

Electrotherapy News

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Hello and welcome to the last 2006 / first 2007 edition of ElectroNews. As ever, there are plenty of electrotherapy related papers to report on – I had to make this selection from over 150 that I have come across since the last edition – and they say that there is no evidence out there on electrotherapy???? Some of it is ‘positive’ and some ‘negative’ . . . but it is all evidence and adds to the total body of knowledge.

Couple of news items before we get going

NEWS 1

Firstly, the CSP Electrotherapy Guidance . . was due for launch in October (06) but has been re-scheduled for early this year now – details below :

The Electrotherapy Clinical Interest are hosting a workshop on **Thursday, 15 February 2007**, to launch the long awaited “Clinical Guidance in the use of Electrophysical Agents”. The main contributors to the document will be lecturing. Venue: Solihull Hospital, West Midlands. The cost will be £35.00 to CIG members, or £50.00 to non-members and copies of the guidance will be available to delegates.

Applications and requests further information should be addressed to:

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NEWS 2

The second item relates to some research we are about to undertake in the field of microcurrent therapy. You may recall, I have made several mentions of this topic in previous editions of the newsletter. There are some really interesting developments in this field, and Leon Poltawski (who will be working with me on this one) is starting a 3 year study into microcurrent and its (potential) effects following soft tissue injury. We have a reasonable amount of literature with regards its effects in bone healing and open wound management (ulcers, pressure sores etc), but this research programme will focus on soft tissue repair and associated issues. If anybody out there has anything useful that might contribute to the research – previous studies – maybe unpublished – manufacturers links or anecdotal material as well as the more obvious references to papers that might be useful, please can you get in touch with Leon (e mail : l.poltawski@herts.ac.uk Tel : +44 (0)1707 284556). We will keep you all updated (web site and mainstream publications) as the research progresses – so watch this space.

NEWS 3

Lastly on the news front, several people have been asking about the new edition of Electrotherapy : Evidence Based Practice. I mentioned in the last edition that it was coming out, and the current estimate is sometime around mid 2007 assuming I can get all the editing done in time. It is looking great (but I would say that!). The work is presented by high profile authors contributing to chapters in their specialist fields, looking at the evidence out there and what implications that it might have in terms of research and clinical practice. There is still some work to do, but will let you know as soon as we have a confirmed date.

CONTENT

OK, so on with the content. As I mentioned earlier, I have had to do a substantial sift to reduce the 150 plus papers down to the selection for this issue – there is actually enough material out there for an issue every month I reckon (if I just had time that is!).

Ultrasound :

- Fracture healing and conventional ultrasound devices
- Ultrasound and phonophoresis
- Ultrasound machines as a potential infection source in clinical practice

Shortwave / Electromagnetic Fields :

- Ultrasound and electromagnetic fields – effect on osteoblasts
- Electromagnetic fields and OA pain
- Pulsed shortwave and metal implants

Laser :

- Cyriax and light therapy for lateral epicondylitis

Interferential :

- Interferential therapy and back pain

TENS :

- Effects of varying TENS frequency and Serotonin release

Electrical Stimulation :

- Three papers on electrical stimulation and stroke
- Electrical stimulation and nerve recovery
- Electrical stimulation and its effect on action potentials
- Recent issues in Iontophoresis
- Cranial electrical stimulation
- Electrical stimulation and muscle related outcomes

Clinical review : Treatment for TMJ problems (including electrotherapy)

Heat & Ice :

- Heat wrap and low back pain
- Ice and ankle sprains
- Cryocuff and TA microcirculation

Tissue Repair :

- Steroids and collagen behaviour

Fracture healing and conventional ultrasound devices

Warden has produced a stream of ultrasound and fracture repair material over recent years – there was one in the last issue – and another one here. The question is often asked of me as to whether it is possible to use a ‘conventional’ ultrasound machine to do the fracture healing type of treatment. Warden has addressed this issue before (Warden et al 1999 ; see web pages for references and summary), but in a new paper (**Warden, S. J. et al. (2006). Ultrasound produced by a conventional therapeutic ultrasound unit accelerates fracture repair. Phys Ther 86(8): 1118-27**) reports the outcome of some experimental (animal) work using a conventional US machine. Essentially, bilateral midshaft femoral fractures were produced in 30 rats. US was applied to one fracture from each animal and the other was treated with an inactive device (placebo intervention). Treatment parameters were 1MHz, 0.1 w/cm², pulsed, and the animals were treated for 20 minutes a day, 5 days a week commencing 1 day post fracture. Those of you that have delved into this literature previously will recognise that this is close – but not exactly the same – as the commonly applied LIPUS parameters. Some animals were killed at 25 days and some at 40 days – looking for the time based trends and the outcomes included bone mass and strength.

The results are summarised thus : There were no significant differences found between the placebo and treatment fractures at 25 days, but at 40 days, the treated fractures had greater bone (mineral) content, an increased bone size, and over 80% more mechanical strength. It does appear therefore than in an animal model, standard US machines CAN deliver the energy in such a way as to produce very significant changes in fracture healing. Warden et al identifies, and I would have done if he had not, that this is an animal / lab experiment, and it would be risky to simply assume that it will transfer to human fracture healing – though one could reasonably expect it to. Further work is needed (predictable) but it is a very useful indication that standard US devices may be able to contribute to this aspect of fracture management – though the idea of doing 20 minutes US a day lacks a degree of excitement! The Exogen and SAFAHS systems which are already out there use a stationary applicator and a patient based / DIY approach to treatment – the patient borrows / loans / leases the machine and delivers the treatment rather than always attending for therapy. Smith & Nephew have come up with a new version of their Exogen machine (<http://ortho.smith-nephew.com/us/node.asp?Nodeld=2865>), but it is time limited and still (relatively) expensive. The full Warden paper is well worth a read. The writing is good, the format easy to follow and some nice

graphics to boot – along with plenty of detail. I am looking forward to the human studies using conventional US devices – as I guess are many of us.

Ultrasound and phonophoresis

I don't usually go on much about phonophoresis (the use of US energy to encourage the migration of drugs into the tissues), but several people have been asking, so when I came across this paper, it seemed like an ideal opportunity to bring one to your collective attention. Hsieh [Hsieh, Y. L. (2006). **Effects of ultrasound and diclofenac phonophoresis on inflammatory pain relief: suppression of inducible nitric oxide synthase in arthritic rats.** *Phys Ther* 86(1): 39-49] has produced an interesting paper following on from another ultrasound and nitric oxide paper published last year.

There is some useful nitric oxide (NO) material in this paper – which I will not try and explain in detail – those that are interested can easily go to the original and work through it, but essentially, NO is synthesised by an enzyme (NO synthase or NOS) which is a free radical associated with inflammatory events (amongst other things). Various forms of NOS have been identified (neural, inducible and endothelial), and it has been shown that it has a role to play in the CNS in relation to pain transmission and inflammatory states. In animal models it has also been shown to relate to persistent inflammatory states and tissue destruction in arthritis.

In this particular work, a spinal cord enzyme (iNOS : inducible NO synthase) which is NOT normally present in the spinal cord was employed as a marker of inflammation and active nociception in the cord.

Essentially, a peripheral arthritis reaction was induced (using a standardised technique) and the levels of iNOS in the cord was evaluated following ultrasound, NSAID phonophoresis or sham intervention in 18 rats. The peripheral (ankle & hind paw) responses were measured and the iNOS levels in the cord also evaluated.

The US or phonophoresis was used 18 hours following the arthritis induction. US was applied at 1MHz, 0.5 W cm², pulse 1:1 (50%) for 5 minutes. The same was used for the phonophoresis which employed a diclofenac gel rubbed into the skin prior to the US treatment. Effectively, there were three experimental groups – US, US phonophoresis and Sham (no US output or NSAID).

Outcome measures were oedema (circumference and diameter of paw and hindfoot) measured pre and 18 hours post arthritis induction, locomotor activity and iNOS immunohistochemistry (details in the paper).

OK, lets get on with the results (all calculated using variations of the repeated measures ANOVA with post hoc). The experimental arthritis produced significant increases in paw oedema and joint measures. The animals in the US and the phonophoresis groups demonstrated significantly raised locomotor activity levels following treatment compared with the sham group. There was plenty of iNOS evidence in the cord following arthritis induction. The effect of the two treatments was to significantly reduce the number of cells staining for iNOS (lots of sub section and laminae analysis in the paper).

Effectively, the experiment shows (confirms) that there is a significant production of iNOS in the spinal cord following this experimental arthritis induction, and that the use of ultrasound and the use of phonophoresis reduces the amount of iNOS present post treatment – a central effect. It also demonstrates a peripheral effect in that animals were more mobile after treatment (both groups).

There is a long discussion in the paper, but I think that this work raises at least 2 issues. Firstly, from what I can see of the results, there was no effective (significant) difference between the US and the phonophoresis groups – i.e. both treatments worked, but there was no advantage of one over the other – which is an interesting finding from a phonophoresis effectiveness aspect. Secondly, that the treatment(s) had a central effect (in addition to a peripheral effect) is potentially valuable and probably needs to be followed up. It is generally thought that US has a LOCAL effect (good evidence for that) but if it also has a central (CNS) effect, there are possible mechanisms to be explored that might result in novel treatment approaches – especially with clinical problems like neuropathic pain etc etc.

Ultrasound machines as a potential infection source in clinical practice

There was some work done years ago up at Birmingham University – though I don't think that it was ever published – concerning the potential infection risk of ultrasound treatment heads. Other related work includes several studies on potential infection risk with interferential electrode sponges (e.g. Lambert et al 2000). This recent paper [**Schabrun, S. et al. (2006). Are therapeutic ultrasound units a potential vector for nosocomial infection? Physiotherapy Research International 11(2): 61-71**]

presents some fascinating information and I would suggest, something that should bring about a change in routine US practice for those who do not, as yet, employ infection control measures in this area.

Given (in the UK anyway) the currency of potential infection risk for patients attending healthcare, it is a timely paper. The research looked at the potential infection risk from US treatment applicators and also interestingly the gel / gel containers.

A decent sized sample of 44 treatment heads and 43 gels were tested initially, with a range of practice environments (health service, practices and residential care facilities) in Australia. Standardised microbiological samples were taken and a threshold set against which the sample could be determined to be contaminated or not. There was a second phase whereby the samples were retested following cleaning with a 70% alcohol swab. In addition to the basic microbial count, the types of organisms cultured (and their resistance to antimicrobial agents) were also evaluated.

The results are of considerable interest, and I would reckon that this paper is well worth a read in full as I can only summarise the essentials here. The contamination levels for the treatment heads and the gels were very similar (27% and 28% respectively). The treatment heads actually came off better in that the majority of the microbial contaminants were those normally occurring on the skin, whilst the gels also showed growth of some opportunistic agents and seemed to be more heavily contaminated. Thankfully (!) no multi-resistant organisms were identified in the sample. Cleaning of the treatment head (70% alcohol swab post treatment) significantly reduced the contamination levels

The results (in a bit more detail) showed that although 27% of the treatment heads were deemed to be 'contaminated', a further 32% yielded micro-organisms, but at a level below the contamination threshold. Similarly with the gels, the 28% contamination was supplemented by a further 23% with micro-organisms below threshold (the gel samples by the way were taken from inside the container lid). There is an interesting breakdown of infective agents and their count – quite puts you off your food by the way!

The useful information concerning what happened following the alcohol swab is impressive. After cleaning with the swab, only ONE of the treatment heads remained contaminated – which was clearly a significant difference.

I would have thought that routine cleaning of the treatment head should become normal practice – if it is not already - after you have read and inwardly digested this paper. Clearly this will deal with the majority of the organisms identified in this study, though one would not suggest that it would deal with everything that might be on offer. The more interesting – and potentially problematic issue – is what to do about the gel bottle? The study made no attempt to try and deal with the micro-organism count in the lid of the gel bottle – but there is an interesting study there just waiting to be done – let me know if you are interested – would love to follow this one up – but maybe the authors of this paper are planning on doing so???

Anyway, if you have just done an ultrasound treatment, not used the alcohol swab and not yet washed your hands enjoy your sandwich!!!

Ultrasound and electromagnetic fields – effect on osteoblasts

Here is an impressive link then between the ultrasound and shortwave sections! Li et al (*Li, J. K et al. (2006). Comparison of ultrasound and electromagnetic field effects on osteoblast growth. Ultrasound Med Biol 32(5): 769-75*) have produced a paper that might be of interest. Predictably (from the title) it is a lab study, and it takes an interesting approach to a critical question – how does energy X (in this case ultrasound or pulsed electromagnetic fields – PEMF's) bring about changes in cellular activity? The group have published extensively on various aspects of this work previously, and those that are interested in cell based lab work will find an excellent summary of the findings to date in the introductory pages. The aim of this particular study was to try and establish the mechanism by which US and PEMF bring about increased osteoblast proliferation, to compare the mechanisms and then to use the results to try and explain how these modalities could enhance fracture repair.

Their basic approach was to use 8 different signal transduction inhibitors – neat approach I think. Effectively, any form of energy delivered to the cells / tissues needs to have an identified mechanism by which it can bring about changes in cell activity. Could be mechanical – thereby using a process of mechanotransduction, could be light therefore using phototransduction etc etc. This is looking at the problem at an even more basic (biochemical) level, and I will leave out the mass of detail in the paper as many of you will have a limited interest in it. Those that are interested (as ever) can go to the original.

Osteoblasts were isolated from rat tissue and then exposed to either US or PEMF energy. The US was delivered with a standard type therapy machine operating at 1MHz, Pulse 1:4, 0.6 W cm⁻² for 15 minutes. The PEMF was delivered using a specialist device – certainly not a classic pulsed shortwave therapy machine. The parameters were set at 7.5Hz single pulse for 2 hours and an electric field intensity of 2mV/cm. These parameters were those that the team had previously identified as being the most effective (there was also a sham group).

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

The results summarise thus : both interventions brought about a highly significant increase in osteoblast viability compared with the control group, but there was no significant difference between the treatment groups (without any inhibitors being applied). When the 8 different inhibitors were tested, there was actually not that much difference between the results, though there was a difference in nitric oxide (link to US paper earlier in this issue) pathway – US cellular activation was inhibited when NO pathway was blocked, but PEMF changes were not.

Intracellular Ca ions appear to be important in both systems – which is good news as I have been talking about this mechanism for several years now, and the discussion follows some interesting arguments. At the end of the day, both ‘modalities’ achieve their transduction by means on intracellular release of Ca ions which sets off intracellular cascade reactions. It is argued that the PEMF has its primary effect at an intracellular level (rather than the more often cited cell membrane level). The US NO pathway effects are compared with the mechanotransduction pathways established elsewhere – which is not too surprising as US is effectively a mechanical (pressure) wave rather than an electromagnetic signal. Some useful links are made by the authors between US mechanical transduction, nitric oxide mechanisms and the changes in VEGF (highlighted in previous newsletters).

Although both US and PEMF interventions increased osteoblast viability, it has been shown that their transduction methods are not exactly the same, with US using more of an NO route and PEMF not. The transduction pathways are similar in that they both result in increased levels of intracellular Ca ions. This is a heavy cell study paper, but I think that it does start to provide decent evidence as to how these modalities are capable of bringing about their physiological and therapeutic effects – moving beyond the speculative. Detail in the paper if you want it, but the take home message would be that both modalities increase osteoblast activity via an intracellular Ca ion mechanism, but how they get there is slightly different.

Electromagnetic fields and OA pain

Next is a much more clinically related paper – and one that does not provide much by way of supportive evidence in this case – for those that say that I only seem to pick on the positive outcomes!

McCarthy et al (***McCarthy, C. J. et al. (2006). Pulsed electromagnetic energy treatment offers no clinical benefit in reducing the pain of knee osteoarthritis: a systematic review. BMC Musculoskelet Disord 7: 51-56***) have produced a systematic review which will be of interest. The authors come from strong research stables at Warwick and Manchester Universities.

It is suggested that Pulsed Electromagnetic Fields (PEMF’s) are used in the management of OA knee, and that there are now sufficient RCT’s in this field to enable a systematic review. The literature from 1966-2005 was considered and the paper identified the search terms, literature sources and methods adopted. Five RCT’s were identified in which PEMF treatment was compared with placebo intervention. Their conclusion was that there was insufficient evidence to support the intervention – it has ‘little value’ in knee osteoarthritis, and does not provide significant pain relief.

The 5 trials accepted for the review were selected from 20 RCT’s and 5 non randomised trials identified from the literature. The majority of these papers failed to meet the reviewers inclusion criteria. There was a total of 276 patients from the 5 trials. Only three of the included trials actually involved pulsed shortwave in the classic therapy sense of the term – mainly because PEMF intervention includes a number of treatment methods and pulsed shortwave is only one of them.

Rather than try and defend an intervention that the RCT's combined results appear not to support, I would raise a couple of points that would be of interest to me. Firstly, and the authors do acknowledge this, the 5 trials did not actually all involve the same treatment. Three were using PSWT and the other two something that is (clinically) quite different. The other point is one that I am currently working on in terms of a review paper – that of dose dependency in electrotherapy. There is little doubt from the evidence that the effects of electrotherapy (in the widest sense) are both modality and dose dependent. An RCT that comes up with a 'no significant effect' result does not equate to a conclusion that the modality is not effective period. If one were to deliver a pain relief drug at the 'wrong' dose, an RCT could easily come to the same conclusion. It may well be that pulsed shortwave is not effective for this patient group but 3 RCT's is not exactly a wide ranging sample, and the doses applied did not come into the equation for the analysis. If all 3 papers involved the delivery of an inappropriate treatment dose, they would (I suspect) still have been included in the systematic review so long as their randomisation etc etc were sound. This is a fundamental issue in electrotherapy research in general. I will let you work on that one, and I will also let you know when I have finished the dose dependency review paper!

Pulsed shortwave and metal implants

The last of the pulsed shortwave papers in this issue is one that will raise an eyebrow or two! It has long been considered that metal in the tissues is a contraindication to many, if not all forms of electrotherapy. I get numerous e mails every week asking if it is ' . . . OK to use TENS to the ankle on a patient with a hip replacement . . . ' etc. There is actually precious little evidence of the feared detrimental effects, but lets confine the story here to pulsed shortwave. The currently cited opinion would suggest that 'high dose' pulsed shortwave and of course continuous shortwave therapies should not be used when there is metal in the tissue concerned. Low dose (non thermal) pulsed shortwave is considered to be OK, though there are many therapists who still do not use any PSWT when there is any metal around.

Seiger and Draper (**Seiger, C. and D. O. Draper (2006). Use of pulsed shortwave diathermy and joint mobilization to increase ankle range of motion in the presence of surgical implanted metal: A case series. J Orthop Sports Phys Ther 36(9): 669-77**

) have produced an interesting paper that might just challenge this view. Although this paper is based on a case series rather than an RCT, it does not negate the fact that this is evidence to add to the pot.

The work investigated the use of pulsed shortwave therapy to the ankle, combined with mobilisations for patients with metal implants in the area. All 4 patients presented with were post trauma ankle stiffness with limitation in dorsiflexion. All had been treated 'conventionally' with a range of interventions, but were left with significant ankle stiffness.

Pulsed shortwave was used at 800 pps and 200 microseconds pulse duration, giving an applied mean power of 48 Watts for 20 minutes (Megapulse II), and this was followed by joint mobilisation techniques and this was followed by an ice pack for 10-15 minutes. The treatment protocol was carried out for 2 or 3 times a week for the first 3 weeks and then twice a week for the next 2 weeks. One patient received 13 sessions, and the others, 8 sessions. There was a follow up measurement of ankle range 1 – 3 months following the final treatment. The primary outcome measures were ac-



tive (non weight bearing) ankle and hindfoot ROM and manual muscle testing. Subjective comments from the patients were also collected (pain, discomfort etc).

All patients demonstrated significant gain in ankle ROM – dorsiflexion, plantarflexion and inversion/eversion. By the end of the treatment period, all patients had achieved ROM to within 5 degrees of the unaffected ankle, and although there was a decrease in ROM at follow up (1-3 months), there was a maintenance of at least 78% of the final post treatment range. These were not 'fresh' clinical problems – ranging from 4 months to 18 years post injury.

The information from the patients recorded during the treatment sessions did not indicate any discomfort, burning or other untoward sensations. There was a reported mild vibration in the area for the first few minutes, but this was not uncomfortable nor did it continue beyond this initial period.

There is an interesting additional item in that one of the patients underwent surgical removal of their metalwork 5 months after treatment and the surgeon was contacted by the researchers and consulted with regards metal or tissue damage at the site. None was identified in the immediate or surrounding bone or soft tissues.

There is no statistical analysis – understandable for a case series with n=4 patients, but there is a wealth of outcome measurement data in the paper. My interest is not just that these patients improved – it was not an RCT, there was no control group and no way to discriminate between the effects of the pulsed shortwave, the mobilisations or the ice for that matter. The interesting thing is that (a) pulsed shortwave was applied with metalwork in the tissues, (b) that it was applied at what would appear to be a relatively high dose in pulsed shortwave terms (48 Watts mean power) which would go against conventional wisdom and (c) on the limited post treatment surgical follow up, there did not appear to be any tissue damage. Patients had not reported any adverse sensations during the treatment nor of any adverse effects after treatment.

Draper has published other papers in this area (pulsed shortwave with metal in the tissues) which are referenced and has carried out a substantial portfolio of research relating to the thermal effects of various electrotherapy modalities. There is clearly more work to do in this area. I would not suggest that we should go and do pulsed shortwave at these doses to everybody with metalwork in the ankle, but maybe it need not be the absolute contraindication that it is at the moment?

Cyriax and light therapy for lateral epicondylitis

Only one laser / light therapy paper this time – there have been a plethora in the last few issues, and I need to get some of the electrical stimulation backlog covered.

A recent paper in Clinical Rehabilitation (**Stasinopoulos, D. and I. Stasinopoulos (2006). Comparison of effects of Cyriax physiotherapy, a supervised exercise programme and polarized polychromatic non-coherent light (Bioptron light) for the treatment of lateral epicondylitis. Clin Rehabil 20(1): 12-23**

pretty much does what it says on the tin!

Lateral epicondylitis is an area where there has been a reasonable amount of clinical research, partly I guess, because it is a difficult clinical problem to manage and partly because various forms of electrotherapy are claimed to be effective. Leaving the latter part of that well to one side for the moment, this study, carried out in Greece involved 75 patients who were allocated to one of 3 intervention groups. This was a controlled study, and patients in each group only received one of the treatments – Cyriax physiotherapy, a supervised exercise programme or light therapy with a device known as the Bioptron 2 which generates polychromatic non-coherent light. The light characteristics

are listed in the paper, but are summarised as being wavelengths in the range of 480 – 3400 nm with 95% polarisation and a power density of 40mW/cm². There is no wavelength profile (i.e. what proportion of the energy is delivered at which part of the spectrum and the suggested energy density delivery of 2.4 J/cm² must be based on a specific time frame, but this is somewhat glossed over in terms of the technical spec. The Cyriax and exercise therapies are fully described in the text.

There was a good outcome measure regime with data collected at the start, at 4 weeks (end of treatment) and then again at one, three and six months after treatment ended. Outcome measures included VAS for pain, a function score and pain free grip strength. Patient drop out was monitored and a research process (CONSORT) flowchart is included in the paper which is (as ever) nice to see.

There is lots of data reported, and I will summarise the essentials rather than go through a blow by blow analysis. The patients were comparable between groups at the start of the process. There was a significant reduction in pain in all 3 groups over the treatment period, though on further analysis, it was shown that the exercise group faired best of all, then the Cyriax and then the light therapy. The difference between the exercise and the other treatments was significant but there was no sig diff between the Cyriax and the light therapy at 4 weeks. Function also improved significantly over the treatment period, though again, not the same for all groups. The sub set analysis came up with the same results as for the pain – exercise best, then Cyriax and then light. No sig diff between light and Cyriax. Pain free grip strength followed exactly the same data trend.

The fact that there was a comprehensive follow up schedule, and that there were not any drop outs from any of the three groups (how did they get that???) was great, but the follow up data analysis was brief to say the least! It certainly looks (from the data table and the results that are presented) that the effects were maintained and that the exercise still comes off the best and still no sig differences between the Cyriax and the light therapy.

There are a number of interesting issues here, and although this was not a true RCT in the classic sense (patients not randomly allocated to groups and no control / placebo group), it does generate some useful data. The light therapy delivered in this particular trial is not actually laser therapy in the classic sense. The light is not monochromatic – it is delivered at multiple wavelengths which is quite different given that a number of researchers (including Karu some years ago) have demonstrated that the monochromasticity is possibly one of the key elements in laser therapies. It certainly looks like all three interventions produced significant improvement, but that given an option, and if only one of them was used in isolation, then a programme of supervised exercise would probably be the winner. Combined treatment approaches would be the clinical norm for most / many of us, but that is even harder to research effectively.

Time to move on the some electrical stimulation reports. There has been a rapidly growing pile of these on my desk over the last couple of months, and much as I could spend pages on each one, I'll have to keep it fairly brief to get enough of them into the available space!

Interferential therapy and back pain

This paper (**Al Abdulwahab, S. S. and A. M. Beatti (2006). The effect of prone position and interferential therapy on lumbosacral radiculopathy. Advances in Physiotherapy 8: 82-87**) looks at the effects of positioning (prone) and positioning combined with interferential therapy in a patient group with lumbosacral radiculopathy. Pain, pain distribution and H reflexes were used as outcome measures in two groups 28 healthy subjects and 28 subjects with this clinical problem. There were

4 different intervention combinations : 3 mins rest in prone : 20 mins rest in prone : 10 mins IFT and 20 mins IFT. The IFT treatments were delivered in the (same) prone position.

Neither the prone position nor the IFT had a significant effect on the H reflex measurements (which were taken as an indicator of spinal nerve root compression) in either group. There was a significant reduction in pain (patient group) with all interventions and also for pain distribution, and the authors argue that this is probably a placebo effect.

The IFT was delivered at a constant frequency of 100Hz at an intensity needed to generate a strong but comfortable tingling sensation. A 4 pole vacuum electrode application was employed. The post hoc stats tests appear not to be fully reported in the paper which is a shame (unless I missed it) but looking at the data tables, it certainly looks like the IFT had a stronger effect on pain than the prone position alone, and the longer the IFT treatment, the more pain relief was achieved.

The reason why the authors suggest that these pain relief effects are probably placebo related comes back to the fact that the interventions did not affect the H reflex, and therefore unlikely to have a physiological origin. I am not entirely convinced by this argument, but could not rule it out. There is almost certainly a placebo effect of all intervention – electrotherapy, manual therapy and everything else – it is there and it is real. That the pain relief should be different between the 4 conditions tested, with more pain relief with the longer interventions would seem to be more than placebo from my point of view, but I have no more proof than the authors. Interesting paper none the less, and some interesting points for debate.

Effects of varying TENS frequency and Serotonin release

Sluka has produced some great material with regards TENS over the years, and this most recent one provides some fascinating insight into TENS mechanisms (**Sluka, K. A. et al. (2006). Increased release of serotonin in the spinal cord during low, but not high, frequency transcutaneous electric nerve stimulation in rats with joint inflammation. Arch Phys Med Rehabil 87(8): 1137-40.**

This is an animal experiment (rats) looking at the difference in TENS frequency on the release of serotonin and noradrenalin in the spinal cord. Serotonin in particular has been linked with various pain mechanisms over the years, and even 20 or more years ago, it was considered to be a likely contender for critical pain neurophysiology pathways. It has been shown that there is effectively a link between serotonin and opioid mechanisms at cord level, and thus this experiment aimed to see whether the use of low frequency TENS [4Hz] (which is purported to operate through an opioid pathway) changed serotonin levels (which would be expected) and to see if high frequency TENS [100Hz] (which is purported to work through a more pain gate bias mechanism) did not have an effect on serotonin levels (expected as it is not primarily an opioid pathway).

Basically, an inflammatory response was generated in the knee (established method) of the 18 rats and 24 hours later, TENS was applied at the appropriate frequency for 20 minutes with electrodes applied at the knee. The device used was a standard clinical TENS unit using a zero net DC pulse form at 100 microsec duration and at the same intensity in both frequency groups (to avoid the potential confounder of intensity effects rather than the frequency effects being looked for). The rats were divided into three groups – 6 each @ high frequency TENS, low frequency TENS and sham TENS.

The serotonin levels were raised post TENS, and there was a sig difference between the low frequency group and the (sham and high frequency) groups. The noradrenaline levels remained unchanged in any group.

The discussion raises some critical points with regards the role of serotonin in pain mechanisms which would be well worth a read for those interested in this field. If I were short of papers to include in this issue, I could spend the next couple of pages on it! The conclusion reached, which appears to be fully supported by the data presented, is that low (but not high) frequency TENS releases serotonin in the spinal cord that activates specific receptors to achieve reduction in hyperalgesia.

There have been several papers over the years that have looked at these mechanisms, mainly in animal models for understandable reasons. This one, I would suggest, adds very nicely to the quality evidence and certainly contributes to our understanding of how different (high – low) frequency applications of TENS appear to have different physiological modes of action.

Electrical Stimulation :Three papers on electrical stimulation and stroke

There have been many papers in recent years looking at various forms of electrical stimulation in relation to neurological issues, and stroke in particular. Historically, most forms of elec stim have been thought of as being inappropriate for CNS lesion patients, but the evidence is certainly stacking up in favour of the alternative view. I will briefly look at this next batch of three for your information.

Yozbatiran, N. et al. (2006). Electrical stimulation of wrist and fingers for sensory and functional recovery in acute hemiplegia. Clin Rehabil 20(1): 4-11.

This paper is another that looks at upper limb function in acute stroke patients, using electrical stimulation and an exercise based programme. 36 patients were recruited to the trial, 18 of whom get a TENS type treatment and 18 that acted as a control. Both groups got 1 hr a day of Bobath type intervention (for 10 days) and the e stim group in addition get 1 hr of stim for the wrist and finger extensors. The outcome measures were related to hand function, kinaesthetic sense and hand movement.

In short, the study demonstrated improvement in kinaesthetic and position sense after the treatment, and there was no significant gain from adding the TENS. Hand movement scores also improved in both groups, but the hand function improvement was only seen in the TENS group.

The TENS treatment to the forearm extensors was provided with a standard clinical TENS device operating with a pair of electrodes over the muscle mass, using a frequency of 2Hz @ 260 micro-second pulse duration (though the pulse duration was set to cycle automatically over a 5 second period, the details of which are not specified (this is in effect a form of pulse duration modulation, one assumes to minimise accommodation, though whether it actually achieves this is somewhat unclear from the evidence out there)

That there is a link between upper limb recovery and sensory capacity (in the widest sense) is not really in doubt. Using the TENS as a supplement to promote sensory capacity and therefore improve function and movement has a good logic. Stimulation at 2Hz is not the strongest sensory stimulus, though it may have had some additional advantage in that the motor nerves would have been stimulated as well. It would be interesting to compare the effects of varying the frequency of the stimulation in this kind of rehabilitation. From my own experience, I would have expected a dif-

ferential effect, but that is anecdotal, and I would not claim to have done any research / trial work in this field – interesting thought though.

The second of the stroke papers is from Archives Phys Med (**Robbins, S. M. et al. (2006). The therapeutic effect of functional and transcutaneous electric stimulation on improving gait speed in stroke patients: a meta-analysis. Arch Phys Med Rehabil 87(6): 853-9**) and as the title suggests is a meta analysis – which should make it especially useful for those of you that are looking for big review material.

The work looks at Functional Electrical Stimulation (FES) and TENS post stroke in relation to gait. Like the systematic review a few articles back, papers were considered from 1966 – 2005 looking for papers where either form of electrical stim had been used and gait speed had been used as an outcome measure.

The quick version of the meta analysis results indicates that the intervention was significantly effective in gaining speed post stroke. When you look at a bit more detail, the following emerge : they found and reviewed 67 full articles (first trawl was just by abstracts) and this came down to n=8 for the final analysis – which is not many given that they started with well over 500, but that is the nature of this type of research. Both FES and TENS studies were found, with more in the FES group. The paper does neatly describe each trial and provides a summary table for quick reference – useful.

The results again are detailed, but the mean effect size was greater for the FES than for the TENS group (which I reckon would match expectation) though it is worth noting that the FES studies were of mixed methodology (some for example were simple single channel devices whilst others used multi channel machines) and there were insufficient TENS trials to run a full effects model analysis. 4 of the trials were controlled and 4 were before-after designs. The conclusions reached by the researchers appear to be consistent with other meta-analyses and reviews which is encouraging.

There is more work needed in this area, but at the moment the compiled research would support the intervention (FES) as being effective in terms of gait speed. I appreciate that gait speed might not be everything so far as clinical recovery is concerned, but the results do not say that FES is ineffective for other parameters – just that gait speed was the parameter of choice for this meta-analysis.

The last of the three post stroke papers is by Yavuzer et al (**Yavuzer, G. et al. (2006). Neuromuscular electric stimulation effect on lower-extremity motor recovery and gait kinematics of patients with stroke: a randomized controlled trial. Arch Phys Med Rehabil 87(4): 536-40**) and is an RCT which should lend it some weight in the view of many.

As you might expect from the title, the trial looked at the effect of muscle stimulation to Tibialis Anterior in terms of motor recovery and gait kinematics. The 25 patients were all within 6 months post stroke and had no voluntary ankle dorsiflexion. They were divided into an NMES (stimulation) group and a control group (n=12 and n=13 respectively). All patients participated in a routine post stroke rehab programme, and the NMES group received, in addition, 10 minutes of stim once daily, 5 days a week for the 4 weeks of the trial (i.e. 20 x 10 min sessions). Interestingly, the applied current was a surged AC current at 80Hz for 10 seconds in a ramped pattern using 2 sec ramp up and 1 sec ramp down – giving a fairly classic pattern even if not from a commonly used machine for this purpose (Sonopuls 992). The rest period following stimulation was 50 seconds, thus one cycle was completed in 1 minute.

The outcome measures included lower limb motor recovery (detailed in the methods) and gait kinematics considering multiple parameters and measures / recorded with a Vicon gait analysis system.

Results were based on the differences pre treatment (1-3 days before) and at the end of the 4 week treatment period. Essentially, patients in both groups demonstrated improvement in lower limb motor recovery, though there was no statistical difference between the groups. Similarly, the gait kinematics data demonstrated improvement, but no significant difference between groups.

The authors conclude that the addition of the NMES (tibialis anterior) to the standard stroke rehabilitation does not provide additional benefit in terms of the outcomes evaluated. There are a number of issues with this intervention study, some of which the authors raise themselves in the discussion. I am not convinced for example that the significant benefits demonstrated by other research groups were likely to be achieved with the stim parameters used here. The device delivers a fairly standard AC stimulation at 80Hz, and I think that the more impressive clinical results data over the last 10 years has come from studies that use specific muscle stimulating devices. These need not be expensive – in fact that are much less expensive than the machine used here, and I have reported in previous editions of this publication some of the key results. There are other issues – for example the 10 minutes a day routine – with one stim cycle every minute, thus the stim group actually got very little stim. If you take the ramp up and ramp down times off the stim period, the accumulative ‘actual’ stim would be 7 out of every 10 seconds x 10 minutes which would give something like 70 seconds stimulation a day. I can’t recall any other paper having demonstrated benefits at this (low) level. It is however a useful study, and maybe if nothing else, it demonstrates that the benefits of elec stim for this patient group needs to be above a threshold level, and maybe this intervention fell below that level?

Electrical stimulation and nerve recovery

Moving away from electrical stimulation in post stroke patients, the next paper goes back to an issue I raised earlier in the year, and one about which I get numerous e mails scattered over a year – electrical stim for denervated muscle. In this 2005 paper by Modlin et al (based in Austria) (**Modlin, M. et al. (2005). Electrical stimulation of denervated muscles: first results of a clinical study. Artif Organs 29(3): 203-6**) were looking (as part of a much bigger project) the effect of electrical stimulation on lower limb muscles in patients with spinal cord injury. The patients all had cauda equine (or conus) lesions and therefore had denervation of the quads, and this had been present for 6 up to 10 years or more. The quads were stimulated with biphasic rectangular impulses of varying durations (1.3 up to 145 milliseconds) and up to 160V (peak to peak) intensity. Without wanting to review all the denervation theory, in order to get a denervated muscle to respond to electrical stimulation, a much ‘larger’ impulse needs to be delivered. The pulse duration and pulse amplitude both contribute to the pulse size. (for those of you that remember, it is the kind of stim we used to do with the IDC type treatments, and is demonstrated with a strength duration curve). It is difficult to tell from this particular report, the full stimulation parameters beyond this information.

There were several outcomes considered including muscle biopsy, CT scans, bone density measurement, skin and mechanical evaluations and in addition a complex neurophysiological assessment (details in the paper).

The stimulation applied appears to be matched to the assessment findings for each individual (rather than a standardised treatment programme). Stimulation varied between 2 and 20Hz and was initially provided for 15 minutes a day up to 20-30 minutes after some time. As the stimulation was ‘increased’ sufficient to generate a knee (extension) movement, weights were added at the ankle to increase the training effect (some additional details in the paper).

The results (looking at a year or more of this stimulation) do demonstrate a change in muscle constitution, and increase in muscle bulk with mean increases of around 30%. The report seems to finish somewhat abruptly after this point which is a shame as with the various outcomes used, I suspect that there is a wealth of further data out there somewhere.

I am sure that there is more to come from this group, as it is certainly part of a larger trial / research programme, so I will go out and look for some more. If you know where there is any – that I appear to have missed – please let me know and I will try and provide updates in future editions.

Electrical stimulation and its effect on action potentials

There has been a long running debate with regards the potential benefit (or not) of electrical stimulation following nerve injury and this recent paper by O'Gara et al (**O'Gara, T. et al. (2006). Continuous stimulation of transected distal nerves fails to prolong action potential propagation.**

Clin Orthop Relat Res 447: 209-13) certainly adds evidence to that debate. It was proposed that if the Wallerian degeneration that takes place distally to the point of a nerve lesion could be eliminated, then the regrowing nerve would only have to 'regrow' though the damaged region rather than all the way to the distal most point of the nerve course with all the concomitant recovery and rehabilitation issues.

The authors employed a rat sciatic nerve injury model and applied various frequencies of electrical stimulation to the distal portion of the nerve and then evaluated the subsequent degeneration, hypothesising that the higher stimulation rates would facilitate longer time to degeneration. The stim rates were 1Hz (n=6), 0.1Hz (n=4) (once every 10 seconds) and 0.01Hz (n=3) (once every 100 seconds). The stimulating wave form was : biphasic, 3-5mA peak to peak (sufficient to achieve a muscle twitch) and pulse duration of 50 – 100 microseconds. Custom made implanted electrodes were used for the stimulation and EMG to the gastrocnemius was the primary outcome measure. Electrodes were implanted on both sciatic nerves but only one was sectioned, though both nerves were stimulated. The known (typical) degeneration of the rat sciatic nerve is between 24 and 36 hours (though it is between 4 and 10 days in humans). If the stimulated nerves continued to function beyond this point, it would be a positive outcome. Stimulation was continuation for the duration of the experimentation. The EMG signals were recorded continuously until a zero response was sustained for 6 hours. Importantly, the integrity of the uncut nerve was always checked prior to sacrifice.

Results : Essentially, the stimulated nerves did no better than normal, and in fact the nerves with the higher stim rate(s) fared worse than those stimulated at the lowest rate, so effectively, the higher stimulation rates hastened the time to failure. None of the nerves propagated nerve impulses for significantly longer than the known degeneration time for this animal model.

There is an interesting discussion in the published paper, but the key finding – that increased stimulation rates increased the rate of action potential failure – is of interest when it comes to the therapeutic intervention that is still practiced by some, i.e. using nerve stim distal to the lesion in order to sustain the traumatised nerve.

I am sure that we might return to nerve / muscle stimulation post nerve lesion, but on the back of this particular (animal) study, there is no evidence that stimulation to the distal nerve segment post nerve injury has a beneficial effect for the patient.

Recent issues in Iontophoresis

I do get a steady stream of e mails asking about issues in Iontophoresis, and much as it is not something that I was involved with on a clinical basis (its use appears to vary greatly from country to country), I do try and keep up with the essential literature, and I have written some materials for the new web pages on this subject (when they get put up on the website). This paper (**Yarrobino, T. E. et al. (2006). Lidocaine iontophoresis mediates analgesia in lateral epicondylalgia treatment. Physiother Res Int 11(3): 152-60**) was to use a case study type approach for a small sample of n=5 patients presenting with a lateral epicondylalgia. Treatment sessions (cold therapy, transverse type fractions and passive stretch) were limited to 3, but between these sessions, a lidocaine iontophoresis was used continuously (24 hour period). The key clinical outcome was a dolorimetric measure over the lateral epicondyle before each session and 1 week after the final (3rd) session.

There was significant improvement between the baseline and the pre session 2 and 3 sessions, both on the dolorimetric and functional (based on interview data) outcome measures. The iontophoresis intervention was a home based programme using a specific piece of equipment which effectively operated between the sessions with the therapist (details about current and electrodes etc in the main paper). It is suggested that the prolonged treatment, but one that utilises a low current density should result in a slower transcutaneous flux, but clearly over longer time periods.

The authors clearly recognise the limitations associated with case study designs in general terms and with some salient issues for this particular trial, but having said that, even without control groups or RCT style designs, the outcome of this work is very interesting.

Cranial electrical stimulation

OK, now for something completely different as they say – cranial electrotherapy for patients with mental health problems – not run of the mill electrotherapy for most of us, but absolutely fascinating none the less. There have been numerous reports over recent years concerning the use of electrical stimulation for patients with depression and memory loss type problems, but this is the first one the I remember concerning aggression. Childs (**Childs, A. (2005). Cranial electrotherapy stimulation reduces aggression in a violent retarded population: a preliminary report. J Neuropsychiatry Clin Neurosci 17(4): 548-51**) has published previously (1988 that I know of and maybe additionally) in this field, but this report concerns the treatment of nine aggressive, retarded patients at a maximum security hospital, all of whom were not responding to conventional care.

A three month programme of cranial electrotherapy was employed, and the paper follows a case study style, but the summary results include a significant reduction in aggressive episodes (by almost 60%), a reduction in seclusions and restraints and furthermore, a reduction in requires sedatives. Importantly, there were no side effects of note and the intervention does offer a potential form of intervention for this difficult to manage client group.

The patients had a range of diagnoses, mental health durations and coexisting conditions, including 4 patients with epilepsy. The stimulation employed was applied through electrodes attached to the ears using a specific low current (max 600 microamps, but was actually applied at a subsensory threshold) at 0.5 or 100Hz applied frequency for up to two hourly treatments daily.

The outcomes evaluated were aggressive episodes, seclusions, restraints and the use of medications in this highly regulated environment. The paper is a most engaging read, and although only a

limited number of the cases are presented in any detail, it is a preliminary report, and the results cover all those involved.

Electrical stimulation and muscle related outcomes

Alex Ward (mentioned in the last newsletter as one of the new authors of Electrotherapy Explained) and colleagues all from LaTrobe University in Australia have evaluated two different current forms in terms of their capacity to generate muscle force (wrist extensors) and also the discomfort associated with the stimulation. (**Ward, A. R. et al. (2006). Wrist extensor torque production and discomfort associated with low-frequency and burst-modulated kilohertz-frequency currents. Phys Ther 86(10): 1360-7.**)

This was an RCT design with 32 asymptomatic subjects drawn from the staff and student populations at the university. The applied currents were essentially a Russian type stimulation and a current that they referred to as a 'Aussie stimulation'. The Russian current stimulation was at 2.5kHz AC with bursts at 50% duty cycle – 10ms ON then 10ms OFF giving an effective bust stimulation frequency of 50Hz. The Aussie stim was at 1kHz AC with a 20% duty cycle (4ms ON followed by 16ms OFF also giving an effective stimulation frequency of 50Hz). Two more conventional electrical stimulation modes were also included in the comparison – both monophasic pulsed, both at 50Hz effective stimulation frequency (20ms cycle) one with pulses of 200 microseconds and one with pulse duration at 500 microseconds. The 200 microsec pulse stim was equated with the Russian stim and the 500 microsec pulses with the Aussie current. There is a clear rationale for these comparisons that are detailed in the preliminary section of the paper.

It was hypothesised that the Aussie current would generate less discomfort than the other current forms though it was anticipated that the force generated would not be significantly different. The Russian stim was expected to be more comfortable than the monophasic pulsed forms but would generate lower levels of muscle force.

The method is nicely outlined in the paper. A within subject design was employed, with all subjects attending for 4 sessions, experiencing all stimulation modes (in a randomised order). Stimulation was applied (subject in control) at maximum tolerance whilst wrist extensor muscle torque was recorded. Applied voltage, applied current and muscle torque were recorded and averaged over three attempts. Subjective opinions from the subjects concerning discomfort of the stimulation were also identified.

As one might expect, there are plenty of results discussed in the paper, and I will only summarise the essentials here : In terms of the force generation, the Aussie current generated the highest and the Russian Stimulation the lowest. Some complex ANOVA and posts hoc analyses showed that the Russian Stim generated significantly less torque than the others stimulation modes. Although the results are statistically significant, when one looks at the actual values, there is not a very big difference between the stimulation modes, and when one considers clinically significant differences, I would not have thought that there was a lot between them – hence the 'discomfort' factor may become even more important.

In terms of the discomfort experienced, the AC stimulation modes (Russian and Aussie) were more comfortable (well, maybe less uncomfortable) than the DC stimulations, which was supported by the statistical analysis.

In terms of the muscle force, the Aussie current was just in the lead, and in terms of discomfort, the Russian stim was the best closely followed by the Aussie current. The authors argue therefore that

the Aussie current provides the ‘best of both worlds’, but in some respects, they would, wouldn’t they????

This trial did not attempt to evaluate their clinical effectiveness in terms of muscle strengthening programmes or rehabilitation with ‘real’ patients. Hopefully this is on its way as it would be of more clinical relevance.

Direct clinical relevance aside, this is an interesting paper and opens up the opportunity for renewed interest in AC type currents as an effective means of achieving muscle stimulation in the clinical environment in contrast to the pulsed DC type currents which appear to be the more popular option at the present time.

Clinical review : Treatment for TMJ problems (including electrotherapy)

OK, a complete change of tack for the next paper which is a clinical review regarding various interventions for TMJ disorders (**Medlicott, M. S. and S. R. Harris (2006). A systematic review of the effectiveness of exercise, manual therapy, electrotherapy, relaxation training, and biofeedback in the management of temporomandibular disorder. Phys Ther 86(7): 955-73.**

There have been several papers along these lines in recent years (an excellent one on Bells Palsy for example), and this paper looks at a variety of treatment approaches, including aspects of electrotherapy, but also includes exercise, manual therapy, relaxation and biofeedback.

Thirty studies were selected for review, and the selection criteria and procedures are identified in the review. Of the 30 studies, 14 related to exercise or manual therapy, 8 to electrotherapy, 7 for relaxation or biofeedback and one looked at a combination of electrotherapy and exercise. The majority of the studies selected were RCT’s (almost predictable in a review of this type) though there were some pre-post test designs, case series and a crossover design. The authors do spend some considerable time in the paper identifying their methods, their rigour and reliability and validity issues (which is always good to see).

In terms of the electrotherapy bits, the laser therapy was the only one that the authors suggest is supported from the evidence that they reviewed. There were a couple of problems with this, though they have done the work and I havn’t, so all credit to Medlicott and Harris for doing the donkey work in the first place. Several of the papers did look at mixed intervention (rather than only one manipulated intervention). The mixed model is much closer to reality for most therapists, though in a review of this kind, it is very difficult to analyse. There are other aspects, but one that concerns me (as it has for some while) relates to the outcome measures employed. There were actually over 75 different measures employed between the thirty papers and the reliability and validity were only reported in a minority of them. Clearly, this limits the strength of the conclusions reached. To base conclusions on results derived from work in which the outcome measures have not been fully evaluated is risky at the very least, and potentially dangerous in terms of jumping to conclusions.

Anyway, if you are involved in TMJ work on a regular basis, you will almost certainly be aware of the paper anyway. For those of you that only see these problems intermittently or occasionally, it is a useful review, well explained and limitations included, and would be worth a read.

Heat & Ice

I get lots of requests for information relating to both heat and cold therapies. These are not my primary research areas though I have done some work in both fields. I have included three recent studies which may have passed you by and may be of clinical interest.

Heat & Ice : Heat wrap and low back pain

Mayer et al (**Mayer, J. M. et al. (2006). Continuous low-level heat wrap therapy for the prevention and early phase treatment of delayed-onset muscle soreness of the low back: a randomized controlled trial. Arch Phys Med Rehabil 87(10): 1310-7**) have previously published in this area, using continuous heat wrap for low back pain (Mayer (2005). Spine J 5(4): 395-403). The 2006 paper is more concerned with DOMS (delayed onset muscle soreness) in the low back post exercise. There have been MANY people who, over the years, have tried to find a way to deal with DOMS type pain, and numerous kinds of electrotherapy have been researched with somewhat mixed results. The study involved 67 'normal' males (no low back pain) who exercised intensively in order to generate a DOMS effect (primarily involving eccentric exercise). Some were pre treated (i.e. as a preventative approach) and some were post treated (i.e. as a way to deal with the problem in a classic treatment sense). There were therefore 2 main groups (preventative and treatment) and within the preventative, there were heat wrap (n=17) and control (stretch) (n=18) groups. Within the treatment group, there were heat wrap (n=16) and cold therapy (n=16) groups.

The main outcome measure was pain (as one might expect) but importantly, it was the **pain intensity** that was the most important measure for the preventative groups and the **pain relief** for the treatment groups. Secondary outcomes included function and disability.

Essentially, the preventative use of heat wrap gave useful improvements in the pain intensity scores, disability and physical function (varying between 45 and 53% better than the controls). In the treatment group, the heat wrap group had much greater pain relief than the cold therapy group (almost 140%) but no differences in disability or function.

It would appear that the long term heat wrap use has both a preventative and therapeutic role in this type of DOMS pain – something that other investigators have not found easy to demonstrate with other interventions. Although for many therapists, dealing with DOMS is not the major issue in a days work, for some, especially those working with sports clubs or practice with those undertaking exercise / training etc, this might be well worth considering. For those of you more concerned with the clinical aspects of back pain, have a look at the 2005 paper (cited in the first paragraph) will see that hot wrap therapy appears to have clinical benefits too.

Heat & Ice : Ice and ankle sprains

It is almost unfair to put this next paper so near to the end of the Electrotherapy News, as it is an excellent paper and a great follow on from a great review by the same authors in 2004. Bleakley (**Bleakley, C. M. et al. (2006). Cryotherapy for acute ankle sprains: a randomised controlled study of two different icing protocols. Br J Sports Med 40(8): 700-5**) looked at using an intermittent versus a 'traditional' ice treatment following acute ankle sprains using an RCT design.

A mixed group of 89 subjects (44 sportsmen and 45 members of the general public) with mild/moderate acute ankle sprains were recruited to the trial who were then randomised to one of two different cold treatment protocols. The standard ice treatment group (n=46) consisted of a standard ice pack applied for 20 minutes every 2 hours (with towelling interface) being a representation of commonly utilised clinical application. The intermittent ice application used the same standard ice pack, but was applied for 10 minutes, then 10 minutes with no pack (room temperature) and then a further 10 minutes ice pack. This protocol was also repeated every 2 hours. Both groups followed the protocol for the first 72 hours. Treatments were self administered by the patients and a diary was kept in order to monitor compliance. A basic exercise programme was included for both groups.

The key outcome measures were pain, function and swelling, recorded at 1, 2, 3, 4 and 6 weeks post injury. A VAS was used for pain, Binkley's lower extremity function scale for function and swelling was recorded with a specific (figure of 8) tape method. Further details of the method, validity of the outcomes and procedures are provided in the paper.

The results are interesting. Function and swelling improved significantly in both groups over the 6 week measurement period, but there was no difference between the groups. Pain similarly improved over the treatment and follow up periods, but there was a significant difference between the two treatment groups, with the intermittent ice treatment group doing significantly better in the first week.

The authors make some interesting and valid points in the discussion which would be worth a read for anybody involved in these type of acute injuries or those that provide advice for patients to self manage in the 'acute' period post injury. The fact that the intermittent ice protocol reduces tissue temperature more effectively and for longer than the standard ice treatment may account, in part at least, for the differences in pain between the groups, though, as ever, there are other possible explanations.

Intermittent ice application appears to offer an advantage, mainly related to pain relief in the first week, and would be worthy of inclusion in the instructions / advice offered to patients, especially given that the other benefits (in terms of swelling and function) were just as good as for the standard ice therapy group.

Heat & Ice : Cryocuff and TA microcirculation

There seems to be an Achilles Tendon paper in just about every issue of this newsletter, and to add to the debate about TA circulation (which I am always happy to join in with), lets try this paper by Knobloch and colleagues (**Knobloch, K. et al. (2006). Changes of Achilles Midportion Tendon Microcirculation After Repetitive Simultaneous Cryotherapy and Compression Using a Cryo/Cuff. Am J Sports Med 34(12): 1953-1959**) is the second paper out in 2006 by this team and involves a group of (26) healthy subjects rather than the more commonly encountered rat or rabbit experimentation. The main area for the investigation is the Cryo Cuff device known to many clinicians by reputation even if not from experience.

The basic investigation involved the repeated application of the Cryo Cuff for 3 x 10 minute periods and a 10 minute follow up phase. The microcirculation of the mid part of the TA was assessed using a laser Doppler device.

None of the subjects (13 men and 13 women) had any TA or ankle problems. One of the key outcomes was the Foot and Ankle Outcome Score (FAOS) and the pre-intervention activity levels and the FAOS scores are explored in the paper. The investigators had previously assessed TA microcirculation during the Cryo Cuff application, but had not included a follow up phase. This work therefore extended their previous work, with a total of 1 hour measurement reported here.

The Doppler system used (details in the paper) evaluated the microcirculation in two different area of the TA, and was a non invasive technique. The system measures haemoglobin levels, oxygen saturation and blood flow (including velocity). The measurements are reported in Arbitrary Units, and although this method has been previously criticised, the authors rationalise why they adopted this approach, and basic reliability results from a previously conducted piece of work suggest that a 5% variability (intrasubject mean) is expected.

The Cryo Cuff technique essentially went like this : water in the system was at 15 degrees C ‘with additional crushed ice in the system’ (which seems a tad vague to me).The probe was placed at 4cm proximal to the TA insertion point in the calcaneus. Baseline assessments were made prior to intervention, and flow measures were taken at 2 and 8mm depths. Following the filling of the system, measurements were taken through continuous monitoring with mean data recorded every 20 seconds. The Cryo Cuff was applied for 10 minutes followed by 10 minutes rest, with this sequence repeated twice further.

The results are complex and reported in some considerable detail. In summary, the tendon O₂ saturation dropped significantly during each cold/compression application, with a significant increase in the recovery period – up to 83% above the baseline level. Superficial capillary blood flow was reduced at each cold/compression cycle and increased during each recovery phase. Interestingly, the deeper blood flow was also decreased during the intervention periods but without the recovery between treatment periods.

The authors conclude that the Cryo Cuff in 3 x 10 minute periods with 10 minutes rest between each resulted in a significant reduction in capillary blood flow. The post capillary filling pressures (detailed in the results) were reduced during compression (which effectively means that there was an increase in venous outflow from the area). It is suggested therefore that these combined results combine to generate beneficial effects on the microcirculation.

Whether you can get to grip with the full data set or not, there is no doubt that there are changes that are demonstrated in TA midportion bloodflow with Cryo Cuff application.

The full results are complex and take some working through. I tried to summarise them in more detail and found myself simply replicating everything that the authors had said, which is a bit pointless when you can read them for yourself!!!

Tissue Repair : Steroids and collagen behaviour

Last paper for this issue relates to one of my favourite old chestnuts of inhibited tissue repair as a result of drug therapy. Haraldsson et al (**Haraldsson, B. T. et al. (2006). Corticosteroids Reduce the Tensile Strength of Isolated Collagen Fascicles. Am J Sports Med 34(12): 1992-1997**) looks at the effect of steroid therapy on collagen fascicles, though it is important to note that this is not a clinical trial and nor does it look at the effect of steroids on the whole tendon – just the isolated fascicles which were derived from rat tail.

Essentially, the collagen fascicles were incubated in either high or low concentration steroid (methylprednisolone acetate – Depromedrol) for 3 or 7 days. There was a control condition in which the collaged was incubated in saline. Following the test period, the fascicules were mechanically tested to failure in a rig and following that, there was a biochemical assessment made (cross links)

The results (in brief) showed that in the low and the high strength steroid solutions, there was a significant reduction in strength compared with the controls at 3 and at 7 days. There was no significant difference between the high and low concentration interventions. The cross linkage results did not demonstrate a significant effect.

The clinical implications are not necessarily directly transferable as whole tendon structure was not evaluated – only single fascicles. It is important to have shown that exposure of collagen fascicles to steroid does most definitely reduce the strength of the material, and this is consistent with numerous previous studies that have demonstrated reduced tendon strength following steroid treatment.

The authors do make some comparisons with the work of others – mainly animal experimentation, but also do relate the failure stresses demonstrated in this work with the stresses applied through the TA for example on strenuous activity. Steroid injection into tendon has been shown to have pain relief type effects, but is also known to weaken the tissue structure and appears to predispose to future damage and possible failure. This experimentation adds significantly to that debate.

OK, so that will be plenty enough for this issue. I hope that something in there was of some interest. The next edition is due in March (or thereabouts), but in the meantime, if you have any queries, issues or papers that you think I have missed and should be included, please let me know (t.watson@herts.ac.uk)

In the meantime, Happy New Year.

Regards

Tim



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