Dynamic myoplasty: Surgical transfer and stimulation of skeletal muscle for functional substitution or enhancement

Pierre Grandjean, MS; Michael Acker, MD; Robert Madoff, MD; Norman S. Williams, MS, FRCS; Jean Woloszko, MD, PhD; Carole Kantor, MS
Bakken Research Centre B.V., 6201 MP Maastricht, The Netherlands; Hospital of the University of Pennsylvania, Philadelphia, PA 19104; University of Minnesota, Minneapolis, MN 55402; The London Hospital Medical College, University of London, London, England, E1 1BB; Tantalus, Inc., Highland Park, NJ 08904

Abstract—Dynamic myoplasty combines muscle transfer with electrical stimulation to provide contractile function that augments or replaces impaired organ function. Dynamic cardiomyoplasty was the first clinical application in which a skeletal muscle, latissimus dorsi, was transferred and stimulated to provide cardiac assistance, a function different from its original one. The problem of early muscle fatigue that was encountered in the initial implementation of the method was solved by training the muscle with electrical stimulation and thus changing its fiber composition. With intramuscular electrodes, the conditioned latissimus dorsi is stimulated in synchrony with the heart muscle. Safeguards are built into the two-channel implanted stimulator to avoid excessively high pulse rates. Clinicians report that 80% of patients with moderate to severe heart failure prior to operation showed a clinical improvement of 1.6 New York Heart Association classes. Alternative methods of providing cardiac assistance that are also being investigated include wrapping the muscle around the aorta, creating a skeletal muscle ventricle, and using the muscle to power an implantable pump. These latter techniques are still under preclinical investigation. Compared with heart transplant, cardiomyoplasty has the great advantage of not being subject to tissue rejection.

The second principal application of dynamic myoplasty is treatment of fecal incontinence through creation of an electrically stimulated skeletal muscle neosphincter (ESMNS). The gracilis muscle of the leg is mobilized, wrapped around the anal canal, and conditioned with electrical stimulation to become more fatigue resistant. To achieve continence, the muscle is continuously stimulated except when the patient wishes to defecate. Overall success rates in achieving continence are 60–65%. Both cardiomyoplasty and the ESMNS technique, and their associated devices, are being refined through ongoing clinical trials.

Key words: cardiomyoplasty, dynamic myoplasty, fecal incontinence, FES, FNS, functional electrical stimulation, heart failure, urinary incontinence.

INTRODUCTION

Myoplasty encompasses a variety of clinical procedures involving transfer of skeletal muscle for replacement or enhancement of body parts. The main uses so far have been for filling facial defects and for breast reconstruction. In dynamic myoplasty, the addition of electrical stimulation allows the transferred muscle also to provide contractile function. The two major applications presently under clinical investigation are dynamic cardiomyoplasty for the treatment of heart failure and dynamic myoplasty for treatment of fecal or urinary incontinence.
Dynamic cardiomyoplasty was the first clinical application in which a skeletal muscle was transferred and stimulated to provide a function different from its original one (1). In this procedure, skeletal muscle can be transferred to reinforce the myocardium. Other approaches form pumping chambers called skeletal muscle ventricles at points along the ascending or descending aorta (2,3). In these cardiac applications, the transferred muscle is paced cyclically in such a way as to synchronize with a selected portion of the cardiac cycle. In treatment of fecal or urinary incontinence, skeletal muscle is wrapped around the anus, urethra, or bladder neck to form a neosphincter. After conditioning, the muscle is stimulated continuously except when the patient interrupts it to defecate or urinate. This paper reviews the technical and clinical experience with these various applications of dynamic myoplasty.

DYNAMIC CARDIOMYOPLASTY

Dynamic cardiomyoplasty is a procedure aimed at treating patients with chronic heart failure, refractory to medical therapy, that severely limits their daily life, a Class III or intermittent Class IV condition, according to the New York Heart Association (NYHA) classification (4). Besides medical therapy, treatment alternatives for severe heart failure are heart transplant, mechanical assists, and dynamic cardiomyoplasty. The last two are still investigational methods. Heart transplant is the gold standard for treatment of Class IV heart disease, but only 8 percent of patients who would benefit from it receive new hearts because of limited donors. In addition, this treatment requires lifelong immunosuppression which may lead to problems with infection, kidney failure, and deterioration of other systems.

In the next 5 to 10 years, we will see advances in various mechanical heart assist devices. The United States Food and Drug Administration (FDA) has approved the first such device, an implantable left ventricular assist system, as a “bridge” until a heart transplant can be carried out. Such devices still require better solutions to the problems created by external power sources and blood-surface interactions, especially when long-term support is considered.

Cardiomyoplasty is the only method of skeletal muscle cardiac assistance currently under clinical investigation. Studies are ongoing worldwide, including the USA, under an FDA investigational device exemption (IDE). Because the method uses autologous tissue, there are no rejection problems, thus no need for immunosuppression, and no need for an external power source.

The major limitation in the initial application of dynamic cardiomyoplasty was the early onset of muscle fatigue as soon as a skeletal muscle was activated at cardiac rates. Pioneering work by Pette (5) and Salmons (6) showed that muscle fiber composition and resulting physiologic and metabolic characteristics (e.g., force, fatigue resistance, contraction, and relaxation times) depend on neural activity. By electrically controlling nerve and muscle activation with functional neuromuscular stimulation (FNS), Peckham, et al. (7) produced increased force and improved the fatigue resistance of atrophied paralyzed muscle (with intact lower motoneurons) in quadriplegic patients.

The possibility of combining knowledge gained in the FNS field with anatomical and surgical considerations suggested a solution to the problem of early muscle fatigue in cardiomyoplasty. First, an innovative progressive muscle stimulation protocol was developed by Carpentier, et al. (4). Second, the latissimus dorsi was selected (8). This muscle is used in dynamic cardiomyoplasty as done today because it is a wide muscle, it is not necessary for normal activity, and it can be easily transferred into the thorax. Latissimus dorsi has its neuromuscular pedicle in its proximal part, making its transfer possible without great disturbance of the nerve or blood supply.

Conditioning Skeletal Muscle for Cardiac Assist

Cardiac and skeletal muscles differ in their response to electrical impulses. A single electrical pulse above a given threshold will cause an action potential to propagate through the entire cardiac muscle mass, producing a smooth muscle contraction. On the other hand, the magnitude and duration of skeletal muscle contraction varies with the characteristics of the electrical pulses: amplitude, width, and interpulse interval (or pulse frequency). Pulse train stimulation grades the magnitude and duration of muscle force by varying the rate of motor unit excitation (temporal summation). Only a train of pulses spaced to produce summation will result in a prolonged (>50 milliseconds) and forceful contraction. The fatigue resistance of stimulated muscle depends on fiber composition, metabolism, stimulus parameters, and duty cycle.

By the early 1970s, Salmons (6), Pette (5), and Peckham (7) had shown that the fatigue resistance of skeletal muscle could be increased by several weeks of low-frequency electrical stimulation. To understand the
source of the aerobic capacity of such conditioned muscle, the group at the University of Pennsylvania studied the bioenergetic correlates of fatigue resistance in conditioned canine latissimus dorsi muscle using the technique of phosphorus 31 nuclear magnetic resonance (P-31 NMR) spectroscopy. An overall small change in phosphorylation with a lack of plateau in developed isometric tension was observed in the conditioned muscle. This was similar to that seen in cardiac muscle during increased energy demand. This study indicated that the markedly enhanced resistance to fatigue of the conditioned muscle is in part the result of its increased capacity for oxidative phosphorylation (9).

Oxygen consumption was also measured directly in conditioned muscle, and compared to its contralateral control. It was found that electrically transformed canine muscle is capable of generating more isometric work while consuming less oxygen per amount of tension developed than its contralateral control muscle. Isometric tension is maintained by turnover of actin-myosin cross bridges without relative movement of the filaments. The rate at which cross bridges cycle determined the energy cost for the maintenance of tension. The fact that cross bridge cycling is more rapid in fast muscle than in slow muscle reflects differences between rates of hydrolysis of the fast and slow isoforms of myosin. Since the conditioned muscle is uniformly slow, a given isometric tension can be maintained at an expense of less ATP (adenosine triphosphate) hydrolyzed. Therefore, less oxygen should be consumed by the electrically conditioned muscle for the same amount of isometric tension developed. These studies demonstrated that the metabolic capacity to do cardiac work is present in electrically conditioned skeletal muscle, and it has the potential to be a functioning myocardial substitute.

Surgical Procedure

Surgery begins with the patient in the lateral position. Through a large incision the latissimus dorsi muscle is freed from its distal insertions, the muscle is lifted, and the electrodes (cathode and anode) are inserted near the nerve branches of the muscle (4). Then, the muscle is inserted inside the thorax through a window made by resection of part of the second rib (Figure 1).

The second phase of the procedure is carried out in the supine position through an incision of the mediastinatum. The muscle is brought under the heart and wrapped, in most cases, in a posterior to anterior wrap, also called clockwise fashion (Figure 1). The muscle is sutured to the myocardium or to the pericardium sack. Patients with chronic heart failure usually have dilated hearts, and the heart coverage that the muscle can provide is sometimes insufficient. In such cases, a piece of the pericardium is used to complete the wrapping.

The procedure is done in different ways depending on the nature of the heart failure. If the heart is only dilated, the muscle is used as a reinforcement; this type of surgery can be done without the use of circulatory support. If the patient is suffering from a large aneurysm, the aneurysm can be resected and the muscle used as a patch, taking care that there is a surface between the muscle and the blood to prevent clotting problems.

Device

The device is an implantable pulse generator (cardiomyostimulator), powered with a single lithium battery and sealed in a titanium case. This second-generation device weighs 60 grams and is connected to

Figure 1.
In cardiomyoplasty, the latissimus dorsi muscle is mobilized, transposed into the chest through the rib cage, and wrapped around the ventricles of the heart. The muscle is then stimulated in synchrony with the heart to augment its pumping function. Permission to reprint this figure has been granted by Medtronic, Inc.
the muscle via intramuscular electrodes. Intramuscular rather than nerve or epimysial electrodes were selected to ensure that a wide range of surgeons would be able to implant the device easily.

Anodic and cathodic electrodes are placed near the nerve branches of the latissimus dorsi. In most patients, there is no need to do nerve mapping, since the nerve branches are clearly visible and can be used as landmarks. The lead is composed of a nickel alloy (MP35N) coil that ends with an electrode coil made of platinum-iridium. The lead is enclosed in a pair of polyurethane sheaths, one of which slides on the other to permit adjustment of the lead length to different muscle sizes. The electrode is inserted inside the muscle near the nerve branches by means of a suture with an attached needle; this is done in such a way as to minimize the energy requirement, taking care not to be so close to the nerve branches as to damage them. Two silicon disks and plastic clips secure the lead in position once the sliding sheath has been adjusted after the positioning of the electrode (Figure 2). For the cardiac channel, a smaller version of the skeletal muscle electrode and lead are used and inserted into the myocardium to ensure proper detection of the heart function.

The muscle is stimulated in synchrony with the heart function by the cardiomyostimulator ("Transform" Model 4710, Medtronic, Inc., Minneapolis, MN) that senses the heart function and paces the muscle with trains of impulses (Figure 2). The device is a two-channel system with one channel dealing with the heart and the other with the transplanted muscle. The heart channel senses the heart and paces it if necessary. The signal from the heart channel is processed via a synchronization circuit that senses the ventricular contraction and, after a synchronization delay, puts out a burst of pulses.

Triggering the muscle by the heart alone may create problems, because most of these patients have very high resting heart rates of 90–100 beats per minute. Stimulating the muscle at this rate could lead to fatigue and consequently muscle damage, depending on the individual patient. This problem has been addressed by providing means to program the device within the following parameters:

1. **synchronization ratio**: enables augmentation or reduction of muscle activity by synchronizing the muscle on different heart beats, for instance, on every three heart beats

2. **muscle upper rate**: enables programming the maximum muscle contraction rate allowed

3. **synchronization upper rate**: enables programming a cardiac upper rate above which the muscle is disabled

4. **adaptive burst duration**: enables the burst duration to automatically adapt to heart rate (burst length decreases as heart rate increases).

In addition, cardiac sensitivity, refractory period, and synchronization delay (time from cardiac event to muscle burst initiation) are programmable to adjust muscle activation to cardiac contraction.

Many characteristics of the stimulator are adjustable: The muscle channel is bipolar and can be turned on and off. The pulse amplitude can be programmed up to 9 V; pulswidth and frequency within pulses can be regulated; burst durations can be adjusted by program-
ming the number of pulses and the pulse interval. A magnet mode has been included to enable the cardiologist to easily assess the unstimulated performance of the heart by applying the magnet to inhibit the muscle channel. The system can be programmed with radio frequency (RF), using standard cardiac pacemaker methods.

A 2-week postsurgery rest period is allowed to give time for the muscle flap to heal and the muscle-heart interface to begin to develop adhesions. Stimulation synchronized on the heart function is begun with single pulses on every other heartbeat. The number of pulses in the burst is then progressively increased every 2 weeks, ultimately producing a burst of 6 pulses lasting about 185 ms (Figure 3). In some cases, after 2 to 3 months of training, the stimulation mode may be switched to pulses on every heartbeat, depending on the patient’s clinical condition. This type of stimulation protocol enables long-term cyclic muscle contraction at cardiac rates. Muscle biopsies in animals have shown that the trained muscle converted to type I fibers (aerobic metabolism). With new knowledge in this field, we may find that complete conversion of this sort is not necessary for adequate function (10).

The device is tuned by changing the synchronization delay. Mitral flow regurgitation is avoided by making sure the stimulation burst never occurs before the mitral valve closure. In patients with more than 3 months follow-up, the settings are typically: pulse amplitude, 4.3 V; pulsewidth, 200 μs; number of pulses, 6; pulse interval, 33 ms; average delay, 50 ms. Average muscle lead impedance is approximately 390 ohms. In order to minimize risks of muscle overuse, most patients are programmed at a synchronization rate of 2:1.

**Evaluation of First Generation Device**

To date, two cardiomyostimulation systems have been developed by Medtronic and used in clinical studies. The first-generation (Medtronic Model SP1005/A) has been described earlier by Grandjean, et al. (11) and is summarized below.

Overall performance of the system has been good in 515 implant-years of cumulative experience. Of 353 systems, 99 have required replacement due to battery depletion (mean time to replacement: 28 months). The longest implant of a single device has been 41 months. There were eight cases of inadequate synchronization, mostly in the first series of devices, caused by inadequate signal filtering. This was solved by retuning the cardiac circuit to adapt to the very weak signals sometimes found in dilated hearts. There were 16 cases of premature end-of-life of the device, mostly happening after patients were subjected to multiple defibrillation shock.

Of 707 intramuscular leads with a total of 1,030 lead-years of cumulative experience, there has been no lead failure (breakage or displacement). The longest implant has been in place for 110 months. There have been only six cases of failure to evoke muscle contraction: as the leads remained integral, it was concluded that perhaps muscle transfer had caused nerve damage. Three of these cases had no recovery of muscle function. Others recovered evoked muscle contraction after 3 to 9 months, indicating nerve damage and subsequent repair. Selectivity of stimulation was good; there has been no lead displacement or breakage. However, there were several cases of lead damage perioperatively when the lead caught in a retractor; such leads were repaired or exchanged during surgery.

In order to meet new requirements as investigation expanded, a new (second-generation) system was developed that is easier to manufacture and meets additional needs realized in early clinical experience. Changes to the cardiac elements of the device are: 1) it uses a demand pacemaker with a larger range of cardiac pacemaking amplitude; 2) it is programmable, and unipolar and bipolar to help in situations where we combine devices (e.g., using it in combination with a dual pacemaker, or implantable defibrillator); 3) its synchronization range has been extended up to 1:8; 4) it may be disabled in case the cardiac rate exceeds a

<table>
<thead>
<tr>
<th>TIME AFTER SURGERY</th>
<th>STIMULATION TYPE</th>
<th>ECG</th>
<th>MUSCLE CONTRACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>WEEK 1-2</td>
<td></td>
<td></td>
<td>NO STIMULATION</td>
</tr>
<tr>
<td>WEEK 3-4</td>
<td></td>
<td></td>
<td>FLAP HEALING, ADHESIONS</td>
</tr>
<tr>
<td>WEEK 5-6</td>
<td></td>
<td></td>
<td>SINGLE PULSES, 2:1</td>
</tr>
<tr>
<td>WEEK 7-8</td>
<td></td>
<td></td>
<td>DOUBLE PULSES, 2:1</td>
</tr>
<tr>
<td>AFTER 2 MONTHS</td>
<td></td>
<td></td>
<td>TRIPLE PULSES, 2:1</td>
</tr>
<tr>
<td>CLINICAL DEMAND</td>
<td></td>
<td></td>
<td>PULSE TRAINS, 2:1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>PULSE TRAINS, 1:1</td>
</tr>
</tbody>
</table>

Figure 3.
Stimulation protocol used for training the transposed latissimus dorsi after cardiomyoplasty.
Changes to the muscle elements of the device are:
1) unipolar and bipolar polarization, 2) adjustable burst length by programming number of pulses and pulse interval, 3) adaptation mode where burst shrinks as heart rate increases to prevent diastolic filling impairment, and 4) upper rate to prevent muscle overstimulation.

Specially developed components make this new device smaller and lighter, and give it greater longevity than the previous generation. Safety features include: 1) runaway protection on cardiac and muscle channels, 2) full protection for electrocautery and defibrillation, 3) special connectors that prevent lead misconnection, and 4) progressive battery depletion indicator which can be monitored by telephone. To retrofit patients with earlier devices, adapters were made for connecting previous leads to new stimulators. Packaging is recyclable and each device has a unique tag that is visible on X-ray.

As of July 1994, this second-generation system had a cumulative experience of 15 implant-years. Of the 58 systems, 13 were replacements of the previous cardiomyostimulators which had reached expected battery depletion. Premature end of life of the device has not been a problem, even in cases of frequent defibrillation. Of 90 leads with a total experience of 30 years, the longest follow-up time has been 8 months. No lead or device failures have been observed.

Clinical Outcomes

In Phase I studies of cardiomyoplasty, surgeons treated 118 patients with various degrees of heart failure using single or multiple procedures (combining myoplasty with ventricular aneurysectomy or coronary artery bypass graft surgery). Preoperatively, the patients were 75 percent Class III and the rest Class IV (confined to bedrest). The average ejection fraction (percentage of blood emptied from the ventricle during contraction) was 20 percent, compared with normal values of 60–70 percent. This low fraction is an indication of advanced heart failure resulting in marked limitation in daily activities and usually poor quality of life.

After cardiomyoplasty, based on subjective clinical evaluations, 80 percent of patients showed a clinical improvement of an average of 1.6 NYHA classes. The hospital mortality of Class III patients was less than 10 percent at most centers. Patients whose conditions were rated Class IV experienced a higher hospital mortality (~30 percent). In most centers, it was concluded that Class IV patients are too sick for this procedure. While there was no overall objective evidence of cardiac function improvement in the total group, two centers showed improved systolic function in subgroups of their patients.

When expansion of the trials was requested, the FDA required a Phase II clinical protocol that would confirm the safety of the first-generation device and better document the efficacy of the cardiomyoplasty procedure. The Phase II trials focused on cardiomyoplasty as an isolated procedure. Between May 1991 and September 1993, 68 patients were enrolled. Their mean age was 57 and most of them (91 percent) were in NYHA class III. Eight patients (12 percent) died prior to discharge from initial hospitalization. At the end of 12 months, 47 percent moved to Class II and 41 percent to Class I. A reference group, not prospectively randomized, was given only medical treatment.

This Phase II trial, a pooled multicenter study, showed objective evidence of systolic improvement: left ventricular ejection fraction rose from 22.7 percent preoperatively to 26.1 percent at 6 months, a small but statistically significant (p<0.05) 15 percent relative increase. At one year, there was no decrease in ejection fraction. Improvements in left ventricular stroke work index and stroke volume were statistically significant. Disappointingly, the maximal increase in oxygen consumption did not reach statistical significance. Again, patients showed a clear improvement of quality of life, as documented in questionnaires. To investigate if cardiomyoplasty improves survival, a randomized trial is currently underway. Deaths in the first year have been mostly sudden, due to arrhythmias in half the cases. This emphasizes the possibility of an automatic defibrillator combined with a myostimulator.

Other Applications Under Investigation

In addition to placing the transferred muscle around the heart, alternate methods of providing cardiac assist are: 1) wrapping the muscle around the aorta (Figure 4), 2) creating a skeletal muscle ventricle (SMV, see Figure 5), and 3) using the muscle to power an implantable pump. These applications are being studied in animal tests. In dynamic aortomyoplasty, the latissimus dorsi is wrapped on the ascending aorta. In the SMV approach, the latissimus dorsi muscle is shaped into a pumping chamber which is then connected, for example, to the aorta. It can be stimulated in a counterpulsation mode, contracting when the heart relaxes. The “aortic chamber” or the SMV
In aortomyoplasty, the latissimus dorsi muscle is wrapped around the aorta and stimulated to contract during cardiac diastole (counterpulsation) to augment diastolic pressure, which will improve coronary perfusion. Aorta during cardiac systole (left bottom) and during cardiac diastole (right bottom). Permission to reprint this figure has been granted by Medtronic, Inc.

empties during the diastolic phase and fills during the systolic phase, augmenting diastolic pressure and decreasing systolic pressure. This decreases the afterload of the heart, decreases wall tension on a failing heart, and improves perfusion of the myocardium. Both approaches aim to reproduce chronically the acute benefits of the intra-aortic counterpulsation balloon pumping that is frequently used in intensive care.

Since the relation of the stimulation to the cardiac cycle is different than with cardiomyoplasty, counterpulsation approaches require a more sophisticated stimulation system. It must account for the facts that heart systolic and diastolic times decrease as heart rate increases, and that the time of onset of muscle stimulation (synchronization delay) and burst duration need to be adjusted as heart rate varies. Medtronic has developed a software-based device, similar to an implantable microcomputer (12). The device function is defined by a program (algorithm), which is loaded into its random access memory (RAM) by RF link. The algorithm permits adaptation of synchronization delay and burst duration as heart rate is changing. Tests of this system have examined the effect of different burst patterns, pulse intervals, and change in number of motor units recruited. Arrhythmia can be detected during muscle stimulation and the pulse burst can be aborted. The system can then wait a few cycles until the heart rate stabilizes and then start to pace again.

Areas for improvement

With any of these cardiac assist approaches that rely on skeletal muscle power, close attention needs to
be given to improvement of muscle graft function, to the biomechanical energy transfer, and to the effect of the electrical stimulation regime. Long-term muscle graft function may be improved by better muscle preparation (preoperative, e.g., voluntary or evoked exercising, passive and active stretch, pharmacological support) and perioperative handling (e.g., to prevent episodes of muscle ischemia). Muscle status and activation need to be monitored closely to evaluate the benefits of such preparations. To this end, research is being performed to monitor graft function by cine X-ray, CT/MRI scans, and echo-Doppler tissue imaging. The activation pattern should maximize power output while minimizing muscle energy expenditure (i.e., minimize fatigue). An ideal way to do this could be to customize training, but that will depend on availability of muscle sensors and feedback methods that are not yet developed. For cardiomyoplasty, improving muscle energy transfer to the heart may require better heart coverage by the transplanted muscle, improved muscle-heart coupling, and improved tissue interface characteristics.

Assessment of the effects of these new protocols necessitates more objective documentation than the clinical assessments used in previous studies (i.e., more sophisticated methods such as pressure-volume investigations by conductance catheter techniques). Some aspects of the extrinsic mechanism of action are discussed above. In addition to continued study of that area, examination is also needed of the intrinsic mechanisms of myocardial recovery: decreasing wall stress, decreasing oxygen demand, cross-vascularization of muscle and myocardium. Finally, the major role played by arrhythmic events in the function of cardiac assist devices strongly suggests the combination of defibrillators with cardiomyostimulators.

DYNAMIC MYOPLASTY FOR FECAL AND URINARY INCONTINENCE

Clinical Problem of Incontinence and Where Dynamic Myoplasty Fits Into Treatment

Fecal incontinence is an underreported condition that affects 2.5 percent of the population (13). One-third of nursing home patients in Minnesota have fecal incontinence1. It is a huge problem in human terms with high direct economic costs and indirect costs that result from the unwillingness of people to leave their homes.

The anal sphincter mechanism consists of the internal sphincter (smooth muscle that provides 80 percent of resting sphincter pressure), the external sphincter (voluntary muscle), and the puborectalis muscle (voluntary muscle). In addition to adequate sphincter function, normal continence depends on mental function, stool volume and consistency, colonic transit, rectal distensibility, anorectal sensation, and anorectal reflexes. Thus, even if the sphincter itself is normal, other abnormalities can cause fecal incontinence.

Among people with a normal pelvic floor, causes of incontinence are diarrheal states, overflow due to impaction or neoplasm, and a wide range of neurologic conditions. For example, the peripheral neuropathy secondary to diabetes results in a 20 percent rate of incontinence among diabetes clinic patients (14). Among people with an abnormal pelvic floor, causes of incontinence include anorectal malformation, trauma, iatrogenic injury (particularly obstetrical injury), and injury to pudendal nerves (due to childbirth or habitual straining). A third group of patients becomes incontinent following anorectal resection for removal of malignancy. In this group, dynamic myoplasty may be done in conjunction with a perineal colostomy to approximate the function of the anus.

Development of Treatment

If there is sphincter injury, surgical repair is effective in restoring continence for solids in about 80 percent of patients (15). If the sphincter is intact, biofeedback has reduced episodes of incontinence by 90 percent in more than two-thirds of the patients treated (15). If these therapies are unsuccessful, colostomy is an effective option, but many patients will not accept it.

In the 1950s, surgeons developed a passive wrap of gracilis muscle around the anal canal (16,17). It failed because the patients were unable to maintain the necessary contraction of the muscle voluntarily and because the muscle fatigued. To treat urinary incontinence, urologists have used a silastic cuff implanted around the urethra and then inflated it from a reservoir of fluid. A similar device has been used for fecal incontinence with some success, but it is not commercially available. For these reasons, research has been undertaken to create neosphincters from skeletal muscle using mainly the gracilis and gluteus muscles.

The electrically stimulated, skeletal muscle neosphincter (ESMNS) was developed as a new option for
patients whose conditions were not amenable to standard available therapy (Figure 6). The ESMNS procedure adds to the passive muscle wrap a neural or intramuscular electrode that stimulates the muscle (18,19). Conditioning with electrical stimulation increases the percentage of type I fibers in the muscle, improving its fatigue resistance. When conditioned, the muscle is continuously stimulated to achieve continence. When the person wishes to defecate, he or she uses a magnet to turn the stimulator off, and then back on afterward.

**Preparation before Muscle Transfer**

In animal experiments with transfer of the sartorius muscle to create a neosphincter stimulated by intramuscular electrodes, the London group (NS Williams) measured flow through a loop of intestine surrounded by the neosphincter. They attached strain gauges to the neosphincter to measure its contraction. After 6 weeks, they observed that the fusion frequency could be significantly reduced, suggesting fiber type conversion. They confirmed this on histology but also observed ischemic damage and considerable reduction in the diameter of type I fibers (20).

Upon observation of distal ischemia of the mobilized gracilis muscle in humans, the London group conducted radio contrast studies of the muscle’s vascular supply in cadavers. These studies showed that the blood supply was not entirely segmental, prompting the colorectal surgeons to do a delay maneuver, similar to that used by plastic surgeons. About one month before muscle transfer, the London surgeons divided the distal blood supply to the gracilis muscle. This has eliminated a greater than 50 percent incidence of ischemic necrosis and perineal sepsis. The Minnesota group (RD Madoff) and the Maastricht (Netherlands) group do not believe the delay maneuver is necessary and therefore do not use it (21).

**Surgical Procedure and Muscle Conditioning**

Dynamic myoplasty for fecal incontinence uses the gracilis muscle because it is easily transposable and not essential to lower limb function. It is a long, thin, ribbon-like muscle whose nerve and blood supply enter proximally. The muscle is mobilized up to that point. Through multiple incisions, the muscle is wrapped around the anal canal and its tendon is secured to the ischial tuberosity. There are two methods of stimulating the muscle. The London group places the electrode on the main nerve to the gracilis at the point where the nerve comes off the obturator nerve and lies on the adductor brevis muscle. This location is well out of the way of contracting muscle, and is thus subject to little tension. In addition, it allows the whole muscle to contract and requires a low voltage to produce contraction.

In the alternative technique practiced by the Minnesota and Netherlands groups (22), the main nerve to the gracilis is identified but not mobilized. Two intramuscular stimulating electrodes (Medtronic Model 4300, Medtronic B.V., Kerkrade, The Netherlands) are woven into the muscle 4 cm apart at the level of the main nerve branches to provide complete muscle stimulation. The leads are tunneled subcutaneously to the lower abdomen where they are connected to an implantable pulse generator (Itrel® 7424, Medtronic, Inc., Minneapolis, MN). Like the London group’s technique, this method also provides muscle stimulation at low voltages that remain stable over the long term.

The treatment protocols also vary with the surgeons performing the procedure. At the Royal London Hospital, the protocol starts with a frequency of 9 Hz and an intermittent pattern of stimulation. Over a period of 8 weeks, the “off” period is gradually reduced until conversion from fast-twitch to slow-twitch muscle has occurred and the muscle is stimulated continually.
Various training regimens that reach the same endpoint seem to be equally good.

At the University of Minnesota, the protocol lasts 14 weeks. The muscle transfer and implant are done at the same time in patients with a pre-existing stoma; but the implant is delayed 6 weeks after the transfer in patients with no stoma in order to limit infection. Muscle training begins at 6 weeks in all patients and continues for 8 weeks, using a frequency of 25 Hz. The duty cycle starts with 0.1 s on and 1.2 s off for the first 2 weeks, and is incrementally shifted at 2-week intervals, reaching 1.0 s on and 0.5 s off in the final 2 weeks. After the training period, muscle is stimulated continuously, typically at 15 Hz.

Demographics and Outcome of ESMNS Treatment of Patients with Complete Fecal Incontinence

At the Royal London Hospital, the first series of procedures were done in patients with intact rectums and used an external stimulator and no vascular delay. Of 11 patients, 5 became continent and 6 procedures failed. In the second series, using internal stimulators and vascular delay, of 16 patients, 15 achieved continence and 1 failed. The overall success rate was 60 to 65 percent, which includes both category 1 and 2 continence (i.e., patients who are continent for liquid and solids: category 1; and solids only: category 2). As there has been a major insult to the sphincter, the continence can never be perfect. Average postoperative anal pressures, measured by manometry, were 60 cm H2O, which is the lower limit of normal.

Also in London, a second group of patients received total anorectal reconstruction following abdomino-perineal excision of malignancy. These patients are examples of the 100,000 colostomy patients in the United Kingdom, and five times that many in the United States, who find an abdominal stoma unsatisfactory and undesirable. In the procedure, the colon was mobilized and transposed 1 to 2 months later. Eight to 12 weeks later, the electrode, lead, and stimulator were implanted and conditioning was begun. The stoma was closed if satisfactory continence and defecation could be shown. Satisfactory anorectal function was achieved in 50 percent of 12 patients.

Konsten, et al. of the Maastricht group were able to restore normal continence with their technique in 65 percent of 26 patients. Biopsy showed an average fiber conversion from 46 percent type I fibers to 64 percent after electrical stimulation (23).

In a multicenter clinical evaluation of dynamic anal myoplasty, 165 patients have been enrolled so far; 90 suffered from idiopathic incontinence, 45 had perineal reconstruction after abdomino-perineal resection for low rectal cancer, and 30 had congenital defects. The preliminary data show that continence could be achieved in more than 75 percent of perineal resection patients, and in more than 60 percent of the incontinent patients. In a study involving 52 patients (most of whom took part in the multicenter study cited above), Baeten reported that 73 percent were fully continent after a median follow-up of 2.1 years (18). There was significant improvement in median time to defecation, and patients reported an improvement in quality of life.

At the University of Minnesota, seven patients with complete fecal incontinence were treated with dynamic graciloplasty and followed for 18 months. Six were female and the mean age of all seven was 45 years. Median duration of incontinence was 8 years (7 mos to 51 yrs). In three patients, the cause was obstetrical or surgical injury; in one patient, cauda equina syndrome; in one, trauma; and in two, imperforate anus. Among six evaluable patients, two became fully continent, three improved markedly, and one remained incontinent. Complications encountered were cellulitis (inflammation of cellular or connective tissue) in four, transient pain in two, and transient edema in one.

Factors that could be adjusted in the ESMNS procedure are the vascular delay, whether or not to create a stoma, the type of electrode and lead, choice of muscle, the wrap configuration, and the conditioning regimen. The effectiveness of vascular delay has been evaluated in a randomized animal study comparing: 1) muscle transposition and then a 6-week wait before the start of stimulation, 2) vascular delay followed by transposition and immediate stimulation, and 3) transposition and immediate stimulation. Preliminary data show that muscle damage occurs in 3), but methods 1) and 2) both minimize muscle damage and provide better functional outcomes (24).

Other Applications Under Investigation

The success of this method for treating fecal incontinence led some investigators to explore a similar approach to restoring urinary continence (25,26). Because this technique involves more complex phenomena which influence the efficacy of the muscle contraction (e.g., muscle wrap technique, location), several surgical
options are being explored (27). In other studies, dynamic myoplasty has been carried out to restore micturition by wrapping innervated segments of the rectus abdominis muscle around the bladder (28).

**Recommendations for Further Research**

The ESMNS represents a promising new therapy for patients with fecal incontinence whose conditions are not amenable to standard therapy. Needed are objective measures of quality of life (18,28) and economic costs of incontinence to show that this is a good therapy. On a technical level, several modifications would improve the device. If it were possible to include a sensory element in the device that would receive afferent information and act in a closed-loop fashion, the device would work in a way more analogous to the native sphincter. Such an artificial reflex activity could eliminate the need for an external magnet.

In the current and future applications of this method, issues of muscle choice and surgical method will need to be evaluated. Muscles other than gracilis (e.g., sartorius, gluteus) may be as good or better. Multiple muscles may produce better results than single muscles. The length of wrappable muscle could be increased if a vascular anastomosis were done. If a nerve anastomosis were done, the muscle could be transferred as a free flap. Arthroscopic techniques may simplify the surgical procedures.

Basic study of transferred muscle and fiber conversion will continue to be important to the refinement of dynamic myoplasty. The inherent force capacity of the transferred muscle is reduced by 30 percent when the tendon is cut (29). Force capacity is also lost when the fibers convert to type I (30). Better understanding of these phenomena is needed to realize the full potential of dynamic myoplasty.

**ACKNOWLEDGMENTS**

This paper arose from presentations at the Engineering Foundation Conference “Neural Prostheses: Motor Systems IV” on July 23–28, 1994 in Mt. Sterling, Ohio. The conference was supported by grants from the Whitaker Foundation, the Paralyzed Veterans of America Spinal Cord Research Foundation, the National Science Foundation, the University of Miami and the Bakken Research Centre. The Public Health Service participated in the support of this meeting under a grant from the National Institute of Child Health and Human Development of the National Center for Medical Rehabilitation and Research, and the National Institute of Neurological Disorders and Stroke. The development of this paper was supported by the Cleveland FES Center, Cleveland OH.

**REFERENCES**


