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News

Firstly, my apologies for the late delivery of this newsletter. I have plenty of material (another 120 papers waiting to be reviewed) but unfortunately, not sufficient time. I have left out 6 papers that I wanted to include in this issue, and I will cram them into the next edition rather than delay publication any further. If you have any comments, suggestion or papers that I appear to have left out and you think should be included, please do let me know (t.watson@herts.ac.uk). Copies of the current and all past editions of the newsletter are available from the web site (www.electrotherapy.org)

News : Web Pages

I have added several items to the ever expanding web site (www.electrotherapy.org) and recent issues include some updates in the Ultrasound section on different Gels, updated references on US and Fracture Healing and an update with regards US for Apomorphine nodules (the trial that we were running).

News : Book Issues

Two items – one news and one a plea I guess! The Electrotherapy : Evidence Based Practice new text is just about to hit the shelf – in the next week I understand. It is available from Elsevier (www.elsevier.com), and there is a link with all the info that you need (ISBN etc etc) from the web pages (go to Modalities then Books, at the bottom of the page). Hopefully the new edition will serve a useful purpose with updated reviews in every chapter on what the evidence does, and does not say about key modalities plus some additional material not in the previous issue.

The plea – well, I am still trying to collect old editions of Electrotherapy textbooks from down the years. I am not asking you to give away your much loved and well thumbed copies of key texts, BUT if you have old editions of texts on the shelf and you are not using them, please do let me know. If you think that it would be too old for me to be interested in, then (a) you would be surprised and (b) e mail me! Funds are limited, but

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even so, completion of the collection is something I would like to do – in part because I am trying to construct some historical review work, and access to old texts would be a real bonus for this. Many thanks to those who have generously responded to the plea on the web front page already – appreciated.

News : Optimising Tissue Repair Courses

As several of you will know, I run a series of courses around the country on the current issues in tissue repair and how it might be influenced by manual therapy, exercise and electrotherapy. I am trying to be sensible and do a limited number of these around the country, rather than lots of courses, each for 20 people in every location possible! Anyway, I am running them through PhysioUK and there is a link to the information about them from the front page of the web site and also from the Courses page. The two remaining dates for this year are April (in London) and November (in Wales).

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Ultrasound – Power Outputs and Radiating Areas

There are two papers in this issue relating to ultrasound and radiating areas of equipment. The first, from a research team in the USA (***Straub, S. J. et al. (2008). Variability in effective radiating area at 1 MHz affects ultrasound treatment intensity. Phys Ther 88(1): 50-7; discussion 58-62.***) deals with an important issue that many of us do not take account of – that of variability (and in fact inaccuracy) of the ultrasound generator that we are using. Several researchers have explored this in the past including Pye et al in the mid 1990's in the UK. This particular study set out to measure the ERA (effective radiating area) of numerous ultrasound treatment heads (66 in fact) from several different manufacturers. All the tested treatment heads were 5cm sq sized and were checked against power output (SAI – or spatial average intensity – in watts per centimetre

squared) and ERA – square centimetres – comparing the ‘official’ values, readout on the machine and the actual values.

This could be an important issue – for example, if research evidence for a clinical effect is demonstrated at a particular dose, but when you apply that dose with your machine, you are delivering something different, it might make all the difference. Most therapists believe that when they set their machine to 1 W cm², that is what they are delivering. This might (or might not) be the case as there is a degree of freedom (for want of a kinder term) in the manufacturing process and an ‘allowed for’ range which means that your machine should deliver an output which is within X % of what you have set. There are several standards and guidelines around, and seeing that this is an International newsletter, I will not even try and go through the standards set for each country but the standard applied in this study was that of the FDA in the USA which allow for a 20% (either way) variation in power output and they do not specify a standard for the ERA other than that the manufacturer should state what the error is (usually around 20 or 25%).

Certainly the previously published evidence (nicely reviewed in the first part of this paper) demonstrates a variability which would surprise most practitioners (though it is not a secret). The Pye study – mentioned above – demonstrated that almost 70% of the tested machines had an output power that varied by MORE THAN 30% from the stated level. It has also been noted that the treatment heads that operate at more than one frequency are (generally) less reliable than frequency specific treatment heads (something that we found when we did the gel tests, published last year and reported in this newsletter). The Straub et al study was specifically looking at the values for 1MHz ultrasound which has not been as widely tested as the 3 MHz frequency.

The experimental method is detailed in the Straub paper, and I’ll not be going through it, but essentially a hydrophone and a wattmeter were used. The 66 transducers were purchased from several manufacturers (anonymously – i.e. the manufacturer was not aware of why they were being bought).

The results show that all the output power values fell within the FDA guidelines (though of course, this allows for a reasonable range in itself) but the ERA’s were very variable, with numerous of them falling outside the manufacturers stated errors (range). It would appear that some manufacturers were consistently better than others – you will have to go read the original to find out which ones were which – but remember that this was a study based in the USA, so the equipment tested was only that which was available in that country). If we take the two extreme values – might not be a scientific analysis, but I am trying to illustrate a point here – with the machine set at a nominal 1.0 W cm², the lowest actual reading was 0.57 and the highest at 1.61 W cm². This may or may not surprise you, but I reckon that this is a wider range than most practitioners would expect. Not only was there a significant variability between manufacturers, but even within – the intramanufacturer difference (up to 53%).

Given that the full data set and test method are described in the paper, I’ll not pursue these details any further, but the important point, and one that is well made from this work, is that the power level that you set on your treatment device may or may not be what you are actually delivering, and although all the transducers fell within the FDA 20% error range, the impact on the actual delivered dose can be quite considerable. Setting 1.0 W cm² but actually delivering as much as 1.6 or as little as 0.5 W cm² is, I reckon, a clinically significant issue. The authors argue, in the discussion, how this might make a difference to the anticipated heating effect. Point taken, but it would (?could) also make a difference to the ‘non thermal’ effect, and thus to the outcome of the treatment. The permitted ‘error’ of the power output AND the ERA when combined can generate variability of up to 150% from the machine read out and still be within the official regulations. The issue is an important one, and I would encourage you to not only look through this paper, but also to

read the commentary from Val Robertson and the author response. Worrying findings, but nice to get the discussion out in the open.

The second paper, from the same research team (though names in a different order!) appeared in Jan 2007 (*Johns, L. D. et al. (2007). Analysis of effective radiating area, power, intensity, and field characteristics of ultrasound transducers. Arch Phys Med Rehabil 88(1): 124-9.*) looking at different technologies to characterise ultrasound fields. The results, like the 2008 paper above, demonstrate considerable variation between different transducers (7 transducers were tested, but at 2 different frequencies). The authors also report an alternative technology to evaluate some of these critical parameters. All the transducer outputs fell within the permitted range, but the variation demonstrated, like in the Straub paper, could have serious clinical implications. One would hope that manufacturers would want to improve the quality of their machine output, but we all want low price equipment, and I guess that higher quality comes at a higher price (I am not into the commercial side of electrotherapy, but it sounds like a reasonable assumption). If manufacturers were forced to calibrate their machines to within, say 10% of the output on the machine, one assumes it would be more accurate, but more costly. The cost-benefit analysis has yet to be done (so far as I am aware), but there is food for thought. I could carry on, but will leave it there for your deliberations

US and Trigger Points

Lets move away from machines and technical stuff and wander off to the use of ultrasound with trigger points in trapezius – evaluated in a clinical trial which I know from e mails received, is what many of you appreciate the most. This paper from a group in Canada (*Srbely, J. Z. and J. P. Dickey (2007). Randomized controlled study of the antinociceptive effect of ultrasound on trigger point sensitivity: novel applications in myofascial therapy? Clin Rehabil 21(5): 411-7*) reports two different US treatments applied to trigger points in Trapezius in 44 patients. This was a relatively straightforward protocol – nothing wrong with that – and the subjects were divided into two groups. One received US at a ‘therapeutic dose (which was 1 MHz, continuous, 1 W cm², 5 minutes to an area 2 x the treatment head – given all the comments in the preceding section - and the ‘control’ group received a ‘nominal’ US treatment at 1 MHz, continuous, 0.1 W cm², 5 minutes, which was expected to have no effect – hence the control label.

Trigger points were identified in clinic patients and they were assessed by palpation and Pressure Pain Threshold (PPT) (the primary outcome measure) before and after the intervention. The recruitment of the patients was a little unclear from the paper – it just says that they were recruited from a pool of patients in a particular clinic. One assumes that they were not attending for specific shoulder rehab, but it is not explicit from the write up. Exclusion criteria were applied, and some 10 patients were excluded following screening. The intervention was a single application of the US (either treatment or control dose) and the PPT was recorded following a particular procedure immediately before and immediately after the 5 minute treatment session. Patients were instructed in PPT outcome reporting and were able to practice using points on the left side (all treatments were to the right trapezius). All OK so far. The statistical analysis was performed twice. The initial analysis included sex as a factor – there were equal numbers of males and females – but was not found to have a significant effect, and thus the results were pooled and the subsequent 2 way ANOVA looked at group allocation and the pre-post PPT levels. The ANOVA identified whether the difference was significant or not and t tests were used to identify where the differences occurred. Strange not to have used a Bonferroni (or similar) post hoc as a part of the ANOVA – they authors do not justify or rationalise this. The patients were blinded as to group allocation, but the therapist doing both the treatment and the assessment were one and the same – thus, single blind is the best that can be claimed.

The results were pretty clear in that there was a significant increase in PPT (i.e. needed to apply a significantly greater pressure in order to reach the equivalent pain levels) after the treatment US – the higher dose. There was no such significant increase in the control (low dose US) group. Both groups were statistically equivalent at the start point (pre US). The mean increase in PPT levels for the treatment group was 44%, though there was no follow up measurements made, and hence, it is not known how long this ‘analgesic’ effect lasted – could have been minutes or hours or who knows?

The discussion considers several interesting issues. The results are consistent with some previous work in this area, but not with others. It is suggested that in one of the previous studies, US was employed in combination with other interventions, which will have confounded the issue. I would agree that in this case, the application of US as the sole intervention will serve to clarify its effect.

The authors consider several mechanisms for the demonstrated effect, but given that they did not have any outcomes other than the PPT, it is not possible to say with any degree of confidence what the mechanism was, but interesting reading. Their conclusion was that US was effective in increasing the pressure pain threshold at a trigger point following a 5 minute application at a particular dose. There are other ways of decreasing trigger point sensitivity (acknowledged in the discussion), but that notwithstanding, this work identifies that US in isolation can have an effect in decreasing the sensitivity of these areas, and might therefore add something useful to the clinical environment. I have some (minor) methodological concerns – the test-retest reliability of the PPT method is alluded to but not really detailed. The source of the patients remains a mystery to me – I have gone back to that section several times, but still can fathom it out – have to guess. The single blind protocol was a shame and why not use a post hoc Bonferroni in the stats? BUT they have demonstrated a significant measurable effect in a clinical population (of undetermined nature) and this is an excellent start point. A comparison of US vs TENS vs Manual Therapy etc would make for a wonderful comparative study – anybody interested???

US and Growth Factors

There has been an increasing interest in the relationship between several therapy modalities and their interaction with various cytokines and growth factors. The fact that these growth factors appear to be critical to the process of repair, this is both a logical and fascinating area. We did not start to use US because we knew about its capacity to influence various and diverse cytokines, but it turns out that this is the case, and a recent paper by McBrier et al from the USA illustrates the point nicely (**McBrier, N. M. et al. (2007). *Therapeutic ultrasound decreases mechano-growth factor messenger ribonucleic acid expression after muscle contusion injury. Arch Phys Med Rehabil 88(7): 936-40.***

This was (predictably) an animal model experiment in a lab setting. Rats (36 of them) were exposed to a bilateral contusion injury to the gastrocnemius (like the Wilkin et al study from 2004). Some animals acted as a control group (n=4) and were not exposed to any injury. The remaining 32 animals were all subjected to a standard ‘drop weight’ contusion injury to both gastrocs, and in every case, the left limb was treated with the ultrasound and the right limb remained untreated. The applied US was at a ‘non thermal’ dose : 3.3MHz using a ‘small’ (1cm²) treatment head, at 0.3 W cm², continuous output, 5 minutes once daily for 4 consecutive days commencing 24 hours after the injury. The treated area was approximately 2 x the size of the treatment head.

A group of 8 animals were sacrificed on each of the 4 post injury days, and both left and right gastrocnemius muscles were excised and subjected to a series of test procedures. This enables the serial changes post trauma

ma to be tracked. The details of the outcome measures and procedures are to be found in the original paper for those who want to know.

The key factor in this study was MGF (Mechano-Growth Factor) which is in fact a local muscle variant of IGF (Insulin like growth factor) which is expressed in response to mechanical changes in the local environment. MGF increases muscle satellite cell activity and protein synthesis, and the potential role for MGF in early muscle repair following injury is succinctly outlined in the introduction to the paper. The researchers used the MGF mRNA marker as a measure of MGF activity.

Using a repeated measures MANOVA, the results (summarised) show that US had an effect on muscle regeneration. Essentially, the effect of the US was to significantly decrease the MGD mRNA expression, but did not have an effect on muscle mass. There was also an effect on the time base identified by post hoc analysis (details in the paper).

There is an interesting discussion following these results. If US is supposed to enhance tissue repair/regeneration, and the MGF levels were actually lower in the US group compared with the controls, there appears to be an inconsistency between the projected outcome and the actual results. The authors suggest a couple of hypothetical reasons why this might be the case (the US actually caused a down regulation of MGF and that the US stimulus was 'too strong' or that the peak effect was earlier than the first measures taken). It is possible (certainly looking at other papers in recent editions of Electrotherapy News) that this is a dose dependent response, and the fact that down regulation of the factor was observed in this instance identifies that US has an effect. Only one 'dose' was tested, and in keeping with other interventions, it is possible that one dose level may increase and another might decrease the same. It might also be possible, as the authors suggest, that they missed the rise (soon after treatment) and by the time they got to take their first measure, the peak had 'gone'.

I am not especially interested in making excuses for a 'negative' outcome – indeed, I would not consider this to be a negative outcome. It was clearly shown that US at this dose has a significant effect on MGF levels in injured muscle tissue. That in itself is important. Whether this is an up or a down regulation MIGHT be a dose dependency or it might relate to a time frame, both of which are questions that can not be answered by this study, and (as ever) further work is therefore needed. Interesting all the same.

Ultrasound Effect on Tumours

There is a continuing controversy with regards the potentially detrimental effects of ultrasound in malignancy and I get as many e mails a week about this as almost anything else. The current guidance (from CSP in the UK and from APA in Australia) that it is considered inappropriate to use therapeutic ultrasound over tissue that is, or is considered to be malignant. It has the potential to increase the rate of cell division – and hence the contraindication. This recent study from the USA (*Wood, A. K. et al. (2007). The antivasular action of physiotherapy ultrasound on a murine tumor: role of a microbubble contrast agent. Ultrasound Med Biol 33(12): 1901-10*) adds some very interesting new evidence to the existing pool of information. Research has previously demonstrated that therapeutic ultrasound applied at a reasonably high dose (but still within 'normal' range) can have an effect of reducing the vascularity of tumour tissue (reviewed and refer-

Seen any interesting papers?

Is there a paper that you have written and ought to be reviewed here?

E mail and let me know electronews@electrotherapyonline.co.uk

enced in the Wood et al paper). The vascular role in supporting tumour growth is well established, and therefore if US can reduce this unwanted angiogenic response, it might have a beneficial effect in reducing tumour growth.

Melanomas in a mouse model were exposed to US at 1.7 W cm² at 1MHz, continuous mode for 3 minutes in this work (lower dose than has been used previously in similar work). The mice (n=22) were injected with melanoma cells and after 2-3 weeks, once the tumour had reached sufficient size, it was isonated with the US (as above) though the 3 minute treatment was not applied in a single application, but rather 3 x 1 minute doses with a 5 minute interval between each. There were several groups of mice including a sham treated group and several different combination of US contrast agent and treatment times. Ultrasound contrast agent was delivered in 3 out of the 4 groups of mice prior to the US (treatment) application. Group 1 got the contrast agent but only sham US. Group 2 got the US but not the contrast agent. Groups 3 and 4 got both the contrast agent and the US, but with different time intervals between the two (15 minutes for group 3 and 60 minutes for group 4). Various vascular measures (using power Doppler) and histology were obtained and in essence the results show that the control group and the US group where no contrast agent had been applied showed no significant change. The 2 groups in which the microbubble contrast agent had been employed showed a significant reduction in tumour vascularity.

The authors argue that the mechanism for the demonstrated effect might be thermal in nature, but also hypothesise that microthermal, or 'non thermal' mechanisms might be responsible – including cavitation. The treatment of tumour tissue with ultrasound is specialist stuff, and although there are several research groups out there undertaking this in both the lab and clinical environments, I would not take it that treatment of tumours with therapy type ultrasound is something is being advocated. The point of this study is that ultrasound CAN have an effect on tumour vascularity – though on the back of the evidence presented here with mouse melanoma type tumours, the use of microbubble contrast agent is also necessary for the effect to be achieved.

US and Heat Shock Proteins

I have mentioned these before (several issues ago I think), but Ethne Nussbaum (who has published some excellent US and other material over the years) has recently published a paper (**Nussbaum, E. L. and M. Locke (2007). Heat shock protein expression in rat skeletal muscle after repeated applications of pulsed and continuous ultrasound. Arch Phys Med Rehabil 88(6): 785-90**) describing the results of some research looking at the effect of US on two of these proteins.

9 rats were treated with either continuous (1MHz: 1 W cm² SATA: 15 mins: 2 cm² treatment head) or pulsed US (1MHz: 2 W cm² SATP: 50% duty cycle (same as pulsed 1:1): 15 mins) on 4 consecutive days to the lower limb muscles in each rat. The muscle temperature and heat shock proteins were evaluated. One limb was treated in each case, with the untreated limb acting as the control. The treatment area was 2 x the size of the treatment head. Animal core temperature was recorded and maintained at a stable (37 C) level. The muscle temperature was measured directly during the final US treatment session. The muscles were excised, weighed and frozen immediately following the final intervention.

Heat shock proteins are considered to offer a degree of protection to cells against adverse physiological conditions. When cells are stressed, heat shock protein (HSP) production is induced (they are also sometimes called stress proteins) and they are thought to have several roles in the living cell, primarily related to protein production and breakdown. There is a brief summary of their actions in the introduction to this paper and several references that the interested reader can pursue. It was hypothesised that repeated US treat-

ments would alter the HSP levels and thus potentially contribute to the understanding of the mechanism of action of this modality.

As with the previous work by McBrier et al, there was no significant change in muscle mass when the US and the control muscles were compared. The core temperatures did not change significantly during or after the US treatments and any changes that were recorded were below those known to be responsible for activation of the stress responses in rat muscle tissues. The local muscle temperatures did increase (significantly) during the US session, but again these changes were not at a sufficient level to generate local heat stress responses.

The HSP levels in the treated muscles increased, but not equally in all muscles and not equally with the two different US interventions. The full details are clearly identified in the original paper, and I will not replicate the full data here. The pulsed mode US generated the greater HSP level changes. The use of pulsed and continuous modes at an equivalent SATA dose was to establish whether the response was a thermal or potentially non thermal response. The authors suggest that given the stability of the recorded muscle mass, and thus the absence of tissue swelling, the response to the US may have been a mechanical rather than a thermal stimulus to bring about HSP induction. The higher temporal peak intensity of the pulsed US is likely therefore to be a key factor in HSP production. The value of therapeutic US in terms of tissue repair may be, in part at least, related to HSP production, and clearly further investigation is needed. HSP's are another interest area for several forms of electrotherapy and it will be interesting to see how this one pans out with future research.

LIPUS and Distraction Callus

LIPUS seems to feature in every issue, and therefore, this one had better not be an exception – but this time, the report is less positive than most of those that I report – though I have always tried to be balanced in the material I include – and try to avoid cherry picking just the juiciest of papers that make electrotherapy sound totally fab! Anyway, on with this paper by Taylor et al from the USA (**Taylor, K. F. et al. (2007). *Low-intensity pulsed ultrasound does not enhance distraction callus in a rabbit model. Clin Orthop Relat Res 459: 237-45***). LIPUS has been previously reported to be effective for a whole range of clinical bone related problems, most noticeably fracture healing, both normal and delayed unions. This particular study was an animal (rabbit) model with a distraction osteogenesis procedure. LIPUS has been used in these lesions previously, but as the authors argue from the start, the optimal parameters have not been determined, and this was key to this study.

Given the conflicting evidence with regards the optimal time to start using LIPUS for distraction work, the authors, working from the other evidence from fracture healing studies, reasoned that early application – i.e. during the distraction phase itself – would be most likely to be effective.

Essentially 44 rabbits were divided into treatment and control groups. All were skeletally mature. A mid tibial shaft osteotomy was performed and then a distraction frame applied. Following a 7 day latent period, the progressive distraction was started at a rate of 0.5 mm every 12 hours for 10 days. The treatment group received LIPUS daily for 20 days whilst the control group were sham treated 20 minutes daily starting on the



first distraction day. Assessment was made with radiography, histology and mechanical testing. There is a detailed description of the procedures and the tests in the paper. The LIPUS was applied at the now familiar dose of 1.5MHz with an SATA on 30mW cm² pulsed for 20 minutes using the Exogen LIPUS system. Animals were sacrificed at either 27 or 37 days post op and both tibiae were excised and stripped of soft tissue. The test procedures are described in substantial detail, but the results (summary) pan out like this Of the original 44 animals, only 32 got through the process unscathed and thus competed the trial with no secondary problems (none of which were related to the LIPUS by the way). There was, at the end of the day, no clear advantage to the limbs from the LIPUS group compared with the controls on radiological, histo or mechanical testing. The authors present a long discussion which evaluates the differences between their results and previous studies that have demonstrated significant benefit for the LIPUS treated groups. It comes down to a methodological argument combining issues of the external fixation employed, the method of mechanical testing and associated issues. At the end of the day, this study did not demonstrate benefit. I would suggest that it puts it in a minority in this regard, certainly when evaluating the overall effects of LIPUS and bone repair. It may well turn out that the system is less effective with distraction osteogenesis than it is with routine fractures and delayed/non unions – but that remains to be seen.

Shock Wave Therapy and Delayed Bone Healing

Wang and colleagues have been mentioned numerous times in this newsletter, and shock wave therapy is something that also appears in just about every issue. I have been getting an increasing number of e mails recently asking about why shock wave is not included on the web pages – well, I am afraid that it is a matter of time – I have LOTS of material, just need time to put it together and then write the web stuff. It is on my 'to do' list I assure you! Anyway, in the meantime, this 2008 paper by Wang et al (**Wang, L. et al. (2008). Extracorporeal shock wave therapy in treatment of delayed bone-tendon healing. Am J Sports Med 36(2): 340-7**) does pretty much what it says on the tin – looks at delayed bone-tendon healing using a rabbit model, and the overall results are significant in favour of the shockwave. Shockwave has been fairly extensively researched in recent years, and it has been reasonably well established as an effective intervention for some chronic soft tissue problems and some bone healing conditions. In this instance, the authors set out to evaluate the effect at a delayed healing at the junction of the patella tendon and the patella.

I will outline the delayed healing model used, but the full details as ever are to be found in the original paper. The key to the procedure was to perform a partial patellectomy (distal 1/3 was removed, and the distal patellar tendon was sutured to the remaining patella BUT a slice of latex was inserted between the tendon and the proximal bone before immobilising for 4 weeks, at which point the latex was surgically removed. This model of delayed healing at the bone-tendon junction appears to be reliable in the rabbit at least! Animals (28 of them) were divided in to shock wave and control groups, and the shockwave was delivered at a single session 2 weeks after the cast and latex sheet were removed. The shockwave dose applied was one that has been previously reported to be effective (0.43 mJ mm²: 4Hz : 1500 impulses). There were numerous outcome measures including radiographic evaluation of the patella tendon complex and measurement of new bone formation, bone mineral status, mechanical testing, measurement of various sample dimensions, tensile strength of the sample through to failure. Histological tests included morphology, collagen fibre alignment, tenocyte density and thickness of the fibrocartilage zone. The mechanism for all tests and equipment used are detailed in the main paper.

The results : There was significantly more new bone formation in the treated tendons at both weeks 8 and 12. The bone mineral content was very substantially higher in the treatment group and the histology showed clear advantages over the no treatment group in terms of the tissue alignment and new bone formation. There were fewer tenocytes at 12 weeks in the treatment group and the fibrocartilage zone was significantly

thicker. Mechanically, the results were significantly better for the treatment group with higher ultimate strength. There are some VERY high percentage differences between groups amongst this data, and it certainly does not appear to be anything like a marginal benefit.

The authors address some interesting points in the discussion, which are there for the interested reader - It does not need me to repeat them. Essentially, having previously demonstrated that Shock Wave Therapy is effective for a variety of clinical soft tissue and orthopaedic problems, this study certainly seems to show significant benefit in a delayed bone-tendon healing model in animals. It would be critical of course to see if this translates into human study effects that are of this magnitude, but it would be impressive if there were! OK, time to move away from ultrasound and variations on that theme, and to have a look at a raft of electrical stimulation papers that I have found in the last couple of months. I came across 40 or more when I did a recent search, and I'll not attempt to get through them all in this issue, but we can have a look at a selection from the pile.

Acu point Electrical Stimulation and Chronic Tension Headache

This RCT design study from a group in Denmark have evaluated the efficacy of a real versus a sham electrical stimulation treatment at a combination several different acupuncture points using a 'pen' stimulator with patients presenting with chronic tension type headache. (**Wang, K. et al. (2007). *Effect of acupuncture-like electrical stimulation on chronic tension-type headache: a randomized, double-blinded, placebo-controlled trial. Clin J Pain 23(4): 316-22.***)

36 patients completed the trail (40 started it) divided equally into a treatment and a placebo (sham treatment) group. The intervention was applied by the patient following a training session. Six acupuncture points were employed for this purpose (bilateral EX-HN5 : GB20 : LI4) and each point was stimulated for 3 minutes, twice daily. The stimulator was a commercial device delivering a patterned stimulation, operating at 2 specific frequencies (2 and 100Hz) switching at 3 second intervals. The square wave stimulus was longer duration at the lower frequency (5ms) and shorter at the higher frequency (0.7ms) applied at 25V and the pen tip was 1mm diameter. The stimulation pattern, output parameters and acu points employed were based on previous research.

There were several key outcomes, based to a greater extent on a patient diary system. Pain duration, pain intensity, headache frequency and medication consumption were recorded for 2 weeks prior to the commencement of the trial – providing a baseline – and then throughout the treatment phase (4 weeks) and then at various follow up points, up to 6 weeks post intervention. A Danish version of the McGill Pain Questionnaire was also employed. Both the therapist and the patient were blinded as to group allocation.

There are a lot of results, but they can be summarised thus : All patients were experiencing a significant number of headaches (more than 16 days a month) and there was no difference between the groups at baseline in terms of age or any key headache related outcome. Both groups demonstrated a reduction in headache duration over the experimental phase, and although the treatment group results were 'better' there was no significant difference between the real and the sham groups. Similarly, the headache intensity and headache frequency reduced over the treatment period, and again, there was no significant difference between the treatment and sham groups. The medication consumption demonstrated a marked decrease in analgesic use for the treatment group, but not for the sham group, which although not statistically significant, did show a 34% difference between groups in mean consumption.

There are a couple of significant limitations to this study – which in fairness the authors have recognised and documented. The relatively small sample size is probably one of the most obvious. There have been other relatively similar studies in 2005 (Melchart et al, Coeytaux et al) with larger samples which would be worth looking at if you are interested in this field. The other important issue relates to the nature of the placebo stimulus. The sham group stimulator provided no current at all. Interestingly patients in this group perceived the same stimulus sensation as those in the real treatment group. The strong possibility that the pen application had a real effect via an acupuncture type stimulus means that the improvement in both groups may be related to the stimulation of the points and that the electrical stimulation and the mechanical stimulation might be ‘equally’ effective – the authors also point out that the fixed intensity of the applicator stimulus might have further confounded this issue. Those who ‘felt’ the stimulus appeared to get a better result, and a machine that delivers a ‘one dose suits all’ stimulus might not be the most effective way to use this clinically.

Anyway, it was a useful trial, and the electrostimulation of these acupuncture points was effective for these patients, as was the mechanical stimulation (the sham group). The real electrostimulation group appeared to get ‘better’ results, but the differences were not statistically significant. That does not negate the value of the intervention, and the use of a variable stimulus strength would appear (to me at least) to be something worth considering.

Lower limb sensory electrical stimulation and gait following stroke

Several of the electrical stimulation papers in the collection relate to stimulation work with neuro patients, and the first of these to be included in this issue (*Yavuzer, G. et al (2007). Effect of sensory-amplitude electric stimulation on motor recovery and gait kinematics after stroke: a randomized controlled study. Arch Phys Med Rehabil 88(6): 710-4*) describes, as one might anticipate, the effect on gait of a sensory level stimulation protocol, applied to the paretic leg of stroke patients.

In short, 30 stroke patients were divided into a treatment and a control group in this double blind RCT. Both patient groups were given ‘standard’ stroke rehab, and the only difference was that the treatment group received in addition sensory level electrical stim to the paretic lower limb daily (5 days a week) for 4 weeks whilst the other group received an equivalent sham stimulation.

The patients were all on their first stroke which was within the 6 months preceding the start of the study. The stimulation was applied for 30 minutes daily with electrodes over the common peroneal nerve and over the tibialis anterior (none of the patients were able to actively dorsiflex their ankle), with stimulation applied using an asymmetric biphasic rectangular pulses at 35Hz at a level sufficient to produce ‘mild tingling’ but not sufficient to bring on a muscle contraction. Clearly the actual intensity of current required to achieve this was different for patients.

The outcomes employed for this work were based around the Brunnstrom stages for lower extremity recovery (scaled 1 – 6) – described in detail in the paper – and several measures of gait kinematics measured with a Vikon system. Outcome measures were not different between the groups at commencement of the study and all patients completed the intervention period. There was a significant improvement in Brunnstrom score over time in both groups, but there was no significant difference between the real and the sham intervention (though there was a marginal difference in favour of the stimulation group). Similarly, there were improvements in gait scores, but similar in both groups.

These results are at odds with several other studies, where significant advantage has been shown in the treatment group, and the authors discuss why these differences might have occurred. Clearly the outcome of this RCT did not demonstrate that the addition of a 'real' stimulation provided any advantage over providing a sham stimulation (most of the other trials have compared stimulation with control – i.e. no stimulation), and there may be some issues relating to the sensory stimulation associated with electrode placement, even if not delivering a current – worth a further look.

Electrical Stimulation for improved hand function post stroke

A further stroke patient research programme is reported by Knutson et al based in Cleveland, USA. This work (*Knutson, J. S. et al. (2007). Improving hand function in stroke survivors: a pilot study of contralaterally controlled functional electric stimulation in chronic hemiplegia. Arch Phys Med Rehabil 88(4): 513-20*) was a small scale study (n=3) of post stroke patients (more than 6 months), using the electrical stimulation to stimulate the fingers extensors in the paretic hand for 2 hours daily (at home) plus some additional hospital based sessions (hour and a half, twice weekly), both over a 6 week period.

The stimulation was delivered using a novel system in which (briefly) the patient wears a special glove on the unaffected side, and when they 'open' the hand, the glove drives a stimulator which is attached to the intrinsic and extrinsic finger and thumb extensors on the affected side (with surface electrodes). The magnitude of the applied stimulation was proportional to the amount of unaffected hand opening. (details in the original). Outcomes were assessed at baseline, at the end of the treatment period and at 1 and 3 months follow up, and included active finger extension range, finger movement control, isometric finger extension test and a 'box and block' test which is a measure of manual dexterity (all described in the paper).

Improvements were noted in finger extension range by the end of the intervention period, though they had diminished by the 3 month follow up point. Similarly, other scores improved during the treatment phase, were largely maintained at 1 month follow up but appeared to decline by the 3 month post treatment point, though not back to baseline levels.

This was a small scale study with no control group and thus can not be as powerful in terms of results as a full RCT style piece of work BUT never the less, the results are encouraging and certainly warrant further investigation and evaluation.

NMES and arm function post stroke

Another case study style paper (*Hedman, L. D. et al. (2007). Neuromuscular electrical stimulation during task-oriented exercise improves arm function for an individual with proximal arm dysfunction after stroke. Am J Phys Med Rehabil 86(7): 592-6*) looks at the potential value of adding a shoulder and upper arm stimulation treatment to an upper limb rehab programme in one individual using a home based intervention. The particular patient in this study was more than 5 years post stroke at the time of the intervention, and in short, although she had some reasonable function of her lower arm, it was her upper arm control that was causing significant problems. Various outcomes were applied before, during and after the NMES intervention and also at a 6 week follow up. The outcomes included the Action Research Arm Test (ARAT) and the upper limb sections of the Fugl-Meyer Assessment, the Quality of Movement subscale of the Motor Activity Log together with various subjective data obtained from an interview with the patient.

The NMES activity was designed to work on the combination of shoulder flexion with elbow extension (to enable the existing wrist and hand function to be realised). A commercial stimulator, operating on 2 channels was used, one for deltoid and one for triceps, and the stimulation was symmetrical biphasic at 35Hz and a pulse duration of 300 microseconds. This stim was associated with particular functional tasks and activities (described in the paper) which the subject was instructed to perform for 3 x 15 minute sessions a day, 7 days a week over the 6 week intervention phase. The subject maintained a diary to see what the compliance was like, and in fact I don't think that it was too bad with 92 of the possible 126 sessions being completed. The ARAT score increased midway through and at the end of the intervention and these changes were maintained at follow up. The Fugl-Meyer scores did not change but the Quality of Movement data showed a perceived improvement in their ability to use the limb during the treatment period and this improved further at the follow up assessment. Contacting the patient at 1 year post intervention, it appears that the improvements were maintained and a much higher level of function was being used.

As with the previous study, I realise the potential limitations of a case study, but these reports are, in my opinion, useful in that they serve to inform areas where further work might be usefully pursued. For those involved in later stage stroke rehab, this would make an interesting read in its own right. For those considering research in this area, it would provide some insight into useful and possibly less useful areas to investigate.

FES and upper limb function post stroke

While on the subject, there was another useful paper along somewhat similar lines, though I did not report it in Electro News when it came out, but it would be worth mentioning here. **Hara, Y. et al. (2006). Hybrid power-assisted functional electrical stimulation to improve hemiparetic upper-extremity function. Am J Phys Med Rehabil 85(12): 977-85.** The relatively small scale study (16 patients, 14 completing) involved an interesting mix of electrical stimulation to the forearm extensors linked to an EMG system that picks up voluntary activity from the same muscles. The stimulator will not activate the muscles unless there is voluntary activity, and the stim is delivered in proportion to the EMG detected. The patients were also given a phenol injected block to specific forearm flexors before the first intervention session.

The stroke patients in this trial were all at least 1 year post stroke and the trial was of an RCT design, but not blinded. The treatment group received a standard treatment plus the FES whilst the control group received standard therapy only over the 4 month study period. The stim intervention was carried out for 40 minutes either once or twice a week

Outcomes included EMG parameters (mainly RMS), active range of motion, a Modified Ashworth Scale score and two clinical tests. The tests and protocols are all described in the original paper. 8 patients ended up completing the treatment group interventions. The EMG data (RMS) and active ROM results showed a significant increase in the treatment group following the intervention compared with the controls and the hand function tests gave markedly better results for the treatment compared with the control group, in whom there was no significant change.

The combination of an EMG integrated electrical stimulation system with a flexor muscle phenol block appears to have given rise to some significant changes in a chronic post stroke group. By using the phenol block to effectively inhibit the 'spastic' flexors whilst using the FES to promote activity in the extensors appears to pay off. There are some points of interest in the discussion, and as ever, I would strongly encourage you to take a look. Whilst it is appreciated that this involves the application of a particular and relatively sophisticated FES stimulation system with the integrated EMG, and also involves the use of median nerve

block with phenol – a combination that is unlikely to constitute ‘normal’ care for most involved in stroke rehab, it does provide interesting further avenues to follow up at some point in the future,

Functional Electrical Stimulation for the Arm Post Stroke

There seems to be a reasonable selection of papers along these lines this time, and this paper from a research group in Canada (Alberta) adds nicely to the group (*Kowalczewski, J. et al. (2007). Upper-extremity functional electric stimulation-assisted exercises on a workstation in the subacute phase of stroke recovery. Arch Phys Med Rehabil 88(7): 833-9).*

Nineteen patients with a mean post stroke period of almost 2 months were divided into low and high intensity FES groups. The work involved both FES and exercise at a workstation, with the high intensity group receiving 1 hour a day for 15-20 days and the low intensity group 15 minutes of sensory level stimulation for 15 minutes a day for 4 days and the high intensity stimulation on the 5th day for an hour. The stimulator was a custom device delivering stim at 50 pulses per seconds of 200 microsec duration, using a biphasic current. The surface electrodes were placed over the wrist and finger extensor muscles on the dorsal forearm, such that hand opening was achieved. The optimal parameters for each patient were established on an individual basis.

The intervention was for 3-4 weeks alongside their regular rehab programme. The activities used at the work station are carefully described in the paper, but effectively, the specialised workstation equipment enabled the quality and time of the activity to be monitored and recorded (reaching, grasping and manipulation tasks). The low intensity stimulation group were included as an effective control, with sensory rather than motor level stimulation for 4 days a week and higher level stim on the 5th day to enable comparison. There was an extensive raft of outcomes including clinical and objective measures. The primary clinical measure (assessed by a therapist blinded to group allocation) was the Wolf Motor Function Test – which looks at motor impairments during ADL type activities. In addition the Fugl-Meyer test was used (as in the previous study) and a Motor Activity Log. Assessments were made pre and post intervention plus at 3 and 6 month follow up. The workstation provided objective kinematic data which was normalised to healthy subjects (described in sufficient detail in the original paper).

The results show a significant difference in the Wolf Motor scores between the high (treatment) and low (control) intensity groups having started from an equivalent point. The significant differences, although identifiable at the 3 month post follow up were effectively lost by the 6 month assessment. The Fugl-Meyer and Motor Activity log scores were not significantly different between groups. The objective workstation scores were significantly better for the high intensity stim group, and this difference became apparent after 3 weeks of intervention and continued thereafter. There were no objective kinematic data for the 3 and 6 month follow up periods.

The outcome of this work illustrated an improvement in both groups, but that the high intensity group did significantly better on the Wolf assessments than the ‘control’ group. The authors provide some excellent

Seen any interesting papers?

Is there a paper that you have written and ought to be reviewed here?

E mail and let me know electronews@electrotherapyonline.co.uk

discussion, and include some comments with regards the difference between statistical and clinical significance.

Electrical Stimulation Types for Stimulated Cycling in SCI Patients

The basic aim of this work was to compare the effectiveness of two different forms of electrical stimulation for otherwise healthy patients with spinal cord injury during a cycling activity (*Szececi, J. et al. (2007). Low-frequency rectangular pulse is superior to middle frequency alternating current stimulation in cycling of people with spinal cord injury. Arch Phys Med Rehabil 88(3): 338-45*).

As has been argued previously in this newsletter, the application of a burst mode of 'medium' frequency stimulation has been claimed to be preferable in that it generates effective muscle stimulation with what appears to be lower levels of discomfort and also claimed differences in fatigue rate and MVC activation. In this study, the efficacy of burst mode 4kHz stimulation modulated at 50Hz was compared with low frequency stimulation at 20Hz at 500 microsec pulse duration, applied to glutei, quads and hamstrings using surface electrodes (details in the paper).

The stimulation was computer controlled such that the appropriate muscle groups were activated in phases appropriate to the crank angles on a cycle ergometer. The stimulation was delivered at between 40 and 80% MVC (patient dependent) and the stim phase was for 1.5 sec followed by 3 seconds rest. The cycle period was for 20 minutes, initially driven by a motor attached to the crank, and then with a progressively increasing electrical stimulation with resistance applied such as to maintain a cycle rate of between 35 and 55 rpm. The isometric torque produced with the low frequency stimulation was significantly greater than with the burst mode medium frequency current and the amount of work done during the session was also greater with the low frequency stimulation. There is more work to be done in this area I have no doubt. The fact that some previous studies have reported that patients generally find the medium frequency currents less irritating may not be the whole story (as ever with these things). The Shannahan et al study reported in a previous issue comparing TENS with IFT for experimental pain relief showed that the TENS had a 'stronger' effect, but that the subjects on the receiving end of the stim preferred the IFT. In this case, the low frequency stim was the more effective (certainly based on torque and work done) though this may not be an ideal group in whom to measure classic pain/discomfort – nice follow up study for somebody somewhere!

Electrical Stimulation and Growth Plate Activity

I get a LOT of e mails every week about whether electrotherapy (under several modalities) can or can not be applied to children, where to put the electrodes and whether it is a contraindication, a precaution or variations on that theme. There have been several studies with some modalities (ultrasound in particular) but there are certainly less published with direct reference to the effect of electrical stimulation. This recent paper from the USA (*Dodge, G. R. et al. (2007). Electrical stimulation of the growth plate: a potential approach to an epiphysiodesis. Bioelectromagnetics 28(6): 463-70*) takes an interesting approach, but one that will certainly add to the continuing debates in the field.

The technique of epiphysiodesis is one used to stop the longitudinal growth of a bone using a surgical intervention. The aim of this work was to see if (in a rabbit model), the effect could be achieved with electrical stimulation as an alternative approach. Rabbits were divided into control, low intensity and high intensity stimulation groups using indwelling electrodes in the distal femur, and the stim was a DC current at 10 microamps (low stim) or 50 microamps (high stim) attached via a commercial bone stimulator for a 2 week period. Given that the main aim of this work was to see if it was possible to deliberately arrest the growth

activity at the epiphysis, the stimulation was clearly for a longer duration than might be applied in the 'typical therapy' setting, but it is whether there is a growth arrest capacity or not that is particular interest for most practitioners.

One leg (right) of each rabbit was used as a control limb thus enabling a differentiation to be achieved whilst allowing for individual differences. Various bone lengths were taken following animal sacrifice (@ 2 weeks) together with AP and Lateral radiographs, followed by several histopathology tests which effectively evaluated to dimensions of the growth plate.

The results showed a significant increase in bone formation in the high current group compared with the low current and controls, and the high current group had a significant retardation in growth (more than 200% compared with the controls and low current groups). The radiographs demonstrated new bone growth in the area of the indwelling electrode in the high current group, a small amount in the low current and no new bone in the controls. The high current group histology showed marked changes (new bony bridge formation and distortion of the epiphysis) and these were not seen in the low current and the controls.

There are a lot more detailed results in the paper, but it is important to note several things. Firstly, this paper DID NOT set out to see if electrical stimulation was detrimental to the epiphysis and therefore should or should not be on some contraindication list. The aim of the work was to evaluate the potential (in an animal model) to deliberately retard longitudinal growth by using electrical stimulation, thus potentially enabling the avoidance of open surgery in the future (which was, in effect, what they demonstrated). Whilst this has important implications in paediatric orthopaedic practice, it MIGHT have implications in therapy practice. The use of various forms of electrical stimulation in children is frequently questioned, with little by way of definitive answers available. The higher of the applied currents was responsible for the strongest effects shown here. The currents were applied for 2 weeks on a continuous basis (not the occasional 10 minutes here and there) and electrodes were inserted into the bone – not exactly a close replication of any standard electrotherapy type application. HOWEVER, the fact that an inhibitory effect was demonstrated at the epiphysis means that it is possible, even if not likely in the therapy world. The use of DC currents was deliberate in that they are the most effective for stimulation of growth and healing responses. This is not evidence that TENS, Interferential and the like are going to inhibit epiphyseal plate growth following an occasional treatment, but never the less, I would think that it is a reasonable contention that electrical stimulation (therapy) in the region of an active growth plate would not be the best idea, and at least should be a strong precaution even if not an absolute contraindication. It is not for me to say what is and what is not a contraindication for treatment A, B or C, but if I was in practice with a younger age patient group, I would want to be mindful of the potentially detrimental effect I could be having. More on this in future I have no doubt, but would be interested in the views of others out there (e mail : tim@electrotherapyonline.co.uk)

Electromagnetics and Nerve Crush Injury

Time to move away from the electrical stimulation modalities and onto a single paper on electromagnetics for this edition. Walker et al (*Walker, J. L. et al. (2007). Electromagnetic field treatment of nerve crush injury in a rat model: effect of signal configuration on functional recovery. Bioelectromagnetics 28(4): 256-63.*) This study does not use a classical shortwave/pulsed shortwave application, but may be of interest for those of you operating in the PEMF arena. The use of pulsed electromagnetic fields has been previously shown to be effective for enhancing the repair response and regenerative rate in various animal models. This work, with rats, evaluated the effect of a sham and three different strength pulsed electromagnetic fields on the sciatic nerve following a crush injury. The exposure to the field was whole body rather than just a local segment (easier with a rat than with a human!) and treatment (at whatever dose) was applied for 4 hours a day

for 5 days (starting on post op day 1) with a solenoid based coil system which generated an induced magnetic field in the animals of 0; 0.03mT; 0.3mT; and 3mT depending on the applied dose for the groups. Outcome assessment was by walking function and a specific 'toe spread' test and measures were taken over a 43 day period. The toe spread test was used (previously published) as this function is affected by both the tibial and peroneal divisions of the sciatic nerve). There were therefore 4 groups (sham plus three treatment groups at different 'doses') and by the end of the work, there were 17 animals in each group.

The results show that there was a good recovery over the assessment period, but that there was no significant difference between groups – all three treatment groups did as well as each other and there was no difference between any of the treatment groups and the sham treatment group.

The authors include a detailed and interesting discussion, mainly focussed around why previous results were showing a definite response in favour of the PEMF groups and this study clearly did not. There certainly appears to be a difference between the 'effective' dose with the in vivo and in vitro work (on which the doses were based), and it is suggested that the effect of PEMF signals, primarily operating through a calcium ion mechanism (the Ca/CaM pathway) in vitro might occur at lower energy levels than in the living animal model. The experimental induction of an axotomy type sciatic injury in a whole animal experimental model might require a different energy for therapeutic benefit compared with an equivalent in vitro model. The discussion includes an interesting mathematical analysis of this proposal if you happen to be interested.

Cryotherapy and Nerve Conduction

Applying cold, in various forms following soft tissue injury, surgery and other painful conditions remains popular in the field of therapy, and I have previously reported several studies which have tried to determine the mechanism and the efficacy of this type of intervention. This study (**Algafly, A. A. and K. P. George (2007). "The effect of cryotherapy on nerve conduction velocity, pain threshold and pain tolerance." Br J Sports Med 41(6): 365-9; discussion 369**) looked at the effect of cold on nerve conduction velocity, pain threshold and pain tolerance on asymptomatic subjects recruited from local sports clubs, and using a within subjects design, compared the response of one treated (cold therapy) ankle with the other which acted as a control. Both the cold and the control conditions were assessed at a single session, though there was a randomisation for which ankle was the control.

The nerve conduction velocity was recorded from the tibial nerve (details provided) and the pain threshold and pain tolerance were recorded at two sites, one at the cryotherapy zone (near the lateral malleolus) and one point around the 4th metatarsal, distal to the treatment zone, but still supplied by the tibial nerve. Both the pain threshold and pain tolerance levels were assessed using a pressure algometer – applying pressure until discomfort was reported (threshold) and continued until it became unbearable (tolerance). The measures were taken at baseline, and then when the skin temp had reduced to 15 and then 10 degrees C (based on other published work) and then on reaching 15 C on rewarming. No problems with this, but clearly, this means that the time taken to reach these temperature changes would not be the same in all individuals. If the actual temperature reached are the critical factor (and previous research would suggest that they are) then this might have an impact on therapy in that most therapists apply cold for a 'time' to get an effect whereas in fact, this might not be the most appropriate dose decision model. Unfortunately, there paper fails to describe in any meaningful way how or where the ice was applied – it appears to be with a pack using crushed ice and it would appear to have been applied somewhere around the lateral malleolus – shame that the detail was not provided.

That notwithstanding, the results show that the mean time taken to reach the 10 C point was around 26 minutes, and there was a significant effect on nerve conduction velocity directly related to the intervention. There were significant differences between ice and control ankle conduction velocities at the 15 C and the 10 C points, and the control ankle conduction did not change significantly during the period (all good stuff). The mean reduction in nerve conduction velocity was around 33% by the time the ankle skin temp had reduced to the 10 degree C point.

The pain threshold and tolerance levels increased significantly in the iced ankle compared with the control ankle (which showed no significant change) with a mean 89% change in threshold and a mean 76% change in tolerance. The measures at the site distal to the ice application also demonstrated significant change with progressive skin temperature drop and compared with the control ankle, with a mean change of just over 70% for threshold and 56% for tolerance.

These results would suggest that the progressive reduction in nerve conduction velocity is likely to be primarily responsible for the changes in pain threshold and tolerance that were demonstrated. The lack of significant change in the untreated ankle would suggest that central mechanisms (not assessed) are less important than the local effects on nerve conduction.

There are some limitations to this study which the authors do not raise – mainly there is almost no description of the cold application (site or method), but the results do demonstrate a clear local effect of cold on nerve conduction velocity, pain threshold and pain tolerance. These effects are related to skin temperature changes, which clearly are linked with points in time, but are not the same for all individuals (the time taken to reduce the skin temperature at 10 C was a mean of 26 minutes but the range was from 20 to 31 minutes – something that warrants further investigation and would be interesting to link this work and some of the other key studies relating time differences in cooling between subjects.

Cold therapy and delayed onset muscle soreness

The second of the cold therapy papers for this issue comes from a group of researchers in Australia and deals with the perennial problem of DOMS (Delayed Onset Muscle Soreness) pain (**Sellwood, K. et al. (2007). *Ice-water immersion and delayed-onset muscle soreness: a randomised controlled trial.* Br J Sports Med. 41(6): 392-7.**

There have been plenty of criticisms over the years with regards DOMS pain. It has been pretty popular in some experimental work in that you can generate a pretty reproducible pain model, but one of the major criticisms is that it is not very much like the clinical pains that most therapists get to treat on a frequent basis. It is however, commonly encountered in sports people, and given the target audience for this journal, seems appropriate. I was also interested in the work, because much of the previously published work that I had read appears to demonstrate that there is not a lot that you can do about DOMS type pain. Ice water immersion appears to be pretty trendy out there (especially in sport) at the moment, though when we looked recently at the evidence (beyond the anecdotal) to support its application, we were unable to find much of quality.

This was good basic DOMS research. A reasonable sized group (n=40) of subjects who were 'untrained' were put through an eccentric quads programme (non dominant leg) such that DOMS was the intended outcome. The trial was an RCT design and double blind, giving it methodology brownie points. The key outcomes included pain, tenderness, swelling, function (hopping), muscle strength and serum marker (CK) were taken pre and then at 24, 48 and 72 hours after the exercise. The subjects were divided into 2 groups – one getting

cold water immersion (3 x 1 minute) or tepid immersion (3 x 1 minute). The iced water was at 5 degree C and the tepid at 24 degree C.

Immediately following the eccentric exercise protocol, subjects stood (up to their ASIS) in either the cold or tepid water (depending on group allocation) for 1 minute, followed by a minute out, this cycle being repeated three times (ohhhhhh how you would want to be in the control group for this work eh??).

There are a lot of results included in the paper, as as I keep saying, I am not trying to replicate the detail of the original papers in this newsletter, just trying to give an accurate summary of the key issues. There were no significant differences between the two groups on any key outcome at baseline – good start. The adopted DOMS protocol worked in that by 24 hours post exercise, there was marked DOMS pain and other features. Apart from the ice immersion group having MORE pain, there were no significant differences between the groups at any time points. The take home message is that on the basis of this cold immersion protocol, there is no evidence of beneficial effect for the therapy – though I have no doubt whatsoever that it will continue to be used in the sports related world. It might of course, be an effective intervention at some other ‘dose’, but using the one minute IN, one minute OUT repeated 3 times regime (which seems fairly popular out there), the measured effectiveness of the therapy has not been demonstrated. The fact that the cold therapy group actually had more pain is discussed as are several key issues about DOMS and associated issues. If you are involved in sport related work, you really should read this. If you have an interest in whether cold immersion is effective or not, you should read it. If you use cold for your patients (DOMS related or not), you should read it and see what the authors have to say. I am sure that the proponents of the cold immersion therapy will have criticisms of the paper, but read it through and see what you think.

OK, so that will do for the moment. The next edition is now scheduled for April/May 2008 and I will start writing now! I will also do my best to include the papers that I had to omit from this issue.

If you have or know of any papers that I appear to have missed, please do let me know and I will get them included somehow if I can. Much as I scan a lot of journals and index systems, I have no doubt that I miss papers and my thanks to those people who e mail and make suggestions – at least I get the impression that somebody reads all this!

E mail : t.watson@herts.ac.uk and don't forget that the Website has some basic material on most, if not all of the key modalities (www.electrotherapy.org).

Regards

Tim

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