Hunterian Lecture

The nutrition of active muscle flaps

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In the last 25 years, techniques utilising muscle flaps to perform mechanical work have become part of the reconstructive surgeon's repertoire. In this paper, these techniques and the particular physiological challenges faced by active flaps are discussed and a sub-classification of muscle flaps into passive and active is proposed. A series of experiments are described that were designed to compare local pO_2 and gaseous perfusion, at multiple sites in pedicled tibialis anterior muscle flaps and controls, using specially developed dual amperometric microelectrodes, in an *in vivo* rabbit model. No significant difference in perfusion was found comparing flaps and controls until the flap's vascular pedicles were divided and ligated, and then flap perfusion fell to zero. Flap and control oxygenation varied significantly only during mobilisation and again, after pedicle division and ligation ($F[13,55] = 12.3$; $P < 0.001$). This absence of functional ischaemia as a consequence of mobilisation in the model used suggests the practice of delaying electrical conditioning of muscle flaps in dynamic cardiomyoplasty and neoanal sphincteroplasty might be unnecessary if these data are reproduced in human latissimus dorsi muscle. The relevance of these findings to free flap oxygen consumption and to the delay phenomenon is discussed.

Key words: Nutrition – Active muscle flaps – Local pO_2 – Gaseous perfusion

What are active muscle flaps?

Muscle flaps can be subdivided according to their blood supply;¹ additional tissues, transfer methods and donor sites. Taylor distinguished the patching of defects with muscle, from the provision of functional repairs.² Operations using native or conditioned muscle flaps to perform work, necessitate formalising this subdivision. The term functional muscle transfer is already used otherwise; therefore, I suggested a sub-classification using the terms 'passive' (classical, defect-filling) and 'active' (work-performing) flaps.³

A muscle flap used in a surgical reconstruction not only to fill a defect but also to perform mechanical work is

an 'active muscle flap'. Current surgical techniques using active muscle flaps in reconstruction include: facial re-animation;⁴⁻⁷ limb reconstruction;^{8,9} neoanal sphincteroplasty;^{10,11} and dynamic cardiomyoplasty.¹² Cardiomyoplasty and sphincteroplasty take active flap reconstruction to a supraphysiological level because they involve conditioning flap muscle electrically to alter fibre phenotype, producing fatigue resistance and the capability of continuous contraction.^{13,14} Flap surgery was limited to static restoration of structure and contour for 2500 years; in 25 years, active flap surgery has made dynamic restoration of function a reality.

Operations using conditioned muscle have accelerated into clinical use at a pace dictated by need and surgical capability, but before investigation of the altered

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physiology that electrical conditioning produces had begun. Initially, animal model failures provoked the inference that 'relative hypoxia' was produced in the flap periphery for 2–3 weeks postoperatively, necessitating a 2-week rest between surgery and commencing electrical conditioning.¹⁵ This practice continued in human cardiomyoplasty, meaning two extra weeks before patients could benefit from their operations. Cardiomyoplasty is limited in its suitability: patients must be ill enough to need heart transplantation, yet well enough to withstand prolonged surgery, then 2 weeks 'flap acclimatisation' before a further 6 weeks of conditioning; so there is a narrow window of opportunity to use it, which might be widened were acclimatisation unnecessary, or some alternative strategy available.

Raw surfaces of viable muscle flaps bleed, so it is reasonable to infer blood and nutrients reach throughout. Alternative causes for flap oxygen insufficiency during the initial phases of conditioning can be considered from available data such as different muscle vasculature in human and animal models and various physiological

insults that result from surgery – especially the demands of converting from glycolytic to oxidative metabolism during conditioning. This paper describes the use of previously developed and validated electrodes¹⁶ to measure muscle oxygenation and gaseous nutrition directly within muscle mobilised both as passive and active flaps to define stimulation protocols which would optimise flap conditioning.

Measuring oxygen and perfusion in vivo

Tissue pO_2 in isolation represents the balance between supply and utilisation. For a particular volume of muscle cells, oxygenation depends on the proximity of oxygenated blood and the diffusibility of oxygen between vessels and tissue. In normal physiology, oxygen delivery approximates to capillary blood flow, but in avascular or ischaemic tissues, the diffusion of oxygen through surrounding tissues, as well as from venous blood and lymph channels, assumes a much greater significance (Fig. 1). In such circumstances, blood flow alone is misleading and a complete assessment of oxygen nutrition can only be achieved by simultaneously measuring pO_2 and diffusive gas exchange (gaseous perfusion) at the same position.

Various techniques exist that can measure pO_2 in living tissue, but each has limitations and none allows simultaneous, continuous measurement of gaseous perfusion at the same. Ultimately, achieving the spatial and temporal resolution necessary in pO_2 and perfusion measurement necessitated miniaturising and validating amperometric electrodes in resting¹⁶ and contracting muscle.¹⁷ Three points about this method are notable: first, it quantifies tissue gas exchange via the wash-in rate of gaseous H_2 , a highly diffusible tracer, rather than a particulate tracer quantifying blood flow. Therefore, it allows the characterisation of pathological oxygen dynamics, especially during ischaemia, when blood flow and nutrient oxygen exchange are unequal.

Second, these microelectrodes measure with a spatial resolution of at least three orders of magnitude better than pulse oximetry, laser Doppler or near infra-red spectroscopy (*i.e.* micrometres rather than millimetres). This is important because it is the physiologically relevant length scale for cells – measuring oxygenation and perfusion within a hemisphere of tissue of volume 0.035 mm^3 which is typically 8 muscle cells in diameter. Tissue oxygenation and perfusion measurement has not previously achieved this degree of precision: compared with the previous best, it is equivalent to being able to see people around the base of Nelson's Column and measure their walking speed, rather than getting a gross measure of the population in 36 km^2 of central London, but still not know if they are distributed evenly in side streets or marching up Whitehall together – see Figure 2.

Figure 1 Physiological and pathological oxygen metabolism in muscle. Under ischaemic conditions, blood supply and oxygen delivery do not equate, concentration gradients alter and unusual diffusive supply occurs.

Figure 2 Three orders of magnitude. The spatial resolution achieved in measurements made using dual amperometric electrodes when compared with other methods is equivalent to the difference in visual resolution when viewing the detailed aerial photograph of Trafalgar Square compared with the 36 km² of central London. (Copyright Simmons Aerofilms, The Astrolabe Cheddar Business Park, BS27 3EB, UK; reproduced with kind permission.)

Third, blinded histopathological assessment of muscle sections from muscles into which electrodes had been inserted and controls revealed minimal tissue damage from electrode placement, even within muscle contracting strenuously for several hours. Indeed, one cell away from the needle track, and comfortably into the zone from which diffusing pO_2 and perfusion are sampled cell architecture was normal and 'control' and 'electrode' muscles were indistinguishable (Fig. 3).¹⁶

Materials and Methods

Electrode construction and validation and the apparatus used in these experiments is detailed elsewhere.^{16,17}

Surgery

Figure 4 is a schematic diagram of these experiments. All experiments were carried out in strict compliance with the *Animals (Scientific Procedures) Act 1986*. Male (4.3 ± 0.3 kg) New Zealand White rabbits were studied. Rabbit tibialis anterior muscle has been used widely as a model in previous conditioning experiments.¹⁸⁻²² Anaesthesia was induced and maintained,¹⁶ operation sites were shaved, disinfected and rabbits were positioned on thermostatically controlled heating pads and draped for surgical access. Pulse oximetry and oesophageal temperature probes were positioned and then each rabbit underwent tracheal intubation and ventilation with air at a minute volume of 1–1.2 l. Hydrogen boluses for tissue perfusion measurements were delivered at the tracheal inlet to minimise residual gases and re-circulation in the anaesthetic circuit.

Four dual electrodes were placed in tibialis anterior muscle under direct vision using surgical loupes and secured in place relative to the muscle by attaching them to the distal tibialis anterior tendon. Reference and counter

Figure 3 Transverse section of a tibialis anterior muscle stained with haematoxylin and eosin. A transverse section of tibialis anterior muscle stained with haematoxylin and eosin at $\times 100$ magnification. The track of the electrode in the centre of the field is surrounded by minimal cell damage. There is slight distortion and extravasation of blood cells around the electrode defect. (Reproduced from Greenbaum AR *et al.* *J Musc Res Cell Motil* 1997; 18: 154 with kind permission from Kluwer Academic Publishers.)

electrodes were positioned in the ipsilateral groin through a transverse incision. The preparation was maintained hydrated by covering incision sites with surgical gauze soaked in warm, physiological saline. Each procedure was terminated with intravenous pentobarbitone.

Results

Baseline tissue measurements

Baseline measurements of intramuscular oxygen and perfusion were made in unmobilised tibialis anterior muscle

Figure 4 Schematic of experimental apparatus and surgery

at rest in 12 rabbits. These measurements confirmed a striking heterogeneity of both pO_2 and perfusion within resting muscle as hypothesised previously, but never demonstrated. Muscle perfusion and oxygenation were not correlated, nor was there correlation between either parameter and fibre type. The actual values of resting skeletal muscle pO_2 and perfusion have been discussed in detail elsewhere.^{16,17}

Mobilised muscle flaps

Pedicated tibialis anterior muscle flaps were raised in six rabbits and compared with unmobilised muscles in seven weight-matched controls.

Steady-state baseline conditions were established and measured for an hour before mobilising the flaps carefully without disturbing the electrodes' positions. Serial measurements of oxygenation and perfusion were made at four points throughout the muscle bed every 10 min for 180 min. Finally, flap pedicles were divided (the point labelled 'devascularisation' in Figs 5 and 6) and measurements were continued for 30 min thereafter. In the control group, measurements of intramuscular oxygenation and perfusion were made in the tibialis anterior muscle every 10–15 min over the same time period.

Statistical analysis

Perfusion and pO_2 data had positively skewed distributions and required a $\log_e$ transformation in order to produce an adequate approximation to a normal distribution before statistical analysis. Comparisons between flaps and controls of baseline pO_2 and perfusion were made using analysis of variance adjusting for multiple observations (readings from each electrode) per animal.

Comparisons between flaps and controls over the time period spanning 30, 60, 90 and 120 min were carried out by repeated measures of analysis of variance, again adjusting for multiple observations. These comparisons were made using the $\log_e$ -transformed percentage changes from baseline.

Summary statistics are presented as geometric means (detransformed $\log_e$ means) adjusting for occasional missing observations using an imputation method.

Findings

Figures 5 and 6 show comparisons between flaps and controls for intramuscular pO_2 and perfusion. Note that pedicle division (devascularisation) occurred only in flaps.

The value of either parameter at $t = 0$ (before flap mobilisation) was taken as a common point of reference (100%). In controls, measurements started at the same time (using the time ventilation commenced as the common reference between groups).

Figure 5 Variation in pO_2 as a percentage of original value in flaps and controls with time. A baseline measurement was always taken before mobilisation of the muscle as a flap and was taken to be the 100% value, as was the equivalently timed measurement in control muscles. This figure shows geometric means and ranges.

Figure 6 Variation in perfusion as a percentage of original value in flaps and controls with time. A baseline measurement was always taken before mobilisation of the muscle as a flap and was taken to be the 100% value, as was the equivalently-timed measurement in control muscles. This figure shows geometric means and ranges.

Starting values: no significant difference existed between flaps and controls at $t = 0$ for either pO_2 ($F[1,11] = 0.06$; $P = 0.80$) or perfusion pO_2 ($F[1,11] = 2.42$; $P = 0.15$).

pO_2 trend: no significant difference in percentage pO_2 existed between the two groups at $t = 30, 60, 90$ and 120 min, ($F[1,11] = 0.086$; $P = 0.77$). Although there was a significant overall decrease in percentage pO_2 with time ($F[3,22] = 14.96$; $P < 0.001$), there was no significant difference between controls and flaps in this downward trend ($F[3,22] = 0.24$; $P = 0.87$).

Perfusion trend: no significant difference in percentage perfusion existed between the two groups at $t = 30, 60, 90$ and 120 min, ($F[1,11] = 1.78; P = 0.21$). Neither was there a significant overall change in percentage perfusion over time ($F[3,22] = 1.87; P = 0.16$), nor did the controls or flaps differ significantly in the trend of their percentage perfusion over time ($F[3,22] = 0.21; P = 0.89$).

Flap mobilisation and devascularisation: no significant changes occurred in control muscles in either percentage pO_2 ($F[9,27] = 1.45; P = 0.22$) or percentage perfusion ($F[9,27] = 1.13; P = 0.38$) at any stage. In flaps, significant changes in percentage oxygenation occurred during mobilisation and devascularisation ($F[13,55] = 12.3; P < 0.001$). No significant change occurred in percentage perfusion during mobilisation ($F[12,50] = 1.57; P = 0.13$) but after pedicle division perfusion fell to zero.

In control muscles, the range in values of pO_2 across the tissue bed was between 10 and 80 mmHg, while perfusion ranged from 1 to 12 ml/s/100 g. In muscle flaps, however, the range in values of pO_2 across the tissue bed was between 46 and 65 mmHg. Similarly, for perfusion the range in values narrowed to between 2 and 7 ml/s/100 g.

Flap mobilisation had no significant effect on intramuscular oxygenation.

Although not pursued in this study, the changes in muscle oxygenation following pedicle ligation suggest future studies quantifying free flap oxygenation and oxygen utilisation during cold ischaemia and after reperfusion.

Oxygenation and perfusion of muscle subjected to different patterns of electrical stimulation

Chronic electrical stimulation causes muscle damage ranging from a few percentage of fibres in muscles stimulated *in situ*,²³ to more than 50% of fibres in muscles used for cardiomyoplasty.^{24,25}

The mechanisms responsible for these changes are unclear, but damage is progressively lessened as the stimulation frequency is decreased between 10 Hz and 2.5 Hz. These experiments, performed in collaboration with Salmons' group,¹⁷ investigated whether the mechanism of this stimulation-induced damage is nutrition related. If muscle damage is a consequence of inadequate oxygen nutrition, a similar gradation in muscle oxygenation and perfusion should be evident during stimulation at 10 Hz, 5 Hz and 2.5 Hz.

Continuous stimulation at different frequencies

The effects of stimulation at 10 Hz, 5 Hz and 2.5 Hz were investigated in the tibialis anterior muscles in six rabbits, the three patterns being presented in random order. Oxygen concentration and perfusion were measured till resting steady-state ($t = 0$ min), at which point muscles

were stimulated continuously for 30 min with pulses of 200 μ s duration at the chosen frequency. Measurements were made every 5 min both during stimulation and for 20 min after stimulation had ceased. The muscle was left to recover for a further 20 min before the next stimulation regimen was applied and so on.

Effect of frequency on perfusion

For all three frequencies, there was a significant decline in perfusion, beginning soon after the onset of stimulation and stabilising at 50% of resting levels. On cessation of stimulation, perfusion recovered rapidly to original resting levels.

Effect of frequency on oxygenation

The difference in pO_2 before contraction began and at any time when a measurement was made, was taken as a measure of the change in tissue oxygenation resulting from the contractile activity of the muscle – the ΔO_2 . A negative ΔO_2 implies less oxygen available to tissue electrodes than before contractions started, suggesting either increased oxygen extraction from the extracellular compartment (possibly because cellular metabolic demands increased), or decreased oxygen supply to the extracellular compartment. The opposite would be true of a positive ΔO_2 .

All three stimulation frequencies produced a rise followed by a fall in ΔO_2 . The maximum ΔO_2 reached was higher, and the subsequent rate of its decline more rapid, for 10 Hz than for either 5 Hz or 2.5 Hz.

Stimulation with increasing duty cycle

Conditioning for both cardiomyoplasty and sphincteroplasty involves delivery of intermittent, high-frequency bursts of stimulation. Since elevated intramuscular pressure during tetanic contractions may be sufficient to obstruct capillary blood flow, tissue nutrition would be expected to be a function of the proportion of the cycle for which the muscle is active and contracting – its 'duty cycle'.

In a second series of experiments, pO_2 and perfusion were measured during intermittent, high-frequency stimulation at duty cycles between 1.3% and 20%. Tetanic contractions of tibialis anterior muscles were elicited with pulse trains of 20 pulses, each of 200 μ s duration, delivered at a frequency of 100 Hz. The interval between pulse trains was varied to generate duty cycles ranging from 1.3% to 20.0% and these patterns were delivered in a random sequence in each of a group of eight rabbits. Muscles were stimulated for 6 min and intramuscular oxygenation and perfusion were measured every 3 min, and for a further 6 min after stimulation had stopped. The muscle was left to recover for 30 min before the next stimulation pattern was applied and so on.

Figure 7 captures the relationship between mean intramuscular perfusion and ΔO_2 during stimulation at different duty cycles. The values are those measured at 6 min (before stimulation was stopped and recovery started). Up until 5%, increases in duty cycle were accompanied by increases in perfusion, but at duty cycles higher than 5% there was no further increase in perfusion and ΔO_2 changed from negative to positive, meaning extra oxygen was not being utilised within cells.

Discussion and future trends

Multi-electrode amperometry

The microelectrode technique used in these studies provides new insight into tissue nutrition. Measurement of pO_2 assesses the availability of oxygen in the extracellular space. Measurement of changes in H_2 concentration resulting from inhalation of a bolus assesses respiratory gas delivery to the tissue. This is the first technique to combine the quantitative measurement of both pO_2 and perfusion simultaneously. Furthermore, it has been validated exhaustively and minimal tissue damage results from electrode placement, even within muscle contracting strenuously for several hours.

Subsequent miniaturisation and development enable simultaneous measurement of tissue pH and lactate as well as pO_2 and perfusion and this process continues.

Measurements in muscles in situ and mobilised as flaps

Direct measurement of intraflap oxygenation and perfusion shows careful mobilisation has no effect on flap oxygen nutrition in this model. Recent studies in rat and pig latissimus dorsi models demonstrate peripheral muscle damage as a consequence distal ischaemia secondary to mobilisation, but less if muscles were pre-conditioned.^{26,27} The rabbit tibialis anterior model, though useful for electrical conditioning studies,^{18–22} is suboptimal in respect of its pattern of blood supply:^{12,28} ultimately, only human studies will provide reliable data.

The stimulated muscle experiments point to further development in active flap physiology and surgery by suggesting the optimum frequency for initial stimulation conditioning is 5 Hz and that there is no advantage to conditioning with a duty cycle of more than 5%. Again, human studies are needed now for further progress.

Finally, in flap mobilisation studies, measurements after division of flap pedicles provide qualitative insight into free flap oxygenation. Future studies during cold ischaemia and after reperfusion will further quantify free flap oxygen metabolism.

In this model, mobilisation as a pedicled flap decreased the range and variability of normally heterogeneous

Figure 7 Variation of mean perfusion and pO_2 with muscle duty cycle. Direct comparison of changes in mean perfusion with changes in oxygenation (expressed as the oxygen difference, ΔO_2) in rabbit tibialis anterior muscles subjected to stimulation patterns of different duty cycle. Error bars denote 95% confidence intervals. Ellipses: perfusion (ml/s/100 g); triangles: ΔO_2 (mmHg). (Reproduced from Greenbaum AR *et al. J Musc Res Cell Motil* 2000; 21: 289 with kind permission from Kluwer Academic Publishers.)

intramuscular pO_2 and perfusion. Oscillatory capillary flow is a well-characterised physiological phenomenon²⁹ and Taylor's group has demonstrated anatomical evidence for choke vessels which might equalise blood flow between angiosomes when oxygen supply is inadequate.³⁰ It may be that the smoothing effect noted in these experiments and illustrated in Figures 5 and 6 indicates a disturbance in the usual intramuscular vascular homeostatic mechanisms during flap mobilisation, so adding qualitative evidence for the role of choke vessels in the delay phenomenon.³¹ Again, further investigation in human muscles is required to support or refute this possibility.

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