REVIEW OF PUBLISHED SERIES ON BLEEDING IN PATIENTS WITH VARICEAL HAEMORRHAGE

	Witzel	Baker[1]	Jackson[2]	Resnick[3]	Conn[4](*)		Paquet[5]†
Study period (mo)	25	72	60	96	144	(72)	36
No of patients	53	115	75+	45	35‡	(24‡)	33
Variceal bleeding	30 (*57%*)	33 (*29%*)	14 (*19%*)	12 (*27%*)	14 (*40%*)	(7 [29%])	29 (*88%*)
Deaths due to variceal bleeding	19	20	7	5	6	(5)	21
All deaths	29	74	21	19	20	(11)	21
% of deaths due to variceal bleeding	66%	27%	33%	26%	30%	(45%)	100%
Alcoholic cirrhosis	81%	?	90%	98%	97%	(100%)	?
Size	31% large	20% large	..	..	2+§	(2·4+§)	3–4 (with erosions)§
Continued alcohol abuse after randomisation	59%	..	52%	54%	69%	(··)	..

*All with ascites. †At risk varices and/or poor coagulation. ‡Includes patients randomised to receive portacaval shunt but refusing operation. §Range + to + + + +.

bleeding episodes, differences in the number and proportion of deaths due to bleeding might be expected. Your 1984 editorial[6] on bleeding varices specifically mentions this point in the context of trials to prevent rebleeding from varices. Witzel et al do not describe how acute bleeding episodes were treated or whether treatment differed between the control group and the sclerotherapy group. Nor is it clear whether the deaths due to bleeding occurred following the first bleeding episode or after multiple episodes. Since no mention is made of the total number of bleeding episodes, it would seem that the first bleeding episode and death were the end points in the study. If this is so the proportion of deaths due to bleeding is still much higher than in all but one of the other studies cited.[1-5] Witzel et al used modified Child's criteria for randomisation. What was the proportion of patients in groups A, B, and C who bled or died? Group A (37%) of their patients would be expected to bleed less and survive longer, than group C patients with ascites.[4] Was the efficacy of sclerotherapy related to these factors? Lastly, 9 patients did not complete the study after randomisation. Was the analysis done on an intention-to-treat basis or were these 9 excluded, and what happened to them?

The conclusion that increased survival in the treated group was due to variceal prophylactic sclerotherapy must be re-examined.

Academic Departments of Medicine and Surgery,
Royal Free Hospital,
London NW3 2QG

A. K. BURROUGHS
G. HAMILTON

1. Baker LA, Smith C, Lieberman G. The natural history of esophageal varices. a study of 115 cirrhotic patients in whom varices were diagnosed prior to bleeding. *Am J Med* 1959; **26:** 228–37.
2. Jackson FC, Perrin EB, Smith AG, Dagradi AE, Nadal HM. A clinical investigation of the portacaval shunt: Survival analysis of the prophylactic operation. *Am J Surg* 1968; **115:** 22–42.
3. Resnick RH, Chalmers TC, Ishihara AM, et al. A controlled study of the prophylactic portacaval shunt: a final report. *Ann Intern Med* 1969; **70:** 675–88.
4 Conn HO, Lindenmuth WW, May CJ, Ramsby GR. Prophylactic portacaval anastomosis: a tale of two studies. *Medicine (Baltimore)* 1972; **51:** 27–40.
5. Paquet KJ. Prophylactic endoscopic sclerosing treatment of the esophageal wall in varices: a prospective controlled randomized trial *Endoscopy* 1982; **14:** 4–5.
6. Editorial. Bleeding oesophageal varices. *Lancet* 1984; i. 139–41.

SIR,—In that German study of prophylactic sclerotherapy for oesophageal varices the main support for such prophylaxis was the very high frequency of initial variceal haemorrhage over 25 months in the control group, ranging from 35% to 83% depending upon variceal size, which was reduced by sclerotherapy.

OUTCOME IN PATIENTS WITH OESOPHAGEAL VARICES WITHOUT PREVIOUS BLEEDING

Variceal size	No	Variceal bleeding	Deaths	
			From variceal bleeding	From any cause
Small	15	1	1	2
Medium and large	42	9 (*21%*)	4 (*10%*)	9 (*21%*)
Total	57	10 (*18%*)	5 (*9%*)	11 (*19%*)

However, in a group of 57 patients we are following up as part of a prospective study with oesophageal varices which have not previously bled, only 10 patients (18%) had a variceal haemorrhage over a mean potential follow-up period of 20·0±7·2 months; of these, 5 died as a direct consequence of bleeding (see table). Of these patients 1 had a recently diagnosed hepatoma and 2 were in Child's grade C before bleeding. Only 1 patient with small varices suffered a variceal haemorrhage. Furthermore, the case fatality rate in our patients (19%) was considerably less than that seen in the German study (55%) over a not dissimilar period of follow-up.

The relatively low bleeding frequency seen in our patients suggests that a considerably longer period of follow-up will be necessary to find out which patients are at sufficient risk of variceal haemorrhage to justify this procedure and its repeated use.

King's College School of Medicine and Dentistry,
London SE5 8RX

PETER C. HAYES
DAVID WESTABY
ROGER WILLIAMS

SIR,—We are convinced by Dr Witzel and his colleagues that prophylactic sclerotherapy is now indicated for large varices. We would, however, be cautious about adopting it for all patients, regardless of the size of their varices. Small varices rarely bleed. Of 97 patients with a first variceal haemorrhage only 11 had bled from small varices (grade II or smaller[1,2]). 87% had bled from large varices (grade III or IV). With small varices the risk of extravasation of sclerosant is increased and complications are commoner.[3] Furthermore, monthly endoscopy, as used by Witzel et al, considerably underestimates the incidence of complications, since deep ulcers are commonest 7–10 days after injection and most have healed by 30 days.

Llandough Hospital,
Penarth, South Glamorgan CF6 1XX

PAUL M. SMITH
JAMES D. R. ROSE

1. Paquet KJ. Prophylactic endoscopic sclerosing treatment of the esophageal wall in varices: a prospective controlled randomized trial. *Endoscopy* 1982; **14:** 4–5
2. Rose JDR, Crane MD, Smith PM. Factors affecting successful endoscopic sclerotherapy for oesophageal sclerotherapy varies. *Gut* 1983; **24:** 946–49.
3 Rose JDR, Smith PM. The natural history of endoscopic oesophageal sclerotherapy complications. *Gut* 1983; **24:** A1003–04.

NON-INVASIVE MAGNETIC STIMULATION OF HUMAN MOTOR CORTEX

SIR,—This note describes a novel method of directly stimulating the human motor cortex by a contactless and non-invasive technique using a pulsed magnetic field. Merton et al[1] have drawn attention to the electrical stimulation of human brain and spinal cord using external electrodes on the skin. Interesting results have been reported on the cortical threshold in Parkinson's disease,[2] on pyramidal conduction velocity in multiple sclerosis,[3] and on pelvic neuropathy related to faecal incontinence.[4]

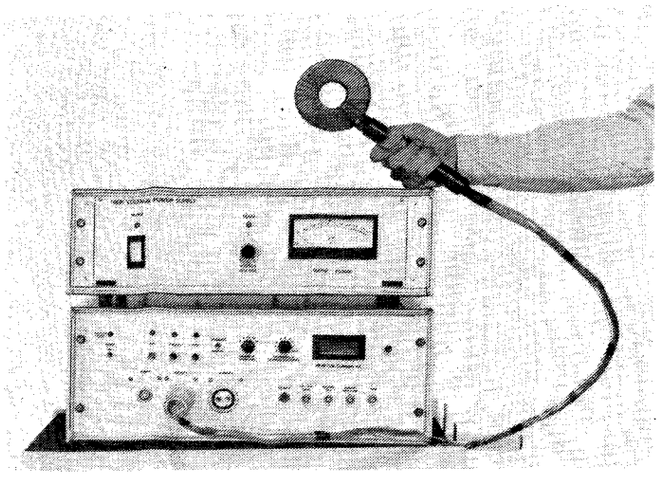

Fig 1—Magnetic stimulator and coil.

Electrical stimulation of the cortex requires careful placement of the surface electrodes and attention to other details of the technique to reduce discomfort to a level acceptable to patients. However, magnetic stimulation of the cortex is pain-free, requires no direct contact with the scalp, is non-invasive, and is easy to use.

Magnetic stimulation of peripheral nerves was reported by Polson et al.[5] The stimulator used on the cortex is similar in concept, but operates with a higher repetition rate of up to one pulse every 3 s and a delivered stimulus of up to twice the intensity. The stimulus is applied via a flat coil, of outside diameter 100 mm, through which a large, brief pulse of current (peak value 4000 A after 110 μs) is passed from a high-voltage capacitor discharge system (fig 1).

When the coil is placed on the scalp, over the appropriate region of the motor cortex, movements of the opposite hand or leg are easily obtained without causing distress or pain. The first cortical stimulations by this method were carried out with P. A. Merton and H. B. Morton at the National Hospital, Queen Square, London. Each twitch, in response to a single stimulus, is accompanied by a muscle action potential (fig 2, upper) just as with electrical stimulation through the scalp or at peripheral sites. Stimulation is assumed to be due to the current induced in the tissue by the rapid, time-varying magnetic field.

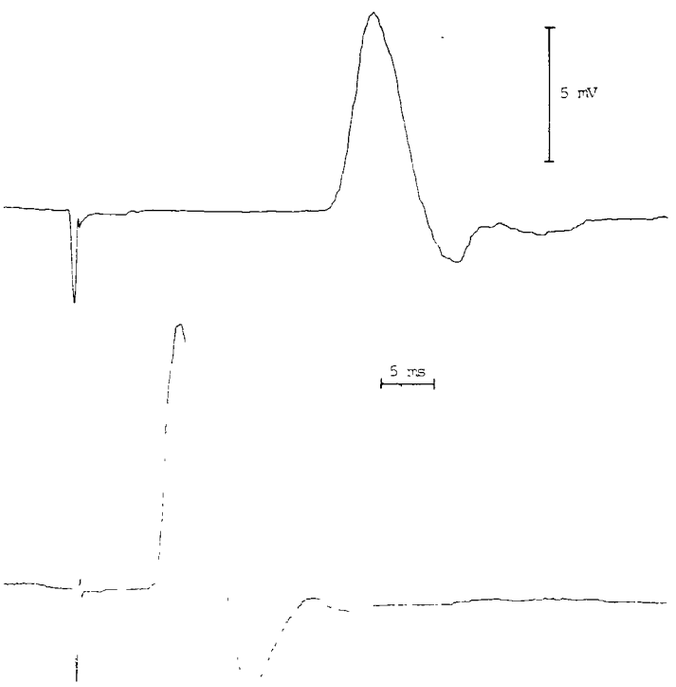

Fig 2—Muscle action potentials recorded from surface electrodes over abductor digiti minimi resulting from magnetic stimulus applied to opposite motor cortex (latency of response 23 ms) (upper) and to ulnar nerve at elbow (latency of response 7 ms) (lower).

The magnetic stimulator will also excite peripheral nerves (fig 2, lower) with minimal discomfort, although where the nerve is superficial it has little advantage over conventional stimulation. However, where a nerve lies deeper—for example, the median or ulnar nerves in the centre of the forearm—magnetic stimulation is readily achievable, whereas conventional stimulation causes considerable discomfort.

Magnetic stimulation of the cortex is particularly effective because of the ability of the field to pass through high-resistance structures. The skull has 8–15 times the resistivity of soft tissues[6] and so offers a considerable barrier to electrical stimuli. Roughly the same size of magnetic stimulus is needed for the motor cortex as for peripheral nerves.

Magnetic stimulation is rapid and easy to use in the clinical environment because it is not necessary to attach stimulating electrodes to the patient. Additionally the coil may be readily moved over the scalp until the desired stimulation site is located. The ability to stimulate corticospinal motor pathways allows their function to be assessed in many neurological conditions or monitored during surgical procedures. Magnetic stimulation is a major advance in the implementation of such studies.

Sheffield University and Health Authority
Department of Medical Physics
and Clinical Engineering,
Royal Hallamshire Hospital,
Sheffield S10 2JF
A. T. BARKER
R. JALINOUS

Department of Electronic
and Electrical Engineering,
University of Sheffield
I. L. FREESTON

1. Merton PA, Hill DK, Morton HB, Marsden CD. Scope of a technique for electrical stimulation of human brain, spinal cord, and muscle. *Lancet* 1982; ii: 597–600.
2. Dick JPR, Cowan JMA, Day BL, Berardelli A, Kachi T, Rothwell JC, Marsden CD. The corticomotoneurone connection is normal in Parkinson's disease. *Nature* 1984; **310:** 407–09.
3. Cowan JMA, Rothwell JC, Dick JPR, Thompson PD, Day BL, Marsden CD. Abnormalities in central motor pathway conduction in multiple sclerosis. *Lancet* 1984; ii. 304–07.
4. Snooks SJ, Swash M. Pudendal nerve terminal latency, and spinal stimulation. In: Henry MM, Swash M, eds. Coloproctology and the pelvic floor. London. Butterworths, 1985: 112–24.
5. Polson MJR, Barker AT, Freeston IL. Stimulation of nerve trunks with time varying magnetic fields. *Med Biol Eng Comput* 1982; **20:** 243–44.
6. Adrian ED, Yamagiwa K. The origin of the Berger rhythm. *Brain* 1935; **58:** 323–51.

SAFETY OF NMR

SIR,—The appearance of an article on the safety of nuclear magnetic resonance (NMR) is welcome (April 20, p 913). However, it is difficult to discuss such a complex topic adequately within the space of a *Lancet* editorial, and the result is omissions and oversimplifications. You give the impression that NMR instruments avoid the problems of static magnetic fields, varying field gradients, and radiofrequency heating and that, providing the patient has no intracranial clips, no pacemaker, and is not in the first trimester of pregnancy, all reasonable precautions have been taken.

The effects of static magnetic fields include changes in EEGs, blood cells, electrolytes, and adrenal glands, delayed wound healing, accelerated allograft rejection, fetal maldevelopment, and reduced tolerance to anoxia.[1-3] Also claimed is longer life, less hostility, and more youthful appearance. Few of these effects have been proved—but few, if any, have been refuted. Some of these effects have been described at fields much less than 2·5 tesla (T). The choice of 2·5 T was to some extent arbitrary and was based principally on the magnitude of Hall effect electromagnetic fields in the aorta.[4] Limiting fields to 2·5 T does not guarantee freedom from harmful effects.

The recommended limit for rates of change of field is based on the current density required to induce ventricular fibrillation in normal myocardium. When the myocardium is abnormal there may be no safe limit.[5]

It is difficult for a manufacturer to produce an instrument which is versatile but which cannot be configured to produce unsafe radiofrequency power levels. Whether such machines should be in clinical use is arguable, but the fact is that they are, and those using them must appreciate the power output of the transmitter, the