form. Juvenile diabetes mellitus with optic atrophy but without diabetes insipidus has been repeatedly reported in the past as a familial recessive syndrome.²⁻⁵ In most of these individuals profound perceptive hearing loss and in some an automatic bladder developed subsequently.

Furthermore, the familial association of Friedreich's ataxia with diabetes mellitus of early onset has been recorded far too frequently to be fortuitous. Recent descriptions are to be found in the papers of N. S. Schlezinger and K. Goldstein⁶ and D. W. Ashby and P. S. Tweedy.⁷ In the former, optic atrophy in addition was recorded in each of two affected sisters; in the latter, while optic atrophy was not a feature of their cases, the authors mentioned that Dr. H. A. Dewar had told them of two sisters with juvenile diabetes mellitus, ataxia, and optic atrophy. In general, optic atrophy is a well-known complication of Friedreich's ataxia.

C. H. Allström *et al.*⁸ reported a recessive syndrome consisting of retinal degeneration (including optic atrophy), obesity, diabetes mellitus of early onset, and perceptive deafness, and this was mentioned in your annotation in 1959.⁹ While these authors claimed that this syndrome was quite distinct from that attributed to Laurence, Moon, Bardet, and Biedl (L.M.B.B. syndrome), S. Solis-Cohen and E. Weiss¹⁰ obtained abnormal glucose-tolerance tests in three of four young sibs with a typical form of this latter condition. Finally M. Fraccaro and F. Gastaldi¹¹ reported an isolated case with all the cardinal features of the L.M.B.B. syndrome, including retinal degeneration with optic atrophy and, in addition, juvenile diabetes mellitus, diabetes insipidus, and ataxia.

While the various associations of juvenile diabetes mellitus seem to occur in many different combinations in these recessive syndromes this may sometimes reflect the clinical interests of the observer rather than true genetical diversity. It will be noted that eye lesions are common to all but cannot be regarded as complications of diabetes mellitus. These conditions are largely unmentioned in textbooks of ophthalmology, and T. Leber¹² states that he had not seen optic atrophy in a wide experience of patients with diabetes mellitus. It must be remembered that at that time the natural history, and therefore the ocular associations, of diabetes mellitus were very different. On the other hand, A. von Graefe¹³ does record that of seven patients sent to him for ophthalmological complications of diabetes mellitus one, a young individual, did have optic atrophy. This report is in the same series of miscellaneous observations as the first description of familial retinitis pigmentosa with congenital deafness, written at a time when von Graefe was 30 years old.

Clifford Allbutt,¹⁴ in discussing a case of diabetes mellitus with optic atrophy in 1871, stated, "Heredity, again, which is known or believed to have an important place in the establishment of glycosuria, points rather to the nervous system, for heredity is so commonly observed in diseases of the nervous system." In view of the developments since that time, only the salient features of which are adumbrated here, this would seem to be an opinion of remarkable perspicacity. Systematic clinical surveys of juvenile diabetics could do much to characterize and delineate these syndromes further and define

their numerical contribution to this heterogeneous entity, which defies any simple unitary genetical explanation.^{15,16} Conversely, systematic studies of carbohydrate metabolism in families with Friedreich's ataxia, L.M.B.B. syndrome, and other related conditions would also be of value. These combined approaches could not fail but throw some light on the problem of diabetes mellitus as a whole.—I am, etc.,

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London W.C.2.

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Uterine Inversion and Cord Traction

SIR,—Uterine inversion was once thought to be a serious risk of pulling on the cord to deliver the placenta. Times have changed and nowadays cord traction is widely used, and presumably is considered safe if practised only during a uterine contraction and at the same time as the fundus is displaced upwards. None the less Dr. T. E. Torbet (25 January, p. 239) describes a case of inversion which occurred despite these precautions and refers us to another reported by Patterson (25 May, p. 1414). In Dr. Patterson's case the precise technique employed during traction is not given, but his description confirms the change which has come over our practice, for he writes: "I applied cord traction in the usual way."

These instances confirm that pulling on the cord whilst the placenta is still in the uterus must involve a risk, admittedly small, of uterine inversion. The accident may depend upon a fundal implantation of the placenta and perhaps even on central insertion of the cord, but who can say if these are present or not whilst the placenta is still inside? I believe that those who practise cord traction whilst the placenta is inside the uterus (and I sometimes do) must accept this risk, and, what is more important, must be prepared to treat the inversion if they produce it.

The uterus which inverts before the eyes of the medical attendant, whether doctor or midwife, must be replaced immediately as Dr. Torbet replaced it. Delay of only a few moments may permit a tight contraction ring to form and shock to develop. Instant replacement in my view takes precedence over anything else. There is no time to scrub up, nor to put on gloves, nor to arrange any kind of anaesthesia; the short delay involved in peeling the placenta off the inverted uterus in Dr. Patterson's case was sufficient to permit a tight contraction ring to form. I have

known several incidences of inverted uteri being replaced at once, the patient being scarcely affected by the event, and I have known others in which there has been delay, shock and collapse have developed, and the patient has been gravely ill.

Cord traction to remove a placenta which we know to be separated and to be lying in the vagina is another matter; it is a much simpler way of removing the placenta than fundal pressure, much less painful for the patient, and the risk of inverting the uterus must be negligible.—I am, etc.,

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Jessop Hospital for Women,
Sheffield 3. C. J. DEWHURST.

Ectopic Pregnancy

SIR,—Your issue of 5 October 1963 has arrived rather belatedly, and I was interested to read the paper on tubal ectopic pregnancy by Dr. C. P. Douglas (5 October, p. 838).

It is rather surprising that in his series he did not observe a physical sign which was so constant in British Guiana (where during my three years as surgeon-specialist we also had an average of two ectopics a week) that in my last year there I gave up doing pelvic examinations and relied entirely on the history and this appearance. The sign is this: a slight, but distinct, abdominal distension below the umbilicus. This sign in leaking or ruptured ectopics was very constant and reliable and I have not yet seen any case not exhibiting it to be subsequently found to be a ruptured ectopic (but note that it is not present before there is a leak of blood into the peritoneal cavity).

It is certainly much safer than puncture of the posterior fornix, and I have never seen it in intestinal obstruction (where the distension is of the whole abdomen, both above and below the umbilicus), perforated appendix, or torsion of ovarian or other pelvic cyst.—I am, etc.,

Sydney, N. N. IOVETZ-TERESHCHENKO.
N.S.W., Australia.

Transverse Myelitis After Vaccination

SIR,—In the smallpox eradication programme mass vaccination is being carried out in this part of the country utilizing the Russian type of dry vaccine for mass vaccination. It is introduced in the forearm by the rotatory lancet method. We wish to report a patient who developed transverse myelitis exactly on the fifteenth day after vaccination.

The patient was a 35-year-old farmer coming from Tindivanam taluk of Madras State in South India. He had been vaccinated about six to seven times in the past with calf lymph vaccine as supplied by the Haffkine Institute, Guindy, Madras. There was a revaccination on 18 August 1963 with Russian dry vaccine. The patient went to work the same day and developed moderate fever that night which remained for the next two days, when he had to remain in bed. Although thereafter he was feeling unwell and feverish he continued to work. On the fifteenth day of vaccination he developed sudden weakness in the two lower limbs and could not walk. He could not pass urine although his bladder was full. When he was admitted to hospital he was found to have flaccid paraplegia with loss of sensation below the eighth dorsal segment accompanied by retention of urine. No neurological abnormality could be detected in the head, neck, and upper limbs. Other systems were completely normal.

On investigation his blood, urine, and stool did not show any abnormality. His venereal disease reference laboratory test was negative in both the blood and cerebrospinal fluid. Cerebrospinal-fluid pressure was normal; it contained 40 mg./100 ml. of proteins and 20 cells/c.mm., almost all lymphocytes. X-ray of spine, chest, etc., were normal. He was diagnosed as a case of transverse myelitis and was treated with prednisolone (60 mg. per day). In the course of one month he recovered almost fully.

There were 13 members in the family, all of whom were vaccinated similarly on the same day with the same vaccine. Most of them had fever but no neurological manifestations. Many persons in the locality were also vaccinated, and although a good many persons had mild to moderate general reactions no one suffered from a similar episode.

Encephalomyelitis occurring after vaccination, and especially after primary vaccination, is well recognized. However, there is no record of transverse myelitis occurring on subsequent vaccination. This was the seventh vaccination of the patient. Although it is quite likely that vaccination had nothing to do with the attack of transverse myelitis, the sequence of events was such that we thought it worth while to give this case publicity through your journal, so that if any reader has come across a similar occurrence he will be able to communicate with us.—We are, etc.,

N. C. SHYAMALAN.

S. S. SINGH.

Medical College,
Pondicherry, India. D. B. BISHT.

Hand, Foot, and Mouth Disease

SIR,—I should like to report an outbreak of "hand, foot, and mouth" disease here. On 2 January I saw a child, aged 3, who had vesicles on the dorsum of both hands, more marked on the fingers than the actual hand, and aphthous ulcers in his mouth, with a surrounding erythema. These were more prevalent over the buccal mucous membranes and less so on the tongue and fauces. Three days later three of his siblings developed an exactly similar rash. None of the children had any constitutional symptoms, and they have all now made an uneventful recovery.

I must confess that I did not know what this rash was when it first appeared. This family lives near Shaftesbury. One of my partners has seen one similar case in Wiltshire.—I am, etc.,

Shaftesbury, Dorset.

JOHN TROWELL.

SIR,—Reading the descriptions of hand, foot, and mouth disease in your correspondence columns recently we were struck with the similarity to a recent case of ours which we had thought to be one of erythema multiforme.

The patient, a man of 29 years, came to see us at the end of December with a reddish maculo-papular eruption on both hands and some superficial ulcers on the buccal mucosa and tongue. Like Dr. K. L. Lipp (7 December, p. 1473), we were struck with the resemblance of the lesions on the hands to early chicken-pox. Two days later a further crop of lesions had appeared on the hands, mainly on the palms, and lesions were now appearing on the feet also. The early lesions on the hands were by now vesicular, and there was bleeding into some of

them. On this day the patient also had a conjunctivitis of his right eye, which resolved without treatment in a few hours. There was no history of recent herpes simplex, or of vaccination. There were no target lesions seen. No virus studies were done, and the oral lesions were treated initially with 1% gentian violet, with little effect.

We have seen no cases among his relatives or contacts, and the patient is now almost completely recovered.

Could it be that some of the cases of hand, foot, and mouth disease described are in fact a mild erythema multiforme, or indeed that hand, foot, and mouth disease is a variety of erythema multiforme? It would be interesting to know if some of the cases where virus studies were not done, or did not show the presence of Coxsackie type A virus, had a history of herpes simplex in the few weeks before the rash appeared.

We would be pleased if anyone could clarify these points for us.—We are, etc.,

Melbourn,
Nr. Royston, Herts.

HUGH R. ERSKINE.

E. F. GRIFFITH.

Parenteral Iron in Pregnancy

SIR,—Anaemia in the last trimester of pregnancy often presents a problem in management. At St. John's Hospital, Chelmsford, every attempt is made to avoid a patient going into labour with a haemoglobin less than 70%.

The usual management is a course of parenteral iron after a full blood examination has confirmed an iron deficiency and a Figlu test has screened for folic-acid deficiency. Until recently a course of parenteral iron has meant that the mother must spend some time in hospital or else attend her G.P. frequently over a period of 1-2 weeks. When mothers have other children at home or live some distance from their G.P. iron injections can be most inconvenient.

To overcome this problem the total dose "imferon" infusion technique has been used for such cases since April 1963. After investigations are completed, arrangements are made to admit the patient to the antenatal ward for one day (9 a.m. to 5 p.m.). The dose of imferon required is calculated from the manufacturer's scale—the average being about 30 ml. (they recommend an extra 10 ml. to be added in pregnancy, but we had overlooked this fact until recently). The dose is divided between two 500-ml. bottles of 5% dextrose. The intravenous drip is erected in the morning, running about 10 drops/min. for the first ½-hour and then increased so that the total infusion is completed in approximately 4-6 hours. The patient goes home the same evening. In the past two months folic acid, 5 mg. t.d.s., has been commenced after the infusion, without waiting for the Figlu result. Initially the total dose was given in 500 ml. of 5% dextrose, and thrombophlebitis was a frequent complication, but by dividing the dose into two 500-ml. bottles this trouble has been overcome. To date there have been two possible reactions with approximately 55 infusions. One patient complained of a burning pain in the arm 15-20 minutes after the drip commenced, and the drip was discontinued with no further ill effects. Another became dyspnoeic and complained of pain in the back of her neck several minutes after the drip commenced. In this case the drip was run-

ning much faster than usual. The tightness in the chest quickly disappeared on stopping the drip and administering subcutaneous adrenaline.

The results of the infusions have been very satisfactory. Of 35 patients so far delivered after treatment, 31 showed a Hb rise prior to delivery. Four showed a further Hb drop in the two weeks after infusion; as they were all within a week of the estimated date of delivery, they were transfused. The average duration of pregnancy at infusion was 36 weeks: the average rise in haemoglobin two weeks after infusion was 10% (12% in the last seven patients who were given folic acid as well). In cases followed over a longer period the rise was progressive, as two examples in the Table show.

Case 1 (infusion at 34 weeks)		Case 2 (infusion at 31 weeks)	
Duration of Pregnancy (weeks)	Hb (%)	Duration of Pregnancy (weeks)	Hb (%)
34 (before infusion)	57	31 (before infusion)	52
36	61	33	60
38	69	35	66
39	76	38	67
		40	76

The aim was to raise the Hb prior to delivery, and patients were not followed closely after the baby was born.

The response to this therapy does not seem any better than to parenteral iron given in divided doses, but reduction in the number of blood transfusions needed antenatally and the convenience to patients makes the procedure well worth while in suitable cases.—I am, etc.,

St. John's Hospital,
Chelmsford, Essex.

G. J. GARTLAN.

Danger on the Road

SIR,—In the *Cyclists' Touring Club Gazette* for October 1904 Constance Everett-Green described the abuse of speed by motorists as "a species of madness." Ever since, those who advocate adequate restrictions on speed have been dubbed "cranks" by motoring enthusiasts, and as suffering from "panic" and "hysteria" by successive Ministers of Transport. For over 40 years motor vehicles in this country have been killing people at an almost constant average of 20 per day; but your editorial points out a change in the significance of the figures. Between the world wars the majority of victims were young children who might thoughtlessly run out into the road after a ball, and elderly or infirm people who were insufficiently active to avoid a fast-travelling car. Now it appears that a majority of the victims are "young male adults and older teenagers" (*B.M.J.*, 11 January, p. 69), who in our "affluent" society are presumably able to acquire a motor-vehicle before they have learned to control the urge for speed. So the motor-vehicle is now to be bracketed with war as a means of exerting a selective destructive action on the cream of the race.

In 30-odd years of general practice I have had occasion to patch up the victims of a good many car smashes—and cannot recall one which was not directly attributable to speed excessive for the prevailing conditions. Your sentence "... the risk of fatal accidents to car occupants and perhaps to lorry drivers