often occur during the phase of recovery at a time when the patient no longer suffers from generalized tetanic spasms and muscle tone is returning to normal, and might be expected to make an uneventful progress towards recovery.

No doubt several abnormalities of function contribute to the genesis of hypotensive episodes in tetanus. In my opinion, hypotensive episodes in tetanus show several features of adrenal insufficiency, reminiscent of mild crises in Addison's disease. It is well known that hypotension and other clinical features may manifest themselves at the onset of such crises before marked biochemical changes become apparent. The slight hyponatraemia recorded in two of the three patients reported is therefore not inconsistent with this explanation.

Accordingly, while supervising medical wards in the Tropics, it was my practice to treat hypotensive episodes in tetanus with moderate doses of hydrocortisone, and obtained a good response to this treatment. In order to prevent the occurence of hypotensive episodes it was my practice to supplement the salt intake of patients suffering from tetanus, so as to decrease the physiological demands on the adrenal cortex and prevent its exhaustion. As soon as the patient was able to take fluids by mouth the salt supplement consisted of substituting one-third strength physiological saline for drinking water. This oral supplement could be started relatively early in the course of the disease, as I avoided when possible the use of curare because of shortage of nursing staff. Such salt supplements are of special importance in the Tropics, where considerable amounts of salt may be lost in the sweat.

With regard to a neurogenic component precipitating hypotensive episodes in the patients described by Dr. Corbett and his colleagues the combination of hypotension and bradycardia in response to visceral stimulation such as tracheal aspiration, followed by increased blood pressure on recovery, would suggest to me vagal overactivity. My reaction, therefore, would be to attempt to maintain blood pressure and cardiac action by the use of parasympathetic blocking agents such as atropine, rather than by the continued use of catecholamines.—I am, etc.,

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Rubella Surveillance: Recent Data from Scotland

SIR,—A detailed account has been presented elsewhere of data relevant to the recent outbreak of rubella in the west of Scotland.[1] In brief, "spotter practitioners" from various areas of the United Kingdom reported to the General Practice Research Unit of the Royal College of General Practitioners 568 cases of rubella for the period ending 7 August 1973, as compared with 807 for the corresponding period of 1972. Notifications to the Registrar General of Scotland revealed early summer outbreaks in the Edinburgh area in 1972 (127 cases in weeks 17-28 compared with 48 in 1973) and in the Glasgow area in 1973 (380 cases in weeks 13-24 compared with 40 in 1972).

The scale of the 1973 outbreak is reflected in the work of the Regional Virus Laboratory, Ruchill Hospital, Glasgow, as follows:

	April		May		June		July		August	
	1972	1973	1972	1973	1972	1973	1972	1973	1972	1973
Total tests	196	420	300	635	339	531	233	394	229	327
Persons tested	122	284	186	391	217	327	146	236	143	208
Suspected rubella in pregnancy	72	158	106	257	115	209	86	161	87	115
Rubella in pregnancy confirmed by tests	3	5	2	4	3	12	2	7	1	3

Because the value of virological tests in the management of suspected rubella in pregnancy (RP) is generally recognized most such cases are investigated, and the above figures for confirmed RP cases from one of the main laboratories carrying out tests in this area probably give a fair measure of the problem. Confirmed RP cases were maximal in June and July 1973, being fourfold higher than in the same month of 1972. This peak is a month later than the peak of rubella notifications to the Registrar General, May-June, weeks 17-24, when the figures for the Glasgow area were nine times greater than in 1972.

In assessing the significance of the increase in RP during the local outbreak of 1973 one may note that rubella was incriminated in only 11 (4·6%) of 239 embryopathies studied in the Glasgow area during 1966-70.[2] If the rubella component increased by between four and nine-fold during the 1973 outbreak embryopathies might be expected to increase by between 13·8% and 36·8% during the period November 1973 and March 1974 in the Glasgow area. However, since virological tests facilitate selective termination of pregnancies when rubella damage is considered highly probable, the observed increase in embryopathies among live births should be less than this and may be insignificant. Observations during the next six months in this area should enable this tentative prediction to be tested.

I thank my colleagues Dr. C. R. Madeley and Dr. C. A. C. Ross of the Regional Virus Laboratory and Dr. D. Reid of the Communicable Diseases (Scotland) Unit for data and assistance in preparing this communication.

—I am, etc.,

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1 Grist, N. R., *Communicable Diseases in Scotland, Weekly Returns*, 1973, No. **35**, 1.
2 Ross, C. A. C., Bell, E. J., Kerr, M. M., and Williams, K. A. B., *Scottish Medical Journal*, 1972, **17**, 252.

Treatment of Diabetic Retinopathy

SIR,—There are two statements in your leading article on the treatment of diabetic retinopathy (25 August, p. 421) with which I do not agree. The first is the opening sentence, "To the diabetic blindness is the most serious complication of his disease." Unless the diabetic values his sight more than his life, diabetic glomerulosclerosis is a more serious complication than retinopathy. The second is the unqualified statement that both types of retinopathy, background and proliferative, cause severe visual disability. In a recent study at King's College Hospital of 92 cases of diabetes of over 40 years' duration 38 had background retinopathy, none of whom complained of disabling loss of vision, while 16 cases had proliferative retinopathy, all of whom had serious visual impairment, six being blind.

Long-standing background retinopathy alone, except in those rare cases in which both maculae are affected, hardly ever causes blindness and is compatible with useful and often reasonably good vision, while proliferative retinopathy carries a far worse prognosis and is the commonest cause of diabetic blindness; this difference should be emphasized.—I am, etc.,

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SIR,—Among the various recommendations for the "Treatment of Diabetic Retinopathy" (25 August, p. 421) I was surprised to note the omission of the potential use of renal transplantation. A recent article describing successful renal transplantation in diabetics[1] notes that this procedure often halts visual deterioration in those young diabetics in whom uraemia and hypertension may lead to a drastic deterioration of vision (as indeed has been recorded in diabetics subject to prolonged dialysis). Surely this treatment merits serious consideration.

I note also omission of any mention of the retinopathy classification of Beaumont and Hollows,[2] which again deserves attention because of the way in which it combines functional and aetiological assessment with ophthalmoscopic observation. Study of their classification leads to other important deductions concerning alternative means of therapy.—I am, etc.,

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1 Kjellstrand, C. M., *et al.*, *Lancet*, 1973, **2**, 4.
2 Beaumont, P., and Hollows, F. C., *Lancet*, 1972, **1**, 419.

Neonatal Jaundice and Maternal Oxytocin Infusion

SIR,—We read the article by Dr. D. P. Davies and others (1 September, p. 476) with great interest. This tends to confirm our initial observation that the incidence of neonatal hyperbilirubianaemia of uncertain aetiology is increased when labour is induced with an intravenous oxytocic agent.[1] Since this observation, originally made at the maternity unit, Fazakerley Hospital, Liverpool, a revised method of induction of labour with a reduced dose of oxytocic agent has been introduced. This has virtually eliminated neonatal hyperbilirubinaemia of unknown aetiology in this hospital.—We are, etc.,

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1 Ghosh, A., and Hudson, F. P., *Lancet*, 1972, **2**, 823.