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 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 embarrassment was 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 reintroduction of intermittent positive pressure ventilation was indicated when PaO2 fell below 70 mm Hg and physical exhaustion was observed. I am, etc.

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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 "swab 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 media and organisms 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 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 environmental control. The results are summarized below:

| Treatment | Total Organisms | Mean Count | Minimum | Maximum | Control
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<tr>
<td></td>
<td>Numbers of tests</td>
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<td>34</td>
<td>10,044</td>
<td>295</td>
<td>291</td>
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"A" 19

We conclude that until positive evidence has been established 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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Septic Shock

SIR.—In your concise yet comprehensive leading article on septic shock (3 January, p. 3) the current controversy on the etiology 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 In addition, endotoxin cannot be incriminated in those cases of septic shock (about 10-15%)2 due to Gram-positive organisms. The lipo-polysaccharide 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 because of the reduced total oxygen consumption and narrowed arteriovenous oxygen difference.3 Although raising the PO2 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,4 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.

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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REFERENCES