

therefore revert to its original name of occupational neuralgia of the radial nerve and its branches at the elbow, or to some other name indicative of the pathology of the condition."

There is also a reference to the radial nerve as the site of compression causing tennis elbow symptoms in a paper written by Mr. Norman Capener in 1966,² in which he described constriction of the posterior interosseous nerve by a tumour, and it was noted that Dr. D. H. Tompsett had carried out similar dissections to those of Mr. Yakoub. It was the publication of this paper which made me decide not to do anything further about the publication of my own work at that time.

However, I think I have established in this letter that the idea of a radial tunnel syndrome is not new. The credit for the original description, as so often in medicine and surgery, belongs to the great surgeons of the past, in this case Sir Charles Bell. It is another "Bell's palsy."—I am etc.,

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- ¹ Roles, N. C., and Maudsley, R. H., *Journal of Bone and Joint Surgery*, 1972, 54B, 499.
² Capener, N., *Journal of Bone and Joint Surgery*, 1966, 48B, 770.

Lithium Intoxication

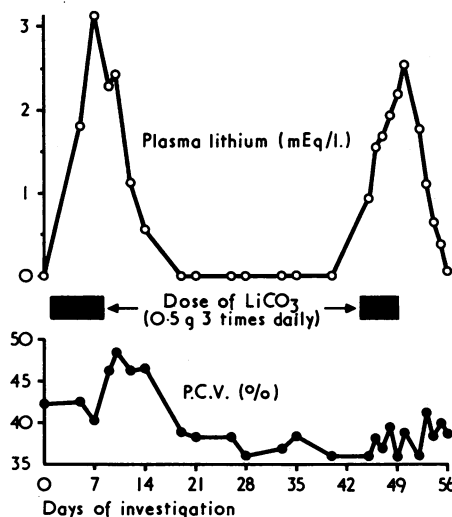
SIR,—I was interested to read the account by Dr. B. von Hartitzsch and others (30 December, p. 757) of neurological phenomena persisting in two of three patients even after reduction of high blood lithium levels with haemodialysis. It is not surprising that the neurological phenomena persisted, for extrapyramidal changes can occur during lithium therapy while the blood levels remain low.

It has been recognized for some time that there is no consistent relationship between the blood lithium level and side and toxic effects. This is presumably because side effects may be associated with a lithium-induced extracellular or intracellular disturbance, or both. When extracellular phenomena develop, usually during the early days of therapy, it is possible that it is the lithium transport system that is responsible owing to its effect upon the cellular membrane, although the blood lithium level remains below 2 mmol/l. This is to be contrasted with the intracellular disturbance, which is usually associated with a major disruption of the electrolyte balance, as manifest by the levels of the electrolytes. In this situation the blood lithium level is above 2 mmol/l.

Taking the upper gastrointestinal tract as an example, the pattern of symptoms can progress along either one of two pathways. Thus it is not unusual for nausea or epigastric fullness to be experienced within three or four days of the commencement of ingestion of lithium, and this sometimes causes the patient to discontinue medication. A blood level taken at this time gives a reading invariably below 1 mmol/l., and the symptoms therefore represent an example of extracellular disturbance. It is not surprising that it occurs, because the lithium ion permeates the body quickly. These gastric symptoms must be contrasted with those associated with the intracellular disturbance, in which the patient is much more likely to be vomiting recurrently. The vomiting tends to persist for several days after the

cessation of lithium treatment and in this situation the blood lithium level will be at least 2 mmol/l. and will rise rapidly.

In the accompanying chart the blood lithium levels of a 75-year-old woman who suffered from severe and recurrent attacks of mania and depression are shown. The first peak, which commenced as soon as lithium



therapy started, was associated with dehydration, as the raised packed cell volume (P.C.V.) shows, and treatment had to be discontinued owing to the appearance of side effects. The second peak, commencing on the 44th day, represents another attempt to give lithium therapy after the dehydration had been dealt with and the P.C.V. had returned to normal. This attempt, however, had to be discontinued on the 49th day because the blood lithium level again began to rise quickly, even before any clinical evidence of side effects had been observed. The chart shows that a reliable and predictable pattern of blood level occurs in some patients so as to allow one to say that in these, at any rate, the high blood lithium levels precede the toxic effects, and if monitored in this way can give an early warning of a developing toxic state.

Contrary to the figures quoted by Dr. Von Hartitzsch and his colleagues, there is no evidence that an optimum level for lithium therapy exists. However, regular assessment of blood lithium levels should be carried out weekly during the first month for the following reasons: (1) it will indicate whether or not the patient is taking the medicine; (2) if untoward effects occur the significance of these can be assessed; (3) if toxic states develop the effect of detoxication therapy can be evaluated more clearly from this original baseline and the likely pathology determined; and (4) regular assessment will indicate when it is thought that lithium equilibrium has been achieved, and it is this state that is thought to be most valuable in the treatment programme. The dosage of lithium should be estimated against the body weight, and if there is any indication of previous renal disease then a lithium loading test is most valuable.

It is to be hoped, however, that the description of these two instances of neurological disorder will not deter doctors from considering lithium therapy in recurrent affective disorders. From its very early days lithium has been recognized as capable, like any other drug, of producing toxic effects.

These risks are well known and their pharmacology has been well described by Schou.¹ They have to be balanced against the risks of suicide and recurring episodes of severe disability. It is, moreover, important to recognize the value of careful selection of patients for treatment, making allowances in the dosage schedule for body weight, dehydration, and renal phenomena. This, together with clinical observation and assessment of electrolyte levels, will do much to reduce the incidence of toxic phenomena.—I am, etc.,

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¹ Schou, M., *Pharmacological Reviews*, 1957, 9, 17.

Cost of Drugs

SIR,—One of the important elements in the high cost of the National Health Service must be the prescribing and dispensing of drugs which are much more expensive than standard preparations but have exactly the same therapeutic effect. Disparities in the price of corticosteroids provide a striking example of the way in which public money is being squandered by doctors who do not take the trouble to find out the cost of the drugs they prescribe.

I know of no evidence that any oral corticosteroid preparation is superior to prednisolone for the treatment of condition such as rheumatoid arthritis and asthma. Prednisolone obtained from the least expensive source costs less than 30p per 100 5-mg tablets (basic N.H.S. price), but most manufacturers charge 42p for the same quantity of their branded product and one firm charges as much as 94p. Equivalent amounts of drugs such as betamethasone (85p), dexamethasone (£2.82), and triamcinolone (£4.70) are even more expensive, and methylprednisolone (£5.14) is the most expensive of all.

The last drug (methylprednisolone) which costs 17 times as much as the least expensive brand of prednisolone, is widely prescribed by general practitioners, although to the best of my knowledge there is no evidence that it is more effective than prednisolone or less liable to cause side effects. Any doctor who prescribes methylprednisolone thus lays himself open to a charge of either ignorance of gullibility, for which the general public has to pay the penalty. Similar examples can no doubt be cited for every other group of drugs, and the total cost to the N.H.S. of such careless prescribing must be enormous.

Members of the medical profession in Britain enjoy the precious right to prescribe any drug, whatever its cost, which they consider to be best for their patients, but they also have an obligation to ensure that this right is not abused.

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Hazardous Wastes

SIR,—All would agree with the importance of the subject discussed in your leading article (30 December, p. 746), but it might be suggested that before preaching to others doctors should first put their own house in order, and this particularly applies to my own specialty, histopathology. Many patho-

logists favour the use of fixatives containing mercuric chloride. These have several advantages, but the waste is usually disposed of down the sink. The biological consequences of mercury pollution are well recognized, and as industry curtails its contribution the role of pathology laboratories will become more significant, for a busy department may discharge up to 50 kg of mercury a year. In a recent article¹ several safe alternatives are outlined, such as the use of zinc chloride or formalin instead of mercuric chloride and the recovery of the mercury as is widely practised by radiologists in the case of silver.—I am, etc.,

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¹ Porter, D. D., *Archives of Pathology*, 1972, **94**, 279.

SIR,—Your leading article (30 December, p. 746) is timely. The Deposit of Poisonous Wastes Act 1972 was drafted in haste and with inadequate consultation. This explains, perhaps, the failure of the Act to recognize and utilize the longstanding and useful partnership of the medical officer of health and the public health inspectorate in the boroughs of Greater London, and instead vests all the enforcement powers with the Greater London Council. The G.L.C. is of course a body with tremendous expertise and we are glad to be able to utilize it. However, since it is the local health authority which is responsible for the public health and is also likely to receive the first intimation of illegal dumping, we feel that amending legislation should be enacted to give concurrent powers to the London boroughs. It is also important clearly to establish the role of the medical officer of health's successor in "community medicine" in these matters.—We are, etc.,

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Rh "Null" is Not Always Null

SIR,—Recently members of two unrelated families with Rh_{null} disease living in West Virginia were brought to our attention. The first family was referred to us for investigation when an Army private, presenting with a marked haemolytic anaemia and splenomegaly, was found to be Rh_{null}. Of seven siblings tested, one sister, aged 25 years, was found also to be Rh_{null}. Further investigations showed evidence of a compensated haemolytic anaemia with a reticulocytosis, mild spherocytosis, and abnormal autohaemolysis and osmotic fragility of the red cells. The cells were M+N+, S=s+, LW-, U weakly positive, and G negative. The parents, first cousins, were of the genotype R1R1 and R1r and were LW+, U+. There was no attenuation of the Rh antigens of the parents or of the six normal siblings. These findings are similar to those in other patients with the rare regulator type of Rh_{null}.^{1,2}

Drs. R. Sanger and P. Tippett kindly examined a sample of blood from the sister; they confirmed the typing results and agreed with us that, although the cells typed as

Rh_{null}, there was evidence of traces of D antigen on the subject's cells. Absorption tests with six different anti-D sera and the subject's red cells showed that they were capable of absorbing anti-D activity. Heat eluates from these Rh_{null} cells reacted with D-positive cells in the antiglobulin test using broad-spectrum Coombs reagent and specific anti-IgG, but not with specific anti-IgA or anti-IgM sera. A small reduction in the anti-D titre was observed in all the absorbed sera. A reduction in the titre of the specific anti-IgG serum was also found. Absorption studies with one example of anti-C and anti-c serum did not result in the removal of antibody activity.

The second person studied was a sister, aged 46, of the patient reported by Dr. P. Sturgeon.³ Typing showed her to be Rh_{null}, LW-, G-, and U+. Unlike the first patient's sister, absorption and elution studies of her red cells with anti-D-containing sera did not show that the antibody had attached to her cells.

This report supports the concept that Rh_{null} disease is not as rare as previously thought and that in some, but not all, patients the presence of the D antigen may be demonstrated by conventional means.—We are, etc.,

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¹ Levine, P., Celano, M. J., Falkowski, F., Chambers, J. W., Hunter, O. B., and English, C. T., *Transfusion*, 1965, **5**, 492.

² Schmidt, P. J., Lostumbo, M. M., English, C. T., and Hunter, O. B., *Transfusion*, 1967, **7**, 33.

³ Sturgeon, P., *Blood*, 1970, **36**, 310.

Effects of Tranquillizers and Hypnotics on Driving

SIR,—We have been studying the persistent effects of hypnotics and tranquillizers, and are therefore interested in the investigation of Dr. T. A. Betts and others (9 December, p. 580) into the effects of different tranquillizing drugs on normal subjects' low-speed driving performance. They have shown that the latter can be impaired by five doses of drug taken over the preceding 36 hours and suggest that this may be important in relation to drug administration in patients. However, they have not investigated the sort of patient for whom these drugs might be prescribed.

Our group at the London Hospital studied normal subjects' cognitive and motor performance, using digit symbol substitution and card sorting, and found that this may be impaired by a single dose of amylobarbitone or nitrazepam given the previous night.¹ Because we doubted whether this observation was relevant to the usual therapeutic situation, we undertook a similar investigation on anxious patients, who received drug or placebo over seven days. These patients were tested 18 hours after the last dose and showed no impairment on the tasks. Electroencephalograms taken on the same occasion showed higher scores for drowsiness after drug than after placebo, but the scores were much lower than those previously obtained in normal subjects. (The results of this investigation are to be published in detail later.)

These results cannot be extrapolated direct to normal driving performance, but

they indicate that tranquillizers and hypnotics may effect the performance of normal subjects and anxious patients in different ways. Dr. Betts and his colleagues recommend that patients who are to take tranquillizers should be warned about possible adverse effects on their driving. However, we feel that neither their investigations nor ours are directly applicable to what in practice is the effect on driving of taking or indeed withdrawing tranquillizers in patients. Our researches strongly suggest that further studies with patients will be required before this question can be answered.—We are, etc.,

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¹ Malpas, A., Rowan, A. J., Joyce, O. R. B., and Scott, D. F., *British Medical Journal*, 1970, **2**, 762.

Two Radiologists—Holland and Britain

SIR,—I opened my *B.M.J.* of 27 January and turned with eager anticipation to page 225 to read of "Two Radiologists—Holland and Britain," but was disappointed when doubts arose as to whether the comparison was between like and like. Many radiologists in Britain would not recognize the life-style and work-style of Dr. Rushton-Wilson.

"Along with his three colleagues he does at least half a day's service work a week—routine barium meals, angiograms, and so on—and he is on duty one week in four. Every day he attends clinical sessions, with the physicians, surgeons, radiotherapists, or specialist groups such as the haematological unit, and usually he goes to the necropsy demonstrations as well."

One session of service work each week is a poor return to give to the populace which pays him his salary. Clinical sessions and visits to the necropsy room are valuable contributions to one's vicarious experience, in which one can discuss other people's work, but are no substitute for personal experience, which at one session a week will be achieved but slowly.

Many of us are doing not less than one half-day of service work each day, five days a week—that, is five half-days each week. A round half-dozen of us within 30 miles of this city are doing just this service load each, each week, and possibly more. By Dr. Rushton-Wilson's standards we should be replaced by 30 whole-time consultants, each doing one half-day a week of service load. As some 90% of the hospital service load of the country is being done in non-teaching hospitals, where radiologists generally work under conditions similar to my own, the consequence would be to wreck the Health Service and introduce a demand for radiologists which would scarcely be met even if all our medical students destined for the hospital services were diverted into radiology.

Were we to reduce our service load to one-fifth of what it is now we would certainly need a day of research each week, if only as "occupational therapy"¹—to keep our brains warm and check that they are still there.

Let us see more "European Counterparts," but may we not see similar articles on con-