Heart failure after a myocardial infarction (MI) is often progressive. After death of the cardiomyocytes, macrophages, monocytes, and neutrophils migrate into the infarct area, initiating the inflammatory response. Infarct expansion then begins to occur because of the activation of matrix metalloproteinases (MMPs), which degrade the extracellular matrix and result in myocyte slippage. This weakening of the collagen scaffold results in wall thinning and ventricular dilation. After the initial inflammatory phase, there is an increase in fibrillar, cross-linked collagen deposition, which resists deformation initiated by the initial inflammatory response. Infarct expansion then begins (1). Evidence suggests that the death of cardiomyocytes results in negative left ventricular (LV) remodeling, which leads to increased wall stress in the remaining viable myocardium. This process results in a sequence of molecular, cellular, and physiological responses that lead to LV dilation. It is suggested that LV remodeling may contribute independently to the progression of heart failure (2).

Cellular transplantation, LV restraint devices, and tissue engineering approaches have emerged as possible alternatives to heart transplantation for the treatment of damaged myocardium. Initial studies focused on the injection of viable cells directly into the infarcted myocardium, a technique which has been termed cellular cardiomyoplasty. More recent approaches include the use of in vitro engineered tissue, which is cultured in vitro and then implanted in vivo, and in situ engineered tissue, which is injected directly into the myocardium. Polymer meshes have also been utilized to prevent LV expansion. This review focuses on the current advances and progress in the use of biomaterials for treatment of MI (Table 1). Biomaterial treatments that have been examined in vivo are covered.

For nearly a decade, researchers have investigated the possibility of cell transplantation for cardiac repair. More recently, the emerging fields of tissue engineering and biomaterials have begun to provide potential treatments. Tissue engineering approaches are designed to repair lost or damaged tissue through the use of growth factors, cellular transplantation, and biomaterial scaffolds. There are currently 3 biomaterial approaches for the treatment of myocardial infarction (MI). The first involves polymeric left ventricular restraints in the prevention of heart failure. The second utilizes in vitro engineered cardiac tissue, which is subsequently implanted in vivo. The final approach entails injecting cells and/or a scaffold into the myocardium to create in situ engineered cardiac tissue. This review gives an overview of the current progress in the growing field of biomaterials for the treatment of MI. (J Am Coll Cardiol 2006;48:907–13) © 2006 by the American College of Cardiology Foundation.
after MI in an ovine model, whereas Sabbah et al. (10) demonstrated similar effects in a canine chronic heart failure model, along with reduction of myocyte hypertrophy, down-regulation of stretch response proteins, and improved sarcoplasmic reticulum calcium cycling. In an effort to more fully decipher the mechanisms behind the CSD treatment, Blom et al. (11) reported a normalized myocyte beta-adrenergic response, reduced myocyte length, increased collagen content, and decreased MMP-9 in a sheep MI model. Clinical studies have also demonstrated the effectiveness of an LV restraint in humans. Konertz et al. (12) reported an improved ejection fraction and reduced LV volume in 27 patients suffering from heart failure 3 and 6 months after receiving a CSD. Franco-Cereceda et al. (13) also reported increased LV function and decreased LV volume in a trial with 8 patients with dilated cardiomyopathy. The length of the study was between 12 and 24 months. Olsson et al. (14) demonstrated continued, gradual improvement in LV volume and function in 12 patients with dilated cardiomyopathy; however, they also reported right ventricular dysfunction and no improvement in cardiac output. Acorn's pivotal clinical trial encompassing 300 patients initially reported that the CSD reduced LV diastolic volume, improved patient quality of life, and reduced the likelihood of additional cardiac procedures (15). However, the significance of the study has been criticized because of the partial recruitment of patients in an unblinded fashion, the influence of missing data, and the decrease in beneficial effects when patients undergoing concomitant mitral valve replacement were separated from the analysis.

Studies examining epicardial polymeric LV restraints have had encouraging results; however, a major drawback with this approach is the surgical procedure required for implantation. There are also some conflicting results as to the real benefit of the CSD in clinical trials, where some measures of cardiac function are improved, while others remain unchanged or deteriorate. Therefore, the results should be taken with some caution, and there exists a need for more long-term results and a more thorough analysis of the exact mechanism behind LV restraint.

**IN VITRO ENGINEERED MYOCARDIAL TISSUE**

Tissue engineering approaches are designed to repair lost or damaged tissue through the use of cellular transplantation and biomaterial scaffolds. Numerous studies have examined...
different scaffolds as well as various culture conditions for creating in vitro engineered myocardial tissue (15–25). Those that have been examined in vivo are discussed in this review. Li et al. (26) first demonstrated the transplantation of cells in a biomaterial scaffold for the treatment of myocardial scar tissue. They reported the survival of fetal cardiomyocytes that were seeded onto a biodegradable gelatin mesh in vitro and implanted onto the myocardial surface in a cryoinjury model; however, the cell seeded grafts did not improve cardiac function. Leor et al. (27) reported both survival and preservation of cardiac function with fetal cardiomyocytes seeded onto an alginate scaffold, which was subsequently implanted in a rat MI model. The grafts were found to be vascularized and the scaffold was completely degraded after 2 months; however, only a small portion of the graft consisted of myofibers. Transplantation of the scaffold alone was not examined, and thus it is unknown whether improvement of cardiac function was a result of implantation of the biomaterial or cell transplantation. Kellar et al. (28) also employed a pre-formed scaffold by using the commercially available Dermagraft, which contains human dermal fibroblasts cultured on a knitted poly(glycolide)/poly(lactide) mesh. Transplantation of the Dermagraft onto the LV resulted in significantly higher ejection fraction above 100 μm (30), as seen with many in vitro engineered tissues. Yet, in a more recent study, this group reported ~450-μm-thick, newly formed myocardium using this approach, which was shown to improve systolic and diastolic function in rats. Five circular grafts were stacked crosswise to obtain grafts of 1 to 4 mm in thickness that were subsequently transplanted onto the epicardial surface of the infarct (31). Although it was not reported, necrosis within the grafts likely occurred because of the significant decrease in thickness after transplantation. Grafts were cultured with increased ambient oxygen and insulin, which may have allowed for the formation of in vitro tissue thicker than the typical 100 μm.

Krupnick et al. (32) also combined cells with a collagen and matrigel mixture. Bone marrow-derived mesenchymal progenitor cells were first suspended in the gel, then seeded onto a porous poly(L-lactic acid) non-woven mesh, and finally reinforced with a layer of poly(tetrafluoroethylene). Instead of implanting the engineered tissue on the epicardial surface, they sutured it into the infarct wall after a ventriculotomy. Aneurysmal dilation did not occur with this approach, which was shown to improve systolic and diastolic function in rats. Five circular grafts were stacked crosswise to obtain grafts of 1 to 4 mm in thickness that were subsequently transplanted onto the epicardial surface of the infarct (31). Although it was not reported, necrosis within the grafts likely occurred because of the significant decrease in thickness after transplantation. Grafts were cultured with increased ambient oxygen and insulin, which may have allowed for the formation of in vitro tissue thicker than the typical 100 μm.

Table 1. Biomaterials for Treatment of Myocardial Infarction

<table>
<thead>
<tr>
<th>Material</th>
<th>Transplantation</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polypropylene</td>
<td>Alone</td>
<td>3–6</td>
</tr>
<tr>
<td>Polyester</td>
<td>Alone</td>
<td>7–14</td>
</tr>
<tr>
<td>In vitro engineered tissue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gelatin</td>
<td>Alone or with fetal cardiomyocytes</td>
<td>27</td>
</tr>
<tr>
<td>Alginate</td>
<td>With fetal cardiomyocytes</td>
<td>28</td>
</tr>
<tr>
<td>Poly(glycolide)/poly(lactide)</td>
<td>With dermal fibroblasts</td>
<td>29</td>
</tr>
<tr>
<td>Collagen type I and matrigel</td>
<td>With neonatal cardiomyocytes</td>
<td>30,31</td>
</tr>
<tr>
<td>PTFE, PLA mesh, collagen type I, and matrigel</td>
<td>Alone or with bone marrow-derived mesenchymal progenitor cells</td>
<td>32</td>
</tr>
<tr>
<td>Collagen type I</td>
<td>Alone or with embryonic stem cells</td>
<td>33</td>
</tr>
<tr>
<td>PNIPAAM (cell culture dish)</td>
<td>Cell sheet of neonatal cardiomyocytes or adipose-derived mesenchymal stem cells</td>
<td>36,37</td>
</tr>
<tr>
<td>In situ engineered tissue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibron</td>
<td>Alone, with skeletal myoblasts, bone marrow mononuclear cells, or pleiotrophin plasmid</td>
<td>45–50,59</td>
</tr>
<tr>
<td>Collagen</td>
<td>Alone or with bone marrow cells</td>
<td>48,51,52</td>
</tr>
<tr>
<td>Matrigel</td>
<td>Alone</td>
<td>53</td>
</tr>
<tr>
<td>Collagen type I and matrigel</td>
<td>Alone or with embryonic stem cells</td>
<td>48,54,55</td>
</tr>
<tr>
<td>Collagen type I and matrigel</td>
<td>Alone or with neonatal cardiomyocytes</td>
<td>56</td>
</tr>
<tr>
<td>Self-assembling peptides</td>
<td>Alone, with neonatal cardiomyocytes, or with platelet-derived growth factor BB</td>
<td>57,60</td>
</tr>
<tr>
<td>Gelatin</td>
<td>With basic fibroblast growth factor</td>
<td>58</td>
</tr>
</tbody>
</table>

PLA = poly(L-lactic) acid; PNIPAAM = poly(N-isopropylacrylamide); PTFE = poly(tetrafluoroethylene).

Rather than seed cells onto a pre-formed scaffold, Zimmermann et al. (29) combined neonatal cardiomyocytes with liquid collagen type I, matrigel, and cell culture medium and then pipetted the mixture into molds to form the desired shape. Upon transplantation onto the epicardial surface of uninjured hearts, the engineered tissue was contractile in vivo up to 8 weeks and was observed to be both vascularized and innervated. In this first study, the single-muscle bundles in the engineered tissue did not increase above 100 μm (30), as seen with many in vitro engineered tissues. Yet, in a more recent study, this group reported ~450-μm-thick, newly formed myocardium using this approach, which was shown to improve systolic and diastolic function in rats. Five circular grafts were stacked crosswise to obtain grafts of 1 to 4 mm in thickness that were subsequently transplanted onto the epicardial surface of the infarct (31). Although it was not reported, necrosis within the grafts likely occurred because of the significant decrease in thickness after transplantation. Grafts were cultured with increased ambient oxygen and insulin, which may have allowed for the formation of in vitro tissue thicker than the typical 100 μm.

Yamada et al. (34) and Okano et al. (35) have developed a unique approach for utilizing a biomaterial for the creation of patches of cardiac tissue in vitro. They utilized a temperature-responsive polymer, poly(N-isopropylacrylamide) (PNIPAAM), which is slightly hydrophobic and cell-adhesive at 37°C but becomes hydro-
philic and cell-resistant at 32°C because of rapid hydration and swelling. Tissue culture plates were coated with PNIPAAm and subsequently seeded with neonatal cardiomyocytes. Once the cells formed a monolayer, the temperature was dropped and the cell sheet was removed intact. Both cell-to-cell junctions and adhesive proteins within the monolayers are preserved, unlike with enzymatic digestion (36). Up to 6 sheets (100 μm) may be layered upon each other to create a 3-dimensional pulsatile cardiac tissue construct without resulting in a necrotic core. More recently, Miyahara et al. (37) transplanted monolayers of adipose tissue-derived mesenchymal stem cells using this cell-sheet technology, which resulted in improved fractional shortening and infarct wall thickness. After 4 weeks, the monolayers had expanded in situ to produce ~600-μm-thick tissue where it was transplanted over the infarct scar. The newly formed tissue consisted of neovasculature, undifferentiated mesenchymal stem cells, and a few cardiomyocytes.

As seen with other in vitro tissue engineering approaches, the majority of external myocardial tissue constructs are limited to a thickness of 100 μm or approximately 6 cardiomyocytes. Studies by both Zimmermann et al. (29–31) and Miyahara et al. (37) demonstrated tissue of approximately one-half a millimeter in thickness in vivo. Although improved culture conditions were attributed to the thicker tissue in the study by Zimmermann et al. (29–31), Miyahara et al. (37) transplanted a cell monolayer that then expanded to form a larger graft in vivo. The monolayer of mesenchymal stem cells produced only a few cardiomyocytes, but this study does demonstrate the in situ expansion capabilities of stem cells in the myocardium. Although these studies offer the hope of regenerating sizable constructs without resulting in a necrotic core. More recently, Miyahara et al. (37) transplanted monolayers of adipose tissue-derived mesenchymal stem cells using this cell-sheet technology, which resulted in improved fractional shortening and infarct wall thickness. After 4 weeks, the monolayers expanded in situ to produce ~600-μm-thick tissue where it was transplanted over the infarct scar. The newly formed tissue consisted of neovasculature, undifferentiated mesenchymal stem cells, and a few cardiomyocytes.

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**IN SITU ENGINEERED MYOCARDIAL TISSUE**

Cellular cardiomyoplasty may be considered the first example of in situ cardiac tissue engineering. Cellular cardiomyoplasty involves the transplantation of viable cells to replace necrotic cardiomyocytes. Although studies have shown some improvement in cardiac performance by using cellular cardiomyoplasty, there are several problems associated with this technique. The current transplantation techniques involve the administration of cells in an aqueous solution administered intravenously, intracoronary or directly injected into the myocardium; however, the techniques are plagued by limited cell retention and transplant survival (38–41). When reported, the number of animals receiving successful grafts is often low. Given that the cells are injected in an ischemic region of the heart, there is also little to no vasculature to supply the implanted cells. Cell survival is thus limited by the lack of retention and vascularization. Another problem associated with the current technique is that the cells are poorly distributed. Cross sections of the infarcted region show clusters of the implanted cells between scar tissue. Conduction through the infarcted region should thus still be a problem, since the cells are in isolated areas and may lead to a proarrhythmic heterogeneous milieu (42). Furthermore, the typical injection technique involves injection of cells in completely liquid solutions and does not give the transplanted cells a temporary matrix to which they can attach. Cellular cardiomyoplasty does not involve the use of biomaterials and is thus not fully covered in this review. Several excellent reviews exist that cover this topic in detail (43,44).

The emerging field of tissue engineering has begun to provide promising alternatives to the typical cellular cardiomyoplasty technique. Although in vitro-engineered myocardial tissue has had some promising results, the limitations described previously led to investigations of a different tissue engineering approach to cardiac repair. This in situ approach utilizes an injectable biomaterial to deliver cells directly into the infarct wall to increase cell survival. Injectable biomaterials can also be utilized in acellular approaches to support the LV wall and prevent the negative remodeling after an MI, or for controlled delivery of therapeutic genes and proteins to ischemic myocardium. An injectable treatment is more minimally invasive than implanting in vitro-engineered tissue or an epicardial patch, and is therefore more clinically appealing.

Christman et al. (45) were the first to demonstrate improved cell survival when transplanted cells are delivered in an injectable scaffold compared to the typical cellular cardiomyoplasty technique. The injectable biopolymer fibrin glue was also shown to induce neovascularization within the ischemic myocardium and reduce infarct expansion. More interesting is the observation that injection of fibrin glue with or without skeletal myoblasts preserved LV geometry and cardiac function in an acute MI model (46). Ryu et al. (47) further demonstrated the beneficial effects of an injectable fibrin glue scaffold by injecting bone marrow mononuclear cells in the matrix. They likewise reported enhanced neovascularization in ischemic myocardium, which was further confirmed by Huang et al. (48). Chekanov et al. (49) also demonstrated improved cardiac function and neovascularization with endothelial cells in a fibrin matrix compared to saline controls; however, injection of fibrin alone or healthy endothelial cells alone was not examined. Therefore, it is difficult to conclude what caused the improvement. Recently, the use of fibrin glue for the treatment of chronic aneurysms resulting from MI has been investigated. Christman et al. demonstrated that the injection of fibrin glue into the aneurysm resulting from an MI restored geometry of the LV and markedly improved LV function (50). Although the improvement of LV function was not sustained at 5 weeks after the injection, arrest of LV dilation and deterioration of LV function occurred.
Other bio-derived materials have also been used for in situ cardiac tissue engineering. Thompson et al. (51) demonstrated successful injection of bone marrow cells in collagen into the myocardium via catheter; however, the injection was done in uninjured hearts, and comparison to cell injection in a liquid solution was not performed. Dai et al. (52) also injected collagen into infarcted myocardium as an acellular treatment. They reported improved LV geometry and cardiac function without increased vascularization compared to saline controls. In contrast, Huang et al. (48) showed an increase in capillary density following injection of collagen. Infiltration of myofibroblasts was also reported. Recently, Leor et al. (53) have suggested that intramyocardial injection of alginate induces neovascularization and improved LV function.

Kofidis et al. (54) examined an in situ approach using matrigel to deliver mouse embryonic stem cells. An LV pouch was formed, similarly to their study using an in vitro approach, and the gel was injected into the area. They demonstrated improved LV function in those animals that received the cell-matrigel mixture compared to those that received either the biomaterial alone or cells in cell culture medium. A further study used this approach to directly inject the cells into infarcted murine myocardium (55). Moreover, Huang et al. (48) demonstrated increased vasculature in infarcted myocardium after injection of matrigel. Zhang et al. (56) used a mixture of matrigel, collagen, and cell culture medium to deliver cardiomyocytes, similar to the system used by Zimmermann et al. (29–31) in vitro, and reported preserved LV geometry and cardiac function.

Davis et al. (57) developed a novel injectable scaffold for the myocardium using self-assembling peptides, which form nanofibers upon injection, creating a microenvironment that is suitable for cell and vessel ingrowth. After injection of the peptides alone into the infarct, progenitor cells expressing endothelial cell markers and vascular smooth-muscle cells were recruited into the nanofibers. Neonatal cardiomyocytes were also injected with the nanofibers and were found to enhance recruitment of endogenous cells. In contrast, little recruitment was seen in infarcted myocardium injected with matrigel.

Taken together, these results suggest that matrigel may be beneficial for delivering cells, but is not ideal for use alone or as a scaffold that promotes in situ regeneration. Self-assembling peptides appear to have great promise in promoting regeneration, but a suitable cell source for myocardial regeneration is needed. Alginate, collagen, and fibrin have shown promise for cellular delivery and regeneration, but their long-term effects have not been examined. Whether their benefits will persist months and years after the scaffold has degraded is unknown. Collagen and alginate are also known to be mechanically unstable in vivo; thus, when used as a stand-alone treatment, an injectable polymer that is stiffer and either non-degradable or more slowly degradable may be more beneficial. Such a scaffold may prevent heart failure by increasing the mechanical strength of the infarct, thereby preventing remodeling and deterioration of cardiac function in a similar fashion to LV restraints. On the other hand, a polymer that is too stiff may induce diastolic dysfunction; therefore, the mechanical properties of the scaffold must be carefully examined.

While the typical tissue engineering approach involves a biomaterial scaffold with cells, such biomaterials may also be employed as delivery vehicles for therapeutic genes or proteins in order to stimulate tissue regeneration. Only a few studies to date have examined this in the myocardium. Iwakura et al. (58) delivered basic fibroblast growth factor via injectable gelatin microspheres and reported increased angiogenesis as well as improved cardiac function. Christman et al. (59) also increased neovascularization in ischemic myocardium by delivering a plasmid encoding the angiogenic growth factor pleiotrophin in fibrin glue. Finally, Hsieh et al. (60) employed self-assembling peptides as a delivery vehicle for platelet-derived growth factor-BB. They reported sustained delivery for 14 days in infarcted myocardium, which decreased cardiomyocyte death and preserved cardiac function compared to either the peptides or the growth factor alone. They also demonstrated a reduction in infarct size. By injecting the therapeutic agent in a biomaterial, a more prolonged delivery profile can be achieved. A scaffold can also act as a gene-activated matrix to increase the transfection efficiency of plasmid DNA (61).

**FUTURE DIRECTIONS AND CONCLUSIONS**

Many cell types and tissue engineering approaches have improved cardiac function in animal models; however, the exact mechanisms of each approach are currently unknown. There are still many questions and issues to be addressed before this technology can be safely applied to patients. For instance, finding the best cell source for cardiac repair continues to remain a major obstacle because of the difficulty in isolating and expanding autologous sources, the ethical issues surrounding certain cell types, and the inability of one cell source to fully replenish all necessary cell types. Furthermore, there is little data examining long-term results. Current studies often last 1 to 2 months, and thus may not be indicative of long-term outcomes.

The use of biomaterials for in situ cardiac tissue engineering is being appreciated as either a stand-alone acellular solution for cardiac repair or a hybrid therapy used in combination with cells or therapeutic agents. Future studies are needed to investigate whether biomaterials can be used to help repair myocardial tissue after an acute ischemic insult and regenerate myocardial tissue in a chronic scar. Biomaterials could be used in situ to increase the wall thickness, restore the geometry, and provide structural support of an injured LV. The body would be its own bioreactor and allow for infiltration of cells within the scaffold matrix to regenerate myocardial muscle and blood vessels. Biomaterials have already been shown to recruit cells into injured myocardial tissue (48,57). To allow in situ myocardial tissue engineering to become a viable option for the
treatment of myocardial injury, engineering of biomaterials to specifically influence the microenvironment of the myocardium will be required. Such materials should be designed to enhance recruitment of progenitor cells for myocardial muscle and myocardial vasculature, and increase durability of improved LV function. Moreover, the formation of muscle bundles with functioning conductive tissue is a necessity.

Another important factor for the future success of biomaterial treatments in the myocardium is the control over the tissue response after implantation or injection. Introduction of a biomaterial into the body can result in a wide range of effects, both local and systemic (62). Implantation or injection results in local injury, which can then initiate an inflammatory response and foreign body reaction. Acute inflammation, which can last from minutes to days, is characterized by the presence of edema and the migration of leukocytes into the tissue. Continued exposure to an inflammatory material can lead to chronic inflammation, which involves the presence of macrophages, monocytes, and lymphocytes. This can be caused not only by a non-biocOMPATIBLE material, but also by a material that is not properly secured in the body. Proliferation of blood vessels and connective tissue also begins to occur at this stage, which then leads to the formation of granulation tissue within 3 to 5 days of implantation. Implantation of a biomaterial can also result in a foreign body reaction, which is indicated by the presence of foreign body giant cells. A greater number of these cells are often seen on the surfaces of rougher or more porous materials compared to those with smooth surfaces. The final phase of the foreign body reaction involves fibrous tissue formation and encapsulation. A biomaterial's reaction at the implant site obviously must be considered when choosing a material for the heart, as chronic inflammation or fibrous encapsulation would impede regeneration. More specific antigen-mediated responses can also occur and should be considered when delivering bio-derived materials containing components from other species. An excessive immune response can even lead to hypersensitivity, which can result in tissue damage due to the release of intracellular chemicals, or excessive thrombus formation. Another concern over the choice of a biomaterial is its affinity for bacterial contamination and subsequent infection at the implant site. Likewise, the ability to properly sterilize a biomaterial is critical.

The reaction of the body to a foreign material should thus be deemed significant when designing biomaterial treatments for the myocardium. The ability of cells to adhere, survive, and migrate within a biomaterial scaffold should also be strongly considered when trying to regenerate tissue. Synthetic hydrophobic materials will almost immediately adsorb proteins that can then mediate cell attachment and spreading. Bio-derived materials such as fibrin glue and collagen natively contain peptide binding sequences that cells can adhere to via integrins. Synthetic materials can also be modified with these peptides as well as other molecules, in order to more closely mimic native tissue (63). These types of materials, termed biomimetics, have not yet been examined in the myocardium, but may prove extremely useful for both improving cell adhesion and viability, and controlling the host response.

Because of the difficulties in finding the appropriate cell source, biomaterial treatments such as external LV restraints or injectable polymers may be more easily realized in the clinic. With the aid of biomaterial scaffolds and a suitable cell source, regenerated myocardium may be achieved. We are thus optimistic that future studies will continue to provide more insights and that the field of biomaterials and myocardial tissue engineering will bring new treatments for those patients with injured myocardium.

REFERENCES


