Loss and Gain in Burns

SIR,—Your leader "Loss and Gain in Burns" (31 October, p. 1090) draws attention once again to insensible water loss through a burn at the eschar stage. Although some thought and investigation has gone into this problem, notably by Carl Moyer¹ in the United States, Burch and Winsor² as early as 1944 had demonstrated that loss of the stratum corneum resulted in a gross increase in the diffusion rate of water through the denuded surface. The eschar, although acting as the protective umbrella so far as organisms are concerned, is positively useless as a protection against water loss.

We are currently engaged on a series of investigations, the results of which we intend to publish shortly, in which by means of an instrument specially devised for the purpose we will be able to measure directly the minute losses through small areas of eschar at different times during the healing phase. It will then be possible to calculate the precise amount of water loss for the particular burn in question at any given time.

We hope by our results, therefore, to be able to be in a position where active resuscitative measures no longer cease with eschar formation but instead the fluid intake is accurately augmented during the first four to five days of the eschar phase of healing in conformity with the insensible loss.—I am, etc.,

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REFERENCES

- ¹ Fallon, R. H., and Moyer, C. A., *Ann. Surg.*, 1963, 158, 915.
- ² Burch, G. E., and Winsor, T., *Arch. intern. Med.*, 1944, 74, 437.

Rubella-disabled Children

SIR,—Much correspondence has occupied your columns concerning the rubella syndrome and how to prevent it. I would like to direct your readers' attention to the problem from a different and most grave aspect.

The two major epidemics of a few years ago resulted in a large number of extremely severely handicapped children who are deaf and blind, and often with additional disabilities. Numerically this group of disabled children represents a far greater disaster than the thalidomide group, yet there is no public outcry, there are no collections of large sums of money on a national scale, no donations for research, no resources available for establishing special centres.

There is an association of parents heroically struggling with limited resources against terrific odds, but the resources are too limited and the odds are too great.

We found, after establishing a small unit at the Centre for Spastic Children, that these children need daily care in a special nursery environment where continuous stimulation can be given, and where all available sensory faculties can be trained by various means, including physiotherapy, auditory training, and training of their partial sight, where it does exist. If this sort of concentrated training were given many of these children would be able to make the grade for education in a school for deaf and blind.

But in most parts of the country these children receive no training at all; in one place they are seen once a month by a peripatetic teacher of the deaf. Several small units, five or six children to a unit, where these children could be treated, observed, and trained, are necessary all over the country. Here study and experience could result in improvement of methods used for training.

But where can one find resources for such a unit? When we see these children at various audiology centres, and the bitter struggle of their parents to do something for them, we feel that Ministries, local authorities, various national organizations for children with different disabilities, and large moneyed trusts are blind and indifferent.—I am, etc.,

Heston, Middlesex.

L. FISCH.

Mongolian Spots

SIR,—May I add a comment to the recent sporadic correspondence on so-called Mongolian spots? Briefly, I have seen these in neonates of darker-complexioned communities in Asia, Africa, Polynesia, West Indies, etc., and also but to a lesser extent in darker Caucasians (e.g., Sicilians, Southern Spaniards). They appear to be localized areas of hypermelaninization which stand out because neonates of all groups are lighter skinned than in later childhood and adult life.

The main practical aspect is to realize their normality and that they "disappear" in the early years, probably by being overlaid as the rest of the skin achieves an increased melanin content.—I am, etc.,

Kampala, Uganda.

D. B. JELLIFFE.

Unusual Twin Pregnancy

SIR,—I wish to present a case of twin pregnancy associated with a rare complication at delivery.

The patient aged 23, para 2—the first pregnancy terminating in a stillbirth at 34 weeks' gestation (the cause being unknown) and the second pregnancy normal—was diagnosed to have a twin pregnancy at the 35th week of gestation, and this was confirmed by x-ray. Although the patient refused to be admitted to hospital for a period of rest, the pregnancy proceeded normally and she was admitted to Cresswell Maternity Hospital, Dumfries, at the 37th week of gestation, in established labour.

After a four-hour normal labour the head of the leading twin was seen to be distending the perineum, and, the head having been delivered, the umbilical cord was found to be twice round the neck. The cord was clamped and cut and the delivery completed. It was then seen that the delivered infant was still attached to the placenta by its cord, and in fact the cord that had been cut was that of the second twin, which was still *in utero*. Before steps could be taken to expedite delivery of the second twin by operative intervention, spontaneous vertex delivery, aided by fundal pressure, was secured within four minutes of the cords being cut. The third stage was completed in 11 minutes. The condition of both twins, males, weighing 4 lb. 12 oz. (2.2 kg.) and 4 lb. 15 oz. (2.3 kg.) was satisfactory.

On close examination of the placenta and membranes it was found that there was only one amniotic sac, and both umbilical cords, measuring 18 in. (0.46 m.) and 23 in. (0.58 m.), were inserted in close proximity to the placenta, which weighed 1 lb. 11 oz. (0.9 kg.).

An extensive scrutiny of the literature showed no reference to a similar case of cord prolapse in a twin delivery and with spontaneous delivery and survival of both infants.—I am, etc.,

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Diabetes and Neuropathy

SIR,—I was most interested in your leading article on "Diabetes and Neuropathy" (10 October, p. 891). You observe that the question must arise whether a polyneuropathy developing in an unstable diabetic on treatment must always be attributed to the diabetes or whether repeated swings to hypoglycaemia might not be a contributory factor. A similar view was expressed by Mulder *et al.*¹ The following two case reports add weight to this suggestion.

Case 1.—A man aged 58 was found to have diabetes mellitus in November 1958, when he was investigated on account of persistent balanitis. He was stabilized on insulin in a dose of 46 units each morning of a mixture of equal parts of soluble insulin and zinc-protamine insulin. After one month on this regimen he commenced to have mild hypoglycaemic reactions before meals and in the mornings. His dose of insulin was reduced in stages to 32 units per day and in January 1959 he was free from hypoglycaemic symptoms. In February 1959 he developed a burning and prickling sensation, described as being most unpleasant, involving the anterior and medial aspect of both thighs and the upper part of both legs. This progressed to involve the groin and buttocks on both sides. There was no objective sensory loss and no motor involvement. By July 1959 there was no improvement despite successive courses of treatment with aneurin, cyanocobalamin, and high-dosage intravenous mixed vitamins. He was having mild hypoglycaemic reactions whenever he was late for a meal and he felt "a bit stupid" in the mornings. All urine samples were free from sugar, so it was decided to reduce his dose of insulin further by stages. By September 1959 it had been possible to reduce his dose of insulin to 8 units per day without recurrence of glycosuria. His symptoms decreased steadily with the reduction in dose of insulin and by the time a dose of 8 units per day had been reached he was symptom-free. Treatment was now changed to tolbutamide 500 mg. per day, and there has been no return of neuropathy in four years.

Case 2.—A man aged 58 presented in March 1957 with a short history of lethargy, loss of weight and intense thirst. Diabetes mellitus was diagnosed and he was stabilized on lente insulin 32 units each morning. After one month on this regimen he commenced to have mild hypoglycaemic symptoms in the morning, and in May 1957 he began to complain of very unpleasant burning pain in both feet "as though the flesh was bare," and of tingling and numbness extending half way up both legs. These symptoms were worse at night and when the feet were warm. There was no objective sensory loss and no motor involvement. There was no glycosuria. His dose of insulin was reduced gradually without recurrence of glycosuria and by July 1957 it was down to 4 units per day, but there was no improvement in his neuropathy. His insulin requirement over the next two months varied from 4 to 8 units per day, but by September 1957 it was possible to control his diabetes by diet alone. From then onwards his neuropathy improved steadily and he was symptom-free by April 1958. There was no recurrence of neuropathy up to his death from coronary thrombosis in August 1958.