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Is alcohol ablation of the septum associated with recurrent tachyarrhythmias?

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Summary

Questions under study: Alcohol ablation (AA) of the septum has been introduced as new therapy in hypertrophic cardiomyopathy (HCM). It was feared that iatrogenic myocardial infarction due to AA may induce re-entry tachyarrhythmias and increase sudden cardiac death.

Methods and results: Twenty-four patients (mean age 52 years) underwent successful AA. Clinical follow-up (FU) ranged from 0.3 to 6.7 years (mean 2.8). One patient died (suicide) 4 years after AA. Left ventricular (LV) outflow gradient (peak-to-peak) decreased (median) after AA from 43 (IQR 25 to 64) mmHg to 1 (IQR 0 to 12) mmHg (rest) (p <0.001) and from 130 (IQR 75 to 165) mmHg to 13 (IQR 0 to 31) mmHg (postextrasystolic) (p <0.001). Transient AV block occurred in 22% (5/24) necessitating temporary pacing. A permanent pacemaker was implanted in 4% (1/24). NYHA-class was 2.5 (IQR 2.0 to 3.0) before and 1.5 (IQR 1.3 to 2.0) (p <0.001) after AA. During FU, 2 pacemakers were implanted due to bradycardia (no AV block). A right bundle branch block was found in 13% (2/24) before and 46% (11/24) after AA (p = 0.003). Non-sustained ventricular tachycardia (NSVT) was observed in 13% (2/16) before and 22% (5/23) (p = 0.46) after AA. Two patients required ICD implantation.

Conclusions: Long-term FU is excellent in HCM after AA. The pressure gradient drops below 25 mm Hg in 95% (23/24) of all patients. Transient AV block occurs in 22% (5/24), but permanent pacemaker implantation is rarely needed (13%, 2/24). Severe NSVT occurs in 13% (2/16) before and 22% (5/23) after AA but ICD implantation is only occasionally required.

Key words: hypertrophic cardiomyopathy; alcohol ablation; tachyarrhythmias

Introduction

Hypertrophic cardiomyopathy (HCM) is an autosomal dominant inherited disease with more than 250 mutations on more than a dozen genes [1]. Typical feature of HCM is an asymmetric septum hypertrophy with outflow tract obstruction in 25–30% of all patients; under exercise the obstruction may occur in almost 70% of all patients due to sympathetic stimulation [2]. These patients are often symptomatic with angina pectoris, dizziness, and syncopes. Sudden cardiac death may occur in obstructive and non-obstructive forms. Treatment modalities are aiming to improve symptoms and prognosis. Another important aspect is to suppress cardiac arrhythmia including atrial fibrillation and non-sustained ventricular tachycardia (NSVT). Medical therapy has been used to improve symptoms using beta-blockers, calcium channel blockers and antiarrhythmics such as disopyramid and amiodarone. Despite aggressive medical therapy, many patients remain symptomatic and have undergone septal myectomy or implantation of an ICD. Surgical myectomy has been the gold standard for treatment of hypertrophic obstructive cardiomyopathy (HOCM) for many years and has been associated

Abbreviations

AA	Alcohol ablation
AF	Atrial fibrillation
BMI	Body mass index
FU	Follow up
HCM	Hyperthrophic cardiomyopathy
HOCM	Hyperthrophic obstructive cardiomyopathy
ICD	Implantable cardioverter defibrillators
LBBB	Left bundle branch block
LV	Left ventricular
LVEDD	Left ventricular end-diastolic diameter
NSVT	Non-sustained ventricular tachycardia
PM	Pacemaker
RBBB	Right bundle branch block
SAM	Systolic anterior motion
SCD	Sudden cardiac death
SR	Sinus rhythm

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with symptomatic improvement and reduction in cardiovascular mortality. More recently, alcohol ablation of the septum with injection of small amounts of alcohol into the first, second or third septal branch was introduced in the late nineties [3]. Alcohol ablation can be done percutaneously without the need of open heart surgery [3–6]. A word of caution has been raised that iatrogenic septal myocardial infarction might cause re-entry tachyarrhythmia [7].

Previous clinical studies have not indicated an increase in arrhythmogenic substrate but the purpose of the present study is to assess cardiac arrhythmia before and after alcohol ablation in a small group of carefully examined patients with hypertrophic obstructive cardiomyopathy.

Methods

Patient population

Twenty-four patients (mean age 52±16 years, 8 women, 16 men) with hypertrophic obstructive cardiomyopathy were included in the present analysis. Patients were selected from a total group of 54 patients with hypertrophic cardiomyopathies seen between 1999 and 2005 at our institution (screening). Genetic screening for determination of the mutations was not done except in one case, which was a family form with severe HOCM. All underwent successful alcohol ablation. Patients were clinically followed for a mean of 2.8 years (4 months to 6.7 years). Body mass index (BMI) was 26.1±5. **Clinical symptoms** were: angina pectoris in 71% (17/24), dyspnoea in 92% (22/24), presyncopes and syncopes in 58% (14/24) respectively 46% (11/24) as well as palpitations in 50% (12/24) (table 1). Due to supraventricular arrhythmias or coronary artery disease every 2nd patient was treated with beta-blockers or calcium channel-blockers and every 3rd with acetylsalicylic acid (table 2). **Inclusion criteria:** patients with symptomatic HOCM (NYHA-class ≥2), refractory to medical therapy and a septal/posterior wall ratio ≥1.5 as well as a pressure gradient at rest ≥30 mm Hg and after provocation ≥60 mm Hg. Amiodarone therapy had to be stopped >2 months and beta blockers >24 hours prior to the procedure. Young age <16 years, pregnancy, AV-block 2 or 3 unless a pacemaker was implanted, and trifascicular block were excluded from the study.

Table 1
Symptoms.

	before	late after (2.8 years)
n	24	23
Presyncope	14 (58%)	11 (48%)
Syncope	11 (46%	1 (4%)
Angina pectoris	17 (71%)	12 (52%)
Dyspnoea	22 (92%)	13 (57%)
Palpitations	12 (50%)	11 (48%)
NYHA class	2.5	1.5

Table 2
Medication.

	before	early (6 months)	late (2.8 years)
n	24	24	23
Acetylsalicylic acid	8/33%	8/33%	8/35%
Beta blockers	15/63%	15/63%	18/78%
Calcium channel blockers	12/50%	9/38%	6/26%
ACE inhibitors	1/4%	3/13%	3/13%
Amiodarone	1/4%	1/4%	3/13%
Oral anticoagulation	2/8%	2/8%	3/13%
Statins	3/13%	4/17%	4/17%
Diuretics	6/25%	5/21%	5/26%

Echocardiography: Transthoracic echocardiography was performed in the parasternal long and short axis as well as apical two and four chamber views. The 2D images provided septal and posterior wall thickness, left ventricular (LV) mass, left atrial diameter, ejection fraction (EF), LV systolic and end-diastolic diameter (LVEDD). The magnitude of LVOT gradient at rest and during provocation with amylnitrate was determined from Doppler echocardiography. Systolic anterior motion (SAM) of the anterior mitral leaflet was detected by 2D echocardiography. Mitral regurgitation was estimated from pulsed Doppler and/or continuous wave Doppler echocardiography.

Exercise testing: 14 of 24 patients underwent treadmill exercise testing. Heart rate at rest and during maximal exercise, working capacity, exercise duration and ECG changes were recorded.

Holter ECG: In all patients Holter ECG was recommended but not carried out in 8 (refused by the patient or the treating physician). 16 of 24 patients were monitored over 24 hours to assess heart rate and arrhythmias using a CardioDay® Version 1.9 (getemed AG, Germany). Specifically, heart rate variability, atrial and ventricular events and tachy-/bradyarrhythmias were determined.

Alcohol ablation

A temporary pacemaker was placed in the right ventricle. After diagnostic coronary angiography and left ventricular (LV) ventriculography, a 6F guiding catheter was placed in the ostium of the left main coronary artery as well as a 4F pigtail catheter (2nd puncture) in the apex of the LV for simultaneous pressure gradient measurements. Next a standard guide wire (Guidant Corp., Santa Clara, CA) with a floppy tip was introduced into the first or second septal branch and an over-the-wire balloon (1.5–2.0 mm diameter) was advanced into a septal branch and inflated with 4 to 6 bar to block the septal artery. Then, contrast material was injected to localise the ablation area under fluoroscopy. Following this procedure echo contrast (Levovist, Schering, Germany) 1 to 2 ml was injected under 2D echocardiography for proper localisation of the ablation site. When the septal bulge or thickened septum was not properly stained, the 2nd or 3rd septal branch was selected and echocardiography repeated until the optimal ablation site was identified. Then, 1 to 3 ml pure (96%) alcohol was slowly injected over 3 to 5 min, while the pressure gradient and the ECG were continuously recorded. The dose of alcohol was chosen according to the response of the pressure gradient, the occurrence of arrhythmias (AV block) and the pain reaction of the patient. If an AV block occurred, alcohol injection was stopped immediately. If pressure gradient did not respond to alcohol ablation, a second branch was ablated (n = 3). At the end of injection, it was waited 4 to 5 min until the balloon was deflated, to prevent alcohol backflow into the LAD. After the interven-

tion, patients were monitored for 18 to 36 hours. The temporary pacemaker was removed after 8 to 12 hours (next morning). Cardiac enzymes (creatine kinase, troponin I) were determined after 6 hours and the next morning.

Follow-up

All patients were asked to undergo a follow-up examination after 3, 6, and 12 months either at the referring cardiologist or at our institution. Mean follow-up was 2.8 years with a range between 4 months and 6.7 years. Echocardiography was carried out at 3, 6, and 12 months (mean 2.2 years). Holter monitoring was done in 23 patients at follow-up examination (mean 2.2 years). Exercise testing was performed in 11 of 23 patients after the mean FU of 2.9 years. One patient committed suicide 4 years after alcohol ablation, therefore late clinical follow-up consisted of 23 patients.

Statistical analysis

Results of continuous variables are given as median with interquartile range. Wilcoxon test for nonparametric paired variables was used for group comparisons. Frequency distributions were assessed with chi^2 test. Differences were considered significant if the two-tailed p value was <0.05. Analysis was performed by using SPSS® Version 11 (SPSS Inc., Chicago, IL).

Results

24 patients were successfully treated with alcohol ablation. The peak-to-peak (invasively measured) pressure gradient decreased from 43 (IQR 25 to 64) mm Hg to 1 (IQR 0 to 12) mm Hg at rest (p <0.001) and from 130 (IQR 75 to 165) mm Hg to 13 (IQR 0 to 31) mm Hg during post-extrasystolic potentiation (p <0.001). A typical recording is shown in figure 1. In 95% (23/24) of all patients the pressure gradient was reduced below 25 mm Hg at rest and 60 mmHg during provocation. As a complication transient AV block occurred in 22% (5/24) between 2 and 30 minutes. In one patient AV block persisted and a pacemaker was implanted after 3 days. A typical recording is shown in figure 2 in a patient who developed AV block during intervention with a pacemaker induced recovery rhythm. Eight patients developed a right bundle branch block (RBBB) after the intervention.

Clinical symptoms

During the intervention 83% (20/24) of all patients developed acute chest pain of moderate to severe intensity. All patients received 5 to 10 mg morphine. Angina was found in 71% (17/24) before and in 52% after ablation (12/23)

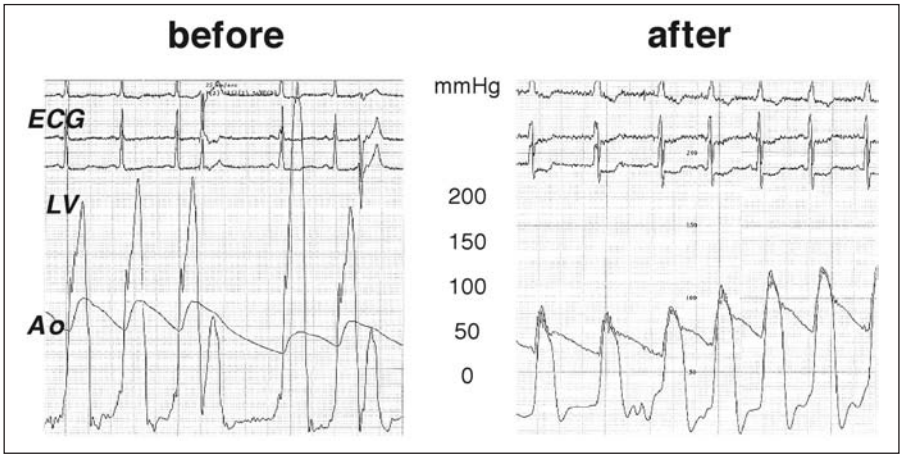

Figure 1

Pressure recording in a patient with severe hypertrophic cardiomyopathy before and after alcohol ablation. Shown are ECG recordings of 3 standard leads as well as simultaneous measurements of LV and aortic pressures. There is a large gradient of approximately 100 mm Hg (post-extrasystolic 170 mm Hg) before but none after ablation.

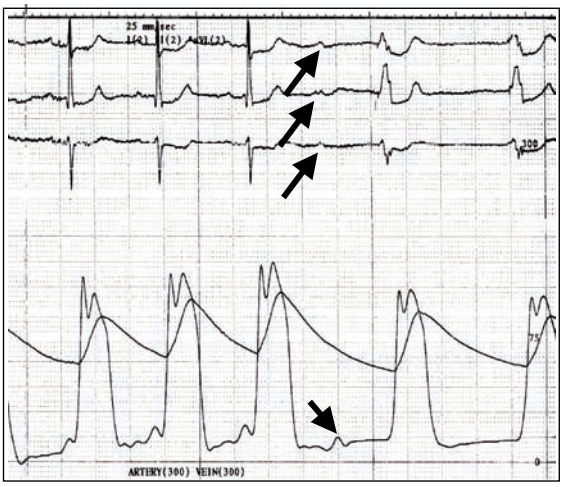

Figure 2

Original recording in a patient who developed AV block III during intervention. Arrows indicate the occurrence of the P-wave in the ECG, respectively A-wave in the LV pressure recording, which is not followed by a QRS complex or LV contraction. The wide QRS complex after AV block III is induced by temporary pacing.

(p = 0.18). Presyncopes and syncopes were reported in 58% (14/24) and 46% (11/24) before and in 48% (11/23) and 4% (1/23) after the intervention (p = 0.46; p <0.001), respectively. Dyspnea decreased from 92% (22/24) to 57% (13/23) (p = 0.006) but palpitations remained unchanged 50% (12/24) (11/23) after alcohol ablation. The median NYHA score decreased from 2.5 to 1.5 (p <0.001) (table 1). Typically, patients reported symptoms in the case of tachyarrhythmias but those with chronic atrial fibrillation reported no symptoms.

Echocardiography

The systolic pressure gradient before ablation was 38 (IQR 22 to 60) mm Hg at rest and 85 (IQR

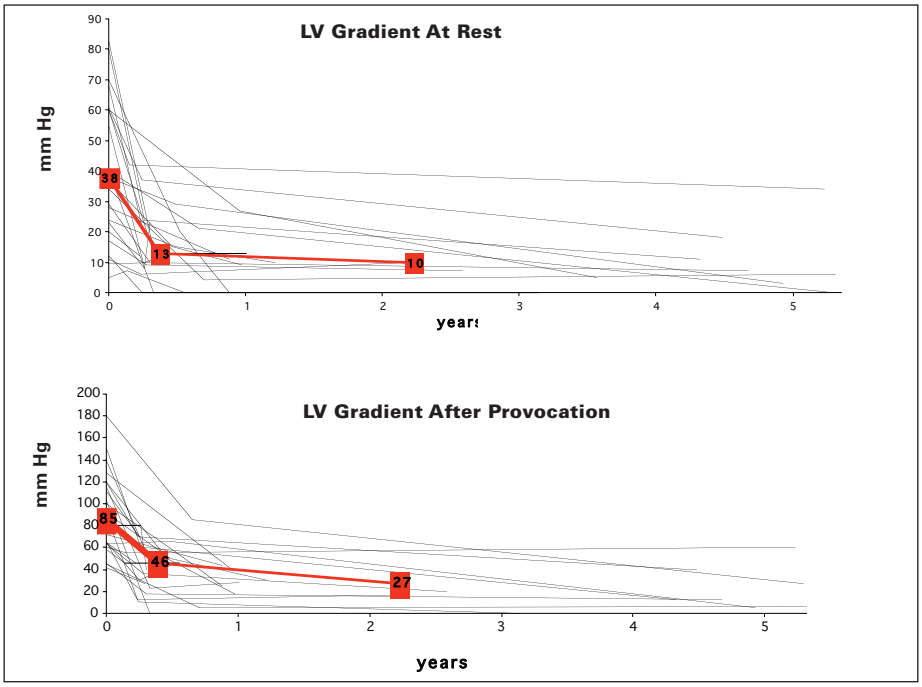

Figure 3

LV outflow tract gradient in patients before and after alcohol ablation (mean follow-up 2.2 years). Data are given at rest (upper panel) and after provocation with amyl nitrite (lower panel). Pressure gradients are measured echocardiographically. Median gradients (red line) decreased from 38 to 13, respectively to 10 mm Hg during late follow-up ($p < 0.001$). The pressure gradient after provocation decreased from 85 to 46, respectively to 27 mm Hg during late follow-up ($p < 0.001$).

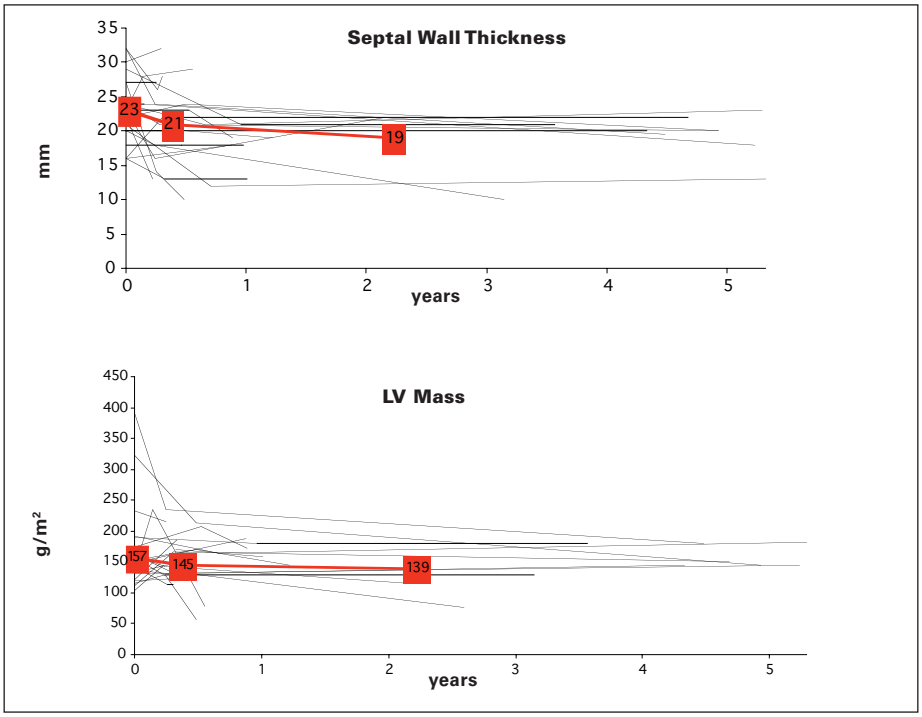

Figure 4

Septal (upper panel) wall thickness decreased in most patients after alcohol ablation. Median septal thickness was reduced from 23 to 21 early and to 19 mm late after alcohol ablation ($p = 0.009$). LV mass (lower panel) decreased from 157 early after to 145 and to 139 g/m² late after alcohol ablation ($p < 0.05$).

64 to 117) mmHg after provocation with amyl nitrite and decreased 3 months after alcohol ablation to 13 (IQR 9 to 20) mm Hg at rest ($p < 0.001$) and 46 (IQR 26 to 57) mm Hg after provocation ($p < 0.001$), respectively. During late FU (2.2 years) the pressure gradient was 10 (IQR 5 to 13) mm Hg at rest ($p < 0.001$) and 27 (IQR 15 to 44) mm Hg after provocation with amyl nitrite ($p < 0.001$) (figure 3). Septal wall thickness decreased from 23 (IQR 20 to 26) mm to 21 (IQR 18 to 24) mm after 3 months ($p = 0.02$) and to 19 (IQR 16 to 22) mm after 2.2 years ($p = 0.009$), respectively. Similarly posterior wall thickness fell from 14 (IQR 12 to 16) mm to 13 (IQR 12 to 15) mm at 3 months ($p = 0.28$) and to 13 (IQR 10 to 14) mm at 2.2 years ($p < 0.004$), respectively. As a consequence, LV mass decreased from 157 (IQR 140 to 185) g/m² (normal: men <125g/m², women <110g/m²) to 139 (IQR 114 to 146) g/m² (11% decrease) during late FU ($p < 0.05$) (figure 4).

Arrhythmias

During routine ECG recording 8% (2/24) of all patients showed atrial fibrillation. 21% (5/24) had a LBBB, 13% a RBBB (3/24) and none an AV block II or III (figure 5). 2.2 years after the intervention AF and LBBB remained unchanged while 46% (11/24) of all patients had a RBBB ($p = 0.003$) and 13% an AV block I, II, III (3/24) (ns). Holter-monitoring was performed in 16 patients before and 23 after alcohol ablation. Non-sustained ventricular tachycardia was observed in

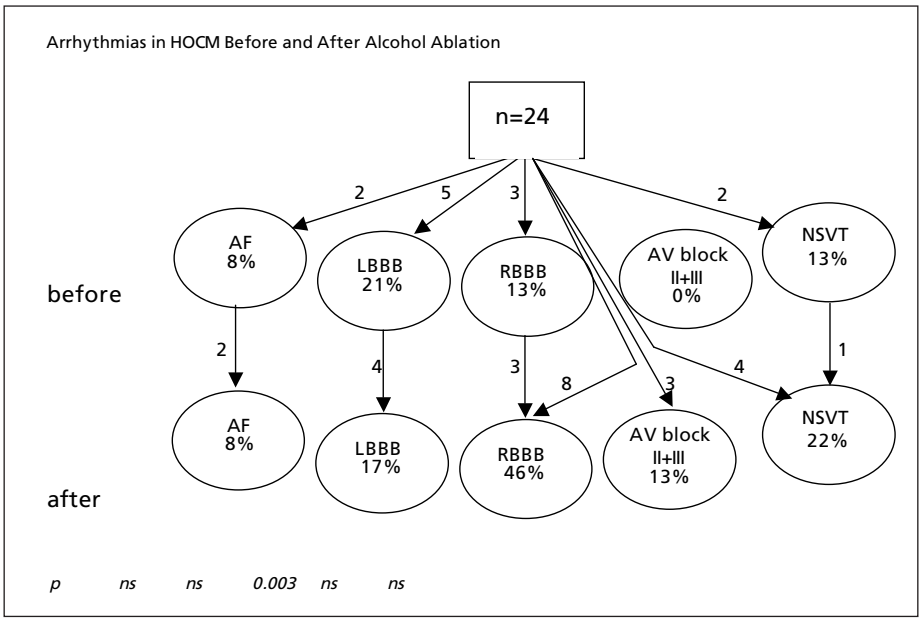

Figure 5

Arrhythmias detected in the routine ECG or Holter-monitoring before and late (mean 2.2 yrs) after alcohol ablation. AF = atrial fibrillation, LBBB = left bundle branch block, RBBB = right bundle branch block, NSVT = non-sustained ventricular tachycardia.

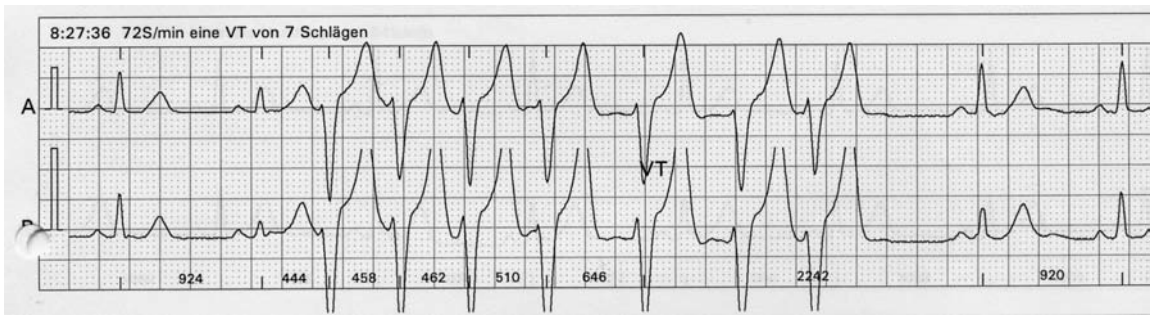

Figure 6

Holter recording of a 43-year old patient 1 year after alcohol ablation. He was treated for 4 years with amiodarone and remained asymptomatic. Five years after alcohol ablation he developed NSVT. He was treated by ICD implantation and received beta-blocker therapy. Amiodarone was stopped.

13% (2/15) before and 13% (2/15) after ablation (table 4) but was higher (22%, 5/23) when all available recordings were examined. SVES were found in 81% (13/16) before, respectively 78% (18/23) after ablation. VES were observed in 75% (12/16) before and 83% (19/23) after the intervention. AV block III was noted in 4% (1/23) respectively AV block II in 9% (2/23) during late FU (table 4). An AV block I was present in 19% (3/16) before and 9% (2/23) after alcohol ablation. As a consequence, in 2 of 23 patients a defibrillator was implanted during FU. A typical pattern of NSVT is shown in Figure 6 in a patient treated with ICD implantation.

Discussion

Alcohol ablation of the septum has become the new treatment modality in hypertrophic obstructive cardiomyopathy with a high success and a relative low complication rate [8–10]. The most dreaded complication is the development of AV block requiring pacemaker-implantation (PM) and prolonged rhythm control after the intervention. In a literature overview (table 4a) 10–14% PM implantations were necessary. However, up to 50% of all studies showed transient AV block although newer studies without bolus injection and lower amounts of alcohol [11, 12] showed transient AV block in 16–21% of all cases (table 4a).

Most of the studies showed a dramatic reduction in systolic outflow tract obstruction after alcohol ablation. Resting pressure gradients in our study were 10 mm Hg during late FU of 2.2 years. A favourable result with regard to elimination of outflow tract obstruction was achieved in 95% of all patients. As a consequence, LV wall thickness decreased by 8% of both septum and posterior wall, whereas LV mass was reduced by 11%. These data indicate that not only the septum but also the rest of the LV show LV remodelling due to the unloading with a decrease in LV pressure gradient of 74% at rest and 68% during provocation. This reduction in LV hypertrophy is probably accompanied by an improvement in diastolic dysfunction with a reduction in LV filling pressure and an increase in filling [13]. The occurrence of a

Table 3
Arrhythmias.

ECG (beats/min)		before	late (1.9 years)		
n		24	24		
Bradycardia (<60)		38%	17%		
Normocardia (60–90)		58%	75%		
Tachycardia (>90)		4%	8%		
Holter ECG		**all before**	**all late**	**paired comparison before**	**late**
n		16	23	15	15
SR		16	23	15	15
AF intermittent		13%	9%	13%	7%
LBBB		31%	13%	33%	20%
RBBB		13%	48%	13%	40%
AV block	**total**	19%	22%	20%	18%
	I	19%	9%	20%	13%
	II	0	9%	0	13%
	III	0	4%	0	0%
SVES	total	81%	78%	80%	73%
	<1000/24h	81%	65%	80%	67%
	>1000/24h	0%	13%	0%	7%
VES	total	75%	83%	73%	80%
	<1000/24h	75%	70%	67%	73%
	>1000/24h	0	13%	7%	7%
NSVT	>5 beats	13%	22%	13%	13%

RBBB in up to 50% and LBBB in up to 20% may counterbalance these beneficial effects by introduction of LV asynchrony. However, a similar number of patients had left bundle branch block before the intervention. Comparable data with regard to outcome have been reported [8, 14–28]. LBBB is a relative contraindication [29] because when RBBB (up to 45%) occurs during alcohol ablation (figure 5) total AV block may appear. In this situation the risk of AV block is in the order of 50% which was not the case in our series.

Risk stratification

Risk stratification has been a major issue in patients with HOCM because there is a small risk of unexpected sudden cardiac death (SCD). Strategies to identify patients at risk for SCD have been manifold and the 5 most important risk factors are [30]:

1. Non-sustained ventricular tachycardia, 2. abnormal exercise blood pressure response, 3. family history of premature sudden death, 4. unexplained syncope, 5. severe left ventricular hypertrophy (septal thickness >30 mm).

In the present study, patients were mildly to moderately symptomatic with moderate to severe outflow tract obstruction. Severe septal hypertrophy (>30 mm) and NSVT was found in 8% (2/24) and in 13% (2/16), respectively. Sudden cardiac death survivors were not present and a family history of SCD was observed in one. Overall risk for SCD was low to minimal in the present analysis.

Arrhythmogenic potential of alcohol ablation

Previous data have indicated that septal ablation may lead to scarring at the ablation site favouring recurrent tachyarrhythmias and non-sustained ventricular tachyarrhythmias (NSVT) [7]. This hypothesis has, however, not been supported by scientific evidence but is pure speculation [31]. In contrast, previous studies have suggested that arrhythmias may even decrease due to the reduction in LV hypertrophy with a diminution of subendocardial ischaemia. The reduction in outflow tract obstruction not only improves exercise capacity [32] and clinical symptomatology but also haemodynamics and diastolic function. Almost all previous studies have shown not only an improvement in symptoms (NYHA classification, dyspnoea and angina pectoris) but also a reduction in palpitations and syncopes [14].

NSVT were observed in the present study in 13% (2/16) before and 22% (5/23) after alcohol ablation. The careful search for arrhythmias after the intervention during FU was responsible for the identification of 2 patients with NSVT who

Table 4 a

a	Alcohol Ablation			Data from the literature			
Author	**year**	**n**	**NSVT**	**AV block transient**	**permanent**	**PM**	**Death**
Chang et al.[16]	2003	224	–	–	14%	–	–
Osterne et al.[17]	2003	18	–	44%	11%	11%	5.5%
Chang et al.[18]	2004	286	–	16%	–	–	–
Gietzen et al.[19]	2004	157	–	–	11%	25%	2.5%
Talreja et al.[20]	2004	58	–	–	12%	12%	–
Faber et al.[21]	2005	242	–	–	10%	10%	1.2%
Fernandes et al.[22]	2005	130	–	–	13%	13%	1.5%
present study	2007	24	22%	21%	13%	13%	0%*
mean			22%	27%	12%	14%	2%

– = no data *1 non-cardiac (death suicide)

Table 4 b

a	Surgical Myectomy			Data from the literature			
Author	**year**	**n**	**NSVT**	**AV block transient**	**permanent**	**PM**	**Death**
Cohn et al.[23]	1992	31	–	–	5%	5%	0%
ten Berg et al.[24]	1994	38	–	–	3%	3%	0%
Schönbeck et al. [25]	1998	110	13%	–	11%	15%	3.6%
Xin et al. [8]	2001	26	–	–	8%	8%	0%
Nagueh et al.[15]	2001	41	7%	–	2%	2%	0%
Firoozi et al.[14]	2002	24	–	–	4%	4%	4%
Minami et al.[26]	2002	75	–	–	8%	–	1%
Talreja et al.[20]	2004	117	–	–	3%	3%	–
Castedo et al. [27]	2004	26	–	–	4%	4%	4%
Ommen et al.[28]	2005	289	–	–	1%	1%	1%
mean			10%	–	5%	5%	1%

– = no data

needed ICD implantation although one of the 2 patients already had NSVT prior to the intervention, the other not. Overall, arrhythmias decreased rather than increased after the intervention (table 3). The only significant change in ECG abnormalities was an augmentation of RBBB from 13% (3/24) to 46% (11/24) ($p < 0.003$). The induction of re-entry tachyarrhythmias could not be documented as it was suggested by some authors [33].

Limitations

Several points of the current study have to be addressed:

1. It is a prospective, non-randomized study in a small group of patients with hypertrophic cardiomyopathy. The disease is not frequent and the data represent experience of our centre in the last 4–5 years. Nevertheless, the data presented in this paper match well with previous published studies [29, 34] (table 4a). Periprocedural complications are within the range of other papers.
2. Previous investigators have expressed their concern that alcohol ablation may induce reentry tachyarrhythmias by scarring of the septum [33, 35]. However, we and others do not believe that this is true. The reported data show excellent outcome after alcohol ablation with very low post procedural complication rates although this is a rather small patient collective. One of the risks of the procedure is the induction of complete AV block which occurred in approximately 1 out of 8 (3/24) patients.
3. Comparisons with previous surgical studies have indicated similar results between surgical myectomy and alcohol ablation [15]. However, comparisons of current literature data suggest that surgical myectomy results in less complete AV block and requires PM implantation only in about ¼ of those undergoing alcohol ablation (5% versus 14%; table 4b). Cardiovascular death was higher in the alco-

hol ablation group but many of the surgical studies did not specify mortality, and the two cohorts have similar mortality ranges between 0% and 5%. Procedural outcomes of surgical or catheter-based therapies are similar but alcohol ablation is less invasive with a shorter hospital stay and lower costs [8].

Conclusions

Treatment of hypertrophic cardiomyopathy has dramatically changed over the last 5–10 years starting with surgical myectomy leading to percutaneous cardiac intervention. Alcohol ablation is a safe and effective treatment with similar results as surgical myectomy. However, PM-implantation for complete AV block may be four times more frequent for alcohol ablation but still remains a rare complication occurring in approximately 1 of 8 patients. The fear of re-entry tachyarrhythmias after alcohol ablation could not be confirmed by the present study but NSVT occurs in 10 to 20% of all patients before and after intervention, and requires an ICD implantation in 1 out of 12 patients (9%). However, the present study is rather small and may not be representative for treatment recommendations because it is not randomised.

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