Preliminary Communication

PULSED MAGNETIC FIELD THERAPY FOR TIBIAL NON-UNION

Interim Results of a Double-blind Trial

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Summary Patients with tibial fractures which had remained un-united for at least 52 weeks were randomly allocated to either active or dummy pulsed magnetic field stimulators and treated in full leg plasters for 24 weeks with a non-weightbearing conservative regimen, as is usual with such techniques. Fractures in 5 of the 9 patients with working machines united and fractures in 5 of the 7 patients with dummy machines also united. These early results of this double-blind trial are compatible with a difference in success rate at 24 weeks on active treatment of +33% to −61% (95% confidence limits) compared with the success rate on the dummy stimulators. The high proportion of fractures uniting in the control group suggests that conservative management of non-union is effective and this may explain much of the success attributed to pulsed magnetic field therapy.

INTRODUCTION

INTEREST in the use of pulsed magnetic fields to accelerate bone fracture repair[1,2] has been stimulated by the reported high success rates in the treatment of longstanding non-unions. Several different systems for generation and clinical application of pulsed magnetic fields have been described.[3] The most widespread clinical system, developed by Bassett et al, has been used on over 11 000 patients worldwide.[4]

An integral part of such treatment regimens is a lengthy period of immobilisation without weightbearing. Little information is available on the frequency of spontaneous healing of longstanding non-unions with conservative management, although it is described by Bassett[5] as being nil. This claim has been challenged and the need for a double-blind trial to separate the effects of electromagnetic stimulation from those of immobilisation has been expressed.[6–8] We have investigated the effect of a pulsed magnetic field on fractures of the tibia un-united at least twelve months after injury. We are attempting to estimate the effect on the proportion uniting by 24 weeks of the pulsed magnetic field component of the overall treatment regimen described by Bassett et al,[9] the fields being specified in detail elsewhere.[3,10]

Because of the unexpectedly slow accrual of patients we report the results from the first interim analysis[11] of this trial.

PATIENTS AND METHODS

Patients were admitted to the trial if they met all the following criteria:

(i) Over a year had elapsed since the fracture of the tibia was sustained.
(ii) Clinical mobility at the fracture site was confirmed by two independent observers during simultaneous radiological examination of the fracture under stress with image intensification.
(iii) A fracture line across the entire width of the tibia was confirmed by two observers after examining at least two radiographs taken from different aspects.
(iv) Two sets of radiographs, taken at least three months apart with the most recent taken on admission to the trial, independently assessed by two observers, showed no improvement.
(v) No operative treatment of the tibia had been given in the six months before the trial.
(vi) The fracture was in the shaft of the tibia 5 cm or more from the knee or ankle joint line.
(vii) Patients were over 18 years of age and gave informed consent.

Patients were excluded if any of the following circumstances applied:

(i) The minimum radiological gap between the bone ends was greater than 0·5 cm.
(ii) There was a screw or other metal partly or wholly in the fracture gap, or internal fixation prevented valid measurement of mobility.
(iii) There was bone disease present.
(iv) There was severe bone sepsis with constitutional effects.
(v) The patient was receiving steroid treatment.
(vi) An external fixator was in position.

Patients were randomly allocated to either an active or dummy stimulator. We used a minimisation procedure[12] to keep the two groups as closely matched as possible. Active machines produced the 1·5 mT peak, 5 ms burst waveform repeated at 15 Hz, as developed by Bassett and described by Barker and Lunt,[3] and drove coils designed to fit around the cast of each patient. Full technical details of the stimulator used may be found elsewhere.[10] Dummy machines differed from active ones only by a single internal connection which diverted their output to an internal load. The orthopaedic staff, the technical staff in the clinic, and the patient were unaware of the machine type supplied for the first 24-week period.

A concealed clock in each machine was used to check that the patient was complying with the recommended treatment protocol. Alarm circuitry indicated any internal malfunction of both types of stimulator. We recommended that patients should use the machines for 12–16 h every day, with a minimum session length of 1 h. Patients were asked to keep a logbook of their usage, which was checked for consistency with the concealed clock. Satisfactory

P. GRESELE AND OTHERS: REFERENCES—*continued*

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compliance was defined as a minimum mean daily use of 10 h with at most 7 days in each 6-week period and at most 14 days in each 24 week period on which the machine was used for 6 h or less.

Clinical Protocol

Patients were seen every 6 weeks for 48 weeks. For the first 24 weeks they were treated with the randomly allocated dummy or active machine. If their fracture had not united by week 24 they were given an active machine for the remaining 24 weeks. The fracture was immobilised with a full leg plaster[13] of 'Baycast' material and the patient was instructed strictly to avoid weight-bearing on the injured leg. Full clinical examinations, as defined below, were carried out at weeks 0, 12, 24, 36, and 48, but the patient was kept in plaster and stimulation continued until week 24 even if union had occurred at week 12. At weeks 6 and 18 (also 30 and 42 if fitted) the plaster condition was checked. The operation of the concealed clock and alarm system as well as patient compliance were checked by technical staff every 6 weeks during stimulation. All staff were unaware of the machine type used during the first 24 weeks.

Full clinical examination was carried out after plaster removal. Static radiographs were taken for use in observer variation studies, the results of which will be reported elsewhere. Mobility in both planes was estimated by two independent observers by mechanical stressing and use of a goniometer held on the limb surface. If lack of mobility was suspected, stress radiographs in both planes were taken and the behaviour of the tibia was observed during stressing by the operator by means of image intensification and by a second observer using a remote monitor. If both were unable to detect movement, during stressing and on the resultant radiographs, then the fracture was defined as clinically united.

The patients were asked to mark on a line, whose ends correspond to "no pain" and "pain as severe as possible", their assessment of the sensation during stressing which was later scored 0–4. Tenderness elicited by pressure over the fracture site was similarly assessed.

If fractures were found to be clinically united at weeks 24, 36, or 48 stimulation was discontinued and a series of controlled axial compression exercises[13] in a full leg clamshell polyethylene splint was started. This was continued for 6 weeks after which the splint was usually discarded and progressive weightbearing without splintage was allowed.

RESULTS

17 patients entered the trial between January, 1981, and November, 1982. In the next 8 months no eligible patients were recruited. 1 of the 17 patients was allocated to the control group, but left the trial at week 18 for personal reasons, without meeting the compliance criteria at any stage. He was reported to have an un-united fracture at 48 weeks.

The remaining 16 patients complied with their treatment schedule (mean daily use over 24 weeks: active 13·4 h, control 13·8 h). Their admission characteristics and main outcome measures are listed in the accompanying table. At the key assessment time (week 24), 5 of the 9 patients allocated to active treatment had clinically united fractures compared with 5 of the 7 controls. The 95% confidence interval for the true difference in the percentage uniting extends from 33% in one direction (active higher than control) to 61% in the other.

In each treatment group all fractures which had become clinically united by week 24 remained so at week 48; all such patients were bearing weight on the unprotected leg by week 36. Pain and tenderness scores tended to decrease over the first 24 weeks (table) with no apparent difference between the groups in the trends over the 48 weeks.

The admission characteristics of the treatment and control groups were in general similar. The 2 oldest patients (aged 60 and 72) were on active treatment. One fracture united by 24 weeks, the other did not. The only patient to have sustained his injury 4 years or more before trial entry (in fact 11 years before) was in the control group and his fracture united by 24

CLINICAL RESULTS OVER 48 WEEKS

Patient	Age	Months since injury	Clinical union at weeks:*				Pain scores at weeks:					Tenderness scores at weeks:				
			12	24*	36	48	0	12	24	36	48	0	12	24	36	48
Active stimulator																
1	60	19	No	Yes	Yes	Yes	2	0	0	0	0	4	4	1	0	0
2	40	33	No	No	No	No	0	1	0	1	2	3	2	2	2	3
3†	43	23	No	Yes	Yes	Yes	0	0	0	0	0	2	2	1	0	0
4†	22	36	Yes	Yes	Yes	Yes	0	0	0	0	0	2	0	1	1	1
5	24	39	No	Yes	Yes	Yes	2	2	0	0	0	3	3	2	0	2
6‡	72	22	No	No	No	No	3	1	1	0	0	3	1	2	0	1
7	24	27	No	No	No	Yes	0	1	0	0	0	1	0	2	0	0
8	38	14	No	No	No	Yes	0	0	0	0	0	0	0	0	0	0
9†	19	12	Yes	Yes	Yes	Yes	1	0	0	0	0	3	0	1	0	0
Dummy stimulator:																
10†	20	17	Yes	Yes	Yes	Yes	4	0	0	0	0	2	3	0	1	1
11	30	13	No	Yes	Yes	Yes	0	0	0	0	0	1	2	1	0	0
12†	30	133	No	Yes	Yes	Yes	1	0	0	0	0	2	1	0	0	0
13	19	16	No	Yes	Yes	Yes	2	1	1	1	1	4	4	3	2	1
14	33	29	Yes	Yes	Yes	Yes	1	0	0	0	0	3	0	0	1	0
15	41	30	No	No	Yes	Yes	1	1	0	0	0	0	1	0	0	0
16	36	17	No	No	No	No	0	0	0	0	1	0	0	0	1	0

*Key assessment time: all patients ununited at this time were placed on known active treatment. †Sepsis present on entry to trial. ‡Female.

weeks. All those patients, in both the active and control groups, who started the trial with active sepsis had united fractures by week 24.

At week 24 all but the first 2 patients were asked whether they thought they had been allocated to a working machine. 8 patients were sure their machines were working (incorrectly in 4 cases) usually because of the perceived clinical improvement, though 1 patient on a working machine claimed to have detected its output. Another 6 could not guess. None claimed to be sure they had a dummy machine. Thus adequate blindness to the treatment allocation appears to have been achieved in the patients.

DISCUSSION

To our knowledge this is the first reported double-blind clinical trial of the efficacy of a pulsed magnetic field in the treatment of non-unions. With only 16 patients, differences in true rates of union would have to be quite large to be detected. However, the presence of the pulsed magnetic field used here must now be seen as an as yet unproven part of the overall conservative management regimen which has united fractures in 10 of these 16 patients within 24 weeks. The treatment effect (the true difference in proportions of fractures uniting at 24 weeks), if it exists, caused by this pulsed magnetic field is unlikely to exceed 33%, as shown by the 95% confidence interval.

At the present rate of recruitment the Sheffield trial will take many years to complete, so it is important that a multicentre trial be mounted requiring, we estimate, about 150 patients.

The results of the active group, with 5 successes out of 9 patients by 24 weeks and 7 out of 9 by 48 weeks, are consistent with the 87% final success rate achieved after a maximum of 22 months of treatment in Bassett's series[13] of 127 tibial fractures (mean treatment time 5·2 months) and also with the many other uncontrolled studies reporting success rates of greater than 50%.[3] All these studies have made the assumption that the success rates that would be achieved with the same management regimen, but without the pulsed magnetic field treatment, would be much lower. Hence the success in achieving union of 5 patients out of 7 in the dummy

group was unexpected. Possible explanations for this result include high efficacy of long-term immobilisation of the limb and avoidance of weightbearing, the placebo effect of the patients "treating" themselves for extended periods each day, the limits on patient activity during waking hours caused by being connected to the machine with a cable approximately 2 metres long, the additional attention they received on their frequent visits to the clinic, or any combination of the above.

There is little information in published reports to indicate the expected success rate of conservative management of longstanding non-unions. However, Watson-Jones[14] strongly recommends extended immobilisation of tibial fractures in full leg plasters for up to 12 months and shows that 5% of the normal population of tibial fractures took as long as 24–48 weeks of immobilisation to achieve union. In a study described as containing a high proportion of multiple, gravely comminuted, seriously contaminated and heavily infected fractures, uninterrupted and prolonged immobilisation was said to have resulted in no cases of non-union.[15] Watson-Jones also refers to studies of other workers, including one of 5000 fractures, who achieved similar results. Nicoll[16] reported a study of 674 tibial fractures treated conservatively, which is often mistakenly quoted as showing that the chance of union after 52 weeks is 0·5%. The study actually shows that 12·5% of those fractures un-united at week 52 subsequently did unite with conservative management and this figure must be taken as a minimum because of the design of his study.

Thus, although long-term immobilisation and conservative mangement is not common practice at present, published reports suggest that union can often be achieved by this regimen even for fractures that have not united for at least 52 weeks. It appears therefore that conservative management of non-union should be reappraised as a treatment choice and that its effect may explain much of the success attributed to pulsed magnetic field therapy.

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Reviews of Books

Handbook of Hypertension

Vol 1—Clinical Aspects of Essential Hypertension. Vol 2—Clinical Aspects of Secondary Hypertension. Edited by J. I. S. Robertson, Western Infirmary, Glasgow. Amsterdam and New York: Elsevier. 1983. Vol 1, Pp 517, Dfl 211, $86. Vol 2, Pp 333, Dfl 188, $79.95.

"Not more books on hypertension", I groaned on being asked to review these two volumes: more groans on discovering that they are only the first two of a six-volume series. Closer inspection revealed that these are multiauthored books, each edited by one or two leviathans of the hypertension world. The overall editors, W. H. Birkenhäger and J. L. Reid, state that "the vast accumulation of biological and clinical knowledge in the field of hypertension has outgrown the limitations of the classical textbook or monograph. Moreover, the subject of hypertension by its very nature is a multi-disciplinary one attracting such diverse professionals as biochemists and public health workers", and believe that a serial handbook is the way to escape from the constraints of a single textbook and to reconcile the interests of generalists and specialists. Unfortunately, such an approach lends itself to the usual disadvantages of a multiauthor text in that it lacks cohesiveness and uniformity of style, there is overlap, and the quality is uneven. Such books cannot be read cover to cover. I tend to go for the familiar first often, I suspect shamefully, to check that my work has been appropriately cited. Less familiar subjects are left for more occasional reading or even ignored.

The combined efforts of many recognised authorities in hypertension are contained here, and clearly these books will be consulted by people with very differing interests, but I suspect that many, like me, will be disappointed by several chapters. For example, chapter 23, on the assessment, investigation, and care of the hypertensive patient, would be a useful review for medical students and possibly some general practitioners, but I doubt that it would satisfy someone engaged in hospital practice or clinical research. Equally, I was disappointed by Lennart Hanson's chapter on the drug treatment of hypertension. It is unbalanced, giving a disproportionate amount of space to beta blockade but dealing sketchily with some of the newer developments, such as angiotensin II converting enzyme inhibitors, which are currently attracting a great deal of attention. He does state boldly that "no attempt will be made to review this topic here" and refers us to the literature. However, I think that an authoritative handbook should give us the distilled wisdom of such an expert. Why else consult the book?

Many of the chapters are re-written versions of well-worn and well-known work. Disappointingly they contain nothing new. For example, chapter 6, by B. Folkow and his colleagues, contains nothing that was not more completely published in his 1978 Volhard Lecture. Must we, therefore, assume that this is the final word on the wall/lumen hypothesis?

Some authors are cast in somewhat unfamiliar roles. Paul Korner has contributed the chapter on cardiac function. With his typical thoroughness he has reviewed those areas which reflect his own research interest but surprisingly does not discuss in detail the role of echocardiography in the assessment of left ventricular mass and function. This technique is probably that most commonly used for such purposes at present, and the clinical journals are full of papers on echocardiographic studies in hypertensives. It is natural for individuals to concentrate on aspects with which they are closely involved, but the result does not meet the requirements for a handbook.

The bold, clear type and the abundance of headings and sub-headings make the book easy to read. The illustrations are plentiful and of the highest quality, and the text, in the main, is well referenced. I doubt whether individuals will purchase this series, but I am sure that the book will find a place in departments of medicine and cardiology and in medical libraries. However, I would be surprised if it went into a second edition.

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