

bleeding tendency cannot be explained on the basis of a lowered VWFR. The disproportionate increase in VIIIc compared with VIIIA and VWFR may represent a non-specific effect (*sic*) analogous to similar results seen after exercise.⁵ Nevertheless, in four patients the ratio VIIIA:VWFR was greater than 1.5, suggesting that the functional capacity of the factor VIII molecule (as assessed by ristocetin cofactor activity) may be impaired in some cases of renal failure.

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- ¹ Kazatchkine, M, *et al*, *British Medical Journal*, 1976, **2**, 612.
² Biggs, R, *Human Blood Coagulation, Haemostasis and Thrombosis*, 2nd edn, p 684. Oxford, Blackwell, 1976.
³ Laurell, C B, *Scandinavian Journal of Clinical and Laboratory Investigation*, 1972, **29**, suppl 124, 21.
⁴ Macfarlane, D E, *et al*, *Thrombosis et Diathesis Haemorrhagica*, 1975, **34**, 306.
⁵ Stibbe, J, *Thrombosis Research*, 1977, **10**, 163.

Music in the hemispheres

SIR,—In referring to the recently published paper of some Japanese authors¹ Minerva suggests (4 June, p 1476) that “the right hemisphere may be dominant for singing” since “cases have been recorded of right-sided lesions causing loss of musical ability while leaving speech unimpaired.” However, impairment of musical ability together with intact speech has also been noted with certain left-sided lesions.² None the less, there is good evidence for believing that singing is primarily subserved by the right cerebral hemisphere. In the rare cases of total left hemispherectomy performed on adults preservation of the ability to sing has been observed despite severe aphasia.³ In addition, intracarotid injection of sodium amytal on the right side has been found to impair singing to a greater extent than injection on the left.⁴

These findings for vocal musical expression are consistent with data from brain-damaged patients which indicate a superiority of the right hemisphere with regard to various receptive aspects of music. Furthermore, a technique known as dichotic listening has enabled these observations to be extended to normal subjects. Among right handers the simultaneous presentation of competing stimuli to the two ears may yield laterality effects related to the nature of the stimuli. Using verbal stimuli, significantly more correct responses are typically obtained in recognition of material at the right ear, while with non-verbal stimuli, particularly musical sounds, there is often an advantage for the left ear.⁵ Such findings are usually explained on the basis of suppression of the uncrossed by the crossed auditory projections to the cortex combined with a relative dominance of the left and right cerebral hemispheres for the perception of verbal and non-verbal stimuli respectively. It should be noted, however, that such effects may be related more to fundamental differences in the processing strategies employed by the two halves of the brain rather than to the intrinsic nature of the stimuli employed, for the same stimuli can give rise to opposite ear advantages depending on the task requirements.

Finally, in connection with the fact that singing may be preserved despite severe

aphasia it is of interest that one patient whose left hemisphere was removed at the age of 10 years was able to sing well only weeks after the operation, yet two years later was still unable, or unwilling, to repeat the lyrics in a normal speaking voice.⁶ This recalls to mind Hughlings Jackson's view that, while “propositional speech” is undertaken by the left hemisphere, the right side of the brain is responsible for “the automatic revival of words.” Yamadori *et al* noted that all but five of their 21 aphasic patients required prompting by the examiner before singing as requested. These authors therefore suggested that the ability to initiate singing spontaneously is more related to the left hemisphere than to the right. Could it be that once primed, as it were, by the left hemisphere, or by explicit prompting, the right half of the brain can run on “automatically,” this process being facilitated in some way by the availability of a familiar melody?

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- ¹ Yamadori, A, *et al*, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1977, **40**, 221.
² Wertheim, N, in *Handbook in Clinical Neurology*, ed P J Vincken and G W Bruyn, vol 4. Amsterdam, North Holland, 1969.
³ Smith, A, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1966, **29**, 467.
⁴ Gordon, H W, and Bogen, J E, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1974, **37**, 727.
⁵ Kimura, D, *Cortex*, 1967, **3**, 163.
⁶ Gordon, H W, in *Hemisphere Disconnection and Cerebral Function*, ed M Kinsbourne and W L Smith. Springfield, Thomas, 1974.

Hyperbaric oxygenation in paralytic ileus

SIR,—The gratifying results of this treatment, reported by Dr R E Loder (4 June, p 1448) have been observed by myself and others.¹ Beside the possible reasons for the improvement that he advances, a purely physical explanation could account for the spectacularly rapid recovery. Boyle's law indicates that at 2 atmospheres the volume of intestinal gas is reduced by 50%, while in the cylindrical form, which occurs in the lumen of the bowel, the diameter is reduced by 29%.² Because of the rapidity of the response it seems probable that the sudden relief of gaseous distension allows circulation in the bowel wall to be re-established and permits peristalsis to recommence.

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- ¹ Watanuki, T, *et al*, “Study on the effects of hyperbaric oxygenation upon intestinal paralysis,” *Hyperbaric Medicine*, ed J Wada and T Iwa. Tokyo, Igaku Shoin, 1970.
² Brown, I W, *et al*, *Advances in Surgery*, 1965, **1**, 292.

Coronary artery spasm

SIR,—Your editorial on coronary artery spasm (7 May, p 1176) mentions facts probably relevant to the mechanism of death while shovelling snow. All five cases reported by Dr R M Whittington (26 February, p 577) had post-mortem evidence of considerable coronary artery atherosclerosis and therefore were liable to react to the cold pressor test of the environment by a rise in blood pressure accompanied by a decreased coronary blood flow from an increase in coronary vascular resistance.¹ The wind blowing on the day of the deaths, apart from increasing the chill factor, may also have caused a bradycardia.² These factors are possibly sufficient in themselves to cause death

without requiring the addition of unaccustomed, vigorous isotonic/isometric exercise or incriminating a stream of cold air down the trachea.³ In fact the heat exchange capability of the respiratory system is such that the inspired gases will be at body temperature by the time they reach the carina.

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- ¹ Mudge, G H, *et al*, *New England Journal of Medicine*, 1976, **295**, 1333.
² Le Blanc, J, in *Circumpolar Health*, ed R J Shephard and S Itoh, p 72. Toronto University Press, 1976.
³ Wells, C, *British Medical Journal*, 1977, **1**, 908.
⁴ Moritz, A R, *et al*, *American Journal of Pathology*, 1945, **21**, 311.

Management of rheumatoid arthritis

SIR,—It is a pity to see in your leading article (30 April, p 1120) emphasis on the treatment of rheumatoid arthritis (RA) yet again placed on a particular group of drugs—the non-steroid anti-inflammatory agents (NSAID)—and on “management by symptoms.” Surely it is now generally accepted that all the group 1-3 drugs in Huskisson's¹ earlier classification have a temporary local effect but no influence on the course of the disease. Little effort need be spent in selecting which drug to use; the patient's preference and cost are what matter. “Management by symptoms” is appropriate only at this stage.

The more difficult problem, which should occupy a correspondingly greater proportion of any survey on this subject, is what to do next. The decision should be made on objective criteria. Serial measurements of serum acute-phase proteins (APR) and the ESR provide a guide.² These measurements are not influenced by NSAIDs, indicating the limitation of this class of drugs. Drugs which do lower APRs include gold and penicillamine, with their characteristically slow effect, and corticosteroids, with a rapid effect; although none of them has been clearly shown to halt disease progression, they probably delay it. Alternatively, progression of RA can be judged by tests of function or by x-rays; but each provides only retrospective information and is not as suitable as APR.

If NSAID do not adequately control symptoms and APR and ESR levels remain high, then disease progression will ensue; some additional treatment is indicated. “Management by symptoms” at this stage will not do; objective measurements are mandatory. Only such measurements can provide the information for efficient monitoring of drug effects and for assessing the relative merits of drugs that could affect disease progression. It may be necessary to resort to more than one such drug either serially or together. We have suggested elsewhere³ a policy, based on serial APR measurements, to cover most eventualities. So long as “management by symptoms” continues to be the goal we fear a proliferation of NSAID instead of the development of drugs with a more profound effect; we also fear ineffective, albeit relatively safe, management of RA.

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- ¹ Huskisson, E C, *Reports on Rheumatic Diseases*, No 54. London, Arthritis and Rheumatism Council, 1974.
² McConkey, B, *et al*, *Quarterly Journal of Medicine*, 1973, **NS 42**, 785.
³ Constable, T J, *et al*, *Lancet*, 1975, **1**, 1176.