R.S.V. Infections and Infant Deaths

SIR,—The recent article by Dr. P. S. Gardner and others (7 February, p. 327) speculating on the pathogenesis in death from respiratory syncytial virus (R.S.V.) infection prompts us to report the following case of infant death with R.S.V. infection.

A male child, 4 weeks of age, died three weeks after discharge from a maternity hospital. During the three weeks at home he suffered from "chest infections" for which he received two five-day courses of Ampiclox (ampicillin and cloxacillin). Two days before admission to hospital the baby stopped feeding and had a "cold." On examination in hospital he was undernourished and dehydrated, with a bulging anterior fontanelle. He died less than 24 hours after admission. Postmortem examination showed infarction of the right cerebral hemisphere, the distribution of which suggested extension of a birth injury. There was no evidence of pneumonia or bronchiolitis, but the finding of mucus in the walls of the main and distal bronchial tree, with inflammatory changes in the walls of the main bronchi, confirmed a diagnosis of acute bronchitis; macrophage exudation was seen in the distal bronchial tree.

Virological investigations were carried out approximately 62 hours after death. Indirect immunofluorescent tests for R.S.V. antigen in lung smears showed abundant fluorescence in predominantly mononuclear cells, but cryostat sections of the same tissue 24 hours later gave negative results. Extracts of lung proved to be anti-complementary in complement fixation tests making it impossible to detect R.S.V. antigen by this method. Immunofluorescence staining of smears and cryostat sections of lung with anti-human globulin conjugate gave negative results. R.S.V. was isolated from the lung extract but not from nasal or tracheal swabs, and postmortem serum contained R.S.V. neutralizing antibody to a dilution of 1 in 16.

These findings of abundant R.S.V. antigen in the absence of human globulin in the lung of this baby support those of Dr. Gardner and colleagues in non-bronchiolitic types of R.S.V. infection. The infection here appears to result from direct virus-cell interaction without the contributory factor of an antibody-antigen reaction, although humoral antibody was present in this case.

Unlike the earlier report of Gardner and colleagues¹ where eight R.S.V. infections in 22 cases of infant death with respiratory signs were detected, this is the only case with proved R.S.V. infection we have encountered in a series of 15 infant deaths with respiratory infection. As our series was studied over a period of prevalence of the virus we conclude that respiratory syncytial virus infection associated with infant death is less common in Glasgow than Newcastle. We are, etc.,

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Mark Akenside 1721-70

SIR,—Dr. R. Haworth's interesting paper (20 June, p. 722) brings more light on a medical man from Newcastle on Tyne. He was, in fact, the son of a butcher and was born in Butcher Bank, which was right in the centre of the old part of the city of Newcastle on Tyne, near where Old Saints Church now stands. Butcher Bank has now disappeared, but near by is a road which has the name Akenside Hill running down towards the Tyne. Probably this place name originates from the distinguished doctor and poet born near by. Also in the Jesmond district, which grew up as a fashionable residential area in Victorian times, there is an Akenside Terrace.

The probability that the physician's personality was difficult and he was unpopular is borne out from local sources. It is said that he was ashamed of his family and of his native place and he seldom visited it when he became famous.¹—I am, etc.,

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Rheumatoid Factor, Vascular Damage, and Hypertension

SIR,—Dr. D. L. Gardner's comments (6 June, p. 602) on our paper about raised serum IgG levels in hypertension (18 April, p. 146) deserve further consideration.

It cannot be reasonably assumed that patients with benign hypertension (that is, without papilloedema) would show no more than hyaline change in their blood vessels. In a series of 50 kidneys obtained from patients with benign hypertension scattered fibrinoid necrosis in afferent arterioles was found in 40% of cases.¹

Pickering has argued for a number of years that malignant hypertension is one end of a continuous spectrum of hypertensive vascular damage and that papilloedema and fibrinoid deposits can occur in any kind of hypertension provided it is sufficiently severe.² The reversal of vascular and retinal changes following treatment of hypertension both in man and experimental animals³ lends support to this view. Furthermore, the clinical entity of malignant hypertension has been blurred over the last 20 years by the widespread use of hypotensive drugs. Thus patients with malignant hypertension can come in and out of papilloedema depending on how assiduously they stick to their hypotensive regimen, and several of our patients fell into this category.

The hypothesis was proposed that denatured tissue, produced by elevated blood pressure or any other means, could evoke the formation of opsonizing autoantibodies which would assist in the elimination of tissue fragments and cellular debris by the reticuloendothelial system. We were careful to point out that our hypothesis was speculative, but in view of the increasing number of autoantibodies being described such a proposition merits further study, and we have extended our observations in this field.

Rheumatoid factor was measured in

hypertensive patients and normotensive blood donors using a modification of the latex test slide technique and commercially available reagents (R.A. test: Hyland Laboratories). The serum was heated to 56°C. for 30 minutes before testing, so as to remove normal serum antiglobulin "heat labile latex fixation factor," which may give rise to false positive results.⁴ The rheumatoid factor at a serum dilution of 1/16 was found in 23 out of 57 hypertensive patients and in 7 out of 47 blood donors ($\chi^2=16.0$, $P<0.001$). Using this technique, the rheumatoid factor was thus present in 40% of severely hypertensive patients and in only 15% normotensive blood donors, and this suggests an association between rheumatoid factor and hypertension. Similar observations have been noted by other workers.⁵

Since the role of the rheumatoid factor has not been fully elucidated for rheumatoid arthritis, one can only speculate as to its function in hypertension. It is possible that the rheumatoid factor is an autoantibody directed against altered gamma globulin.⁶ If such is the case, then its presence in hypertension is not surprising, since elevated levels of serum IgG are also found in this condition.—I am, etc.,

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Consultancy in Radiology

SIR,—Dr. R. Eban (20 June, p. 736) raises the question of the requirement of a higher qualification from applicants for consultant posts in radiology. The East Anglian Regional Hospital Board has, I believe, always been mindful of the advice issued by the Ministry of Health in 1948 that "the determination of status cannot depend solely on the possession of postgraduate qualification" but has also been guided by the views expressed by college representatives at advisory appointments committees.

Some colleges and faculties have issued general advice on the qualifications and experience that they would expect to find in candidates for consultant posts. The Faculty of Radiologists has not in fact done so; but the Board consider they have a responsibility to maintain the standing of the specialty, in comparison with others, by giving preference to those who have attained a standard set up by the specialty's own Faculty.—I am, etc.,

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