Editorial

Dynamic cardiomyoplasty: time to wrap it up?

Fifteen years ago Larry Stevenson, in his memorable lectures, pointed to the great sheet of muscle that is the latissimus dorsi, and proposed that this potential power source should be harnessed for the purpose of circulatory assistance, and put to much better use as a cardiac muscle in patients handicapped and dying of heart failure.[1] Salmons and others[2 3] had demonstrated that this fast twitch fatiguable muscle retained within its genome the potential to be transformed into a slow contracting, fatigue resistant phenotype, similar to cardiac muscle. The logic seemed inescapable. It seemed inevitable that this resource would be used routinely to rescue the many with shortened miserable lives because of end stage heart failure.[4] The combination of advances in muscle physiology, electronic engineering, and experimental surgery,[5–7] culminated in 1985 in the first successful use of stimulated skeletal muscle to aid the circulation[8] in a patient with a left ventricular defect following excision of a tumour. That patient has survived—unlike the operation that saved her life. What happened?

Four UK units embarked on well thought out and carefully planned collaborative trials of what became known as dynamic cardiomyoplasty. In this application the muscle was mobilised on an intact extrathoracic neurovascular pedicle, wrapped around the heart, and stimulated to contract in time with ventricular systole. None of the four units are currently performing this surgery. About 35 patients were operated on in the UK, of whom none from our unit are still alive. The experience was similarly dismal in the other units. Experience in the USA is similar with very little reported evidence of success. Of the many European centres originally performing this surgery, only one has published any encouraging data in the past four years,[9] with the results inviting the comment that the authors had learned from the experience of earlier trials to select fitter candidates for the surgery, and in particular those with only modest cardiomegaly. Animal and clinical experiments also explored using skeletal muscle ventricles, constructed to beat in series with the heart,[10 11] and procedures generating counterpulsation where muscle is wrapped around either the descending or ascending aorta (aortomyoplasty).[12 13]

Cardiomyoplasty was taken up with enthusiasm in Brazil, a country with many young patients with end stage left ventricular failure caused by Chagas' disease. The early results there were better than those achieved in Europe, a difference later attributed to the different nature of the disease being treated. In fact the progress of some patients in this subgroup was said to be spectacular.[14] Chagas' disease does not result in the thin walled ischaemic ventricle so commonly seen in cardiology practice in the developed world, but rather a dilated but well perfused and possibly recoverable myocardium. Interestingly, in spite of the encouraging reports that appeared in the surgical literature, there is no mention of cardiomyoplasty in the papers on Chagas' disease in the current edition of *Heart*.[15 16]

Several factors have contributed to the failure of what was seen to be a very promising advance. One is that a skeletal muscle works by shortening in a linear fashion, reducing its length and pulling. Once wrapped around the heart, it has to produce radial shortening to generate pressure, and 90% of the power expended to produce linear work is wasted.[17] Furthermore, it has to compress the ventricle during systole when wall tension is greatest and, like the ventricle, it has to rely on diastolic perfusion for its own blood supply. In addition, the application of the relevant Lames equations or even Laplace's law quickly demonstrates that the larger the volume of the wrapped chamber the less efficient is the muscle in producing ejection or even ventricular support within the timespan of the cardiac cycle. For all of these, and other, reasons cardiomyoplasty is grossly inefficient. Aortomyoplasty might achieve better results, working on the much smaller aortic diameter, and contracting in diastole to create counterpulsation.[18] Skeletal muscle ventricles, when properly designed and configured, should also be capable of overcoming the fundamental inefficiencies of cardiomyoplasty.[19] The use of linear actuated artificial pumps powered by more remote, and more powerful, muscles than latissimus dorsi may now be a distinct possibility.[20]

The muscle transformation process, achieved by current pacing protocols, has also been a disappointment. They result in muscle with a power output too low to deliver useful work in the circulation.[21 22] An additional problem is the inefficient use of the muscle bulk available for work, with the more powerful, better perfused proximal portion not even reaching the heart.

The problems that originally concerned clinicians remain. There are two operational problems: the period of eight weeks needed to train the muscle, during which it is being paced but is not yet ready for work (better protocols may help to overcome this problem); and the so called vascular delay, which is the time between the mobilisation and recovery of the muscle, designed to allow some recovery of the blood supply to the muscle's extremities from the vascular pedicle at the proximal end. Furthermore the muscle actually wrapped around the heart is relatively ischaemic and may even tamponade the failing heart. Candidates who are truly end stage may not survive this initial two months, including one or more operations.

The original concept that we all have a large muscle which can readily be relieved of its current duties to perform the more vital task of powering the circulation has not lost its appeal. For the time being however, it has been a disappointment and much more radical and perhaps lateral thinking is required.

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