CORRESPONDENCE

Correspondents are asked to be brief

Tunnel Tactics

SIR,—Your leading article, "Tunnel Tactics," (15 June, p. 573) on median neuritis and the carpal tunnel syndrome was both informative and pragmatic. Our frequent encounter with this condition in the Workers' Compensation Board of British Columbia confirms your observation of symptoms proximal to the wrist. Acroparaesthesiae and night discomfort are also frequent symptoms.

While you mention nerve conduction studies as an established procedure you relegate this to a role primarily valuable only in research or when disability is great, preferring injections of hydrocortisone or a therapeutic test of immobilization. You conclude that most carpal tunnel syndromes appear to be benign and often self-limiting, yet, on the other hand, you stress the importance of early surgical decompression ("simple and effective") to avoid permanent nerve damage.

It is with respect to this most important aspect that nerve conduction and electromyographic studies must be considered in the early stage. Mild compression or neurapraxia of a peripheral nerve is a reversible condition if not prolonged. The many surgically treated cases with unfortunate persisting numbness and muscle weakness are the sequelae of unnecessary delay in obtaining decompression. Once Wallerian degeneration occurs peripherally with degeneration of the axons and their myelin sheaths recovery will never be complete.

Objective parameters have been established in nerve conduction studies and give indications for surgery (or otherwise). Other conditions such as a more generalized neuropathy or the occasional pronator syndrome in which the median nerve is compromised more proximally can be excluded. Sometimes a vague discomfort in the wrist can be traced to the ulnar nerve. An E.M.G. can also assist in the diagnosis of cervical root involvement. Since the widely accepted technique for median nerve conduction examination is relatively simple and brief and since it is of prime importance to relieve neurapraxia early it is hoped that you will draw attention to and encourage this procedure rather than lure your readers into a false complacency, believing that mere "reassurance and simple conservative measures" are all that are required, only later to find the neurological deficits irreversible.—I am, etc.,

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SIR,—The words "spontaneous mechanical pressure" in the first sentence of your leading article, "Tunnel Tactics" (15 June, p. 573), reveal a deplorable lack of insight. There is no such thing as *spontaneous* compression of the median nerve at the wrist. In that type of case, so common in middle-aged women, a space-occupying lesion can always be found and is responsible for intermittent pressure on the nerve and its vestigial artery against the transverse ligament. It consists of moderately hypertrophied tenosynovial membrane, the presence of which can be fully appreciated only by examination of the whole flexor compartment in a bloodless field.

I reported this finding in 18 consecutive cases of "spontaneous" compression at the great orthopaedic congress held in London away back in 1952, but it attracted little attention. In the report of the proceedings[1] the term "synovitis with effusion" was unfortunate, because the histological appearances of the thickened sheaths are normal and there is never an appreciable effusion (compare incipient rheumatoid or tuberculous tenosynovitis). The only possible cause of the hypertrophy of the tendon sheaths is an increase in frictional resistance caused by a degree of stenosis of the tunnel so minor that it cannot be detected by inspection or measurement. The synovial lesion gives rise to physical signs such as general weakness of grip, limitation of full flexion and extension of the fingers and wrist, slight swelling of the digits, and occasional vague fullness above the wrist. The thickened paratenon is highly vascular, and a further increase in its volume from the peripheral vasodilatation that accompanies deep sleep accounts for nocturnal pain and paraesthesia. Complete division of the transverse ligament enlarges the tunnel and allows the hypertrophy to resolve, so giving lasting relief. Partial thenar atrophy from localized interstitial fibrosis of the median nerve is a separate phenomenon caused by the change in position of the paratenon with every extension movement of the fingers and wrist.

In 1959 I wrote an annotation[2] covering these and several other points and, apart from a few mistakes in spelling, I would stand by every word of it. It seems to have escaped the attention of most writers on the subject. After gaining a much wider experience I would now go much further and say that in every case of carpal tunnel syndrome, no matter what the aetiology, the median nerve is the innocent victim of pressure by synovial tissue in some kind of disorder. Diagnostic tests and therapeutic measures should therefore be directed more at the sheaths of the flexor tendons than at the median nerve as you counsel in your article.—I am, etc.,

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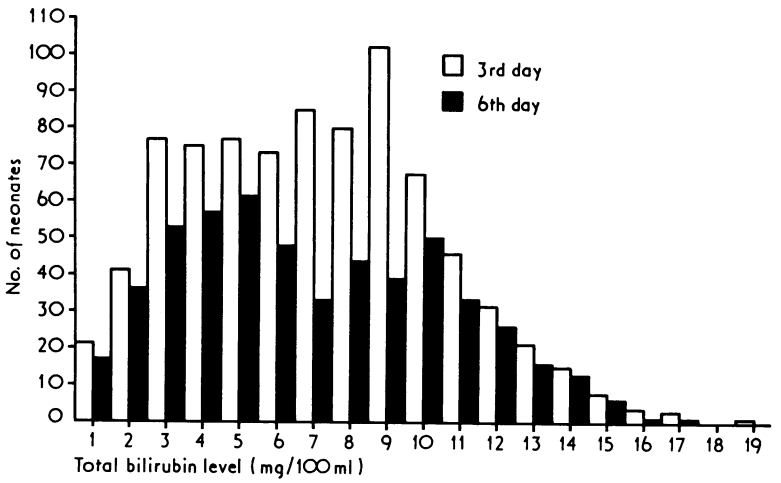
1 Nissen, K. I., *Journal of Bone and Joint Surgery*, 1952, **34**, 514.
2 Nissen, K. I., *The Postgraduate Medical Journal*, 1959, **35**, 379.

Oxytocin in Labour and Neonatal Jaundice

SIR,—In their excellent paper (27 July, p. 228) Dr. S. R. Gould and his colleagues failed to show an association between the use



of oxytocin in labour and an increase in the *mean* bilirubin concentration in the neonates. Our results[1] obtained from examination of *mean* bilirubin concentrations in a prospective study of 1,353 labours and neonates agree with the relationship they have shown, but we differ from the conclusion drawn. Possible explanations for this come to mind and deserve comment:

(1) In the study by Dr. Gould and his colleagues serum bilirubin concentrations were estimated on blood taken from the cord at birth and from the baby on the sixth day of life. We suggest that the timing of these blood samples was not ideal for the purpose of this study. It is not to be expected that the bilirubin concentration would be raised in cord blood specimens, whereas by the sixth day of life it can be shown that it has passed its peak and is decreasing. The figure shows the distribution of mean total bilirubin concentrations on the third and sixth days of life in our series of 815 neonates resulting from labours induced or accelerated with oxytocin. It will be seen that on the third day the peak incidence of neonates is associated with a bilirubin concentration of 8 mg/100 ml whereas by the sixth day the peak incidence of neonates has a bilirubin concentration of only 5 mg/100 ml. This suggests that in most neonates the bilirubin concentrations are falling by the sixth day. A similar trend is reported by McConnell *et al.*[2] who found that on the fifth day serum bilirubin concentrations were 32% lower than on the third day in bottle-fed infants.

(2) Several workers[1-4] other than Gould and his colleagues have tried to show an association between a raised *mean* neonatal bilirubin concentration and the maternal administration of oxytocin. With the exception of Davies *et al.*[3] none have yet found a significant relationship between the use of maternal oxytocin in labour and the *mean* bilirubin concentration of the neonates. Even Davies *et al.* claimed only that the association was of minimal significance ($P < 0.05$, method of statistical analysis not stated). However, after labours induced by amniotomy and oxytocin we have shown that the mean total dose of oxytocin received by the mothers whose babies were normal (bilirubin concentration < 12 mg/100 ml) was only 9·91 U, whereas mothers of neonates with hyperbilirubinaemia (bilirubin concentration > 12 mg/100 ml) received a mean total dose of 18·77 U of oxytocin. This difference between the total doses of oxytocin administered is highly significant (*t* test: $P < 0.001$). Furthermore, we found that the proportion of neonates who developed hyperbilirubinaemia increased in direct relation to the increasing total dose of oxytocin administered. This association was also highly significant (Wilcoxon's test of differences between mean ranks: $P < 0.001$).

Our results support the association between the use of oxytocin in labour and the subsequent development of mild neonatal jaundice, though other factors are involved.—I am, etc.,

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1 Beazley, J. M., and Alderman, B., *Journal of Obstetrics and Gynaecology of the British Commonwealth*. In press.
2 McConnell, J. B., Glasgow, J. F. T., and McNair, R., *British Medical Journal*, 1973, 3, 605.
3 Davies, D. P., *et al.*, *British Medical Journal*, 1973, 3, 476.
4 Ghosh, A., and Hudson, F. P., *Lancet*, 1972, 2, 823.

Minocycline and β-haemolytic Streptococci

SIR,—Resistance of β-haemolytic streptococci to the tetracyclines can be a clinical problem.[1-3] Therefore it is of interest that a new analogue of tetracycline, minocycline (Minocin, Lederle), is now available with reports of its activity against tetracycline-resistant streptococci.[4,5] To compare in vitro this activity with that of tetracycline 63 β-haemolytic streptococci were obtained from a variety of clinical sources. Dilutions of each antibiotic were made and added to 10% horse-blood agar (Oxoid, Base No. 2) to yield culture plates with a range of final antibiotic concentrations from 0·3 to 20 μg/ml. The test organisms were grown overnight at 37°C in Todd-Hewitt broth (Oxoid) and were diluted 1/50 before each was "spotted" on to an antibiotic-free control plate and on to each antibiotic-concentration plate. After overnight incubation at 37°C the inoculated plates were examined for the presence or absence of growth.

With ordinary dosage of tetracycline (250 mg six-hourly) maximal blood levels of 2-4 μg/ml may be expected,[6] whereas for minocycline at recommended dosage (200 mg "loading" and 100 mg 12-hourly thereafter) the mean serum concentration over a six days' course is between 1·53 and 2·30 μg/ml.[7] Therefore to assess potential resistance among the test organisms "threshold" minimum inhibitory concentration values were adopted of 2·5 μg/ml tetracycline and 1·25 μg/ml minocycline, with the results shown in the table.

No. of Strains Tested		No. (%) of Strains inhibited by: Tetracycline ≤2·5 μg/ml		Minocycline ≤1·25 μg/ml	
Group A	33	16	(48)	22	(66)
B	15	2	(13)	2	(13)
C	9	5	(55)	5	(55)
G	6	3	(50)	3	(50)

In vitro minocycline seems to have some advantages over tetracycline against group A β-haemolytic streptococci but not against strains of groups B. C, or G. However, it is debatable whether or not this represents any real advantage in vivo.—I am, etc.,

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1 Garrod, L. P., Lambert, H. P., and O'Grady, F., *Antibiotic and Chemotherapy*, 4th edn., p. 151. Edinburgh, Churchill Livingstone, 1973.
2 Robertson, M. H., *British Medical Journal*, 1973, 4, 84.
3 Fallon, R. J., *British Medical Journal*, 1973, 4, 300.
4 Steigbigel, N. H., Reed, C. W., and Finland, M., *American Journal of Medical Science*, 1968, 255, 179.
5 Garrod, L. P., Lambert, H. P., O'Grady, F., *Antibiotic and Chemotherapy*, 4th edn., p. 161. Edinburgh, Churchill Livingstone, 1973.
6 Garrod, L. P., Lambert, H. P., O'Grady, F., *Antibiotic and Chemotherapy*, 4th edn., p. 154. Edinburgh, Churchill Livingstone, 1973.
7 Frisk, A. R., Tunevall, G., *Antimicrobial Agents and Chemotherapy*, 1969, 8, 335.

Sun, Wind, Sand and the Skin of Children

SIR,—Your leading article (13 July, p. 72) and the letter from Dr. E. J. Moynahan (10 August, p. 410) prompt report of the observation that infants suffering from atopic lesions of the skin or napkin dermatitis need not suffer exacerbation of their symptoms when involuntarily exposed to the desiccating action of sun, wind, and sand when on holiday.

The mothers of 11 patients (age range 10 months to 6 years) with symptoms currently in remission expressed anxiety, from previous experience, that exacerbation would occur during forthcoming seaside holidays. This group and five other children (age range 4 months to 2 years) were questioned about the provision of adequate sun protection (clothing, hat, "sun cream") and relevant advice was given—this generally reinforcing previous sensible practice. In addition, application of Parfenac cream (bufexamac 5%) morning and evening was prescribed, all 16 children having previously achieved the current remission of their symptoms with this preparation (maximum time in remission seven months, minimum one month).

When seen subsequently 12 children (75%) had remained symptom-free, 3 (18·75%) had had a flare-up equivalent to the original presenting condition, and 1 (6·25%) reported a small eczematous area in a new site (the knees) which had lasted for the duration of the holiday only. The three with a severe flare-up returned about 75% of their prescribed ointment and all were from the group who had not previously experienced a flare-up of symptoms under holiday conditions. All the remainder returned quantities suggesting that the preparation had been used as prescribed.

In addition to the advantage of being a non-steroid, this anti-inflammatory agent has a bland, moisturing base that is not painful on application and consequently is generally well tolerated by children. While sensible clothing and gradual exposure remain the best method of avoiding the adverse effects