on it is essential that there should be a sanction, such as automatic reinstatement of any suspended doctor if the health authority is dilatory. Furthermore, in making these time limits one key stage is missing—that between the doctor being suspended and the health authority deciding that a prima facie case exists against the doctor. This stage can take vears because health authorities will recklessly suspend doctors on the basis of rumour and then have to labour hard to find something, anything, to justify the suspension and to avoid legal redress by the aggrieved doctor. Nevertheless, we agree with Dr Forsythe in welcoming the proposals for a graded treatment for graded degrees of misconduct.

Dr Forsythe seems to think that the paragraph 190 procedure is a true appeal mechanism. But the Minister of Health has recently ignored the verdict and dismissed a doctor who won his appeal. Surely the verdict of any appeal committee should be binding until overturned by a higher authority? Because the NHS is a monopoly employer all hospital doctors must have the right to appeal to the Secretary of State against any dismissal by a health authority. That is not the case at the present. Any doctor can be summarily dismissed without warning on any charge and is denied the right of appeal. This interpretation of the law has recently been upheld in the High Court.

The joint working party still favours secret trials of doctors—Why? The disciplinary hearings of the General Medical Council are heard in public, and as a result the profession is held high in the public esteem for its efforts in keeping its house in order. Lord Justice Brown in his judgment of a disciplinary case against a doctor in 1986 stated: "The maxim that justice must not only be done but must manifestly be seen to be done applies in full measure" (Crown Office 595/86). In that judgment he was only following article 6 (i) of the European Charter of Human Rights. All inquiries of suspended doctors should be held in public. Only the guilty have something to fear, and in condoning secret trials the profession will, rightly or wrongly, be seen as conspiring against the public weal.

The Society of Clinical Psychiatrists has produced a code of conduct about suspended doctors. The main thrusts of the proposals are that, except in an emergency, doctors may not be suspended until after investigation, that suspensions should follow the Advisory, Conciliation, and Arbitration Service code of practice, that the NHS should not usurp the function of the General Medical Council in deciding if a doctor is professionally incompetent or guilty of professional misconduct, that HM (61) 112 should be abolished, that the verdict of any appeal mechanism should be binding on both sides, and that all changes should conform to the European Charter of Human Rights and to the European employment laws.

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1 Forsythe M. Real progress with disciplining doctors. Br Med $\mathcal J$ 1988;297:1487-8. (10 December.)

Lectures and lecturers

SIR,-The thoughts expressed by Professor Michael Shepherd raise some interesting questions.1 Taking as his text "Why lecture?" quotation from an essay by Virginia Woolf, he considers various teaching situations and seems to adopt the view that lectures are redundant. In this, of course, he has the distinguished support of Alice in Wonderland, who told her tutor, "'I think I should understand that better,' Alice said very politely, 'if I had it written down: but I'm afraid I can't quite follow it as you say it."

The question which has been worrying me over the holiday is why do audiences continue to assemble for lectures offered by learned societies, by postgraduate medical centres, and at numerous meetings throughout the year, and why do speakers continue to appear? A film or television recording can offer an alternative to the presence of a lecturer but only if the material is suitably adapted for the medium; a recording of a man reading his notes - perhaps with a few slides on the screen - is no substitute.

The paper concludes with a quotation from Dr Johnson, suggesting that reading a book is better than attending a lecture on the subject. This is not borne out by experience. After lecturing for some years on a (non-medical) subject I was encouraged to write a book on it; rather than there being a reduction in the number of times I am invited to address meetings, requests have quadrupled since publication. Professor Shepherd touches on one advantage of a lecture over a book-the presentation can be related to the particular needs of the audience. Indeed, the speaker can benefit too because questions and discussion during a lecture can highlight those parts of the talk which could be improved.

Doctors, it seems, will continue to attend meetings expectantly, even though they have often been disappointed by lectures in the past.3

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- 1 Shepherd M. Lectures and lecturers. Br Med J 1988;297:162-3 (24-31 December.)
- Carroll L. Alice's adventures in wonderland.
- 3 Essex-Lopresti M. Illuminating an address: a guide for speakers at medical meetings. Med Educ 1980;14:8-11.

Drug Points

Widespread tremor after injection of sodium calcitonin

Drs J I CONGET, J VENDRELL, I HALPERIN, and E ESMATJES (Endocrinology Unit, Hospital Clinic, University of Barcelona, Barcelona 08036, Spain) write: Synthetic calcitonins are effective for diseases associated with an increase in bone resorption and turnover-for example, Paget's disease, hypercalcaemia of malignancy, and primary hyperparathyroidism. Side effects occur in up to 10% of patients, and, although often mild, they are severe enough to stop treatment in over a third of patients.12 We here describe a case of widespread tremor after subcutaneous administration of salmon calcitonin

A 35 year old insulin dependent diabetic woman was admitted with a three week history of painful ankle. After clinical and radiological evaluation we diagnosed reflex dystrophy of the ankle. Rest and salmon calcitonin were prescribed. All biochemical and haematological values at admission were normal. Within about 90 minutes of the first subcutaneous injection of 100 IU of salmon calcitonin (Sandoz) the patient developed a widespread fine tremor. It affected the head and the arms and legs, which were equally affected, though the tremor was most evident on the distal parts of the limbs. The tremor lasted about an hour. Similar tremors occurred after subcutaneous injection of the same dose of another batch of the same calcitonin and another make of salmon calcitonin (Armour). Tremor did not occur after placebo. Serum calcium concentrations before and during (90 minutes after the injection) the episodes of tremor were normal.

The most common side effects of salmon calcitonin include gastrointestinal symptoms (nausea, vomiting, abdominal pain, diarrhoea) and vascular symptoms such as facial flushing and tingling of the hands. Erythema and pain at injection sites, polyuria, rash, and an unpleasant metallic taste have also been described. They usually occur within one hour of injection and may be diminished by use of the subcutaneous rather than the intramuscular route.3 In our patient the timing and repeated appearance of widespread tremor after subcutaneous injection of salmon calcitonin suggest that the association was not fortuitous. There is evidence that during the process of producing synthetic calcitonins impurities (erroneous peptides) can appear, which vary with the batch and the make. Some side effects of these products therefore relate to these substances. In our patient, however, we observed widespread tremor after several batches and two different makes of salmon calcitonin, but the serum calcium concentration was normal throughout. On the other hand, the calcitonin gene encodes the precursor peptides for calcitonin and calcitonin gene related peptide. The calcitonin pathway predominates in the C cells of the thyroid and the gene related peptide in the nervous system. The gene related peptide shows greater homology with salmon calcitonin than the human calcitonin, and this homology has been invoked to explain the putative effects of salmon calcitonin on the nervous sytem.56 This point is very attractive as a pathogenic hypothesis in our case, although the role of calcitonin gene related peptide in normal human physiology is controversial. Nevertheless, widespread tremor should be considered as an additional side effect of salmon calcitonin treatment.

- 1 McIntyre I, Evans IMA, Hobitz HHG, Joplin GF, Stevenson JC. Chemistry, physiology and therapeutic applications of calcitonin. *Arthritis Rheum* 1980;23:1139-47.
- 2 Singer FR, Fredricks RS, Minkin C. Salmon calcitonin therapy for Paget's disease of bone. Arthritis Rheum 1976;23:1148-54.
- 3 Gennari C, Passeri M, Chierichetti SM, Piolini M. Side effects of synthetic salmon and human calcitonin. Lancet 1983;i:594-5.
 4 Bangham DR, Zanelli JM. Side effects of calcitonins. Lancet
- 1983;i:926-7
- 5 Tschopp FA, Henke H, Petermann IB, et al. Calcitonin generelated peptide and its binding sites in the human central nervous system and pituitary. Proc Natl Acad Sci USA
- 6 Morris HR. Panico M. Etienne T. et al. Isolation and characterization of human calcitonin gene-related peptide. Nature 1984:308:746-8.

Thrombocytopenia induced by angiotensin converting enzyme inhibitors

Drs B GROSBOIS, D MILTON, C BENETON, and D JACOMY (Department of Internal Medicine and Drug Surveillance Laboratory, Hopital Sud, Rennes, France) write: The angiotensin converting enzyme inhibitors are known to induce leucopenia and neutropenia, particularly in patients with renal insufficiency and autoimmune diseases. Thrombocytopenia is usually associated with aplastic anaemia,2 but there are only two reports of pure thrombocytopenia.34 Two sisters with idiopathic hypertension were treated with angiotensin converting enzyme inhibitors, which induced thrombocytopenia after nine days and eight weeks of treatment. We assessed the likelihood of drug induced thrombocytopenia using a scale for chronological (C=0-3) and semilogical (S=0-3) features produced by a French consensus

Case 1-A 76 year old woman with no history of allergy or autoimmune disease was admitted in March 1985 with mucosal and cutaneous purpura which had appeared 24 hours earlier. A blood count showed severe and isolated thrombocytopenia (1×10⁹/l); a bone marrow aspirate showed megakaryocytes. She had received quinidine (100 mg/day) since 1983 and enalapril (20 mg/day) for the previous 10 days. After withdrawal of these drugs, a platelet transfusion, and treatment with prednisone (50 mg/day for five days) her platelet count became normal (229×10⁹/l). HLA typing was A2 B8 DR3/A9 B13 DR7. She was discharged with no treatment and her platelet count remained