of "simple concussion." Once frank signs of clot compression have appeared, time subsequently spent in the x-ray department looking for skull fractures is precious time needlessly squandered. In the specific instance of possible penetrating wounds of the cranium, these are notoriously "invisible" even on first-class standard projections of the skull. Patients suspected of such injuries are better referred without delay to a neurosurgeon.

It is common, though not universal, practice to x-ray the cervical spine at some stage in patients with cranial trauma, especially those who are or have been unconscious. Only too often the view obtained is a lateral projection that fails to show C6, C7, or T1. Only too often such films are assumed to exclude injury and further views are not requested.

Lastly, positioning of the patient to secure adequate skull x-rays may be prejudicial to a co-existing but unsuspected cervical spine injury, particularly in an unconscious or otherwise injured casualty. In such cases it is arguable that neck x-rays should take priority over head x-rays.

We suggest that these criticisms of some of Mr. Fowler's original points support rather than undermine his basic argument that it is better to exercise good clinical judgement than to depend on radiographs.—We are, etc.,

A. M. JONES.
P. J. E. WILSON.

Departments of Radiology and Neurosurgery, Morriston Hospital, Swansea.

Vasectomy

SIR,—Now that vasectomy seems firmly established as a reputable, efficient, and perfectly legal method of birth control, may I beg my fellow general practitioners to refer suitable patients directly of the surgeon of their choice as they would for any other operation? If the doctor does not know of a suitable surgeon in his vicinity he will have no difficulty in finding one. There is no longer any need for the Simon Population Trust to be involved except in unusual circumstances.

With the limited but devoted staff and hampered by financial stringency we are hard put to it to deal with a vast correspondence at present running at the rate of more than twelve hundred letters a week.—I am, etc.

L. M. JACKSON,
Honorary Director,
Simon Population Trust
Voluntary Sterilization Project.

Crediton, Devon.

* *A leading article on the legality of sterilization appears at p. 704—ED., *B.M.J.*

Unnecessary Spectacles

SIR,—It remains an erroneous idea that one may go blind or damage the eyes from wearing glasses, not wearing glasses, or wearing the wrong glasses.

Ophthalmologists do commonly encounter cases similar to that which Mr. O. Gayer Morgan (17 January, p. 175) describes where, for various reasons, a totally incorrect refractive prescription has been worn with no disability or even awareness of the situation on the part of the patient, Yet our practices are not littered with the accommodational cripples which should have resulted from the unnecessary prescription of spectacles. The surprisingly rare examples which do crop up usually respond rapidly to orthoptic treatment. Why did this particular case not also respond similarly? Gross accommodational deficiency does occur in people who have never worn glasses.—I am, etc.,

M. J. GILKES.

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Polyarteritis

SIR,—I was interested in Dr. D. N. Golding's three cases (31 January, p. 277). This condition is usually referred to as cutaneous polyarteritis nodosa, and it bears the same relationship to systemic polyarteritis as cutaneous lupus erythematosus does to systemic lupus erythematosus. It affects only the skin, skeletal muscles, and peripheral nerves, and is usually self-limiting. Of the eleven patients seen at St. Bartholomew's Hospital over a period of 20 years none has died of the disease, and in two only is there any persisting activity, both in the skin.

More important is the fact that in 100 consecutive cases cutaneous lesions histologically typical of polyarteritis nodosa were found in these 11 cases alone. A positive skin biopsy in this disease appears therefore to be diagnostic of the cutaneous and not the systemic type, and should be interpreted accordingly.—I am, etc.,

PETER BORRIE.

London W.1.

Psychiatric Social Worker

SIR,—Having been a psychiatric social worker before I was a general practitioner, I was interested in Mrs. Joan Brandon's Personal View (7 February, p. 361).

I think part of the difficulty is linguistic. The original psychiatric social worker tutors were trained in America, and the working language has, I think, retained a transatlantic flavour. Mrs. Brandon talks about the "skills" of the social worker (six times, always in the plural) as though these are a collection of assorted assets rather than a basic body of professional knowledge and competence. She goes on to say that it is as meaningless to ask what a social worker is, as what a doctor is—apparently because there are specialists in each profession. But one can assume that any doctor who is registered can use his five senses and a few tools, as well as his knowledge and experience, to diagnose and treat the commoner ailments; and to know when further consultation is necessary.

I disagree with Mrs. Brandon's final point. Surely there is such complete agreement between doctors and trained social workers about respecting a patient's confidence that it would be a waste of time to discuss it? Doctors have long worked closely with district nurses. They do a great deal of social work. The good ones are extremely good, and they have a common language with the doctor, which saves time and possible misunderstanding. I think there could well be a place for a psychiatric social worker too, but it would be better to find some general practitioners who agree and some psychiatric social workers whose employers would release them part-time, and then discuss how the actual individuals concerned are going to make it work—rather than discussing generalities at conferences. Given good will and a compatible sense of humour, it should work.

One final point; could social workers find a better name? I think it's a pity that almoner was dropped for the medical social worker. Like doctor and nurse, it had changed its meaning, but, also like them, everyone understood its current usage. If it hadn't been dropped, "community almoner," as opposed to "hospital almoner," might have been possible.—I am, etc.,

PHILIPPA WIGAN.

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Arthus Reaction and Pneumonia

SIR,—Dr. P. S. Gardner and colleagues (7 February, p. 327) speculated on the pathogenesis of respiratory syncytial virus infection in infants, and left the problems aired but open. Some recent work which I have been doing with Dr. R. B. Heath (Virology Department, St. Bartholomew's Hospital) seems to give positive support for the implication of an Arthus or type 3 allergic reaction in recovery from such infections and offers some explanations for these problems.

We have been studying the sequence of immunological events in tissues, serum, and bronchial secretions which follow a primary non-fatal parainfluenza virus infection in mice. The experimental model used has been reported elsewhere, (1) and this work is being prepared for publication. Our observations are as follows.

Within two days of the infection there is an influx of immunoglobulin-containing cells into peribronchial and perivascular oedema fluid (Fig. 1). This process accelerates very rapidly at about day five and subsequently the cells organize into lymphoid-like areas (Fig. 2). These lymphoid aggregations persist for at least 50 days after the infection. IgA- and IgA-containing cells are present in a ratio of roughly 1:1, while IgM-containing cells are present in less significant numbers. Compared to the IgA:IgG cell ratio of about 1:10 in spleen and lymph nodes, these findings strongly suggest some preferential production of IgA in lung tissue. Initially virus is almost completely confined to cells of the bronchial mucosa and reaches maximum intensity in this location at days 3 to 4 (Fig. 3). There is then a dramatic change which seems to correlate with the accelerated arrival of immunoglobulin-containing cells in the lungs. The infected cells

are abruptly shed into the lumen with an intense inflammatory exudate (Fig. 4).

Immuno-electrophoresis shows that bronchial washings contain only albumin and IgA before infection. An inflammatory exudate appears two to three days after infection, is at its peak at about the fifth day, and then slowly subsides. At this time IgA, IgG, and other serum proteins are present. IgM was not detected. A month after infection the protein constituents of the bronchial secretions return to normal but with a suggestive increase of IgA. Specific antibody was first detectable at days 7-8 and appeared in serum and secretions at the same time. During the inflammatory stage the antibody in secretions was both IgG and IgA and at one month was mostly IgA. The antibody in serum was present in all the immunoglobulin classes but at one month was mostly IgG.

These findings would fit well with an Arthus reaction occurring at about day five, followed by or associated with the sudden shedding of the infected cells into the bronchial lumen. Conventional light microscopy confirms the initial polymorphonuclear infiltrate one would expect. It is not unreasonable to assume that a gradient between available antigen and early local antibody could exist to produce the necessary moderate antigen excess in the presence of complement in the inflammatory exudate and thus to provide the conditions necessary for a classical type 3 allergic reaction to occur. There is no convincing evidence that either secretory or serum IgA can fix complement or mediate an Arthus reaction. These attributes are definitely associated with IgG antibodies. It is possible that local IgA antibodies present at the time of antigen release could modify the extent of such a reaction. The ability to make IgA antibodies in man is variable and there is probably a group of physiological "slow starters" in IgA production. (2) In any event adult levels are not usually achieved before three years of age. Maternally derived antibodies are all IgG and the usual long-term response to parenteral vaccination procedures is also IgG. In neither case are these antibodies likely to be found in a healthy respiratory tract, but will appear only following inflammation.

I would therefore suggest that a widespread Arthus reaction occurs in these infants after considerable virus replication and inflammation, and the resultant cellular debris then causes airways obstruction in an infant's smaller bronchioles and may cause anoxia and death. It may be that the picture of pneumonia, rather than being the manifestation of some entirely different allergic mechanism as Dr. Gardner and colleages suggest, could be due to aspiration of this cellular debris to alveolar level.

Part of this work is supported by an M.R.C. grant.

—I am, etc.,

G. BLANDFORD.

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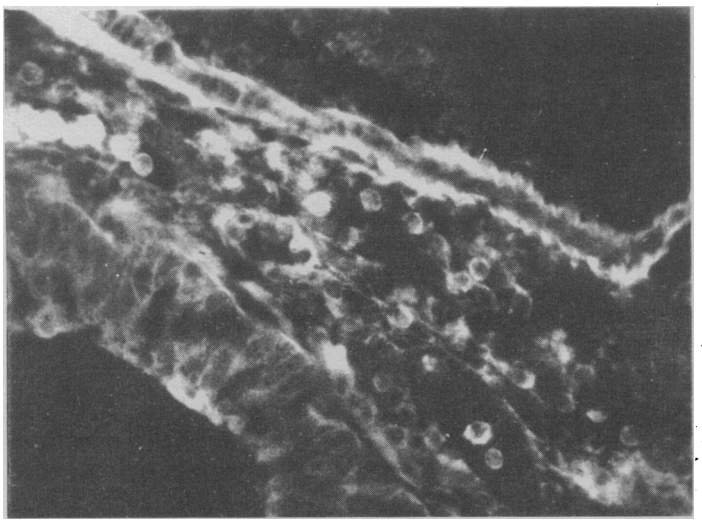

Fig. 1

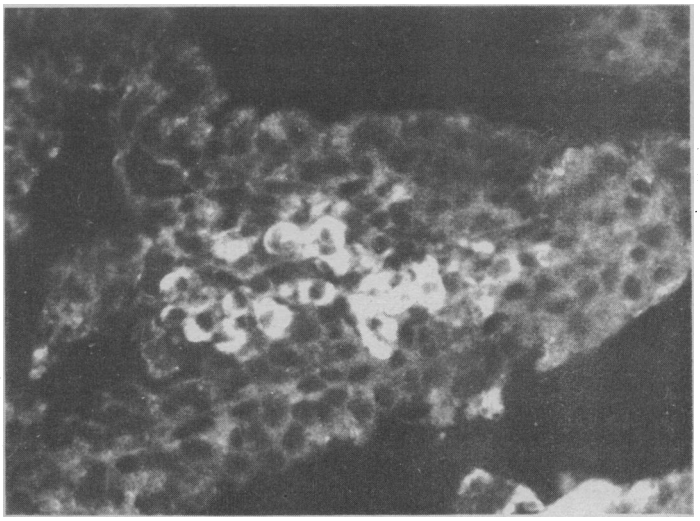

Fig. 2

Indirect fluorescent antibody staining of IgA-containing cells in mouse lung: (Fig. 1) 2 days post infection, (Fig. 2) 12 days post infection (×540).

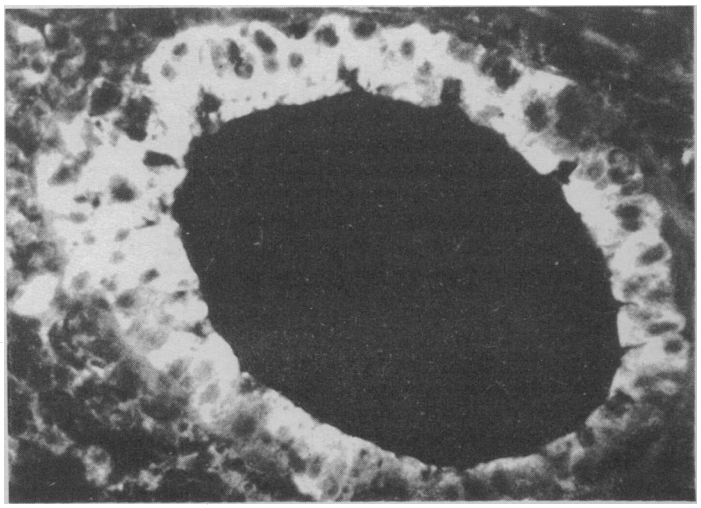

Fig. 3

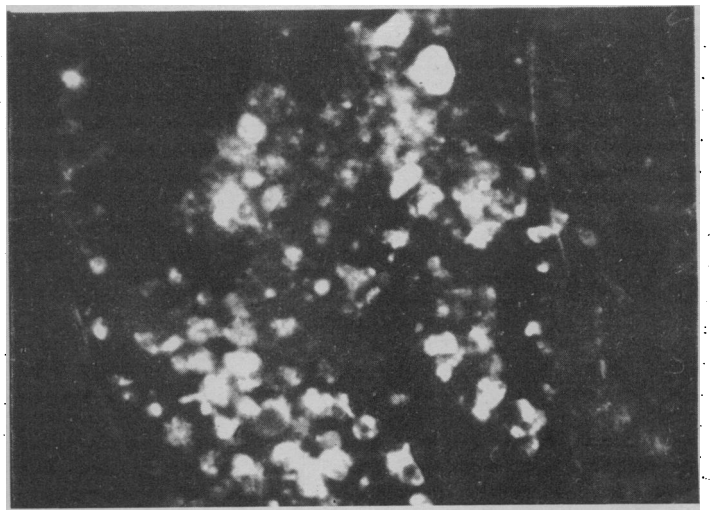

Fig. 4

Fluorescent antibody staining of Sendai virus antigens: (Fig. 3) 4 days post infection, (Fig. 4) 5 days post infection (×540).