DOWNLOAD PDF SURGICAL REMODELING IN HEART FAILURE

Chapter 1 : Surgical Ventricular Restoration to Reverse Left Ventricular Remodeling

Although a favorable outcome has been reported in selected patients, this method is currently not recommended for treatment of heart failure because of high surgical failure rates. The present paper reviews some of the relevant literature regarding surgical left ventricular remodeling in heart failure.

This is an open access article distributed under the terms of the Creative Commons Attribution License http: This article has been cited by other articles in PMC. Abstract Heart failure is one of the major health care issues in the Western world. An increasing number of patients are affected, leading to a high rate of hospitalization and high costs. Even with administration of the best available medical treatment, mortality remains high. The increase in left ventricular volume after a myocardial infarction is a component of the remodeling process. Surgical Ventricular Restoration SVR has been introduced as an optional therapeutic strategy to reduce left ventricular volume and restore heart geometry. So far, it has been established that SVR improves cardiac function, clinical status, and survival in patients with ischemic, dilated cardiomyopathy and heart failure. Since its first description , SVR has been refined in an effort to standardize the procedure and to optimize the results. This review will discuss the rationale behind surgical reversal of LV remodeling, the SVR technique, its impact on cardiac function and survival, and future expectations. Myocardial infarction, Left ventricular remodeling, Surgical ventricular restoration, Systolic function, Diastolic function. The incidence and prevalence of heart failure continues to increase due in part to an extended average life expectancy and morbidity and mortality remain high despite improvement in treatment. Heart transplantation remains the treatment of choice for patients with medically refractory end stage HF [4 , 5]. However, the need for immunosuppression and the paucity of donors have greatly restricted the selection criteria, leaving many patients and physicians seeking other options. Medical therapy, targeted to block the neurohormonal pathway, has dramatically improved the survival of HF patients by slowing the progression of the disease [6 - 8]. Despite the application of the best available medical therapy, the percentage of patients suffering from signs and symptoms of HF still remains high. This supports the concept that HF progresses independently of neurohormonal activation due to an abnormal and excessive increase in left ventricular LV volume. This theory, the biomechanical model of HF, was first proposed by Mann and Bristow [9]. The concept of a biomechanical model of HF introduces the need for optional strategies aimed at reducing LV volumes, and restoring heart geometry. Surgical ventricular restoration SVR has been introduced to restore LV shape, size, and function in patients with ischemic dilated cardiomyopathy and HF. The technique, initially introduced by Dor [10] and Jatene [11], has been refined over the last ten years in an effort to standardize the procedure and optimize the results. This review will discuss the rationale to surgically reverse LV remodeling, the SVR technique, its impact on cardiac function and survival, and future expectations. Remodeling occurs in several clinical conditions, including myocardial infarction MI , cardiomyopathy, hypertension, and valvular heart disease. Myocardial infarcts MI , particularly large, transmural infarcts, result in a number of structural changes involving both the infarcted and non-infarcted zones [12]. LV remodeling usually begins within the first few hours after an MI and may progress over time. It is accompanied by a secondary volume-overload eccentric hypertrophy of the non-infarcted remote regions that should counteract the increased wall stress and reduce the stimulus for further dilatation [13 , 14]. However, a negative balance, related to the infarct size and the degree of myocardial cells loss, towards LV enlargement may result in loading conditions that promote further dilatation and global ventricular dysfunction [12]. Structural and geometric ventricular changes proceed along with increased myocyte stress, neurohormonal activation, collagen synthesis, fibrosis, and remodeling of the extracellular matrix, resulting in further deterioration of cardiac function [15]. Since LV wall tension is directly proportional to the LV internal radius and pressure, and inversely proportional to wall thickness, any intervention to optimize this relationship would be beneficial in terms of improving wall compliance and reducing filling pressure. Optimization may also be beneficial in terms of enhancing the contractile performance of the LV by increasing the extent and velocity of systolic fiber shortening [16]. This technique, completed during cardioplegia, involved opening the ventricle in the center of the depressed area

and performing a thrombectomy when indicated. Then, exclusion of the dyskinetic or akinetic LV free wall proceeded with an endoventricular circular suture passed through the fibrous tissue above the transitional zone. In the event of recurrent ventricular arrhythmias, cryotherapy was applied at the transitional zone. A Dacron patch lined with pericardium was secured at the junction of the endocardial muscle and scarred tissue, thereby excluding non contractile portions of the LV and septum. The excluded scar was folded over the patch to assure hemostasis. Myocardial revascularization was performed before reconstruction, making sure to revascularize the proximal left anterior descending segment in addition to performing mitral valve repair when indicated. To avoid excessive resection, leaving too small a residual volume, Dor introduced the use of an intraventricular balloon. It was removed before closure of the ventricle. Later, the procedure was adopted by many skilled and creative surgeons without real standardization, making results difficult to compare. McCarthy described a no-patch, double purse-string suture technique [19]; Mickleborough described a tailored scar excision, with septoplasty when indicated for dyskinetic septum, and modified linear closure [20]; Menicanti adopted a technique that is similar to the Dor procedure except for the use of a pre-shaped mannequin TRISVR TM, Chase Medical Richardson, TX , which is illustrated in Fig. The mannequin is useful when the ventricle is not extremely enlarged to reduce the risk of the residual cavity being too small. It is also useful when the transitional zone between scarred and non-scarred tissue is not clearly demarcated, as in dilated cardiomyopathy and recent MI.

Chapter 2 : Management of heart failure - Wikipedia

Heart failure is a major cause of death in the western population. Five-year survival of patients in heart failure is below 50% and patients in NYHA class III and IV have a 1-year survival below 50%.

Advanced Search Abstract Maladaptive remodeling is associated with impaired prognosis in heart failure, and prevention of remodeling is an established therapeutic target. However, it is much less clear whether remodeling may be reversed once it has developed. In the last decade, anti-neuroendocrine therapy with ACE inhibitors, and even more potently, beta-blockers, was shown to improve surrogate markers for reverse remodeling, such as ejection fraction EF , ventricular volumes, and mass. For beta-blockers, reverse molecular remodeling was also shown in biopsy specimens on the cellular and subcellular level. Both moderate endurance training and continuous positive airway pressure CPAP therapy in heart failure patients with sleep apnea induce reverse remodeling. Cardiac resynchronization therapy improves exercise capacity and quality of life in patients with ventricular dyssynchrony and is clearly associated with geometrical and functional reverse remodeling over time. Whether this translates into improved survival remains to be demonstrated. Surgical approaches for reverse remodeling, such as mitral valve replacement, aneurysmectomy, and volume reduction Batista procedure have been developed, but may also be associated with high perioperative mortality. Mechanical unloading of the failing ventricles by left ventricular assist systems LVAD induces well-characterized reverse remodeling on the cellular and subcellular level. However, persistent functional improvement in a significant subset of patients that would allow weaning form the device is still under debate. Novel, complementary approaches, such as gene transfer or stem cell therapy are under pre-clinical and clinical investigation. Taken together, reverse remodeling can be induced by pharmacological and non-pharmacological therapy and may serve as a surrogate parameter for therapeutic success in the individual patient. Since maladaptive remodeling is associated with poor prognosis, identification of novel strategies to reverse this process remains a promising target. Reverse remodeling , Heart failure Introduction Heart failure remains a major clinical and economic health care problem, with increasing prevalence mainly related to better survival from myocardial infarction and increased longevity of the population. Despite improved therapeutical options, reduction in mortality from heart failure only marginally contributed to the average 3. Remodeling and reverse remodeling The progression of heart failure is associated with left ventricular remodeling, which manifests as gradual increases in left ventricular end-diastolic and end-systolic volumes, wall thinning, and a change in chamber geometry to a more spherical, less elongated shape Fig. This process is usually associated with a continuous decline in ejection fraction. The concept of cardiac remodeling was initially developed to describe changes which occur in the days and months following myocardial infarction. Atlas of heart failure, The process of cardiac remodeling is influenced by hemodynamic load, neurohumoral activation, and other factors still under investigation. The myocyte is the major cardiac cell involved in the remodeling process. Other components include the interstitium, fibroblasts, collagen, and coronary vasculature Table 1 ; relevant processes also include ischemia, cell necrosis and apoptosis. Functional polymorphisms in modifier genes relevant for disease progression may impact on the remodeling process. Despite myocyte hypertrophy, this leads to defective contractile function, which may contribute to further progression of myocardial remodeling.

Chapter 3 : Batista procedure - Wikipedia

Therefore, surgery of end-stage heart failure using conventional techniques has gained new interest after the introduction of the Batista operation. However, a clear surgical identification of the patients to be included in this group is lacking.

Lifestyle changes[edit] People with CHF are educated to undertake various non- pharmacological measures to improve symptoms and prognosis. Sleep apnea is an under-recognized risk factor for heart failure Weight reduction â€" through physical activity and dietary modification, as obesity is a risk factor for heart failure and left ventricular hypertrophy. Monitor weight â€" this is a parameter that can easily be measured at home. Rapid weight increase is generally due to fluid retention. Weight gain of more than 2 pounds is associated with admission to the hospital for heart failure [2] Sodium restriction â€" excessive sodium intake may precipitate or exacerbate heart failure, thus a "no added salt" diet 60â€" mmol total daily intake is recommended for patients with CHF. Fluid restriction[edit] According to a review in , there is apparently no evidence of benefit of fluid restriction in patients with clinically stable heart failure otherwise receiving optimal pharmacological treatment. Some drugs which increase heart function, such as the positive inotrope milrinone , lead to increased mortality, and are contraindicated. Angiotensin II receptor antagonist therapy also referred to as AT1-antagonists or angiotensin receptor blockers , particularly using candesartan , is an acceptable alternative if the patient is unable to tolerate ACEI therapy. It is also believed that angiotensin directly affects cardiac remodeling, and blocking its activity can thereby slow the deterioration of cardiac function. Combined therapy did reduce HF-related hospital admissions with an absolute risk reduction of 4. Thus, combined therapy does not improve mortality and may slightly increase morbidity. Diuretics[edit] Diuretic therapy is indicated for relief of congestive symptoms. Several classes are used, with combinations reserved for severe heart failure: Eplerenone is specifically indicated for post-MI reduction of cardiovascular risk. If a heart failure patient exhibits a resistance to or poor response to diuretic therapy, ultrafiltration or aquapheresis may be needed to achieve adequate control of fluid retention and congestion. The use of such mechanical methods of fluid removal can produce meaningful clinical benefits in patients with diuretic-resistant heart failure and may restore responsiveness to conventional doses of diuretics. It is also thought that catecholamines and other sympathomimetics have an effect on cardiac remodeling, and blocking their activity can slow the deterioration of cardiac function. Positive inotropes[edit] Digoxin a mildly positive inotrope and negative chronotrope , once used as first-line therapy, is now reserved for control of ventricular rhythm in patients with atrial fibrillation; or where adequate control is not achieved with an ACEI, a beta blocker and a loop diuretic. The inotropic agent dobutamine is advised only in the short-term use of acutely decompensated heart failure, and has no other uses. The mechanism of action is through inhibiting the breakdown and thereby increasing the concentration of cAMP similar to beta adrenoreceptor agonism, resulting in inotropic effects and modest diuretic effects. The related drug eplerenone was shown in the EPHESUS trial [27] to have a similar effect, and it is specifically labelled for use in decompensated heart failure complicating acute myocardial infarction. While the antagonism of aldosterone will decrease the effects of sodium and water retention, it is thought that the main mechanism of action is by antagonizing the deleterious effects of aldosterone on cardiac remodeling. Recombinant neuroendocrine hormones[edit] Nesiritide , a recombinant form of B-natriuretic peptide, is indicated for use in patients with acute decompensated heart failure who have dyspnea at rest. Nesiritide promotes diuresis and natriuresis, thereby ameliorating volume overload. It is thought that, while BNP is elevated in heart failure, the peptide that is produced is actually dysfunctional or non-functional and thereby ineffective. Vasopressin receptor antagonists[edit] Tolvaptan and conivaptan antagonize the effects of antidiuretic hormone vasopressin , thereby promoting the specific excretion of free water, directly ameliorating the volume overloaded state, and counteracting the hyponatremia that occurs due to the release of neuroendocrine hormones in an attempt to counteract the effects of heart failure. The EVEREST trial, which utilized tolvaptan, showed that when used in combination with conventional therapy, many symptoms of acute decompensated heart failure were

significantly improved compared to conventional therapy alone [28] although they found no difference in mortality and morbidity when compared to conventional therapy. This treatment modality may alleviate symptoms, improving quality of life, and in some trials has been proven to reduce mortality. In the remaining two thirds of patients who have a QRS complex duration of ms or less , CRT may actually be harmful. The CCM mechanism is based on stimulation of the cardiac muscle by non-excitatory electrical signals NES , which are delivered by a pacemaker -like device. CCM is particularly suitable for the treatment of heart failure patients with normal QRS complex duration ms or less and has been demonstrated to improve the symptoms, quality of life and exercise tolerance of heart failure patients. Although ICDs deliver electrical shocks to resynchronize heart rhythm which are potentially distressing to the patient, they have not been shown to affect quality of life. Another current treatment involves the use of left ventricular assist devices LVADs. LVADs are battery-operated mechanical pump-type devices that are surgically implanted in the upper part of the abdomen. They take blood from the left ventricle and pump it through the aorta. LVADs are becoming more common and are often used in patients waiting for heart transplants. Surgery[edit] The final option, if other measures have failed, is heart transplantation or temporary or prolonged implantation of an artificial heart. These remain the recommended surgical treatment options. However, the limited number of hearts available for transplantation in a growing group of candidates, has led to the development of alternative surgical approaches to heart failure. These commonly involve surgical left ventricular remodeling. If heart failure ensues after a myocardial infarction due to scarring and aneurysm formation, reconstructive surgery may be an option. These aneurysms bulge with every contraction, making it inefficient. Cooley and coworkers reported the first surgical treatment of a left ventricular aneurysm in In the s, Vincent Dor developed a method using a circular patch stitched to the inside of the ventricle the endoventricular circular patch plasty or Dor procedure to close the defect after excision. The STICH trial Surgical Treatment for IschemiC Heart Failure will examine the role of medical treatment, coronary artery bypass surgery and left ventricle remodeling surgery in heart failure patients. Results are expected to be published in [needs update] and It involves removal of a portion of viable tissue from the left ventricle to reduce its size partial left ventriculectomy , with or without repair or replacement of the mitral valve. At 3 years only 26 percent were event-free and survival rate was only 60 percent. Mesh-like constraint devices such as the Acorn CorCap aim to improve contraction efficacy and prevent further remodeling. Clinical trials are underway.

Chapter 4 : Surgical left ventricular remodeling in heart failure.

72 Ann Thorac Cardiovasc Surg Vol. 16, No. 2 () Review Surgical Remodeling of the Left Ventricle in Heart Failure W. Jack Wallen, MD, PhD, and Vivek Rao, MD, PhD, FRCSC.

Advanced Search Abstract Patients with ischaemic cardiomyopathy and left ventricular LV systolic dysfunction represent the highest-risk population with heart failure HF. The cornerstone of treatment remains guideline-driven medical therapy, which is associated with significant improvement in survival and quality of life. The most commonly considered surgical interventions are coronary artery bypass graft surgery, at times combined with surgical ventricular reconstruction SVR and surgery for mitral valve regurgitation. Surgical ventricular reconstruction has been introduced as an optional therapeutic strategy aimed to reduce LV volumes through the exclusion of the scar tissue, thereby restoring the physiological volume and shape and improving cardiac function and clinical status. This review will briefly discuss the rationale to surgically reverse LV remodelling, the technique, and the indications, to the best of our knowledge, coming from the Center with the largest worldwide experience. Indeed, HF is a syndrome with a broad spectrum of heterogeneous symptoms and signs caused by cardiac dysfunction and resulting in a wide range of clinical expressions. However, beyond this early stage, the remodelling process is driven predominantly by eccentric hypertrophy of the non-infarcted remote regions, resulting in increased wall mass, chamber enlargement and geometric distorsion. Furthermore, mitral regurgitation MR may occur as a consequence of the LV remodelling worsening the prognosis. In addition, ventricular dilatation results in annular enlargement, which further increases valve incompetence. Since LV wall stress is directly proportional to LV internal radius and pressure and inversely proportional to wall thickness, any intervention to optimize this relationship would be beneficial in terms of either improving wall compliance and reducing filling pressure or, as wall stress is a crucial determinant of afterload, in terms of enhancing contractile performance of LV by increasing the extent and velocity of systolic fibre shortening. Finally, although the matter of functional chronic ischaemic MR, in terms of whether, when and how it should be corrected is still considerably controversial, it should be pointed out that surgical ventricular reconstruction SVR offers either the possibility to repair the mitral valve through the LV opening or the potential of improving mitral functioning by reducing LV volumes and papillary muscles distance which is a main determinant of functional MR. After that, the ventricle is opened with an incision parallel to the left anterior descending artery, starting at the middle scarred region and ending at the apex. The cavity is inspected and any thrombus is removed if present. The mannequin is useful in giving the surgeon the correct position of the apex and in maintaining the long axis of the ventricle in a physiological range 7. The exclusion of dyskinetic or akinetic LV free wall is performed through an endoventricular circular suture passed in the transitional zone. The ventricle is closed over the mannequin respecting the longitudinal diameter; if the dilatation involves also the inferior wall, a short plication of the inferior wall is performed to avoid amputation of the apex. The final result has to be an elliptical shape of the ventricle; when the dilatation starts close to the aortic valve, a running suture is conducted from the inner of the ventricle over the mannequin towards the apex. The mannequin is deflated and removed before completing the closure of the ventricle. The positioning of the patch is crucial in determining the residual shape of the new ventricle. To this aim, we pay attention to positioning the patch with an oblique orientation, towards the aortic outflow tract. When indicated, mitral valve is repaired through the ventricular opening with a double arm stitch running from one trigone to the other one, embedding the two arms in the posterior anulus of the mitral valve. To avoid tears of the posterior leaflet of the mitral valve, the suture is reinforced with a Teflon strip. A restrictive mitral annuloplasty with a ring implantation may be performed in selected patients, when the LV opening is not big enough to have a good exposition of the mitral valve. Tailored approaches The surgical procedure as described above is usually performed to reverse LV remodelling after an anterior MI. Surgery for the posterior aneurysm generally involves a patch to close the neck of dilatation. The series is changed over the course of 25 years of experience, either in terms of number of patients treated or for type of patient. The decline in the number is mainly due to the advances in the treatment of acute MI, which had also an impact on survivors, in

the meaning that patients with the classical dyskinetic remodelling of the apex decreased while we observed an increase of patients showing LV chambers severely distorted with a more global LV dilatation. In the former group, the indication to perform SVR is clear, while in the latter more caution is required for the lack of a well-defined zone of transition between the scarred tissue and the remote myocardium. To this regard, the use of cardiac magnetic resonance CMR with late-gadolinium enhancement LGE for the detection of myocardial scar has gained a major role in the patient selection, allowing the exclusion of those patients for whom the final result is expected to be unfavourable.

Chapter 5 : Left Ventricular Reconstructive Surgery (Modified Dor Procedure) | Cleveland Clinic

In this review article, we describe the most common surgical procedures currently used to reverse or arrest remodeling of the left ventricle in patients with congestive heart failure (CHF).