Static magnetic field effects on the sagittal suture in Rattus Norvegicus

S. Camilleri, LDS (Eng.), MSc, and F. McDonald, BDS, MSc, M.Orth.MIBiol
London, England

Twenty-day-old Wistar albino rats were exposed to static magnetic fields by placing a neodymium-iron-boron magnet over their sagittal suture. Cellular activity was monitored by the uptake of tritiated thymidine in control, north, south, and unoperated animals at 1, 3, 5, and 10 days (n = 10 per group). A total of 160 animals were used for this part of the study, with the animals examined 1, 3, 5, and 10 days after surgery. Bone remodeling was examined by tetracycline fluorescence with 10 animals allocated to 5- and 10-day periods for north and south poles (n = 10 per group) and control experiments. This consisted of the placement of unmagnetized alloy, similar in size and shape to the magnets, and also included unoperated animals (n = 5 per group). A total of 60 animals were used for the tetracycline study and were examined at 5 and 10 days after surgery. While the tetracycline examination revealed very little change, the thymidine reflected a reduction in thymidine uptake subsequent to placement of the magnet, reaching a maximal effect at 3 days and returning to a normal value thereafter. This questions the potential of static magnetic fields affecting cell mitotic activity as previously reported. (AM J ORTHOD DENTOFAC ORTHOP 1993;103:240-6.)

The effects of electric and magnetic fields on biologic tissues have been investigated as far back as the eighteenth century. However, electrical phenomena were disputed, until the experiments of Yasuda et al.,[1] which showed subperiosteal deposition of bone in relation to an electric current. Bassett et al.[2] worked on a parallel course, and further developed a noninvasive use of pulsed magnetic fields to deliver magnetic energy to the tissues. This idea has been patented and has been used with remarkable success in the treatment of nonunions and pseudarthrosis of bone. However, clinical controls are difficult to achieve.

Magnetic fields of sufficient magnitude have been shown to affect various biologic systems at organ, tissue, cellular, and subcellular levels. Experiments have shown that electric and magnetic fields may have separate effects on cells.[3] There are problems in distinguishing the effects of electric and magnetic fields. A pulsed magnetic field will generate an electric current in the tissues. This generation is based on the physics of charged ions moving with Brownian motion in a field producing electrical charge. Because of the complexity of tissue responses, even if this effect is on nonbony tissue, biochemical mediators may be released that ultimately overwhelm the response of cells to these other effects. While these effects may appear negligible when studying magnetic effects in vivo, in in vitro studies the influence becomes significant.

The development of rare earth magnets with extremely high field strengths offers a possible alternative means of applying magnetic fields of sufficient magnitude. A problem in conducting an in vivo experiment is the application of the magnet to the tissues for a sufficient length of time. Undue distress of the animal has to be prevented. In addition, the animal must not be able to disturb to remove the magnet.

There have been very few experiments in the literature on the effects of static magnetic fields on bone growth and remodeling, and none have investigated the possibility of different poles having different effects. These studies have used magnets implanted in the tissues. This leads to many problems, notably that:

1. Postoperative infection is a constant problem and may affect the pattern of bone deposition.
2. The magnetic field may influence the healing process, thereby masking the result.
3. The magnets, if not protected, are highly susceptible to corrosion by tissue fluids and disruption of their magnetic domains. The susceptibility to corrosion is dependent on the alloys used and their relative positions in the electrochemical series. The simple fact that dissimilar metals are used allows galvanic cells to develop and destroy the domains within the magnet. This

From the Department of Oral Biology (Physiology/Orthodontics).
This work was supported by the Wellcome Foundation, England.
Copyright © 1993 by the American Association of Orthodontists.
0889-5406/93/$1.00 + 0.10 8/1/35014

is avoided clinically by impervious coatings, e.g., a steel sleeve.

4. Little is yet known about the possible local and systemic toxic effects of the alloy used in the construction of the magnet. Specific problems may be encountered by toxic concentration in certain organs.

Magnets have been used in dentistry for several years as an aid to denture retention and are now being used in orthodontics to apply force to teeth.[4-6] These authors have used magnetic forces to facilitate orthodontic movement. In particular, Blechman[7] was able to demonstrate that magnets deliver forces without undesirable effects on the vertical dimensions. However, in a clinical situation it is difficult to control the many variables associated with the craniofacial region. Therefore it was proposed to investigate the effects of a static magnetic field (SMF) on the growth and remodeling of bone as a baseline for further work.

Magnetic fields may affect cells in a number of ways.

1. Theoretically, enzyme systems that involve free radical intermediate stages may be affected by magnetic fields as these will exhibit diamagnetic anisotropy. An example is the increase in reaction rate of the metalloenzyme catalase when exposed to a 6.0 magnetic field.[8] In a review of the literature, Tenforde[9] listed the articles dealing specifically with the effects of SMF on various enzyme systems. While this could affect some of the results seen, no attempt could be made for physical explanation of the magnetic sensitivity of some of the systems examined. Magnetic fields have been described as having a "stirring" effect on ionic reactions.[10] It is possible that some reactions could be speeded up by this effect, especially those carried out in vitro.
2. Paramagnetic oxygen molecules may be redistributed in the presence of a magnetic field gradient.[10] Although this has not yet been demonstrated to have any noticeable biologic consequences, it may help explain the apparent effects of SMF on growth and development, as a high oxygen concentration is toxic to developing tissues.
3. The lamellar phospholipids of the cell membrane are weakly diamagnetically anisotropic, the hydrocarbon chains of the macromolecules probably contributing to most of the anisotropy.[11] For the rod-like macromolecules to align in a SMF, the magnetic interaction energy must exceed the thermal interaction energy of the system. For individual molecules, the field strength would have to be enormous.

Early studies on the effect of static magnetic fields suggested that exposure may lead to such abnormalities as pyknosis,[12] depressed respiratory rate,[13] malignant transformation,[14] and delayed healing.[15] More recent studies, often using higher field intensities and longer exposure times, have not been able to replicate the results. Tsutsui et al.,[16] Cerny,[17] and Esformes[18] studied the effects of SMF generated by cobalt-samarium (CoSm) magnets in vivo and in tissue culture. They all reported no significant differences between control and experimental groups. The tissue culture experiments of Tsutsui et al. seem, however, to be based on one sample only. Frazier et al.[19] repeated Malinin's experiment and found the culture technique to be responsible for the malignant transformation.

The aims of this study were (1) to determine the effects of a static magnetic field on the pattern and rate of bone deposition, with a tetracycline bone marker, and (2) to determine the effect of a SMF on thymidine uptake with tritiated thymidine as a nuclear marker. The thymidine would give an assessment of the mitotic activity of the cells, being incorporated into the nucleus during the cell cycle. The tetracycline study would give an estimate of the synthesis carried out by the cells.

Both these aims would be able to establish a baseline from which further examinations are undertaken. It was also decided to use static fields to eliminate the effects of change in field and thus change in load on a tissue. It has been demonstrated that bone remodeling in particular is load dependent.[20]

MATERIALS AND METHOD

The tetracycline study

The study was performed with rats of the Wistar albino strain. They were approximately 20 days old at the start of the study and weighed an average of 25 gm each. The average weight at 25 days was 75 gm. The study was carried out when the skull of the young rat is still growing in width,[21] and the animals could be separated from the dam to prevent cannibalism. In addition, the suture had entered its period of definitive form.[22]

Fixation of the magnets* over the suture used cyanoacrylate adhesive (Loctite, Herts, UK.). This adhesive has long-

*Material: Neodymium Iron Boron. Composition: $Nd_2Fe_{14}B$.
Composition by weight: Nd = 26.7%, Fe = 72.3%, B = 1.0%.
Finish: Nonmagnetic tin. 15 to 20 μm thick.
Maximum energy product 245 kJ/m^3.
Curie temperature 315° C.
Magnet shape: 9.5 mm diameter ×3.2 mm bore × 1.6 mm thick.
The residual induction was 1.5 T.
These magnets have the highest magnetic energy per unit volume available commercially and so were used in preference to the CoSm magnets.
Magnet Developments, Swindon, U.K.

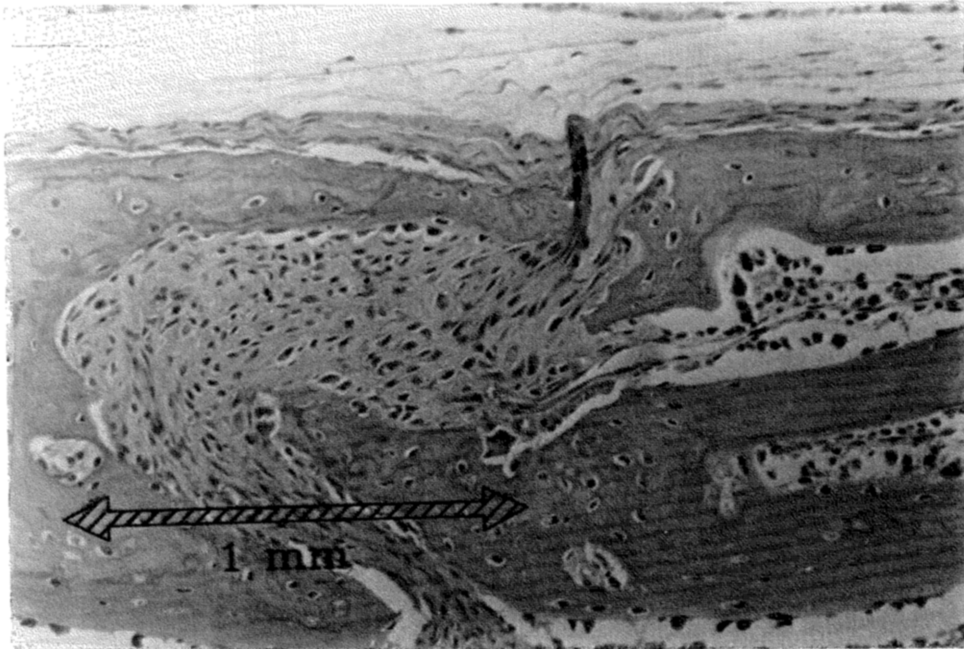

Fig. 1. Hematoxylin and eosin coronal section of rat sagittal suture demonstrating normal histologic appearance of tissue elements. Despite placement of the magnet with cyanoacrylate adhesive, no inflammation can be seen.

term carcinogenic effects but for the period of the study was considered suitable. In addition, any effects as a result of the adhesive could be compared with the untreated animals. The magnets used retained their flux for the duration of the experiment. Previous magnets lost flux significantly.

There was no removal of overlying fur. No anesthesic was necessary for this procedure, and the animals did not appear distressed in any way. At the time of surgery, the animals were injected intraperitoneally with 25 mg/kg body weight of oxytetracycline in sterile water. Examination of the scalp of rats who had lost their magnets, presumably through grooming, showed no signs of inflammation or ulceration, the skin appearing pink and healthy. The tissues of the animals killed at the end of the experiment showed no signs of gross inflammation. Fig. 1 shows a hematoxylin and eosin slide of a rat sutural area after the magnet has been in place for 5 days. The magnet, although at distance from the suture (0.5 mm) would deliver a magnetic field strength of 100 mT at the suture (Fig. 2). This was considered sufficient for the purposes of the experiment to link the study to the effects of nuclear magnetic resonance (NMR) scanning. Nuclear magnetic resonance (NMR) scanning consists of two basic types of scan, a high and a low field. Obviously, this only relates to the low NMR fields. Certain types of imaging use high fields in this order of magnitude. In addition, it is already a clinical presumption that increasing the flux will increase the speed of tooth movement. This is despite the varied reports mentioned that show a decrease in bioeffect. Measurement of the magnetic flux and the field in air confirmed that, with an air gap of 0.5 mm, 100 mT could be measured. The distance was from the magnet face to the probe, where a constant reading of 100 mT on the Gauss meter (Magnetic Developments, Swindon, United Kingdom) was recorded. These recordings were produced by a Hall effect Gauss meter supplied by the manufacturers of the magnets (Fig. 3).

The animals had access to food and water ad libitum and were kept at a constant temperature and humidity with a light/dark cycle of 12 hours. The cages the animals were housed in were constructed of a plastic base and a nonmagnetic aluminum top to avoid any extraneous magnetic flux changes.

Ten animals were allocated to north and south groups, and five to control and unoperated groups. A total of 60 animals were used for the tetracycline study. The control animals had identical pieces of demagnetized metal glued to the scalp. They were to be killed at 5 and 10 days. Shorter time intervals would not have produced enough growth to be valid in comparison to the errors of the method of measurement.

Another group who were not operated on for both the thymidine study and the tetracycline study were examined.

After the experimental period they were killed by CO_2 asphyxia. The animals were then decapitated, and the magnets peeled off. An acrylic resin dummy was placed to mark its position. This was necessary to locate it on the ground sections. The heads were placed in a solution of 10% formalin with a citrate buffer.

The specimens were embedded in methacrylate resin

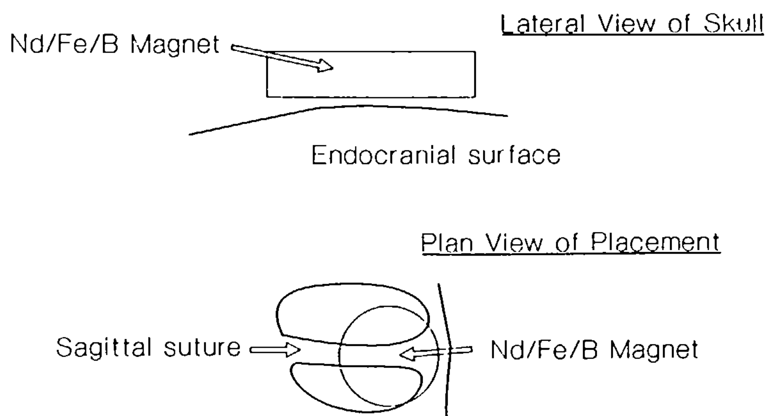

Fig. 2. Diagrammatic representation of placement of magnet. The circular magnetic has one face north, and the other south. The field placed closest to the cranium can be varied by inverting the magnet.

(L.R. White, Guilford, U.K.) according to the procedure recommended by the manufacturers. Sections were obtained with a water-cooled rotary diamond wheel approximately 450 μm thick. All cuts were made in the coronal plane and were cut to a thickness of 500 μm to avoid distortion. These were numbered on both sides to enable measurements to be taken from both sides of the section. The sections were examined under high power with a Leitz microscope that used incident ultra violet light and an exciter filter to show tetracycline epifluorescence.

Measurements were carried out with an eyepiece micrometer. The specimens were viewed under high power (x120) and converted to millimeters after standardization with a stage micrometer. The use of high power made visualizing areas of fluorescence easier and reduced the error in measurement.

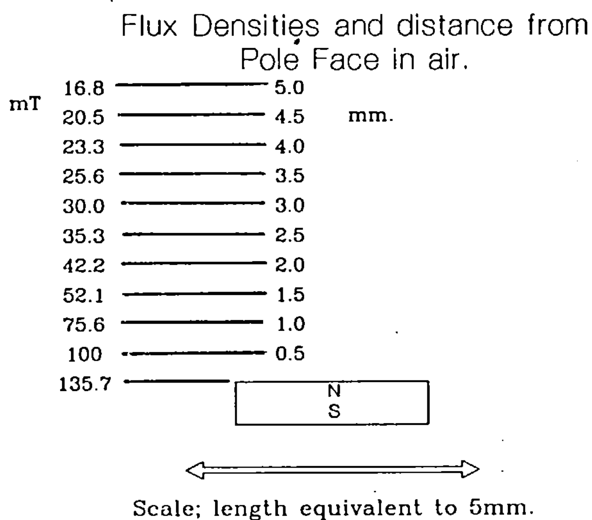

Fig. 3. Variation of flux from face of magnet as determined with Hall effect probe.

The autoradiographic study

Thymidine has been shown by Amano et al.[23] to be incorporated into the DNA, being a specific precursor. The rats used were the Wistar albino strain of *Rattus Novegicus* as used in the tetracycline study. After positioning the magnets with adhesive as described previously, the animals were labeled 1 hour before killed at 1, 3, 5, and 10 days after surgery. The dose of tritiated thymidine used was 0.5 μCi/gm of body weight and with an activity of 5 μCi/mmol. This was administered intraperitoneally. The animals were killed an hour later, decapitated, and the skin dissected away from the skull around the magnet, leaving that part of the skin immediately under the magnet undisturbed. The heads were then fixed in formol saline for at least 7 days.

A total of 10 animals per time period were allocated to north, south, control, or unoperated animals (a total 160 animals were used in this part of the study).

Stepped serial sections at 0.5 mm levels were taken. Five sections were taken at each level. One section was to be stained with hematoxylin and eosin. Two sections were to be counterstained with eosin and used for autoradiography. Two sections were to be kept as reserves.

In this study the specimen was counterstained after the exposure of the slides. Ilford K5 emulsion was used for the exposure. Cell counting was then carried out with a Zeiss standard research microscope at a magnification of x250. The

Table I. The results of the tertracycline measurements (millimeters)

Magnet	Days after surgery 5	10	p value
Control	1.159 ± 0.590	1.176 ± 0.627	0.43
North	1.217 ± 0.467	1.272 ± 0.543	0.52
South	1.382 ± 0.418	1.196 ± 0.601	0.18
Untreated control	1.095 ± 0.478	1.251 ± 0.448	0.28

Mann-Whitney and Student *t* test confirm no significant differences between the groups.
Mean and SD shown.

cells that were included as labeled were those that had a count of five grains and above, located over the cell nucleus. All cells were labeled in the specimen that had taken up the thymidine. However, only those associated with the suture, which appeared to be showing the staining characteristics of fibroblasts, were examined for labeling. To determine the labeling index, the total number of cells in the suture that were labeled were counted and divided by the number of cells seen in each section. This was expressed as a percentage.

$$\text{Labeling Index} = \frac{\text{Number of cells with } +5 \text{ grains}}{\text{Total number of cells}} \times 100\%$$

A reproducibility study was performed by determining the index of 30 slides and repeating this 2 weeks later. The results were compared by using the Student *t* test, and the correlation coefficient determined.

RESULTS

The tetracycline study

There was no significant difference in growth between the north and south groups (Table I). Nor was there any difference between the experimental and control groups. The majority of the experimental and control animals exhibited a pattern of remodeling similar to that described by Young.[24] There was marked fluorescence on the endocranial plate, indicating continued bone deposition. The fluorescent endocranial line was thicker in the parietal than in the sagittal region. The signs were consistent with the calvarium remodeling to become flatter as it grows. This takes place chiefly by bone deposition on the endocranial surface of the center and the ectocranial surface of the periphery. There is a small amount of resorption occurring at the center of the ectocranial surface, the delicate, highly curved bones becoming progressively embedded in their thicker, flatter successors. There was no significant difference between the north, south, and control groups. The mean growth increment was assessed as the thickness of bone seen beyond the label in areas where bone deposition was known to occur.[24] Therefore the mean growth increment was 0.14 mm over the experimental period.

Thymidine uptake

The reproducibility study for the autoradiography showed no significant difference at the 95% level, and the correlation coefficient was 0.987. The results, however, showed a depression of activity maximal between 3 and 5 days, with a tendency to return toward normal at 5 days for both the north and south groups. The control group showed a constant level of activity throughout the experiment, with a statistically significant difference with the Mann-Whitney nonparametric tests. There was no difference between the north and south groups (see Table II).

DISCUSSION

These findings are in support of those of Feinendegen and Muhlensiepen[25,26] who demonstrated reduced activity of thymidine kinase in mice exposed to intense magnetic fields.

The theories of Dellinger[5] and Kawata[6] are, however, not substantiated by this level of field. They claim, although indirectly, that the magnetic field surrounding the appliances increases bone turnover and facilitates treatment.

A major variation between their use of the appliance and this model is that forces varied with time. As can be seen from the field measurements, distance dramatically effects the field. To obtain the most from the magnet-based appliance, the exact effects of magnets have to be established. It is difficult to interpret findings from clinical results. The magnet field may affect the craniofacial musculature and/or may need additional factors such as force applied simultaneously. All this is conjecture.

If remodeling is enhanced, then an increase in mitotic activity should occur. However, differing cell populations are being examined. On the one hand, osteoblasts are responsible for the bone deposition and are found close to bone. These can prove difficult to isolate in histologic specimens and so may not have influenced the overall mitotic activity. In the case of thymidine uptake, the cells are found in the midline of the suture. In these cell types, there was a decrease in uptake of thymidine. However, fibroblast cell turnover is enhanced during normal remodeling, and so it could be expected that the cell mitotic rate should increase.[25] This is not seen to be the case when considering the uptake of thymidine. The experiments of Papatheofanis and Papatheofanis[27] also indicate that magnetic fields do not affect bone mineralization in the short term. Bruce et al.[28] claim that the magnetic field enhances fracture healing. Again, the initial reduction in labeling

Table II. The labeling index of cells of the sagittal suture

Magnet face	Days after surgery			
	1	3	5	10
Control	1.556 ± 0.605	1.640 ± 0.943	1.863 ± 0.709	1.731 ± 0.674
North	1.659 ± 0.872	0.693 ± 0.321	1.138 ± 0.749	1.453 ± 0.597
South	1.484 ± 0.451	0.724 ± 0.367	1.672 ± 0.654	1.564 ± 0.793
Unoperated control	1.432 ± 0.587	1.574 ± 0.672	1.653 ± 0.608	1.640 ± 0.591

n = 10 in each group, percent labeled cells.
Student *t* test and Mann-Whitney confirm the following;
1. At 1 and 5 days no difference exists between all experimental groups ($p = 0.56$).
2. There is a significant reduction in labeling of cells at 3 days for north and south groups when compared with the controls ($p < 0.05$).
3. There are no differences between north and south groups at 3 days ($p = 0.42$).
Mean and SD shown.

index, followed by a gradual return toward normal values, would not be consistent as both phenomena are associated with increased cell turnover. Bruce et al.[28] also suggest that tissue maturity is augmented. The opposite seems to be the case here if cell division is delayed.

Evidence does exist that magnetic fields may affect growth and development. Recent experiments on guppies,[29] chick embryos,[30] and nematodes[31] show a decrease in the developmental rate between the control and experimental groups.

Feiendiegen and Mulensiepen[25,26] conclude that the action of SMF on thymidine kinase is to depress its activity. Thymidine kinase phosphorylates thymidine. This is a rate limiting step for the entry of the precursor into DNA. They propose that this effect is due to the action of the field on the intracellular membrane to which the enzyme is bound. Furthermore, as oxygen radicals are trapped by the membrane, alteration of the intracellular membrane may influence the intracellular radical concentration. Thus enzymes that lack radical intermediate stages may be affected in this manner. Besides, this apparent effect of SMF on DNA could very well explain the decrease in rate of growth and development observed in the experiments previously mentioned.

Bruce et al.[28] in their experiments were somewhat similar to this study, in that rare earth magnets were applied to observe the effect in vivo. However, the magnets were implanted, possibly introducing additional variables, such as postoperative inflammation and infection. Furthermore, the flux densities reported are different than those used in this experiment, nearly five times lower. This may be due to the distance between the magnet and the tissue under investigation. Whether any of the magnets exhibited corrosion, with resultant loss of power, was not stated in the article. This would be expected after implantation for any length of time.

Another variable is the length of time during which the experiment was conducted. All the experiments quoted were carried out over a period of at least 10 days, the experiments of Bruce et al.[28] extending over 4 weeks. In this study difficulty was experienced in getting the magnets to stay in place for more than 5 days, the rats tended to dislodge them as they grew larger and stronger. Thus there is no record of longer-term effects. It is possible that, having initially depressed cell turnover, a rebound to above normal values may take place after 5 days.

The nature of the effect of the magnetic field is still speculation. Nevertheless, the weight of evidence points toward the cell membranes as influenced by the field. As has already been discussed, biologic membranes orient in a magnetic field of sufficient intensity. This orientation could be sufficient to alter the properties of the membrane, e.g., by distorting pores or by covering or uncovering active sites. Enzymes bound to the membrane could very conceivably have their reaction rate altered. For tritiated thymidine, alteration in the reaction rate of thymidine kinase, a membrane-bound enzyme would limit the entry of the molecule into DNA and affect the number of cells entering the "S" phase. This would be manifested by a reduced labeling index.

A "relaxation time" has been decribed,[25,26] i.e., the time taken by the subject to return to normal values after removal from the field. This would be due to the molecules disorienting by normal thermal energy. A long relaxation time is indicative of large molecules, such as phospholipids, being affected. The relaxation time was inversely proportional to the exposure time. Feinendiegen[25] postulates that this may be due to the responding structure altering in such a way to make a return to the relaxed state easier. This observation may help explain the tendency to return to normal seen in this study. It is possible that the structure aligns slowly, altering its properties. As the rest of the struc-

ture responds and realigns in a different direction, its properties return to normal.

CONCLUSIONS

Static magnetic fields do not seem to affect bone growth to any significant degree over the period under study. However, thymidine uptake is inhibited to a significant level.

A possible explanation could be that the exposure to the field in some way inhibits cell division. This is eventually exhausted, which allows a return to normal. Free movement of the subject in the field negates the effect. This would indicate that a large, fixed structure is affected as movement here would result in no net orientation of the molecules.

The aim was to examine an arrangement of fibrous tissue and bone surfaces with a similar cellular content to a healing fracture. The study must question the validity of static magnetic fields and their use in fracture healing. There is a need for a clearer understanding at the cellular level of the effects of static magnetic fields.

REFERENCES

1. Yasuda I, Noguchi KA, Sata T. Dynamic callus and electric callus. J Bone Joint Surg 1955;37A:12-92.
2. Bassett CAL, Pawluk RJ, Becker RO. Effects of electrical current on bone in vivo. Nature 1964;204:652-4.
3. Goodman EM, Sharpe PT, Greenbaum B, Marron M. Pulsed magnetic fields alter cell surface. FEBS Lett 1986;199:275-8.
4. Dellinger E. A clinical assessment of the Active Vertical Corrector; a non surgical method for skeletal open bite. AM J ORTHOD 1986;89:428-36.
5. Kawata T, Hirota K, Sumitani K, et al. A new orthodontic force system of magnetic brackets. AM J ORTHOD DENTOFAC ORTHOP 1987;92:241-8.
6. Blechmann AM. Magnetic force systems in orthodontics: clinical results of a pilot study. AM J ORTHOD 1985;87:201-10.
7. Blechmann AM, Smiley H. Magnetic forces in orthodontics. AM J ORTHOD 1978;74:435-443.
8. Haberditzl W. Enzyme activity in high magnetic fields. Nature 1967;213:72-3.
9. Tenforde TS. Chapter VII. In: Gandolfo M, Michaelson SM, Rindi A, eds. Biological effects and dosimetry of static and ELF electromagnetic fields. New York: Plenum Press, 1985.
10. Aceto H, Tobias C, Silver J. Some studies on the formation of magnetic fields. IEEE Trans Magnetics 1970;6:368-73.
11. Rosenblatt HC, Yager P, Schoen PE. Orientation of lipid tubules in a magnetic field. Biophys J 1987;52:595-301.
12. Mulay IL, Mulay LN. Effect of a magnetic field on Sarcoma 37 ascites tumor cells. Nature 1961;190:1019.
13. Reno VR, Nutini LG. Effect of mägnetic fields on tissue respiration. Nature 1963;198:204-5.
14. Malinin GI, Gregory WD, Morelli L, Sharma VK, Hench JC. Evidence of morphological and physiological transformation of mammalian cells by strong magnetic fields. Science 1976;194:844.
15. Gross L, Smith LW. Effect of magnetic fields on wound healing in mice. Fed Proc 1961;20:164a.
16. Tsutsui H, Kinouchi Y, Sasaki H, Shiota M, Ushita T. Studies on the CoSm magnet as a dental material. J Dent Res 1979;58:1597-606.
17. Cerny R. The reaction of dental tissues to a magnetic field. Aust Dent J 1980;25:264-8.
18. Esformes I, Kummer J, Livelli TJ. Biological effects of magnetic fields generated with CoSm magnets. Bull Hosp Jt Dis Orthop Inst 1981;41:81-84.
19. Frazier ME, Andrews TK, Thompson BB. In vitro evaluation of static magnetic fields. In: Phillips RD, Gillis MS, Kaune WT, Mahlum DD, eds. Biological effects of extremely low frequency electromagnetic fields. Springfield, Virginia: Thomas Springfield, 1979.
20. Hert J, Lisková M, Landa M. Reaction of bone to mechanical stimuli-Part 1. Continuous and intermittent loading of tibia in the rabbit. Folia Morphol (Praha) 1971;19:290-300.
21. Young RW. Autoradiographic studies on postnatal growth of the skull in young rats injected with tritiated glycine. Anat Rec 1962;143:1-13.
22. Moss ML. Growth of the calvaria of the rat. The determination of osseous morphology. Am J Anat 1954;94:333-356.
23. Amano M, Messier B, Leblond CP. Specificity of labelled thymidine as a deoxyibonucleic acid precursor in radioautography. J Histochem Cytochem 1956;7:153-5.
24. Young RW. The influence of cranial contents on postnatal growth of the skull in the rat. Am J Anat 1959;105:383-415.
25. Feinendiegen LE, Muhlensiepen H. Magnetic fields affect thymidine kinase in vivo. Int J Radiat Biol 1985;52:469-79.
26. Feiendiegen LE, Muhlensiepen H. In vivo enzyme control through a strong stationary magnetic field. The case of thymidine kinase in mouse bone marrow cells. Int J Radiat Biol 1987;52:469-479.
27. Papatheofanis FJ, Papatheofanis BJ. Short term effect of exposure to intense magnetic fields on haematologic indices of bone metabolism. Invest Radiol 1989;24:221-3.
28. Bruce GK, Howlett CR, Huckstep RL. Effect of a static magnetic field on fracture healing in a rabbit radius. Clin Orthop 1987;222:300-6.
29. Brewer HB. Some preliminary studies of the effects of a static magnetic field on the life cycle of the *Lebistes reticulatus* (guppy). Biophys J 1979;28:305.
30. Bersani F, Zini Q. Preliminary observations: the effects of a static magnetic field on chick embro's growth. In: Gandolfo M, Michaelson SM, Rindi A, eds. Biologic effects and dosimetry of static and ELF electromagnetic fields. New York: Plenum Press, 1985.
31. Peeling J, Lewis J, Samoiloff M, Bock E, Tomchuk E. Biological effects of magnetic fields: chronic exposure of the nematode *Panagrellus redivivus*. Magn Reson Imaging 1988;6:655-60.

Reprint requests to:
Dr. Fraser McDonald
Department of Oral Biology (Physiology/Orthodontics)
Floor 22
Guy's Tower
St. Thomas St.
London SE1 9RT