Alcohol septal ablation for obstructive hypertrophic cardiomyopathy
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Chapter 10

Discussion
The present thesis, current knowledge and practical implications

In this Chapter, the data presented in the thesis – combined with relevant previous literature – are discussed in terms of the following four types of context: first, the effectiveness of ASA in terms of gradient and symptom reduction; second, the risk of periprocedural complications – with a focus on PM implantation – after both ASA and surgical myectomy; third, survival and ventricular arrhythmia after ASA and myectomy; and fourth, a comparison between a young and elderly age group in terms of effectiveness, periprocedural risks and survival after ASA.

In addition, limitations of the definitions used in the current literature for comparison of ASA and myectomy are discussed, as well as the differences between the current guidelines and recommendations and the results of this thesis. A discussion of the overall “performance” of ASA and a glimpse into the future conclude this thesis.

Symptomatic improvement, gradient after the procedure and reintervention

ASA and surgical myectomy are both treatments that aim to improve symptomatic status by relieving obstructed flow in the LVOT of patients with HCM. Both a meta-analysis and recent studies have found short-term symptomatic improvement to be good after both treatments (1-3). In Chapter 6.1, using a questionnaire administered to patients who had undergone either ASA or myectomy, we found that this beneficial effect was sustained at long-term follow-up. We also found that annual admittance for heart failure was uncommon and the average ejection fraction was good both after myectomy and ASA (61± 11 % vs. 63 ± 8%) (Chapter 6.1). However, despite the fact that ASA and myectomy are comparable with respect to long-term symptomatic status, we found that gradients after ASA were slightly higher than those after a myectomy procedure (19 (10-42) mmHg vs. 10 (7-13) mmHg, P < 0.001). This difference was also seen in a previous meta-analysis (2). The importance of residual gradients after ASA was underscored by the small but significant predictive value of the post-procedural gradient on all-cause mortality (HR1.01, 95% CI 1.00-1.01) that is demonstrated in Chapter 9.

During the first series of ASA procedures performed in 1994, it was decided that the first septal branch should be used for the induction of septal infarction. However, as exemplified in Chapter 3.1, the highly variable septal coronary anatomy may limit the technical possibilities of the operator, which perhaps explains the higher gradients found after ASA (Chapter 6.1). This formed the basis of our research in Chapter 3.2, where we found that outcome was far less likely to be successful if the ablated septal branch was more than 20 mm
distal from the base of the septum and at the same time a more proximal septal branch was present and not ablated.

Understanding of this association between underlying septal anatomy and a successful ASA requires further clarification. Since the origin of the LAD is situated in the atrioventricular groove, it is also situated at the level of the beginning of the very basal part of the interventricular septum. The distance from the origin of the LAD to the origin of the ablated septal branch is thus a surrogate for the distance from the base of the septum to the entry point of the ablated septal branch. In the case of unfavourable septal coronary anatomy, a proximal septal branch is not amenable for ablation, so the procedure must take place in a more distally located branch. This can result in a more distal infarct location, thereby providing a possible explanation for the higher gradients seen after ASA.

In order to better understand the magnitude of the influence of the location of the infarction, in Chapter 4 we performed an analysis of CMR images from patients who had undergone ASA. A successful outcome was seen in all patients with a basal infarction, whereas patients with a more distal infarction had a higher risk of an unsuccessful outcome. On the other hand, larger infarct size measured with CMR had no influence on outcome after ASA. This again illustrates the importance of the location of the septal infarction and that the correct choice of the septal branch used for ablation plays a crucial part in this. Striving for a small basal infarction seems to lead to the best results in ASA.

Even though NYHA class after ASA and myectomy are comparable at long-term follow-up, reinterventions (usually a second ASA procedure) are necessary in about 8% of patients, which is considerably higher than for myectomy (2%) (Table 1). This emphasises the importance of aiming for the most effective ASA procedure.
**Periprocedural complications**

ASA was introduced as an alternative to surgical myectomy with the advantage of being less invasive and thus more attractive for some patients with symptomatic obstructive HCM. Despite its more invasive nature, surgical myectomy is still considered to be safer by many clinicians and investigators, in the United States in particular. In Chapter 6 we therefore investigated the outcome of ASA and myectomy with special attention for complications associated with ASA and myectomy in a “real world” single-centre cohort study. Periprocedural mortality was found to be around 1-1.5% after both ASA and myectomy. When complications were investigated in detail, more complications were found after surgical myectomy, mostly due to post-operative bleeding. Also the length of in-hospital stay was longer after myectomy (Table 1). In an accompanying editorial, the main reason for the complication rate being higher after myectomy than after ASA was suggested as being limited experience (Chapter 6.2). Indeed, complication rates for myectomy were found to be lower in a highly specialised centre such as the Mayo Clinics (4). Nevertheless, differences between centres in patient characteristics (age, co-morbidity such as presence of coronary artery disease) make this type of comparison difficult. And even though all complications after myectomy presented in Chapter 6.1 were treated successfully, when this single-centre study compared periprocedural complications and length of in-hospital stay, the results favoured ASA, indicating that its less invasive nature could be beneficial.

Periprocedural complications after ASA were also investigated in two different age groups in Chapter 8. Not unexpectedly, compared with younger patients, elderly patients had a higher rate of complications after ASA, which in fact supports the notion that ASA should also be considered for younger patients.
Table 1: Current results of both procedures

<table>
<thead>
<tr>
<th></th>
<th>Myectomy</th>
<th>ASA</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Effectiveness</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptomatic relief</td>
<td>95.5%</td>
<td>92%</td>
<td>0.43</td>
</tr>
<tr>
<td>Gradient at follow-up (mmHg)</td>
<td>10 (7-13)</td>
<td>9 (10-42)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reintervention</td>
<td>1.6% (0.6-2.6)</td>
<td>7.7% (0.6-2.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Periprocedural mortality</td>
<td>1.1%</td>
<td>1.3%</td>
<td>0.75</td>
</tr>
<tr>
<td>Periprocedural VT/VF</td>
<td>0.2%</td>
<td>2.2%</td>
<td>0.055</td>
</tr>
<tr>
<td>PM implantation</td>
<td>4.4% (2.6-6.2)</td>
<td>10% (7.8-12.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>All complications*</td>
<td>28%</td>
<td>14%</td>
<td>0.004</td>
</tr>
<tr>
<td>In-hospital stay (days)</td>
<td>9 (4-6)</td>
<td>5 (4-6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Survival</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall survival</td>
<td>1.4%/year</td>
<td>1.5%/year</td>
<td>0.78</td>
</tr>
<tr>
<td>SCD + ICD shock</td>
<td>0.49%/year</td>
<td>0.41%/year</td>
<td>0.47</td>
</tr>
<tr>
<td>SCD - ICD shock</td>
<td>0.47%/year</td>
<td>0.34%/year</td>
<td>0.16</td>
</tr>
</tbody>
</table>


PM implantation after ASA and myectomy

Both after myectomy and after ASA, nodal and infranodal pathways can either be interrupted due to resection of septal myocardium or damaged due to the induction of a septal infarction. There are however some clear differences between the two procedures: after myectomy a left bundle branch block can be found and after ASA a right bundle branch block is usually found. A logical explanation for this difference is the nature of the resection after myectomy, i.e. left-sided septal myocardium is removed, leaving the right bundle branch intact. In contrast, ASA usually creates a transmural infarction involving the right side of the septum (>90% of cases, Chapter 4), thus damaging the right bundle branch. Two possible explanations for why AV block occurs in some patients, but not in others, are preprocedural vulnerability of the AV conduction system, and differences in the size and location of the excised myocardium or induced septal infarction. The exact reasons are however unknown.
In a recent meta-analysis, the post-procedural rate of pacemaker implantation was found to be twice as high after ASA than after myectomy, largely due to the low rate after myectomy in the studies included in this analysis (Table 1). In Chapter 6.1, the rate of pacemaker implantations (9%) observed after myectomy was higher than the rate reported in this meta-analysis. Experience and volume of patients may explain this difference, as also suggested in Chapter 6.2. The rates are for instance higher than the 2.4% reported in a highly specialised U.S. centre (i.e. Mayo Clinics)(4). One must realise however, that the 9% rate of PM implantation found in Chapter 6.1 is a “real world” figure, that is in line with the findings of two other studies – one study extracted data from a large U.S. database and the other from a highly specialised centre (Cleveland Clinics) – which both reported a 9% pacemaker implantation rate (5,6). Apparently, pacemaker rates after myectomy vary in publications and also amongst specialised centres.

Another important issue is that there are clear indications that the actual risk of PM implantation in the long-term is higher than the periprocedural risk reported in the current literature after both ASA and myectomy. For instance, the 9% post-operative risk of having a PM implanted after myectomy in the Cleveland Clinics increased to 20% during an eight year follow-up period (6). Also after ASA, the occurrence of late permanent AV block has been reported (7,8). However, studies on this topic are limited.

Factors that predict whether some patients do and others do not require a PM after ASA and myectomy are described in Table 2. A higher age, pre-existing LBBB (for ASA) and a pre-existing RBBB (for myectomy) can be seen as indicators of a pre-existing vulnerable conduction system which leads to a higher risk for the need for a PM. In Chapter 6.2, a lack of surgical experience was suggested as being a possible factor influencing the increased rate of PM implantations. Indeed, a detailed description of what techniques an experienced surgeon uses in order to prevent complete heart block, or how periprocedural TEE can be used might be helpful for other centres performing myectomy. Unfortunately these data are not widely available, although risk factors and some periprocedural techniques that are associated with lower rates of PM implantation have been described for ASA (6,7,9-12, Table2). For example, as described in Chapter 5, whereas the dosage of ethanol did not predict the need for a PM, a slower injection of ethanol instead of bolus injection as well as a lower number of ablated septal branches are known to be associated with a lower need for a PM (Table 2).
Table 2: Risk factors for permanent AV block

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Myectomy</th>
<th>ASA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-existing LBBB a,c,e</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>Pre-existing RBBB a,c</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td>&gt;1 procedure b,f</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>&gt;1 septal branch ablated b,c</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Alcohol bolus* c</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Advanced age c,d,g</td>
<td>?</td>
<td>++</td>
</tr>
</tbody>
</table>


Survival and ventricular arrhythmia

A recent meta-analysis has shown excellent survival rates after both ASA and myectomy (Table 1) (13). The occurrence of ventricular arrhythmia has however been a concern ever since the conception of ASA. Case reports and studies of ICD carriers have shown that ventricular arrhythmia does occur after ASA (14-18). A word of caution was even given when the rate of cardiac death due to ICD shocks was found to be higher after ASA than after myectomy (19). In Chapters 6, 7 and 9, we therefore investigated the occurrence of ventricular arrhythmia as well as survival after both myectomy and ASA. In the single-centre study described in Chapter 6, the annual rates of SCD after ASA and myectomy were both low (0.7% vs. 1.4%, P=0.15), as were the annual rates of death due to any cause: 1.5% after ASA and 2.2% after myectomy (P=0.25). In the multi-centre study described in Chapter 7, the annual SCD rate was 0.96%/year after ASA, 0.75%/year after myectomy and 1.0%/year in a control group (P = 0.40). After multivariate analysis, the risk of SCD after ASA was estimated to be significantly higher (HR 2.1 (1.02–4.39), p=0.04). However, 40% of cases of SCD after ASA consisted of an appropriate ICD shock in this study as this was included in the definition of SCD. Considering that appropriate ICD shocks do not necessarily affect survival, this can explain why overall survival was comparable after ASA and myectomy in this multi-centre study (1.9%/year vs. 2.0%/year, P=NS). In the European ASA registry (Chapter 9), an all-cause mortality rate of 2.4%/year and an SCD rate of 1.2%/year was found in 1275 patients after ASA. A new finding was the independent prediction of mortality
by residual obstructive gradients after ASA, although this effect was rather small (HR 1.01, 95% CI 1.00-1.01; p=0.047).

In summary, even after acknowledging that VT/VF may occur after ASA, the incidence of VT/VF is very low and does not affect survival after ASA.

Young versus elderly age group after ASA
American guidelines state that if myectomy is viable, ASA should not be performed in younger patients. In Chapter 8, effectiveness, periprocedural risks and survival after ASA were compared between a younger (43 ± 8 years) and an older age group (64 ± 6 years).

After the procedure, symptomatic improvement in terms of NYHA class and reduction of the LVOT gradient were comparable in both age groups. However, periprocedural complications in terms of complete AV block and pacemaker implantation were more common in older patients than in the younger age group. Survival rates and risk for ventricular arrhythmia after ASA was found to be independent of age; both young and elderly patients had a survival rate and risk of cardiac death that was similar to their age-matched control groups.

The findings in Chapter 8 do not support the notion that ASA should only be performed in the elderly, though a very young age group (<40 years) has not yet been investigated in terms of periprocedural complications, long-term complications effectiveness, and long-term survival.

Limitations of comparing the two techniques and problems with current definitions
A randomised controlled trial comparing ASA and surgical myectomy would be the desired method to ultimately decide which technique is preferable. However, such a trial has not yet been performed, for several practical reasons, one of them being the necessity of including a high number of patients (20).

Another major issue when comparing ASA and myectomy is that there is no consensus on the definition of success after both ASA and myectomy. Different definitions have meanwhile been used, for instance reduction of the invasive gradient, reduction of the echocardiographic gradient at follow-up and also reduction of the echocardiographic gradient at different time intervals, not to mention symptomatic improvement.
In order to avoid underestimation of cardiac death, in some studies the definition of cardiac death is stated as being the occurrence of an appropriate ICD shock and death due to unknown causes. Obviously, this in turn leads to an overestimation of death due to ventricular arrhythmia.

In conclusion, due to unresolved issues concerning different endpoints, different indications used for the two techniques and the lack of a randomised controlled trial, it remains difficult to directly compare ASA and myectomy and to decide which technique is preferable.

**ACC/ESC guideline recommendations and impact of the current thesis**

The American College of Cardiology’s 2011 guidelines on treatment of HCM state that surgical myectomy should be performed in experienced centres and is the first line of treatment of symptomatic obstructive HCM with a class IIa recommendation. ASA should be regarded as an alternative only for patients in whom surgery is contra-indicated due to serious co-morbidities or advanced age – with a class IIa recommendation– and when the patient expresses preference for ASA after thorough informed consent – only as a class IIb recommendation (21). At variance with the above American guidelines, the more recent European HCM guidelines, issued in 2014, give a class Ib recommendation for both ASA and myectomy as a treatment option. In these European guidelines, surgical myectomy is regarded as a first line treatment (class I recommendation) if other lesions are present that warrant surgery (e.g. mitral valve repair/replacement) (22).

As summarised below, the data presented in this thesis do not fully support the recommendations of the American guidelines; instead, they favour and supplement the European guidelines.

First, Chapters 3.2 and 4 show that knowledge of septal coronary anatomy can either assist the operator in choosing the correct septal branch and basal location of the infarction or can lead to the conclusion that coronary anatomy is not suitable for ASA and that the patient should be referred for surgical myectomy instead. Therefore, favourable septal coronary anatomy should be incorporated into the guideline recommendations that favour ASA.

Second, In Chapters 6 and 7, long-term survival was found to be favourable after both techniques, while both therapies were also equally effective. These results do not support the preference for surgical myectomy given in the American guidelines.
Third, Chapter 8 does not support the recommendation in the American guidelines that ASA should be confined to older patients, since results in younger patients are also just as good for ASA as for myectomy. It should be noted that for children and adolescents ASA needs further study before ASA can be recommended for this specific age group.

Fourth, the heart team should consist of a surgeon experienced with myectomy, an interventional cardiologist experienced with ASA, and an imaging cardiologist. In order for the heart team to make a decision, they must have at their disposal the complete history of the patient, a coronary angiogram (evaluation of suitable septal anatomy and presence of coronary artery disease), an echocardiogram (evaluation of distribution of left ventricular hypertrophy, SAM, concomitant valvular disease) and a CMR report (including the CMR images if echocardiography image quality is inferior). The conditions that give preference to either ASA or myectomy are listed in Table 3 and may aid the heart team in their decision making.

Fifth, the fact that rates of periprocedural complications, effectiveness and long-term results are now available for both types of procedures (Chapter 6 and 7), facilitates a process of shared decision making between patient and treating physician, which is at variance with the class IIb recommendation for ASA as stated in the American guidelines of 2011. After the heart team has decided that both procedures are feasible, a process of shared decision between patient and treating physician should be initiated.
**Table 3:** Criteria that give preference to either myectomy or ASA

<table>
<thead>
<tr>
<th></th>
<th>Myectomy</th>
<th>ASA</th>
</tr>
</thead>
<tbody>
<tr>
<td>childhood/adolescence</td>
<td>++</td>
<td>--</td>
</tr>
<tr>
<td>Age&lt;40</td>
<td>+</td>
<td>+/-</td>
</tr>
<tr>
<td>Surgical necessity*</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>pre-existing LBBB</td>
<td>+</td>
<td>+/-</td>
</tr>
<tr>
<td>pre-existing RBBB</td>
<td>+/-</td>
<td>+</td>
</tr>
<tr>
<td>pre-existing PM</td>
<td>+/-</td>
<td>+</td>
</tr>
<tr>
<td>Increased surgical risk#</td>
<td>+/-</td>
<td>+</td>
</tr>
<tr>
<td>Moderate AOI</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Septal anatomy unsuitable</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

*coronary artery disease requiring surgery or valvular disease requiring surgery (including mitral valve disease with a central or anterior directed mitral insufficiency not due to SAM; elongation of the AMVL; or anterior displacement of papillary muscles).

#based on known risk factors (age, hypertension, DM, history of vascular disease, severe pulmonary disease, poor mobility and previous cardiac surgery).

Performing and improving the ASA procedure

In summary, the left coronary artery and the septal anatomy are visualised via arterial access using a guiding catheter and contrast injections in multiple views. The first accessible septal branch is cannulated and an over-the-wire balloon is inserted. After inflation of the balloon, concentrated alcohol (95%) is injected in order to induce a septal infarction.

This section summarises how the findings of Chapters 3-8 can be used in practice for optimal efficacy in terms of gradient reduction, minimising periprocedural complications and prevention of ventricular arrhythmia (see also Table 4). This summary includes several tips and tricks that were demonstrated during a EuroPCR 2015 learning session on ASA.

Optimal effectiveness: determining the infarct location using MCE and septal anatomy

After injections are made into the left coronary artery (usually a right cranial view), the target septal branch is cannulated and an over-the-wire balloon can be introduced. In the first series of ASA procedures performed in the 1990s, temporary occlusion of the septal branch, by inflating the balloon, was used to induce temporary ischaemia in order to evaluate a pressure drop of the LVOT gradient. However this pressure drop had limited predictive value (23) and was replaced by myocardial contrast echocardiography (MCE). The use of MCE was associated with a reduction in complications (prevention of remote infarction, e.g. in right ventricle) and better outcome after ASA. However, in Chapter 4, CMR revealed that an undesired distal infarction may still occur despite the use of MCE. In order to prevent residual gradients after ASA, the coronary angiogram should be thoroughly inspected. The definitive injection of ethanol should not be given until inspection of the septal coronary anatomy identifies the correct septal branch to be used for ablation and this has been corroborated by MCE showing the correct perfusion area. As shown in Chapter 4, the operator should strive for a small infarction at a basal location and choose the correct septal branch. For septal branches that are difficult to reach, a venture device can be used.

Two markers of success that can be used during the procedure are as follows: 1) The MCE shows whitening/infarct location in the correct basal location (Chapter 4). 2) The coronary angiogram shows the correct choice of septal branch (no proximal septal branch and/or the distance from the base of the septum tot the ablated branch is <20 mm) (Chapter 3).
Prevention of tamponade
Tamponade due to temporary pacing is a serious complication that has also led to the periprocedural death of a patient, as described in Chapter 6. This complication can be prevented by the insertion of a regular temporary pacemaker-lead via the subclavian vein by a dedicated electrophysiologist in the PM lab before the procedure (24). This technique will also most likely reduce PM lead infections or loss of capture, relative to a temporary PM lead inserted via the groin.

Prevention of alcohol leakage
Leakage of ethanol is the second risk of periprocedural death (Chapter 6) and/or remote infarction. The following factors for preventing alcohol leakage were described during the learning session on ASA at EuroPCR 2015:
1) Choose the correct balloon (short (6-8 mm) and slightly oversized in diameter).
2) Check for complete balloon apposition using contrast injections, both antegrade and retrograde (injection behind the balloon).
3) Give a low-dose injection of ethanol (1-3 ml total, or amount according to septal thickness (0.08 ml/mm)).
4) Inject the ethanol very slowly (rate 0.5-1 ml/min).
5) After ethanol injection, flush with 0.3-0.5 ml saline injection.
6) Wait at least 5 minutes before deflation and removal of the balloon.

Prevention of vascular bleeding
Radial instead of femoral access can reduce vascular complications. Although the insertion of a second catheter for continuous gradient monitoring is difficult with this technique, this can also be monitored using echocardiography (24).
Prevention of ventricular arrhythmia

In general, survival after ASA is excellent, with a very low occurrence of ventricular arrhythmia. Survival is also comparable to that in a non-obstructive HCM population (Chapter 7). Nevertheless, case reports and a single-centre study have shown that malignant ventricular arrhythmia may occasionally occur. Suggestions for preventing ventricular arrhythmia were made in Chapters 4 and 5, and are aimed at limiting infarct size:

1) Strive for a small infarction at a basal location (based on septal anatomy and MCE).
2) Avoid large diameter septal branches (instead use a more distal side branch ablation under guidance of MCE).
3) Avoid injecting too large a volume of alcohol.
<table>
<thead>
<tr>
<th>How to</th>
<th>Technical approach</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimise gradient reduction</td>
<td>combined use of MCE and inspection of septal coronary anatomy, striving for basal infarct location</td>
</tr>
<tr>
<td>Prevent tamponade</td>
<td>insertion of temporary (subclavian) pacemaker by a dedicated electrophysiologist</td>
</tr>
<tr>
<td>Prevent alcohol leakage</td>
<td>- balloon choice (6-8mm short, slightly oversized in diameter)</td>
</tr>
<tr>
<td></td>
<td>- check balloon apposition using antegrade and retrograde contrast injection</td>
</tr>
<tr>
<td></td>
<td>- low dose of alcohol (1-3 ml, or 0.08ml/mm of septum)</td>
</tr>
<tr>
<td></td>
<td>- slow alcohol injection (rate 0.5-1ml/min)</td>
</tr>
<tr>
<td></td>
<td>- slow flush with 0.3-0.5 ml saline</td>
</tr>
<tr>
<td></td>
<td>- wait &gt; 5min before deflation/removal of the balloon</td>
</tr>
<tr>
<td>Prevent bleeding</td>
<td>radial instead of femoral access</td>
</tr>
<tr>
<td>Prevent ventricular arrhythmia</td>
<td>- low alcohol dosage (1-3 ml, total)</td>
</tr>
<tr>
<td></td>
<td>- small infarct size, while striving for basal location</td>
</tr>
<tr>
<td></td>
<td>- avoid large diameter septal branches (using side branches instead)</td>
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</tbody>
</table>
How to inform the patient
If the heart team considers both procedures to be feasible, the preference for one of the two procedures should be decided jointly by the treating physician and the patient. The results of this thesis and data from the literature enable the patient to be fully informed about the risks and benefits of both options. Information that can be discussed with the patient is presented in Table 3, and, if available, centre-specific results should also be provided.

In summary, one should evaluate whether or not the patient has understood the following facts: the nature and invasiveness of both techniques, the good long-term survival after both ASA and myectomy, the effectiveness of both techniques for treatment of symptoms, the higher risk of a PM implantation after ASA relative to myectomy, and the greater need for reintervention after ASA; and, last but not least, the clearly less invasive nature of ASA relative to myectomy.

Future perspectives
ASA and myectomy have both evolved into techniques that have excellent long-term survival and a very low risk of SCD. Further improvements in survival are therefore difficult. There are however unresolved issues that do warrant further research.

ASA is still associated with slightly higher gradients after the procedure, as well as a higher need for a reintervention and a higher rate of PM necessity compared with myectomy. The development of periprocedural techniques that can prevent these complications is indicated. For instance, case reports on periprocedural use of CT or three-dimensional echocardiography have been presented but need further development and study (24,25). Combining knowledge on the location of conduction pathways and exact location of the septal infarction may allow us to reduce the numbers of patients requiring a pacemaker.

A case report and case series have recently been published on a new treatment of obstructive HCM using a mitraclip (26,27). The mitraclip is used to stop the SAM in a direct manner, thereby limiting obstruction in the LVOT. Due to its relatively non-invasive nature, and possibly even lower complication rate (no risk of PM placement), this technique is as promising as ASA and myectomy at the time they were first performed. Future research is required to establish its role as either a conjunctive technique or as a stand-alone technique.
Reference List:
5. Panaich SS1, Badheka AO2, Chothani A3, Mehta K4, Patel NJ5, Deshmukh A6, Singh V7, Savani GT7, Arora S1, Patel N1, Bhalara V1, Grover P7, Shah N5, Elder M1, Mohamad T1, Kaki A1, Kondur A1, Brown M1, Grines C1, Schreiber T1. Results of ventricular septal myectomy and hypertrophic cardiomyopathy from Nationwide Inpatient Sample [1998-2010]. Am J Cardiol. 2014;114(9):1390-5.


