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 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 "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 1½ 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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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 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,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 Walter² 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