Electrotherapy News

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Hi there and welcome to the latest missive. There are some really impressive papers in this issue (but I guess I always think that!). Just before we start, couple of 'news' items for you. Sponsorship of ElectroNews has been taken over by EMSPhysio Ltd – to whom I am grateful. I am happy to try and keep this update going, to review the papers etc etc, but it is not free as such, and without sponsorship, it would simply stop. EMSPhysio have kindly agreed to support the venture for the next year, so all being well, there should be a continuation for the foreseeable future.

The new edition of Electrotherapy Explained is out – some of you may have seen it already. John Low and Ann Reed (the original authors) have been joined by Val Robertson and Alex Ward, and in fact Robertson and Ward are now the lead authors. The text, now in its 4th edition, is published by Elsevier which should make it reasonably easy to get hold of (if having trouble, try www.elsevier.com) ISBN is 0-7506-8843-2. There is a 12th edition of Electrotherapy : Evidence Based Practice coming out next year (2007) and I am editing that edition (between doing the web and writing newsletters . . . . Looks good from this end at the moment, so watch this space!

I was trying to update the web site over the summer – got some of the text updated and new links etc all done, but the major overhaul and complete restructure did not get done I am afraid – not that it makes any difference to content I appreciate – just trying to make it more accessible and compliant with regulations etc etc. Still working on it and with any luck should not be too long.

There seems to have been a flurry of publications of mine out in the last couple of months. I have only really picked on one of them for inclusion in this newsletter (Al Mandeel and Watson 2006) – seems a bit cheeky to get all my own stuff in here – but the others, including papers on Tissue Repair (Watson), one on Electrotherapy and Tissue Repair (Watson), one on physiological changes with manual therapy (Moulson and Watson) and one that I may have mentioned before on Long-wave Ultrasound and Heating (Meakins and Watson) are all listed on the web pages. There is a paper coming out (Poltawski and Watson : expected Jan 07 – to be confirmed) which has investigated the differences in transmission characteristics of US coupling gels – does it make any difference which one you use? I'll let you know as soon as it comes out and include a summary of it in the newsletter. No point doing it now as you'll not be able to access the paper – it has been accepted (Ultrasound in Medicine and Biology) but currently not even available online as a 'forthcoming paper'.

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Contents for Vol 2 Issue 3

Ultrasound, NSAID's and Ligament Healing

The first paper in this issue is one that I got just too late to go into the last one, though I did promise it was coming. Warden et al (***Warden, S. J. et al. (2006). Low-Intensity Pulsed Ultrasound Accelerates and a Nonsteroidal Anti-inflammatory Drug Delays Knee Ligament Healing. Am J Sports Med 34(7): 1094-102***) have produced an excellent piece of work looking at the potential influence of these two interventions that are commonly employed in relation to soft tissue injury. Warden, as you may well appreciate, has published a range of papers concerning ultrasound and fracture healing amongst others, but this particular piece aimed test the hypothesis that low-intensity pulsed ultrasound accelerates ligament healing, a nonsteroidal anti-inflammatory drug delays healing, and the nonsteroidal anti-inflammatory drug inhibits the beneficial effect of low-intensity pulsed ultrasound. This would be consistent with the prevelant evidence out there, and would be in keeping with the information I have collated on both US and NSAID's. This was a laboratory experiment using a rat injury model.

60 adult rats underwent bilateral transection of their knee medial collateral ligaments. There were basically 2 different drug groups in the trial – one got the NSAID (celecoxib) combined with what is in effect a carrier solution. The others got just the carrier solution. The drug was delivered 5 days a week. Following drug administration animals were given low dose pulsed ultrasound to one knee and 'inactive' ultrasound to the other knee. Animals were sacrificed at intervals (2, 4 and 12 weeks) and the mechanical properties of the ligaments tested. Ultrasound was applied with a LIPUS device (very similar to those employed in the fracture healing studies – see web site) delivering at 1 MHz, 0.01 W/cm-2 pulsed (effectively) at 1:4 at 100Hz, 20 minutes daily, 5 days a week. [This dose is VERY low but was based on Warden's previous work – again see web site - /modalities / ultrasound / fracture healing for further information – or just read Warden (2003) Sports Med 33;95-107]

The experimental design resulted in 4 different Rx combinations – Carrier + placebo US, Carrier + Real US, NSAID + Sham US, NSAID + Real US. In summary, the results showed that after 2 weeks

of treatment, ligaments treated with active low-intensity pulsed ultrasound (LIPUS) were 34.2% stronger, 27.0% stiffer, and could absorb 54.4% more energy before failure than could ligaments treated with inactive LIPUS. The ligaments from the NSAID group could absorb 33.3% less energy than could ligaments from the Carrier group. There were no ultrasound or drug effects after 4 and 12 weeks of intervention. Interestingly ultrasound and drug intervention did not interact to influence ligament mechanical properties at any time point.

Warden et al conclude that low-intensity pulsed ultrasound (LIPUS) accelerated but did not improve ligament healing, whereas the NSAID delayed but did not impair healing. When used in combination, the beneficial low-intensity pulsed ultrasound effect was cancelled by the detrimental NSAID effect, and reading through the paper in some detail, this seems to be an appropriate conclusion. The authors further suggest that low-intensity pulsed ultrasound after ligament injury may facilitate earlier return to activity, whereas NSAID's may elevate early reinjury risk.

This would be a good paper to read through the detail rather than just the abstract, but essentially it does appear to demonstrate beneficial effects of very low dose ultrasound – lower than most therapists would deliver in routine clinical practice – and this is in keeping with numerous research papers over recent years. It also demonstrates that NSAID application following soft tissue injury does have an inhibitory effect on the rate of healing – see web site for loads more references.

Ultrasound and Ligament Repair

Another 2006 paper on a similar version of the ligament / ultrasound research theme is provided by Leung et al (***Leung, M. C. et al. (2006). Therapeutic ultrasound enhances medial collateral ligament repair in rats. Ultrasound Med Biol 32(3): 449-52***). The research employed a rat model (as did Warden), and 36 rats had their medial collateral ligament transected. The primary outcome measure was TGFbeta (TGF-â) levels (rather than the commonly applied mechanical testing). TGF-â is a cytokine that has been shown to be of some considerable importance in tissue repair following injury / trauma (see web pages – tissue repair for summary). The ultrasound was delivered at 3MHz pulsed 1:4 using an under water application technique. Treatments were of 5 minutes duration, and different intensities were compared (0.0W cm-2, 0.5 W cm-2 and 2.3 W cm-2). Treatments were carried out daily for either one, 5 or 10 days. There were therefore in effect, 9 experimental groups with 8 animals in each (3 different intensities – control, low and high) and 3 different durations (1,5 and 10 days).

The results showed that TGF-â was not detected in the 1-day group. In the 5-day and 10-day groups, the levels of TGF-â were significantly up-regulated in the high-dose subgroup ($p < 0.05$). The 10-day group also registered a significantly higher expression of TGF-â than did the 5-day group ($p < 0.05$). The authors propose that these results suggest that pulsed US therapy may enhance ligament repair by up-regulating the extent of TGF-â in a high-dose application. Long-term treatment with this therapy could obtain further improvement.

Their conclusion would certainly be consistent with their results, but it is of considerable interest (to me anyway) that in this case, it was the high dose of US that achieved the most significant results (and for that matter, the more treatment sessions the better). The Warden study reported above used a very low dose but also demonstrated a significant outcome – the difference between them was from 0.01 to 2.3 W cm-2, both using a rat medial collateral injury model. Now there may have been some differences in the way in which these doses were reported, but even so, this is an apparently massive differential. It would be interesting of course to use the TGF-â outcome measure with the Warden design and the mechanical testing with the Leung design to get a more complete picture, but I guess more rats and a whole lot more expense would be involved.

Ultrasound and Fracture Healing

OK, another couple of US papers before we move on to another modality, but in keeping with previous newsletters, it would be difficult not to include a fracture healing paper, so try ***Malizos, K. N. et al. (2006). Transosseous application of low-intensity ultrasound for the enhancement and monitoring of fracture healing process in a sheep osteotomy model. Bone 38(4): 530-9***. Malizos actually has 2 papers out this year – the other one is more of a review paper [Malizos, K. N. et al. (2006). Low-intensity pulsed ultrasound for bone healing: An overview. Injury **37**(Suppl 1): S56-62] but is also a good paper on the subject. Anyway, in the Bone paper, the authors aimed to look at two things : (a) to investigate the application of transosseous low-intensity pulsed ultrasound (LIPUS) on the enhancement of fracture healing and (b) to demonstrate the ability of transosseous ultrasound propagation to monitor the healing process

The researchers used a sheep model (makes a change from rats) and generated a midshaft tibial osteotomy which was fixed with an external device. The ultrasound was applied using implanted US transducers (not, I appreciate a realistic proposition for most therapists) and animals were allocated to either a treatment or a control group. The active US group received US at an almost predictable dose (for fracture healing), in keeping with almost all other work done in this field [1MHz, 0.03 W cm-2, pulsed 1:4 at 1kHz, 20 minutes daily]. Tissues were tested at 100 days post operatively. I'll not go into massive detail here – plenty of information in the full paper if you want it – but essentially the outcome measures included radiographic analysis, bone density and mechanical (3 point) testing.

The results demonstrated significantly higher probability of radiographic healing for the animals in the treatment group ($P = 0.009$) and there were also significant differences for Bone Mineral Density, breaking load, strength, stiffness and Young's modulus – all in favour of the US group. The secondary intention of the study – to look at the potential value of the US as a fracture healing outcome measure - was also clearly demonstrated.

I have included the paper in the newsletter in that it adds to the body of evidence with regards the application of US in fracture healing. I appreciate that the majority of you will not be using implanted US transducers in your patients, but that does not detract from the value of this piece of work.

Ultrasound and Bone-Tendon Healing

On a similar theme, but with a bit of a twist is a recent paper from Hong Kong ***(Lu, H. et al. (2006). Low-Intensity Pulsed Ultrasound Accelerates Bone-Tendon Junction Healing: A Partial Patellectomy Model in Rabbits. Am J Sports Med.34(8); 1287-1296***).

Following the same rationale as the previous Warden paper, it is argued (quite reasonably) that LIPUS (low intensity pulsed ultrasound) has been demonstrated as effective in fracture healing, so does it have an effect at the bone-tendon junction. The experimental work employed a rabbit injury model with 48 rabbits undergoing a partial patellectomy. Some animals were treated with ultrasound whilst others acted as controls. Animals were sacrificed at various intervals between 2 and 16 weeks. The US, started at 3 days post operatively using the Exogen device (1.5MHz, 0.03 W cm-2 pulse 1:4 20 minutes daily as one might predict – seems to be the standard dose in these trials). Treatment was made daily up to the time of sacrifice and was delivered through a window cut in the immobilisation cast using a standard gel couplant. A range of outcome measures were em-

ployed focusing on radiographic evidence, biomechanical testing and histology (plenty of detail in the paper).

The radiographic measurements showed significantly more newly formed bone at the patellar tendon-patella healing junction in the ultrasound group compared with the controls at weeks 8 and 16. Histologically, the ultrasound group at weeks 8 and 16 showed improved tissue integration, characterized by trabecular bone expansion from the remaining patella and regeneration of fibrocartilage layer at the patellar tendon-patella healing junction. Fluorescence microscopy revealed earlier bone formation in the ultrasound group when compared with the controls at weeks 8 and 16. Mechanical testing showed significantly higher failure load and ultimate strength in the ultrasound group compared with controls at week 16.

The authors conclude that low-intensity pulsed ultrasound was able to accelerate bone-to-tendon junction repair and therefore could be used in the clinical environment to accelerate bone-to-tendon junction repair and facilitating earlier rehabilitation.

A nicely constructed piece of research and it certainly adds to the swiftly growing volume of material in this field. For anybody interested, there are the best part of 40 references to chase up covering many of the papers that I have been citing for some time plus some others which are well worth a look at.

Ultrasound and Chronic Low Back Pain

The last US paper for this edition is much more clinically focussed. ***(Mohseni-Bandpeia, M. A. et al. (2006). A prospective randomised controlled trial of spinal manipulation and ultrasound in the treatment of chronic low back pain. Physiotherapy 92(1): 34-42)***.

The trial was an RCT design comparing the effect of manipulation and exercise treatment with ultrasound and exercise treatment. The trial was of good basic size with 120 patients with Chronic LBP recruited and allocated to either a manipulation / exercise group or an Ultrasound / exercise group. The exercises employed were the same for both groups in that they were generated from PhysioTools software, but were matched to the individual needs of the patient. The manipulation group (detailed in the paper) followed a Maitland type approach, but did include a high velocity thrust – not just mobs – and patients were seen between 2 and 7 times, mean of 4 sessions. The ultrasound was delivered at 1MHz, continuous using between 1.5 and 2.5 W cm-2 for between 5 – 10 minutes. Patients received between 3 and 11 sessions – mean of 6 sessions.

Outcomes (blinded) included a raft of measures including pain intensity, functional disability, ROM, EMG and endurance capacity. These measures were taken throughout the treatment programme and also at a 6 month follow up.

In summary the results showed that following treatment, there was a significant improvement in pain, ROM and function in both groups (not included in the abstract by the way). Patients in the manips group demonstrated a significantly greater reduction in pain and functional disability and greater ROM (short term). There were some differences in the EMG outcomes between groups, but not for other EMG elements. At the 6 month follow up (questionnaire) results show that both groups still demonstrated significant improvement compared with pre-treatment, but there was a demonstrable advantage to patients in the manips group.

The authors conclude that although there were improvements in both groups (which there clearly were), patients receiving manipulation/exercise showed a greater improvement compared with

those receiving ultrasound/exercise at both the end of the treatment period and at 6-month follow-up

There are more details of the results and analysis in the main paper, but apart from anything else, it shows the importance of reading the whole paper rather than just the abstract. In this case, the abstract focuses on the advantage of the manips group over the US group (which is fine – it is a part of the results) but almost fails to mention the fact that the US group made significant improvements over their pre-treatment levels.

Anyway, I am not trying to rubbish the results because the manips groups did better than the US group – far from it. There are a couple of important points to make and then I'll move on. One is that US combined with exercise has a measurable and significant beneficial effect for this patient group. Manipulative therapy appears to have additional benefits. One would most like to know the answer to two additional questions – which can not be answered by this design. One is, what would happen to a control group – or a group that only got the exercise – in other words, a treatment that the patient could do on a DIY basis and therefore not need to 'attend' therapy? It would be interesting to know if either the manips or the US offered any advantage over and above the tailored exercise regieme. The other thing that would be of interest, is what happens if one adds an US intervention to the manips – the patient is coming for Rx anyway, so if whilst they were there, does adding the US Rx (few minutes extra) add anything to the outcome?? Clearly, one would need a far larger trial to be able to answer these issues, but can't deny that it would be interesting to know that answer to that! I do, by the way, have some issues with the US dose (which is a bit vague) being based on a 1985 textbook and not being related to any research based parameters – but I'll save that for another day).

I do have a couple more US papers, but will save them for the next edition before you start thinking that I need to change the name of this to UltrasoundNews!!!

Ice and Electromagnetic Fields post Fracture for Pain and Swelling

A 2005 paper that looks at an area previously considered in the UK (Buzzard et al 2003). The research (although not from this year – apologies) also comes from the Hong Kong camp with Cheing as the lead author ***(Cheing, G. L. et al. (2005). Ice and pulsed electromagnetic field to reduce pain and swelling after distal radius fractures. J Rehabil Med 37(6): 372-7)***.

This was a clinical rather than an animal / lab based study to which 83 patients were recruited, all of whom had a 'straightforward' distal radial fracture with no real complications. They had all been immobilised for 6 weeks in POP and were divided into 4 groups – ICE and PEMF, ICE and Sham PEMF, PEMF alone or Sham PEMF – effectively the control group. The PEMF applied was using a Pulsed Magnetic Field Therapy System (from Australia it seems), delivered at 50Hz, intensity of 99 gauss for 30 minutes. The ice was applied with a standardised ice pack to the dorsal wrist area for 30 minutes. Sham PEMF applications delivered no energy to the patient, though the machine appeared to be working. All patients were also given an exercise programme to be conducted at home – details in the paper. I couldn't see (but may have missed it) which order the combined ice / PEMF was delivered in i.e. ice first or PEMF first – not sure that it matters, but I am aware that there are

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

plenty of stories around out there with regards the sequence / order of these combined treatment. Anyway, I will let you know if I get to find out – am lecturing in Hong Kong later in the year, and from the programme, it looks like this paper is on the presentation list, so maybe, more details to follow

Outcome measures included a VAS for pain, swelling measured by volumetric displacement (much better than the good old circumferential measures) and also ROM using a standard goniometer. The authors argue that this is a reliable measure so long as reference markers are used. There are papers out there that would challenge this, but that is for another day! Measures were recorded at the start of the trial and then before the intervention on days 1, 3 and 5.

The results indicate that at day 5, a significantly greater cumulative reduction in the VAS and the ulnar deviation ROM was found in the combined ice & PEMF group than the other 3 groups. For volumetric measurement and pronation, participants in the combined Rx group performed better than subjects in the control group but not those in the ice and sham PEMF group.

The authors conclude that ice is effective (with exercise) for this patient group, but that the addition of a PEMF Rx to ice therapy produces better overall treatment outcomes than ice alone, or pulsed electromagnetic field alone in pain reduction and range of joint motion in ulnar deviation and flexion for a distal radius fracture after an immobilization period of 6 weeks.

There are some interesting issues raised in the discussion, including questions with regards treatment dose, therapeutic windows – an issue close to my own current work – see the updates 'Current Concepts in Electrotherapy on the web if interested - and also raises the sequence issues I mentioned above.

Pulsed Shortwave in Clinical Practice

OK, so this time I will include one of my own papers – not for advertising – just for information I guess! ***Al-Mandeel, M. and Watson, T. (2006) An audit of patient records into the nature of pulsed shortwave therapy use. International Journal of Therapy and Rehabilitation 13(9);414-419***.

This is the first paper of several relating to an extensive piece of work done on Pulsed Shortwave – this one looking at where it is used in the clinical environment, what kind of things it is used for, parameter settings and record keeping. The research programme that followed looked at both laboratory and clinical studies with PSWT, but this was the preliminary work to set the scene. A random selection of hospitals from around the country were selected, and from trawling the records available, we were able to identify to what extent PSWT was actually used (average was about 11% of all treatments) and for what type of clinical problems (massive range, but predominantly musculoskeletal problems, OA and fractures). Treatment parameters that were employed by therapists were also evaluated with the aim of using this data to inform the later lab and clinical trails. Interestingly, the majority of treatments were delivered either once a week or on a single occasion. Treatment was most commonly delivered for 10 minutes and trying to establish the commonly used 'progression' of treatment turned out to be very problematic. It was interesting that these commonly used 'settings' did not seem to match the (limited) research evidence published to date.

As an addendum to this evaluative audit, we were able to look at the treatment records and found (somewhat disturbingly) that not one of the 192 treatment records examined actually included all the information that one would need to know in order to meet documentation (and hence) legal standards. This is not to suggest that record keeping with PSWT is any worse than for any other intervention – we did not look at the others – but of some concern none the less, especially given

current level of litigation (real or threatened), and even without bringing litigation into the equation, it is worrying that it would appear not to be possible to pick up a set of treatment notes and follow through the treatment with confidence. Interestingly, the one thing that all notes did include was the treatment time.

Anyway, the research continued from this point and went on to include a full survey relating to practice and then an extensive laboratory and finally a clinical trial. These papers are in preparation and will let you know where and when they get published. The key findings from this paper are that pulsed shortwave, as a therapeutic modality, is still reasonably widely used in the clinical setting in England, that it is considered to be effective by therapists, but that the quality of the patient records leaves something to be desired.

Change tack now, off to several papers concerned with electrical stimulation in various forms

TENS and Postural Sway

This is a healthy subject study that evaluates the effect of TENS on postural sway ***(Dickstein, R. et al. (2006). TENS to the posterior aspect of the legs decreases postural sway during stance. Neurosci Lett 393(1): 51-5)***. Thirty subjects were tested under 4 different TENS application conditions – no TENS, Bilateral TENS, Unilateral TENS to left / right. Subjects were on a force platform. Various sway parameters were recorded in a reasonably complex design, with each subject being tested under each condition in each of three blocks, with randomised sequencing. Vision was allowed in some cases and not in others. TENS was applied with surface electrodes over the gastrocnemius muscle(s) using stimulation at 100Hz, 200ìsec duration, bipolar zero net DC stimulation set at the sensory threshold. Tests were for 30 seconds with a sufficient rest period between tests to prevent fatigue. The force plate and associated software was used to determine the average sway velocity of the centre of pressure.

In short, the results demonstrate that the application of TENS brought about a decrease in postural sway, in addition to a decrease in the absolute values of maximal and minimal medio-lateral and anterior-posterior velocity.

Thus, similar to sub-threshold random electrical noise, it appears that the application of low-amplitude TENS to the lower limbs decreases postural sway during stance. So far as I can see from the results, there was no significant difference between the various TENS groups) although there was a trend for the (L) side stimulation to be more effective. The difference was between the TENS groups and the control (no TENS).

There have been suggestions in the literature, and a couple of case study papers that have proposed this type of intervention as a component of neuro rehab programmes (stroke patients in particular) and the authors suggest that this could be an avenue for future work plus the critical question as to whether the beneficial effects last longer than the stimulation period – which would be of some importance in the clinical environment I would suggest. Anyway, it is an interesting pa-

per that adds to our knowledge in this field and provides a platform for more clinically focused research to follow.

Interferential and Analgesia

As has been identified previously, Interferential is not exactly the most comprehensively researched of the electrical stimulation modalities, so it makes for a nice change to be able to include a couple of useful IFT papers in this edition.

The first one, from a German research group, looks at the analgesic efficacy of IFT for patients with psoriatic arthritis ***(Walker, U. A. et al. (2006). Analgesic and disease modifying effects of interferential current in psoriatic arthritis. Rheumatol Int: 1-4)***. It has been argued that IFT can influence the behaviour of psoriasis, possibly via a cAMP related mechanism, and this group set out to investigate whether IFT provided analgesia and / or disease modifying outcomes. It was a case series, and was in effect a pilot study rather than a full blown RCT. Nine patients with psoriatic arthritis were recruited (peripheral joint involvement – not axial). Interferential was delivered using a 2 pole application method, with immersion in a water bath for the small joints of the hands and feet. Treatments were done at home by the patient every day with a morning 5 minute session at 100Hz and an evening 5 minute session at 10Hz. The current was applied a just above the sensory threshold. Treatments were carried out daily for the 16 week trial period.

Outcome measures included VAS, morning stiffness, various joint assessments, SF-36, radiographic scores and blood tests (all detailed in the full paper).

The results show that the IFT improved SF-36 assessed body pain, but not other SF-36 subscales. Morning stiffness, tender joint counts, and physician assessed disease activity improved. In contrast, visual analogue scale assessed pain, CRP and ESR measurements were unchanged. MRI of the most affected hand or foot documented a tendency towards worsened tendinitis, soft tissue swelling, and new joint space narrowing and erosions. Bone scintigraphy showed a trend towards deterioration. New joints became inflamed within treated sites. It was concluded that IFT has analgesic effects in psoriatic arthritis, but does not have significant disease modifying effect.

When I first came across the paper, the results were consistent with what one might have predicted, and was not all that surprised, but it was certainly interesting to see if there was anything happening beyond the analgesic effect. This does not mean that IFT is a waste of time for these patients pain relief is a mighty fine thing – it is just that the capacity for the stimulation to modify the disease process has not been demonstrated (even though there is some reasonable evidence that it can do so in psoriasis).

Interferential and Different Models of Pain

Two papers that have looked at the effect of IFT on different models of experimental pain – the first of which ***(McManus, F. J. et al. (2006). The analgesic effects of interferential therapy on two experimental pain models: cold and mechanically induced pain. Physiotherapy 92(2): 95-102)*** comes from an established electrotherapy research group in Australia.

McManus et al were concerned with the effects (or not) of IFT on two different experimental pain models – cold induced and mechanically induced pain protocols. It has been argued that testing the efficacy of electrotherapy modalities (not just interferential) in lab based pain studies may have lim-

ited direct relevance to clinical pain in that the subjects are otherwise 'normal' and that there is a limited equivalence of clinical vs experimental pain. This has been argued by several authors over the years. Anyway, this study set out to evaluate the efficacy of IFT in these two different pain models using an RCT design in a lab based, healthy subject trial..

Twenty pain-free participants were recruited and each participant was exposed to the two methods of pain induction (adequately described in the full paper) on different days. The outcome measures were primarily the appropriate thresholds for the pain test in question; being cold pain threshold (time to first sensation of pain), intensity and unpleasantness measured on a visual analogue scale (VAS); mechanical pain threshold (tolerance to pressure) and unpleasantness (VAS).

The IFT was delivered using a 5000 Hz carrier, 2 pole delivery using self adhesive electrodes with modulation set at 100Hz. The IFT was delivered to the forearm (anterior / posterior). The experiment was of 60 minutes duration, with 6 x 10 minute experimental cycles, and the IFT was delivered for 30 minutes starting 20 minutes into the procedure. The procedure is well written up and useful time series graphics make the process clear

Essentially, the IFT produced similar effects on the threshold (first sensation of pain) for both cold and mechanical pain. The thresholds were significantly increased and the percentage changes in both were similar, as were their standard deviations. These results indicate that the analgesia provided by IFT is similar whether the origin of pain is cold or mechanical, and suggests that IFT can affect pain from a range of origins.

The authors note that the effect of IFT on other measures was not as pronounced. With mechanical pain, neither pain tolerance (maximum tolerable pain) nor unpleasantness was significantly altered. With cold pain, both intensity and unpleasantness showed a small but statistically significant change, and they therefore conclude that the cold and mechanical pain models are equally effective experimental tools to investigate electroanalgesia. Furthermore, it is proposed that these findings also suggest that future research should not induce pain beyond threshold when using the cold and mechanical pain models, as little additional information is gathered whilst subject discomfort and the risk of tissue damage is increased – something that I am sure will be considered a bonus by the (willing) subjects in this type of research if the recommendation is adopted.

There are some excellent points raised in the discussion, which combined with a well presented piece of research would make this an essential read for anybody concerned with lab based experimental pain research, or indeed anybody wanting to read through a clearly presented piece of lab work.

Interferential and Experimental Inflammatory Pain

The last of the interferential papers I'll describe in less detail – not because it is any less important, but simply on the basis that space is disappearing fast in this issue and there are a couple more topics to cover yet.

Jorge et al ***(Jorge, S. et al. (2006). Interferential therapy produces antinociception during application in various models of inflammatory pain. Phys Ther 86(6): 800-8)*** used an animal model of inflammatory pain and aimed to evaluate the effectiveness of IFT in reducing inflammatory pain and oedema in rats. There were 2 different inflammatory models employed a Formalin based nociceptive induction – generates a tonic inflammatory pain – and a Carrageenan type mechanical hyperalgesia (details provided in the text).

Inflammatory responses were induced in the hind paw using an established technique (formalin based or carrageenan based), and the interferential was applied to the hind limb using a 4000Hz carrier signal, modulated at 140Hz at a sensory amplitude of 5mA for 1 hour.

Pain (flinch response) mechanical hyperalgesia and oedema were used as outcome measures (again detailed in the paper). There were several groups of animals tested *IFT pre Formalin, *Sham IFT pre Formalin, *IFT post Formalin, *Sham IFT post Formalin, *IFT post Carrageenan, and Sham IFT post Carrageenan.

As you might expect with so many experimental groups, the analysis was 'interesting' and I'll only summarise the results here – you can work your way through the full data as it is all there!

- The IFT significantly reduced the formalin-evoked nociceptive response when applied to the paw immediately after but not before the formalin injection.
- IFT application at 2 hours after the carrageenan injection significantly prevented a further increase in carrageenan-induced mechanical hyperalgesia only immediately after discontinuation of the electrical current application.
- The antinociception induced by IFT was not attributable to a reduction in inflammation because IFT did not significantly reduce the edema induced by either formalin or carrageenan.

The authors propose that the results suggest that, despite its short-duration effect, IFT is effective in reducing inflammatory pain and should be considered primarily for use in the control of acute inflammatory pain.

It is interesting to note that the IFT was effective in relation to pain management, but did not make a significant difference to the inflammatory state or secondary signs of it – like the oedema. It has been suggested that IFT has a direct effect on oedema – something for which I have never managed to find much by way on convincing evidence. It may well achieve a result like this by either pain reduction and therefore more movement possible, therefore oedema reduction, or similarly, activation of local muscle pump, increased local blood flow and thus reduction in oedema. There have been some interesting propositions over the years with regards the potential role of neurogenic inflammation (Levine et al), though this has not been investigated with regards IFT so far as I am aware. There is a possibility that various forms of electrical stimulation can influence the inflammatory events rather more directly – maybe we can come back to this in future editions, but at the present time, and limiting the discussion to IFT, it looks like its primary effect is in relation to pain management – in keeping with the other evidence out there (and in fact, in keeping with the findings of the Walker et al paper above).

Magnetic Fields and Osteoblasts

There is a growing interest in magnetic field type therapies, and the evidence that various forms of magnetic fields are capable of having what might turn out to be a therapeutic effect in the tissues continues to be published. A recent paper by Huang et al ***(Huang, H. M. et al. (2006). Static magnetic fields up-regulate osteoblast maturity by affecting local differentiation factors. Clin Orthop Relat Res 447: 201-8)***. Has evaluated – in a fairly classic cell study – the effects of static magnetic field exposure to a particular cell type – in this case osteoblasts. Numerous authorities have argued that whilst dynamic magnetic fields might just have therapeuric potential, a static field is unlikely to do so as it is similar in many respects to the magnetic field that we are constantly exposed to on a day to day basis. There are plenty of arguments either way, and this is probably not the place to run through the pro's and con's of each side of the debate. Anyway, this paper reports the effect of a static magnetic field on osteoblasts, looking at how the applied field could result in

cell activity changes. Cells were exposed (continuously) to a static magnetic field for between 12 and 72 hours (at 0.4T) and in addition to microscopy, outcomes included levels of TGFâ (again), Type I Collagen and other cytokines and compounds. The paper clearly takes the reader through the microscopic and other findings but in summary, it was shown that the TGFâ levels, Type I Collagen, Osteopontin levels and alkaline phosphatase were significantly affected by the field and the microscopy results showed greater differentiation features than the controls.

Whilst for some this will not be too surprising, the authors do appear to have shed some additional light on how the previously demonstrated cell differentiation effects of static magnetic fields might be achieved – not least of which would be through changes in local regulatory factors. In some ways this is somewhat removed from clinical practice, but there are a couple of brief but important points : Firstly, magnetic fields are already being extensively used in various therapies, with increasing numbers of positive outcome studies. This paper makes a contribution to our knowledge on how the applied energy might be achieving these clinical results. Secondly, it does help to show that static (in addition to dynamic) magnetic fields have the capacity to influence cells and tissues, opening up further avenues for research in the future.

Endogenous Currents and Tissue Repair

Last couple coming up, the first of which, numerous people have e mailed me with saying have I seen it etc etc. ***Zhao, M. et al. (2006). Electrical signals control wound healing through phosphatidylinositol-3-OH kinase-gamma and PTEN. Nature 442(7101): 457-60***.

Naturally occurring (endogenous) electric fields are an interest area of mine and have been for some while. They were a part of my own PhD work some years ago, and I have had the opportunity to write and present on them numerous times. They are a fascinating area for research and I have been surprised that they have not attracted that much attention. Zhao et al are known in this field, and have provided some attractive new evidence that the endogenous fields that occur following injury have a definite role to play in the tissue repair process that follows.

They show that electric fields, of a strength equal to those detected endogenously, direct cell migration during wound healing as a prime directional cue and furthermore, that by manipulating endogenous wound electric fields, they were able to affect wound healing in vivo.

Some of the detail gets a bit hairy for me – don't know about you guys – but if I work at it, I can follow it well enough. The essential findings were that the endogenous electric currents acted as directional cues in both cell movement and wound healing studies, and the authors argue, that this is on a similar basis to chemotactic responses. The authors have, amongst other things, identified some of the genetic aspects of cell movement in response to electric currents, and may well help to explain something else that I have been concerned with for some time – which is why some people appear to have 'flat batteries' when it comes to inhibited or delayed repair – it may be genetic after all – watch this space – I have no doubt that there is more to come!

Tendinopathy and Biomechanics

Last one for this issue – before you get fed up with reading this lot – back to tissue repair and the controversy over tendinopathy. Wang et al ***(Wang, J. H. et al. (2006). Biomechanical basis for tendinopathy. Clin Orthop Relat Res 443: 320-32)*** have produced a useful review paper that looks at both the mechanical components and some of the biochemistry of tendinopathy. The de-

bate continues with respect to whether there is, or is not, an inflammatory component to this common clinical problem. Whist I am not sure that this paper actually resolves any of the most problematic controversies, it does make for a useful read for those concerned with its management. There is a useful review of some of the more salient theories, the ambiguity of some of the research published, and what I thought to be a useful consideration of microtrauma and associated inflammatory responses. To some extent, the authors come to the almost predictable conclusion that further research is needed, but in getting to that point, they do manage to review an extensive range of material, and even if I (personally) might disagree with some of their conclusions, for anybody working with tendon injury, damage or pathology, it would constitute a useful read, and possibly a good discussion paper for a journal club (or equivalent).

OK, so I think that will be enough for this issue. Hopefully something in there was of some interest, and I already have the start of a pile of papers for the next edition – due around December sometime.

As previously, if you have any comments, suggestions for papers that I should have included or anything else for that matter, please do e mail and let me know – and many thanks to all those that have done so to date – feedback is always welcomed.

Regards

Tim