

purity aside, there is at least one practical reason for paying as much attention to the chemical language as to the English language in articles in *The Lancet*. Incorrect usage or spelling is unlikely to enhance the accessibility of a paper in computer searches based on key words.

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*This letter has been shown to Dr Winship, whose reply follows.—ED. L.

SIR,—Your correspondent's criticism is valid and it reflects a proof-reading oversight on the part of authors and editor. In our manuscript's original form the standard abbreviation CpG was used throughout, to denote the deoxy (cytidyl) 3':5' guanosine dinucleotide moiety, more loosely referred to by Professor Gray as "cytidylguanosine". However, the Editor insisted that this standard abbreviation in the title and at first mention in summary and main text be expanded into a more detailed chemical name (cytosine phosphoguanidine), despite our strong objections. The only strictly correct way of naming a chemical compound fully is by its chemical formula (preferably accompanied by its structure); anything else is merely an abbreviation of some form. The Editor argued that more of his readers would recognise his abbreviated way of representing this dinucleotide moiety than the commonly used abbreviation CpG. I contend that a higher proportion of *Lancet* readers know the four bases of DNA as A, G, C, and T rather than as adenine, guanine, cytosine, and thymine, and would understand the abbreviation CpG, based upon the IUPAC one letter system,¹ better than cytosine phosphoguanidine. Having had such a confusing abbreviation imposed upon us we may be forgiven for overlooking the minor error of "phosphoguanidine" appearing as "phosphoguanadine"—indeed, *The Lancet* made the same error, having it right on the contents page but not in the article.

I suggest that in these days of computer-aided literature searches *The Lancet* should review its policy of not using standard abbreviations such as CpG, especially in titles. If not, literature searches by computer may miss potentially important articles in the journal.

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1. IUPAC: IUPAC Commission on Biochemical Nomenclature (CIBN). Abbreviations and symbols for nucleic acids, polynucleotides and their constituents. *J Mol Biol* 1971; 55: 299-310.

*We thank Lois Ann Colaanni, of the National Library of Medicine, Bethesda, Maryland, for testing the accessibility of "CpG" in the MEDLINE index. One search yielded 92 titles but not Dr Winship's paper. However, a computer-retrievable abbreviation may not be understood by all readers. We now accept that the paper's title should have read "Detection of Polymorphisms at Deoxy(cytidyl-3',5'-guanosine) dinucleotides (CpG) and Diagnosis of Haemophilia B Carriers". The NLM has been informed.—ED. L.

FISH OIL IN OSTEOARTHRITIS

SIR,—There has been much interest in the use of fish oils in the treatment of rheumatoid arthritis,¹ since the effectiveness of eicosapentaenoic acid (EPA) in the alleviation of the symptoms of rheumatoid arthritis has been demonstrated in several hospital-based studies.^{2,3} An inflammatory component in osteoarthritis is well established.⁴ However we know of no data on the use of EPA in osteoarthritis, and we report a pilot study to investigate the effect of EPA as an adjunct to ibuprofen in the treatment of osteoarthritis in general practice.

We studied 21 women and 5 men, aged 52-85 years, with a clinical diagnosis of osteoarthritis made by their general practitioner, confirmed by radiology in 22 (85%). These patients had symptoms after taking ibuprofen 1200 mg daily for at least two

MEAN VISUAL ANALOGUE LINE SCORES

	Pain			Interference with activities		
	Mo 1	Mo 6	Difference	Mo 1	Mo 6	Difference
EPA	42.8	27.1	15.7	33.7	23.5	10.2
Placebo	44.0	34.8	9.2	38.2	34.1	4.1

weeks before entry to the study. They were then given 10 ml of EPA or placebo oil daily in addition to ibuprofen for six months. The two treatment groups did not differ significantly at the outset or conclusion of the study with respect to mean daily dose of both oil (either EPA or placebo) and ibuprofen.

The patients assessed pain and interference with everyday activities on separate 100 mm visual analogue lines each day of every fourth week (table). The average scores for both these indices at week 24 were strikingly lower in the EPA than the placebo group. Although this difference was not statistically significant, we believe that these findings more than justify a full-scale clinical trial into the effect of EPA in osteoarthritis.

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ROSANNE SHIRALI
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4. Altman R, Gray R. Inflammation in osteoarthritis. *Clinics Rheum Dis* 1985; 11: 453.

IS TANNIN REALLY BACTERICIDAL?

SIR,—Your June 3 note (p 1279) refers to Dr M. Toda and colleagues' report that extracts of tea and coffee inhibit the growth of bacteria causing gastrointestinal disease. The components of the beverages producing this bactericidal action being unknown, tannin seemed a good candidate, in your opinion. Tea does contain tannin but the fact that coffee contains very little excludes the possibility of tannin being the relevant component. Both coffee and tea stimulate gastric acid secretion and increased acidity in the stomach may be the bactericidal factor. In the 1850s Max von Pettenkofer swallowed a large amount of a culture of the cholera bacillus in an attempt to disprove the, in his opinion, biased bacteriological theory of Robert Koch. Surprisingly enough nothing happened to him—but later on his opponents found out that he was suffering from gastric hyperacidity.

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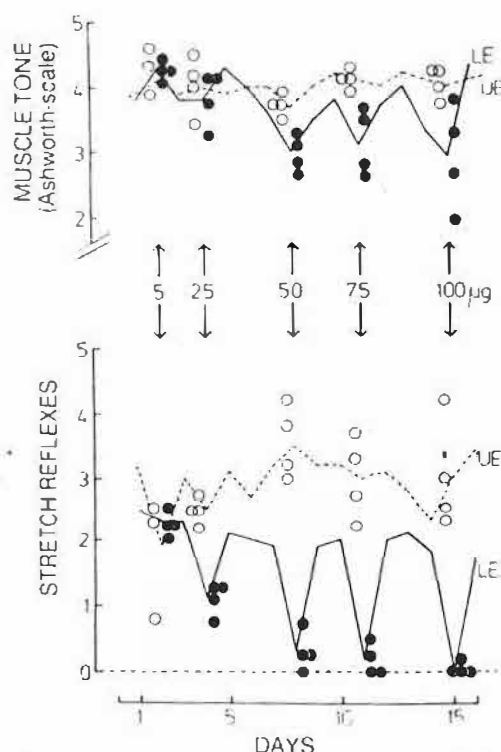
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INTRATHECAL BACLOFEN FOR INTRACTABLE SPASTICITY DUE TO SEVERE BRAIN INJURY

SIR,—As Perin and colleagues^{1,2} have demonstrated, intrathecal baclofen acts favourably in severe spinal spasticity. However, very little is known about its effectiveness in chronic intractable spasticity due to severe brain injury.

We report a 29-year-old man with diffuse brain oedema and intracerebral haematoma after severe head and brain injury. He had all the characteristic features of an apallic syndrome.³ Despite high doses of oral baclofen and tizanidine and physiotherapy, extreme spasticity of both legs and arms developed. Five months after the accident intrathecal application of baclofen was started. Increasing test bolus doses of 5, 25, 50, 75, and 100 µg were given at the same time in the morning (figure). Spasticity and selected reflexes of both arms and legs were assessed daily by two independent examiners. This assessment showed an apparently dose-related attenuation of stretch reflexes, lasting up to 9 hours, and a slight reduction in muscle tone of the lower, but not the upper, extremities (fig) probably attributable to the site of application.⁴ No adverse effects



Muscle tone and stretch reflex measurements and dose of intrathecal baclofen.

Lines = averages, circles = individual measurements; UE = upper extremities, LE = lower extremities.

Upper: 5 = rigid, 1 = no increase; ○ = movements in shoulder, elbow, wrist; ● = movements in hip, knee, ankle.

Lower: 5 = clonus; 0 = no reflex; ○ = tendons of biceps, triceps, brachioradialis; ● = tendons of quadriceps, triceps surae.

were seen, but because of the very small effect on spasticity of the 100 µg bolus dose neither a further increase of dose nor continuous intrathecal infusion was felt to be justified.

The lack of clinical improvement in our patient with cerebral spasticity is in contrast to the beneficial effects observed in spinal spasticity.^{1,2,5,6} This finding might reflect differences in the underlying pathological processes. The term cerebral spasticity covers various conditions caused by lesions rostral to the brainstem precipitating especially disabling antigravity patterns in arm flexor and leg extensor muscles. The general view is that this type of spasticity results from unbalanced facilitation and inhibition of the spinal cord by the damaged or disconnected cerebral cortex.⁷ In the dosage used by us without success in cerebral spasticity, intrathecal baclofen has been shown to diminish effectively and reliably increased muscle tone due to spinal cord lesions. This finding seems to indicate that reduction of excitatory input of primary afferent fibres to alpha-motoneurons at the spinal level, essential for baclofen's effectiveness in spinal spasticity,⁸ is less important for alleviation of spasticity of supraspinal origin.

However, successful treatment of cerebral spasticity with intrathecal baclofen has been reported by Dralle et al⁹ in a 4-year-old boy with long-standing hypoxic apallie syndrome showing typical antigravity patterns in arms and legs. An initial test bolus dose of 50 µg effectively eliminated spasms and reduced muscle tone of the upper and lower extremities. This is a surprisingly high dose in view of the fact that maximum test bolus doses of 50 µg⁶ or 75 µg¹ are used in adults with spinal spasticity. Perhaps, however, higher intrathecal baclofen concentrations are needed to be effective in cerebral spasticity, a notion that also seems to be consistent with our findings. But great care is indicated. As do patients with spinal spasticity,¹ those with cerebral spasticity might show pronounced interindividual differences in sensitivity to

intrathecal baclofen. The striking results in spinal spasticity should encourage exploration of this promising new approach for the treatment of intractable disabling cerebral spasticity.

We thank Prof H. Glossman for his help in obtaining baclofen, which was kindly supplied by Ciba-Geigy, Basel.

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BARORECEPTOR RESET WITH NITROPRUSSIDE AND DRUG-RESISTANT HYPERTENSION

SIR,—We report two cases of resistant hypertension (ie, hypertension unresponsive to a good multidrug regimen) successfully managed by periodic intravenous nitroprusside infusions.

In April, 1988, a 44-year-old man was admitted to hospital in a hypertensive crisis (260/150 mm Hg), with left eye blindness, frontal headache, and palpitations. Investigation ruled out a secondary origin for his hypertension, and primary hypertension had been diagnosed 6 years earlier. At the time of admission the patient was on a regimen of guanethidine, frusemide, spironolactone, atenolol, and enalapril, which had been started 6 months earlier, after several other drug regimens had failed. While he was on this five-drug regimen his blood pressure (BP) had ranged between 150/95 and 170/110 but in the week before admission his BP increased progressively despite a good compliance, as judged by pill counting and by an interview with his wife. The exacerbation in his hypertension was not preceded by administration of other drugs such as a non-steroidal anti-inflammatory agent or by changes in life-style and diet.

His BP fell to 160/105 after 300 mg diazoxide intravenously but further attempts to control BP by diazoxide were unsuccessful.

Conventional regimens having apparently been exhausted, we tried a new approach to the control of this man's resistant hypertension. Since upward resetting of the depressor reflexes has been described^{1,2} we speculated that a downward reset might improve pharmacological control of raised BP. We tried to induce such a reset by the intravenous infusion of nitroprusside over 36 hours; the rate of the infusion was regulated to maintain the BP around 130/80. 3 days after the infusion of nitroprusside the patient's BP remained at 160/95 or less and the patient was discharged without alteration in the previously reported therapy. His BP stayed between 160/100 and 140/90 for 22 days but then rose and a second cycle of nitroprusside was given. The benefit lasted for 27 days. So far nineteen such cycles have been necessary, and their therapeutic effect has ranged from 19 to 31 days.

Similar results have been obtained in another hypertensive patient who started the periodic nitroprusside infusions in July, 1988. In this patient, the induction of baroreflex reset was demonstrated by the method of Eckberg et al.³ However, we cannot exclude the possibility that as yet unknown mechanisms other than