follow up in only two patients. Thus in most patients the infarcted region affected the right bundle branch. The late occurrence of incomplete left bundle branch block in the other patient may be explained by resolution of the peri-infarction oedema of right bundle branch and fibrosis of the initial part of the left bundle branch.

Complete atrioventricular block requiring permanent DDD pacing developed in one patient, in whom right bundle branch block and a prolonged PR interval (220 ms) were recorded before ablation. This was trifascicular block, and the AV node was most probably intact. The artery supplying the AV node is most commonly a branch of the right coronary artery and sometimes of the left circumflex artery, but never a septal branch of the left anterior descending artery. At the three and six month follow up of this patient, Holter recordings showed a dominance of sinus rhythm with right bundle branch block when the pacing frequency was temporarily reduced to 50 beats/min. It therefore remains to be determined whether pre-existing bundle branch block is a contraindication to septal alcohol ablation. Knight *et al* described transient complete heart block in four of 18 patients, but five patients in this group had a permanent pacemaker implanted at the time of the procedure.[2] In other series permanent DDD pacing was necessary after the intervention in 16% to 20% of patients.[4–6]

New bundle branch block is reported to be the most common ECG change in 52% to 61% of patients.[2 4–6] These studies report no data on how many patients had bundle branch block before the procedure. After septal alcohol ablation, more than 50% of patients develop right bundle branch block. In contrast, left ventricular septectomy often creates left bundle branch block, which was thought to mimic atrioventricular sequential pacing.[7] However, right ventricular pacing affects left ventricular kinetics differently from spontaneous left bundle branch block,[8] and good clinical and haemodynamic results of DDD pacing in HOCM are not related to the fact that paced QRS complexes resemble left bundle branch block.[9] In this context, the right bundle branch block which often develops after the catheter procedure, causing changes in left ventricular kinetics, seems to have minimal effect on early and mid-term results. We did not find any relation between the reduction in pressure gradient and the development of permanent bundle branch block.

Isolated natural occlusion of the septal branch or branches has not, to the best of our knowledge, been described. We have never seen this in our catheter laboratory (where we do 1300 coronary angiographies a year). Therefore the ECG pattern of infarction caused by isolated occlusion of a septal branch is unknown. The ECG signs of myocardial infarction induced by alcohol ablation were not uniform in our nine patients. The most common acute ECG change was the development of right bundle branch block (all patients). Fresh ST segment elevation in leads V1 to V4 was present in five of the nine patients (55%), and new Q waves in four (44%). However, the localisation of new Q waves was not related to ST segment elevation, as shown in table 3. In anterior infarctions, where the proximal left anterior descending artery is occluded (together with the septal branches), right bundle branch block is a rather uncommon ECG pattern.

Prolongation of QT and QTc intervals has been reported in HOCM, especially in patients with serious ventricular arrhythmias.[10 11] In our group, the QTc interval was longer than in healthy controls both before and after the procedure.[10 12] In a study reported by Seggewiss *et al*, temporarily lengthened QTc intervals could be observed in all patients.[4] QTc prolongation as a result of ablation is at least partly caused by prolongation of the QRS complex. To avoid this influence we also analysed the behaviour of the JT interval. In contrast to QTc, JTc did not show significant changes.

Several investigators have suggested that increased QT dispersion is associated with malignant ventricular arrhythmias in patients with hypertrophic cardiomyopathy.[10 13 14] In our group, QT-d both before and after the procedure was greater than in healthy subjects, in whom values from 30 (10) ms to 51 (17) ms are reported.[10 12 13] Ablation did not significantly influence this variable. It should be noted that our patients were free from serious ventricular arrhythmias before and after the procedure, as shown by the clinical course and by repeated Holter monitoring. However, arrhythmic complications have been reported in two other series.[2 4] In one of these, one patient developed ventricular fibrillation after profound bradycardia during arterial sheath removal two hours after the procedure. Another patient developed several episodes of ventricular tachycardia in the first six hours after the intervention, presumably as a result of alcohol leaking down the main lumen of the left anterior descending artery.[2] In the second series two patients developed ventricular fibrillation before the alcohol injection owing to mechanical irritation by the catheter or the guidewire. Another patient died eight days after the procedure from uncontrollable ventricular fibrillation.[4]

Alcohol septal ablation in HOCM induces significant changes in the resting ECG in most patients, despite occlusion of a relatively small artery. However, the infarct related artery supplies the origin of the ventricular conduction system. Based on acute and mid-term ECG changes it is not possible at present to determine whether alcohol septal ablation carries a risk of ventricular arrhythmias or sudden cardiac death. Additional prospective studies are required to show whether electrocardiographic indices can be used for risk assessment after alcohol septal ablation in obstructive hypertrophic cardiomyopathy.

1 Sigwart U. Non-surgical myocardial reduction for hypertrophic obstructive cardiomyopathy. *Lancet* 1995;**346**:211–14.
2 Knight C, Kurbaan AS, Seggewiss H, *et al*. Nonsurgical septal reduction of hypertrophic obstructive cardiomypathy. Outcome in the first series of patients. *Circulation* 1997;**95**: 2075–81.
3 Kornacewicz-Jach Z, Gil R, Wojtarowicz A, *et al*. Acute results of an alcohol ablation of the septal artery in obstructive hypertrophic cardiomyopathy. *Kardiol Pol* 1998;**42**:105–12.
4 Seggewiss H, Gleichman U, Faber L, *et al*. Percutaneous transluminal septal myocardial ablation in hypertrophic obstructive cardiomyopathy: acute results and 3-month follow-up in 25 patients. *J Am Coll Cardiol* 1998;**31**:252–8.
5 Seggewiss H, Faber L, Gleichman U, *et al*. Septal branch occlusion in hypertrophic obstructive cardiomyopathy: acute results in 45 patients [abstract]. *Eur Heart J* 1997;**18**(suppl):468.
6 Gietzen F, Kuhn H, Leuner C, *et al*. Acute and long-term results after transcoronary ablation of septum hypertrophy in hypertrophic obstructive cardiomyopathy [abstract]. *Eur Heart J* 1997;**18**(suppl):468.
7 Schulte H, Bricks W, Loesse B, *et al*. Prognosis of patients with hypertrophic obstructive cardiomyopathy after transaortic myectomy. *J Thorac Cardiovasc Surg* 1993;**106**: 709–17.
8 McAreavey D, Tripodi D, Epstein N, *et al*. Dual chamber pacing relieves outflow tract obstruction in hypertrophic cardiomyopathy despite preexisting left bundle branch block [abstract]. *J Am Coll Cardiol* 1993;**21**:123A.
9 Kappenberger L, Liacle C, Daubert C, *et al*. Pacing in hypertrophic obstructive cardiomyopathy. A crossover study. *Eur Heart J* 1997;**18**:1249–56.
10 Dritsas A, Sbarouni E, Gilligan D, *et al*. QT interval abnormalities in hypertrophic cardiomyopathy. *Clin Cardiol* 1992;**15**:739–42.
11 Fei L, Slade A, Grace A, *et al*. Ambulatory assessment of the QT interval in patients with hypertrophic cardiomyopathy: risk stratification and effect of low dose amiodarone. *Pacing Clin Electrophysiol* 1994;**17**:2222–7.
12 Jordaens L, Missault L, Pelleman G, *et al*. Comparison of athletes with life-threatening ventricular arrhythmias with two groups of healthy athletes and a group of normal control subjects. *Am J Cardiol* 1994;**74**:1124–8.
13 Mioreli M, Buja G, Melacini P, *et al*. QT interval variability in hypertrophic cardiomyopathy patients with cardiac arrest. *Int J Cardiol* 1994;**15**:121–7.
14 Buja G, Mioreli M, Turini P, *et al*. Comparison of QT dispersion in hypertrophic cardiomyopathy between patients with and without ventricular arrhythmias and sudden death. *Am J Cardiol* 1993;**73**:973–6.