cord blood and from venous samples on days 4 and 7 in infants born after surgical induction of labour and intravenous oxytocin infusion. A control group of neonates was also studied. Haemolytic disease was excluded in all infants by appropriate investigation. The mean birth weight, gestational age, incidence of operative delivery, and breast-feeding on discharge was similar in both groups. The results are summarized in the table.

	Mean Biliburin in mg/100 ml ± S.E.M.		
	Cord	Day 4	Day 7
Oxytocin Group N = 13	2·308 ± 0·233	5·285 ± 0·94	3·69 ± 0·91
Control Group N = 14	2·507 ± 0·277	5·35 ± 0·8	3·50 ± 0·560

The mean total bilirubin in cord blood was lower in neonates delivered after oxytocin-induced labours, but this difference was not statistically significant. No difference between mean bilirubin levels on days 4 and 7 was apparent. Furthermore, we could find no correlation between the total dose of oxytocin infused and neonatal bilirubin levels.

Though the results of our study are in conflict with the authors' findings, we would endorse their view that any possible association between maternal oxytocin infusion and neonatal jaundice should be viewed in the proper perspective, and a small rise in bilirubin level is unlikely to be of importance in a healthy term baby of normal birth weight.—We are, etc.,

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Congenital Syphilitic Nephropathy in an Adopted Infant

SIR,—In view of the rising incidence of syphilis, the current interest in congenital syphilitic nephropathy, and recent moves to change screening procedures for adopted children, we would like to report an instructive case.

A 16-week-old illegitimate boy awaiting adoption was admitted to this hospital for investigation of haematuria and proteinurea. His natural mother was apparently well, the pregnancy and delivery were normal, and a maternal Wassermann reaction at eight months' gestation was reportedly negative. Neonatal examination had been normal, but subsequently there were feeding difficulties. At the age of 13 weeks he developed proteinuria and haematuria following an upper respiratory infection.

Examination revealed hepatosplenomegaly and moderately enlarged kidneys but no oedema, lymphadenopathy, or snuffles. Hypoalbuminaemia, heavy but highly selective proteinuria, raised plasma IgM levels, and femoral subperiosteal new bone formation were present. Serum cardiolipin W.R., Reiter complement fixation test, *Treponema pallidum* immobilization test, serum V.D.R.L. slide test, and IgG and IgM fluorescent treponemal antibody tests were all positive.

A diagnosis of congenital syphilitic nephritis was made and the patient was successfully treated with a three-week course of procaine penicillin. The natural mother was traced and found to have no clinical evidence of syphilis but weakly positive serology. She was given a course of penicillin treatment. The prospective adoptive parents' serology was negative, and in spite of this illness they have subsequently adopted the child.

In this case maternal exposure to infection ceased after the child was conceived and no antibiotic was administered during pregnancy. At eight months' gestation maternal serology must have been positive and the negative W.R. was either an error or reflected an insufficiently sensitive test. For routine screening the V.D.R.L. slide test would seem preferable, and in those at particular risk repeating it in the last trimester, with a subsequent test for cord-blood IgM fluorescent treponemal antibody, would seem to be indicated.

In the light of this case we wish to emphasize the need to consider syphilis in infantile nephritis or nephrosis, even with a negative prenatal history, and feel that the medicosocial implications of failure to achieve antenatal diagnosis in a subsequently adopted child justifies careful reappraisal of syphilitic screening procedures in pregnancy. —We are, etc.,

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Infantile Herpes Zoster

SIR,—In reply to Dr. G. D. W. McKendrick and S. C. Raychoudhury (11 August, p. 352), we regret omitting any reference to their paper.[1] This was not intended to mislead, but in the space available in a short annotation it was not possible to refer to all the relevant literature.

The special importance of our case of herpes zoster in a young child was not only the exposure to varicella in utero but also the virological confirmation of the diagnosis, which is absent from most similar published reports. In stating the two hypotheses for the pathogenesis of herpes zoster we merely wished to indicate that both are considered possible by some.[2,3]—We are, etc.,

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1 McKendrick, G. D. W., and Raychoudhury, S. C., *Scandinavian Journal of Infectious Diseases*, 1972, **4**, 23.
2 Berlin, B. S., and Campbell, T., *Journal of the American Medical Association*, 1970, **211**, 1831.
3 Horsfall, F. L., and Tamm, I., *Viral and Rickettsial Infections of Man*, 4th ed. p. 918. London, Pitman, 1965.

Muscle Cramps during Maintenance Haemodialysis

SIR,—We welcome the paper by Dr. G. R. D. Catto and others (18 August, p. 389) on the treatment of muscle cramps in haemodialysed patients with Slow Sodium tablets. The neglected problem of dialysis cramps is now beginning to receive the attention it deserves.

With reference to their data, however, we should like to point out that a reduction in the mean frequency of cramps from 54·7 to 40·7%, though statistically significant, is perhaps less significant to the patients, as 40% is still considerably troublesome. It is also perhaps misleading to express the difference of 14% as a percentage of the 54·7% and refer to this as a "reduction of 26%." Most people would be inclined to call a change from 54·7 to 40·7% a reduction of 14%. The same criticism applies to their description of the change in severity from 5·4 to 3·8 arbitrary units as a "reduction of 30%." Moreover, judgement of the severity of the cramps, when related to a 10-point scale, must have been difficult for the patients. Within so wide a scale a change from 5·4 to 3·8 units must have been small in terms of the relief afforded.

Notwithstanding the marginal nature of the improvement, Dr. Catto and his colleagues claim that the administration of up to 14 tablets of Slow Sodium on each dialysis day is a better way of controlling cramps than "an increased sodium concentration in the dialysate for all patients." We, on the other hand, recommend[1] the use of isonatric dialysis (dialysate sodium concentration of 145 mmol/l.), combined with appropriate ultrafiltration. Isonatric dialysis with controlled ultrafiltration brought about a reduction in the incidence of cramps from 55% to 13% in our patients,[2] and the current incidence is $< 15\%$ (extreme range per patient nil to 35%). Thus, the frequency of cramps is noticeably greater using the conventional dialysis, even with Slow Sodium, than when using controlled isonatric ultrafiltration.

Isonatric dialysis is compatible with complete and satisfactory control of blood pressure.[3] In fact, hypertension has not been a problem, either long-term or short-term, despite what might seem to some as more adverse clinical conditions in Dundee —namely, only 16 to 18 hours of total dialysis time per patient per week, divided into two sessions. The inter-dialysis weight gain of 3 to 5 kg is partially a consequence of the restricted dialysis time available. Contrary to the belief of Dr. Catto and his colleagues our patients do not complain of thirst; they revert to near normal, old-established, patterns of fluid intake. Medical injunctions to alter eating and drinking habits may work in the short term but are rarely effective for long.

Dr. Catto and his colleagues seem to value the ability to "tailor" the number of tablets to each individual patient's needs, but the practicalities of tailoring the number to individual needs (presumably retrospectively) could be difficult in a busy dialysis unit. The prospect of swallowing up to 14 tablets each dialysis seems daunting to us. Multiple pill-swallowing is also surely more prone to error than the use of a standardized dialysate composition which, with a "physiological" concentration of sodium, has proved in our experience to be suitable for all patients. Certainly it seems contradictory to be deliberately removing sodium from the patient with one hand (dialysis with low sodium concentrations) while feeding him sodium with the other. It would have been interesting to know the plasma sodium concentrations of their patients and how often it was monitored. Even 24 hours after dialysis using a dialysate sodium concentration of 135 mmol/l., plasma sodium concentrations can be as low as 131 to 133 mmol/l.[4]

We continue to believe that, in the course of haemodialysis treatment, the removal of accumulated dietary sodium is most easily accomplished by the net transfer of the "surplus" extracellular fluid during controlled isonatric ultrafiltration.[1] This single process can be closely monitored during dialysis through the parallel change in body weight. A concomitant of this straightforward technique is a marked reduction in the prevalence of dialysis cramps. The conventional approach with hyponatric dialysate (sodium approximately 130 mmol/l.) involves the unmonitored dialytic transfer of