department. The R.M.P.A. very much regret that this degree of independence has not been given to the Hospital Advisory Service.—I am, etc.,

WILLIAM SARGANT,
Chairman,
Public Relations Committee, R.M.P.A.
London W.1.

REFERENCE

1 *Report of the Committee of Inquiry into Allegations of Ill-Treatment of Patients and other Irregularities at the Ely Hospital, Cardiff*, Cmnd. 3975. London, H.M.S.O., 1969.

The Influenza Epidemic

SIR,—Dr. J. N. Andrews (17 January, p. 171) invites doctors in other areas to give the salient features as they have affected patients during the influenza epidemic. My observation of cases bears a remarkable similarity to his in that children were rarely affected; the illness was severe and of sudden onset; prostration was much greater than in any other epidemic I have experienced; the prolongation of the illness was proportionate to the age of the patient; the cough was persistent and completely resistant to medication; and many younger patients presented with severe abdominal pain. On the other hand, I met no cases of cyanosis.

A common finding was of one-sided basal crepitations in the older patient. They were given either tetracycline or erythromycin, yet *x*-rays of the chest a week or so after clinical recovery showed no traces of an inflammatory condition. Not a single case of influenza occurred in any of my "at risk" patients who had been vaccinated against influenza; the vaccinations were given during the month of October 1969.—I am, etc.,

L. G. R. HULL.
Doncaster,
Yorks.

SIR,—Dr. J. Fry (17 January, p. 171) hopes that "a profile of this epidemic will be built up and published by the Department of Health and Social Security." I think that the Department would be better employed in telling people how to treat the disease.

During my 30 years' experience of influenzal outbreaks in general practice I have been increasingly impressed by the ignorant impotence of the public in the face of this recurring scourge. When one reflects on the death rate resulting from even a minor epidemic such as we have just encountered, it is obvious that influenza is a major public health hazard, the last surviving counterpart to the great plagues of mediaeval times.

The man in the street regards influenza as a sort of severe cold. Encouraged by T.V. advertisements, he "fights" the infection with antipyretics while remaining ambulant as long as possible, thus dissipating his reserves. He has no notion of the toxic processes involved, and gets up as soon as his temperature is normal. "I was getting weak from lying in bed, doctor," or " I can't stick the bed any longer; it's making my back worse"—and these cases inevitably relapse. We doctors are far too busy to argue with and educate the public during an epidemic, but surely this is an eminently suitable field for a five or ten minutes' T.V. film, suitably repeated. How much unnecessary work we should be saved, and how much morbidity and depression avoided.

Finally, it is impossible to forecast the severity of an attack in the early stages and my observations apply only to normally fit people. Obviously, any pre-existing weakness reduces the individual's resistance, and such cases may well call for our utmost skill in treatment.—I am, etc.,

MUNGO B. HAY.
Bridge of Weir,
Renfrewshire.

SIR,—We read the letter from Dr. G. S. Crockett (17 January, p. 171) with considerable interest and were struck by his finding of "extremely low levels of potassium in almost every case" of influenza he has investigated in the current outbreak. This observation surprised us, as we have estimated the plasma electrolytes in the majority of our influenza cases and did not recall finding a significant incidence of hypokalaemia. However, prompted by Dr. Crockett's observations, we have made a detailed analysis of our cases and the following information has come to light.

Since Christmas 1969 we have admitted 26 cases of influenza (Table, Group 1) the diagnosis being based on clinical features and substantiated by a fourfold rise in complement-fixing antibodies to *Myxovirus influenzae* A and/or the isolation of this virus from throat swabs. In those cases where virus has been isolated the agent had been confirmed as Influenza A2 virus (Hong Kong variant) by the Reference Laboratory at Colindale. The age of these patients has ranged from 7 months to 75 years, and the degree of clinical illness has been quite variable; nevertheless, clinical features similar to those described by Dr. Crockett were found in the more seriously affected. In 16 (61·5%) cases plasma electrolytes were estimated at admission, and this figure includes all those graded as severely ill, but, as is shown in the accompanying Table, we have been unable to confirm the presence of marked hypokalemia.

Furthermore, we have analysed 67 cases (Table, Group 2) of virologically proved influenza admitted to the same unit between January and April 1969. In view of the fact that these were infected by a similar strain of virus comparisons with them should be valid. Out of 67 cases 44 (65·7%) had the plasma electrolytes estimated, and this group also showed marked variation in the clinical spectrum and a similar age distribution. However, once again no evidence of significant hypokalaemia was apparent.

In summary, an analysis of 93 cases of virologically proved Influenza A2 admitted to this unit has been made. In 60 cases plasma electrolytes were determined in the acute phase of the illness and in 8 instances minimally depressed plasma potassium levels were found. These results do not correlate with those of Dr. Crockett, and it is of interest to postulate why hypokalaemia was found in such a high proportion of the Kettering cases, as there appears to be a clinical similarity with at least a proportion of the Leeds patients and the same strain of influenza virus is involved. We did wonder whether complicating features such as preceding diuretic therapy, steroid therapy, etc., had influenced the findings. However, in view of Dr. Crockett's observations further studies of the plasma potassium levels in this condition may well be warranted.—We are, etc.,

Virologically Proven Cases of Influenza A2 (Hong Kong variant)

	Group 1 December 1969–January 1970	Group 2 January–April 1969
Total number of cases (Deaths in brackets)	26 (2)	67 (7)
Number with plasma electrolyte estimations (% of total in brackets)	16 (61·5%)	44 (65·7%)
Number with potassium level in the normal (3·6–5·3 mEq./l.) range	11	40
Number with potassium level outwith the normal range	5*	4†
Number diagnosed on serology (fourfold or greater rise in C.F. antibody)	12	14
Number diagnosed by virus isolation from throat swabs	8	38
Number diagnosed by serology and virus isolation	6	15

*Actual potassium levels: 2·9, 3·2, 3·3, 3·5, 5·7 mEq./l. †Actual potassium levels: 3·3, 3·4, 3·4, 3·5 mEq./l.

J. STEVENSON.
B. K. MANDAL.
Department of Infectious Diseases,
Seacroft Hospital, Leeds.

M. H. HAMBLING.
Public Health Laboratory,
Leeds.

SIR,—During the recent influenza epidemic 18 cases of influenza and respiratory failure were admitted to the resuscitation units in the Portsmouth group of hospitals. The ages of these patients ranged from 29 to 77 years. Of these, four had no previous chest diseases, eight had chronic bronchitis, three had asthma, one had pneumoconiosis, and two were diabetic and were on insulin. The majority of the patients when admitted were deeply cyanosed, semicomatose, or comatose, with tidal volume and Pa_{O_2} as low as 100 ml. and 32 mm.Hg respectively. Death from severe hypoxaemia was imminent if resuscitation measures were not taken.

In the resuscitation units the following treatments were given: oxygen therapy, bronchotracheal lavage, intermittent positive pressure ventilation with oxygen-enriched air via oral cuffed Portex tube, the use of antibiotics, bronchodilator drugs, steroids if indicated, sedatives, muscular relaxants, and intravenous therapy. Seven out of 18 patients were tracheostomized.

The length of time the 18 patients were

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ventilated varied from 2 hours to 264 hours. The total ventilation time for the 18 patients was 2,088 hours. At the peak period of the epidemic 8 ventilators were used at one time. Out of 18 patients 5 survived. Of the 13 patients who died 10 died with fulminating pneumonia. Two diabetic patients, with blood sugar up to 1,300 mg./100 ml. despite peritoneal dialysis, gradually deteriorated and died. One died after an acute episode of status asthmaticus

In surveying these cases the following findings were of interest: Cerebral vascular episodes were found in three patients during acute viraemia. One patient had epileptiform fits during severe hypoxaemia and viraemia. Hyperkalaemia and a high blood urea were found in severe acidotic patients on admission, while hypokalaemia was the common finding after prolonged treatment of this group of patients.

In weaning the patients off the ventilators, the call for reinstitution of intermittent positive pressure ventilation was indicated when Pao_2 fell below 70 mm.Hg and physical exhaustion was observed.—I am, etc.,

K. T. TCHEN.

St. Mary's General Hospital,
Portsmouth.

Bacteria and Cadmium-treated Fabrics

SIR,—Recently a variety of cotton fabrics treated with cadmium have been offered to hospitals. It is claimed that bacteria falling on this type of cloth are killed. This note reports our investigations into the efficacy of the treated cloth.

The methods used are novel and should give a more accurate measure of the numbers of organisms present than the traditional "sweep plates." Chemically treated materials which are claimed to act at ambient relative humidity cannot be tested either in fluid media or on the surface of agar, since the chemicals diffuse into the medium and are being tested in solution. The following method tests "dry killing."

Experiment 1.—Samples of the treated cloth together with control cotton sheeting were suspended on cradles in the patients' beds and left overnight. The samples were removed for bacteriological examination and stretched across a plastic ring of 14 cm. internal diameter. The procedure used was to sweep the taut cloth over the input tube of a slit sampler. The organisms removed were impacted on to the surface of nutrient agar in the usual way. 30 litres of air was drawn through in $1\frac{1}{2}$ min. while the surface was moved slowly round so that the whole surface was covered three times. The material was now removed and 20 litres of room air sucked through and the organisms impacted on to the opposite half of the plate as an environment control. The results are summarized below:

	Treated Cloth	Control
Number of tests	34	21
Total organisms	10,044	6,115
Mean count	295	291
Mean count environment control	4·0	4·5
By unpaired "t" test	P = 1·0	

The implant varied widely from patient to patient, from 21 only on one piece to 3,696 on another, and an attempt at a more even seeding of the fabrics was therefore made.

Experiment 2.—the test samples and controls were horizontally displayed in a cubicle in a chessboard pattern. One of us undressed and dressed again in the middle of the area. The desquamated skin liberated was allowed to settle for one hour. All the pieces of cloth were then collected, put into plastic bags and left at room temperature for 24 hours. Counts were done as in the previous experiments. Results:

Controls	17,	42,	49,	65,	87,	93,	98,	99,	100
Tests	24,	46,	48,	54,	63,	69,	91,	98,	103

P = 0·15 "t" 1·59

We conclude that until positive evidence has been published of the effectiveness of this material its introduction into the hospitals of Britain should be delayed, because of both the increased cost and the possibility of allergic sensitization taking place. We have not investigated the effect of the treatment in rendering cloth resistant to rotting in humid climates.—We are, etc.,

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R. R. DAVIES.

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Septic Shock

SIR,—In your concise yet comprehensive leading article on septic shock (3 January, p. 3) the current controversy on the aetiology and management of this growing clinical problem was discussed. There are, however, two aspects of the problem on which the article was misleading.

Not only are there differences between different species in their response to endotoxin, as you point out, but there are important differences in any one species between the effects of endotoxin and those of whole live Gram-negative organisms. In the dog, for example, which has been the most extensively investigated model, live organisms do not cause the precipitous hypotension, the rise in portal pressure, and rise in total peripheral resistance, all of which occur after endotoxin.[1 2] In addition, endotoxin cannot be incriminated in those cases of septic shock (about 10-15%[3 4]) due to Gram-positive organisms. The lipopolysaccharide from the cell walls of Gram-negative bacteria is therefore contributory in many cases to the pathogenesis of clinical septic shock, but it is certainly not the exclusive cause.

In the paragraph discussing therapy, you state that "To the extent that the transport of oxygen is disturbed it is rational to correct reduced oxygenation of tissues by supplying oxygen . . . ." In those cases with the severe lung lesion transport of oxygen to the tissues is impaired, but in most patients with septic shock the tissues are unable to extract oxygen normally, as witnessed by reduced total oxygen consumption and narrowed arteriovenous oxygen difference.[5 6] Although raising the Po_2 of the inspired gas would seem to be indicated clinically, it is ineffective in raising the total oxygen consumption. To explain the mechanism of this paradox, many authors have postulated peripheral arteriovenous shunting,[7-9] but work in progress in this laboratory suggests that the basic lesion is the peripheral cell's inability to utilize oxygen. Biochemical evidence is already available that sepsis and endotoxins disturb cellular metabolism.[10-12]

If this work is substantiated, it will be necessary to adopt the new concept of cellular damage as the *cause*, not the effect, of the haemodynamic problem in septic shock.—I am, etc.,

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SIR,—Your leading article "Septic Shock" (3 January, p. 3) contains either an important omission or an error. I think you may wish to draw the attention of your readers to this, which is also to be found in the primary reference to which the article refers.[1]

The article makes certain recommendations as to the levels of central venous pressure which are appropriate for the administration of intravenous infusion in septic shock. The values for central venous pressure which the author quotes are "10 to 12 cm. of water" and "10 to 15 cm. of water." These values appear to be incorrect. Either the units are in error, in which case they should have been millimetres of water, or the reference level used for the measurement was somewhat unusual, in which case it is particularly unfortunate that this was omitted.

McGowan and Walter[2] stated that "when the central venous pressure is −5 cm., or below in shocked patients a rapid infusion of a suitable fluid should be given intravenously to raise the central venous pressure to the upper part of the normal range—that is, —3 to —4 cm. which can be assumed to be adequate for normal cardiac output." They used the sternal angle as their reference level.—I am, etc.,

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**The reference level for which 10-12 cm. water could be considered a normal central venous pressure is the interior of the right

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