To understand the role of surgery in the treatment of patients with a ventricular aneurysm, one must first consider the criteria used to define the presence of an aneurysm. Angiographically, an aneurysm may be defined as an area of the ventricle which is demarcated from the surrounding chamber by hypokinesis, akinesis, or dyskinesis. An angiographic "aneurysm" may relate anatomically to an obvious sac of thin scar (classic pathological definition of an aneurysm) or it may correspond to a region of mixed scar and viable muscle which may be of variable thickness. In the latter case, it may not be obvious whether this region would benefit more from revascularization with the hope of recruiting hibernating myocardium and improving regional wall motion, or from resection with ventricular reconstruction. Preoperative perfusion scans and positron emission tomography have been used in attempts to separate scar tissue from viable nonfunctioning myocardium. Echocardiography and nuclear magnetic resonance may help to assess the extent of thinning in the target area. However, in these cases, we believe that the final decision with regards to appropriate intervention can best be made at the time of surgery. With direct examination of the heart, if the area in question is scarred and significantly thinned, resection is indicated.

For aneurysm repair, we prefer a modified linear closure technique. This approach is applicable for all types of aneurysms (broad-based or narrow-necked, true or false). Advantages of this technique are that it is relatively simple, it provides reproducible results, and it can be adapted for a variety of intraoperative situations (calcified or noncalcified). The entire repair can be safely performed in the decompressed beating heart. This approach decreases ischemic time in patients who often have significant compromise of ventricular function and diffuse triple vessel disease. In our opinion, the only situations in which a more complex type of endoaneurysmorrhaphy repair technique may be advantageous is in cases of acute infarction where friable tissues may make a linear closure difficult or impossible to perform.

Indications for Aneurysm Repair

In patients with a chronic ventricular aneurysm, poor prognosis has been associated with increased aneurysm size, decreased global ejection fraction, and more extensive coronary artery disease. In these patients, modern medical therapy can be very effective in controlling symptoms and patients live longer before hemodynamic decompensation occurs. However, once decompensation begins, deterioration may be rapid and patients may die before appropriate investigation can be done and the possibility of surgical intervention entertained. For these reasons, we believe patients who develop an aneurysm after a myocardial infarction should be followed closely and considered for surgery when signs of decompensation first occur. These signs may include symptoms of angina or congestive heart failure, the appearance of ventricular arrhythmias, or recurrent embolic episodes. Even in asymptomatic patients, we believe that aneurysm resection should be considered if there is critical coronary anatomy (left mainstem stenosis or triple vessel disease) or evidence on serial noninvasive testing of failing left ventricular function (ie, increasing diastolic dimensions, decreasing ejection fraction, or increasing mitral regurgitation). Surgery in these cases should be performed before the patient reaches the stage when transplantation is the only reasonable alternative.

All patients with a left ventricular aneurysm should undergo cardiac catheterization to assess the extent of coronary artery disease. Biplane ventriculography is essential to evaluate left ventricular size and shape. Whether or not the patient is a candidate for surgery depends on the function of left ventricular cavity outside the aneurysmal segment. If at least one area of the myocardium is functioning (ie, basal constrictors anteriorly and/or posteriorly) the patient should be considered for surgical repair. In general, the more discrete the left ventricular aneurysm and the more dyskinetic the aneurysmal segment, the lower the operative risk.

In patients with a left ventricular aneurysm, some degree of mitral regurgitation is often present. Preoperative echocardiographic studies often indicate 2+ mitral regurgitation when none was suspected on the angiogram because dilution of dye in the enlarged ventricle makes it difficult to see the regurgitant jet. In aneurysm patients, the degree of mitral regurgitation is critically
dependent on left ventricular geometry and loading conditions. Repair of the aneurysm ± revascularization may result in significant improvement in valve function by one of three possible mechanisms: (1) decreased annular dilatation secondary to decreased left ventricular size; (2) improved function of the valvular apparatus caused by realignment of the papillary muscles secondary to aneurysm repair; or (3) improved function of ischemic papillary muscle caused by revascularization. In our experience, provided the aneurysm is fairly discrete, the larger and the closer it is to the mitral valve apparatus, the more likely surgical repair is to favorably influence the degree of mitral regurgitation. Mitral regurgitation in itself is not a contraindication to aneurysm excision. Only when severe mitral regurgitation is combined with severe global left ventricular dysfunction would transplantation be considered a better option.

**Surgical Technique**

1. Left ventricular aneurysms should be approached through a midline sternotomy. When extensive pericardial adhesions are encountered, mobilization of the heart should be kept to a minimum until cannulation of the aorta and the atrium have been accomplished. Double venous cannulation is recommended for most aneurysm repairs. An aortic vent is inserted for de-airing purposes. In patients with coronary artery disease who require bypass grafting, adequate distribution of cardioplegia to the left ventricle is insured by retrograde delivery. Cold blood cardioplegia is used. The right ventricle is perfused antegrade either through the aortic root or through a right coronary artery graft.
After cannulation, adhesions are dissected and the heart is mobilized. (In the case of contained free-wall rupture or "false aneurysm," final dissection into the aneurysmal sac should be delayed until after going on cardiopulmonary bypass.) In many cases, with clear-cut scarring and thinning, the extent of the aneurysm is obvious. In others, inspection reveals an area of mixed scar and viable muscle with no obvious thinning. After placing such patients on cardiopulmonary bypass with decompression of the left ventricle, an area of dimpling or collapse may become obvious. If thinning of the infarct area is not obvious, simple needle aspiration, as shown in this figure, can be used to determine the degree of thinning and the need for resection and repair.
3 Once the presence of a resectable area of thinned wall has been confirmed and before opening the aneurysm, the patient is placed in Trendelenburg and the aortic vent is opened to gravity. Stay sutures are applied to the epicardial surface of the scarred area. The sutures are elevated and a sponge is held over the proposed incision site to protect the operating team as the thinned area is opened.

4 In patients with intraventricular clot, the edges of the incision are retracted with Babcock clamps and the clot is mobilized and removed in one piece if possible. A simple teaspoon can be used in this maneuver to scoop out the clot. A flexible sucker is used to decompress and empty the left ventricular cavity while clot is being removed. A left ventricular vent is inserted through the right superior pulmonary vein only after inspection insures that no thrombus remains. In this way, fragmentation and possible embolization of thrombus is avoided. The vent is placed on gentle suction.
Any obvious thinned transmural scar is excised. With the heart open and beating, the surrounding edges are palpated and assessed for contractility. Areas capable of significant contraction or wall thickening in the unloaded state are not resected but revascularized whenever possible, whereas areas that do not contract are considered for excision.

Before final trimming, the size and shape of the remaining left ventricular cavity is evaluated. When the residual chamber is of relatively normal size and shape, linear closure can be easily accomplished. In patients with extensive coronary artery disease, marked chamber dilatation, diffuse hypokinesis, and distortion of ventricular shape (spherical versus conical), it is not possible to restore the ventricular cavity towards normal size or shape with any repair technique. In such patients, for linear closure to be accomplished without distorting left ventricular geometry (specifically the relationship between papillary muscle and the septum) a portion of the nonfunctioning wall may have to be left behind. In these difficult cases, the final resection margins are determined with these considerations in mind.
In patients with marked thinning of the septum or an obvious septal aneurysm, a patch septoplasty should be performed using bovine preserved pericardium. (A) A coronal section of the heart after resection of an anterior aneurysm reveals thinning of the anterior septum which requires reinforcement with a pericardial patch. (B) The patch is applied to the left ventricular aspect of the septum and sewn in place to the surrounding normal myocardium on three sides with 4-0 prolene. (C) Anteriorly, the patch is incorporated into the linear ventriculotomy repair.
Aneurysm excision ± septoplasty are performed on the open beating heart. Using the principles of excision as previously described, in many cases the excised specimen is composed of a mixture of infarcted and viable muscle and it is not possible to leave behind a rim of fibrous tissue as described in the classic description of aneurysm repair. In such cases, closing sutures have to be placed through fairly thick areas of myocardium. (A) The thinned edges of the aneurysmal sac are retracted with Babcock clamps. The limits of the resection margins have been determined by palpation and the thinned noncontractile area is being excised.
The excision has been completed and the edges of the resection margin are illustrated. The incision is closed with mattress sutures of 2-0 prolene buttressed by felt strips. As the diagram shows, sutures are placed further apart on the tissue than on the felt so as to plicate the length of the incision in the closure. This technique helps to restore the shape of the ventricle towards normal.
Once all of the sutures are in place, the vent is placed on gravity to establish a column and then clamped. Starting at each end, sutures are tied leaving an area for de-airing in the center of the closure. Blood is left behind, the lungs are ventilated, and the patient is rotated from side to side as blood and air are ejected through the de-airing site. When de-airing is completed, the final sutures are tied. The heart is then decompressed by opening the vent to gravity while the closure is reinforced with a continuous over and over suture to ensure hemostasis. By performing the entire repair without cross clamping, ischemic time is kept to a minimum. This may decrease perioperative mortality and morbidity especially in those with severe triple vessel disease. Following aneurysm repair, in those requiring aortocoronary bypass grafting, the aorta is cross clamped and cardioplegia is performed using a combination of antegrade and retrograde delivery as previously described. Diseased arteries are bypassed whenever possible. We revascularize the proximal portion of the left anterior descending (LAD) even if the distal vessel has been amputated in the repair. Revascularization of even a small part of the septum may be important in improving short-term and long-term results in these patients.
In most patients with an inferior aneurysm, the principles of tissue resection and repair are identical to those described for anterior aneurysms. In some cases, however, thin scar extends up to the level of the mitral valve apparatus and it would be impossible to excise the thinned area and reapproximate the edges without plicating or distorting the valve ring. In these patients, principles of linear closure are modified in the following way: (A) The apex of the heart is elevated. (B) The proposed incision is indicated.
(Continued) (C) The aneurysm has been opened and the edges are retracted. The thinned portion of the posterior wall is indicated (shaded area). The proposed resection margin is outlined by the dashed line. (D) Most of the thinned area has been resected, the remaining edge to be closed is shown in cross section. To accomplish the repair without distortion of the mitral valve annulus and to reinforce the closure close to the valve, a pericardial patch is used. It is attached first along the mitral annulus. In this diagram, the patch is elevated to show sutures in the thinned scar (shaded area) which is closed posteriorly as a separate layer.
After tying the sutures in the thin layer of scar, the pericardial patch is reflected posteriorly to cover and reinforce the repair. The patch is attached laterally and medially to relatively normal myocardium. The apex of the patch is incorporated into the posterior linear repair which is accomplished as previously described. (F) In the completed posterior repair, an over and over continuous suture is used to complete the closure and ensure hemostasis.
Aneurysm Resection in Patients With Ventricular Tachycardia

Aneurysm patients often present with ventricular arrhythmias. Electrophysiological studies in these patients have suggested reentry mechanisms in a fixed anatomic substrate are responsible for the arrhythmias whereas chamber dilatation, increased wall tension, and resulting subendocardial ischemia may play a role in initiating the ventricular tachycardia (VT). Results of extensive intraoperative mapping have shown that most arrhythmias in these patients arise from a subendocardial or midmyocardial focus, usually at the junction between scar and nonscarred myo cardium. Such foci correspond pathologically to sheets of surviving muscle fibers mixed with extensive areas of scar. In patients with VT and a ventricular aneurysm, the target area for ablation almost always lies outside the area of resectable transmural scar and is most often found on the scarred septum. Such patients can be considered for map-directed VT ablation at the time of aneurysm repair. In centers where mapping is not available or for high-risk patients (age >65 years), a visually directed septal intervention will provide effective control of arrhythmias in most cases. Our approach in these patients is as follows: Once the aneurysm is opened and the extent of septal scarring has been determined, sharp dissection is used to excise a thin layer of endocardial scar from the left side of the septum. After removal of this sheet of scar, overlapping cryolesions (−60°C for 2 minutes) are applied along the perimeter of the excision in order to isolate possible deep septal foci. If the septum has been significantly thinned by the endocardial excision, a patch septoplasty is performed as previously described.

Even in aneurysm patients with no clinical history of ventricular arrhythmias, a preoperative electrophysiological study will often reveal inducible VT. In these patients, if the arrhythmogenic substrate is not modified by the aneurysm repair, the potential for arrhythmias during follow-up is significant. For this reason, Vincent Dor has advocated septal intervention in all patients with an aneurysm. In my opinion, whether septal procedures as previously outlined should be performed to minimize the risk of future arrhythmias in patients with septal scarring and no previous history of arrhythmias is an issue yet to be resolved.

Results Achieved With Aneurysm Repair Using the Modified Linear Closure Technique

Using these principles of patient selection and the operative technique of modified linear closure, aneurysm repair can be accomplished with a low operative mortality and morbidity. We recently reported a consecutive series of 92 patients in whom the operating room mortality was 3% despite the fact that a high proportion of these patients presented with diffuse coronary artery disease, advanced left ventricular dysfunction, and ventricular arrhythmias. This mortality compares very favorably with others reported in the literature using linear or more complex repair techniques. We believe that the linear closure technique, which can be accomplished without aortic cross clamping, is advantageous because of its simplicity, reproducibility, and limitation in ischemic time. In our series, only 18% of patients required intra-aortic balloon pump support and there were few perioperative complications including only one perioperative stroke. We believe that our approach with removal of all LV thrombus before insertion of an LV vent is important in minimizing the risk of stroke.

In our experience, most patients are symptomatically improved postoperatively. Using nuclear angiographic (MUGA) data we have provided objective evidence of improved LV function. In addition, echocardiographic data available in a limited number of patients showed significant decrease in LV dimensions and

Perioperative Management

After aneurysm resection, high filling pressures are usually required to maintain an adequate cardiac output. Patients with low output despite adequate filling are treated with an intra-aortic balloon pump and inotrope support.

In the postoperative period, patients who have been on long-term amiodarone therapy present a particularly difficult problem because they are susceptible to severe Adult Respiratory Distress Syndrome (ARDS). To prevent this complication, amiodarone should be discontinued at least 4 weeks before surgery whenever possible. Excessive volume administration should be avoided and FIO2 should be kept as low as possible during the pump run and throughout the perioperative period. In these patients, daily chest radiographs and serial arterial blood gas determinations should be monitored very closely. If the patient becomes hypoxic or pulmonary infiltrates appear, vigorous diuresis and broad spectrum antibiotics are indicated.

After aneurysm resection, patients are not routinely anticoagulated. Postoperative echocardiograms are performed and only if significant clot is seen are patients put on coumadin for three months. If significant postoperative ventricular arrhythmias occur they are treated aggressively with intravenous amiodarone therapy. Need for chronic amiodarone can be reassessed at 3 months based on Holter monitoring.

COMMENTS

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mitral regurgitation postoperatively. Interestingly enough, there was no correlation between postoperative ejection fraction or postoperative ventricular size (end diastolic dimension) and functional class. Possible reasons for this discrepancy have been previously discussed. In our series, in which patients were followed for 10 years, there were 15 late deaths as a result of congestive heart failure. Actuarial survival including hospital deaths was 88% at 1 year, 86% at 2 years, and 80% at 5 years.

With respect to the patient subgroup with ventricular arrhythmias, using a map guided approach to VT ablation, 74% of patients had a completely negative postoperative electrophysiological study. All those with positive tests were discharged on amiodarone. In this subgroup, during follow-up, freedom from sudden death was 96% at 5 years and overall survival was 80% at 5 and 8 years.

In summary, our experience with the modified linear closure technique for aneurysm resection has shown that aneurysm repair can be performed even in patients with severe coronary artery disease and poor ventricular function ± ventricular arrhythmias with a low operative mortality, good symptomatic improvement, and excellent 5-year survival. This technique is relatively simple, reproducible, and can be modified when necessary to effectively deal with a number of intraoperative variables. We believe that our results support more liberal indications for aneurysm excision. We would recommend this procedure even in asymptomatic patients with critical coronary anatomy and in those with relatively minor symptoms but objective evidence of deteriorating left ventricular function (increasing diastolic dimensions, increasing mitral regurgitation, or decreasing ejection fraction). We believe that earlier surgical intervention in these patients should be considered before they decompensate to the point that cardiac transplantation or an implantable assist device represents the only reasonable option.

REFERENCES


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