Patient	Dosage of Clindamycin	Time of Specimens	Serum level	Sputum level
1	300 mg.	2 hours	12·0 µg./ml.	4·025 µg./ml.
2	,,	,,	7·6 µg./ml.	7·0 µg./ml.
3	,,	,,	Not done	0·91 µg./ml.
4	,,	,,	3·1 µg./ml.	2·1 µg./ml.
5	,,	,,	Not done	2·2 µg./ml.

It would seem therefore that serum and sputum levels can be achieved sufficient to inhibit *H. influenzae*.—I am, etc.,

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Another Paraquat Fatality

SIR,—We wish to record a further instance of fatal paraquat poisoning. So far as we can ascertain, this is the tenth reported fatality caused by this widely used herbicidal preparation.[1-4] In December 1965 a 42-year-old male accidentally drank a small quantity of "weed-killer" from an unlabelled stout bottle. It was later estimated that the quantity of fluid consumed was about three-quarters of a teaspoonful. The nature of the substance could not be immediately ascertained, but it was subsequently identified as Gramaxone 19% w/w paraquat ion) by the laboratories of I.C.I. Ltd. On admission to hospital a few hours after taking the poison, the patient complained of a burning sensation in the throat and repeated vomiting. Despite immediate gastric lavage, prophylactic penicillin, and abundant oral and intravenous fluids, there was progressive clinical deterioration. At three days the tongue was severely ulcerated and two days later haemoptysis occurred. On the eighth day jaundice, pyrexia, tachycardia, and a blood urea of 375 mg./100 ml. were recorded. The patient died some hours later.

The outstanding necropsy findings included focal myocardial necrosis, severe pulmonary haemorrhage and oedema, dense eosinophilic alveolar membrane, and proliferation of fibroblast-like cells in the alveolar septa.

It is apparent from this and other reports that the lethal dose of paraquat may be very small. Further fatalities will only be avoided by proper care in the storage and use of this valuable but potentially dangerous compound. A detailed and illustrated clinico-pathological report is being prepared for publication.—We are, etc.,

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Drugs in Depressive Illness

SIR,—I was interested to read the article by Dr. A. M. Porter (28 March, p. 773). In his discussion he concluded that his work provided evidence to suggest that the milder forms of depression may be effectively treated with support and a placebo. One of the difficulties he mentioned in carrying out a drug trial with placebo was the easy breach of the cipher when drugs like imipramine were used because of their side-effects, and suggested the possibility of adding atropine to the placebo tablets to avoid this difficulty. This possibility has been discussed previously in a critical view of antidepressant drug trials.[1] This review also mentioned that most depressed patients get better anyway, and the patients who improve after being prescribed tablets have done *post hoc*, but not necessarily *propter hoc*.

In a double-blind drug trial carried out in this hospital for which preliminary results are now available, 54 patients entered the trial and of these 15 were treated with a tablet containing amitriptyline and perphenazine (Triptafen), 20 with perphenazine (Fentazin), and 19 with a placebo containing atropine. Each of the patients was randomly allocated to one of the treatments and assessed on three occasions in terms of 15 symptoms plus an overall rating. The trial and its analysis are still continuing.

Degree of Improvement	Triptafen	Fentazin	Placebo
Worse	2	1	5
Unchanged ..	2	4	3
Improved	0	0	4
MuchImproved	1 } 11	4 } 15	5 } 11
Recovered	10	11	2

χ^2 (2 d.f.) = 1·52

χ^2 was evaluated after pooling the "worse" and "unchanged" categories.

The Table shows the distribution of patients according to their degree of improvement at the final assessment. A statistical analysis shows that $\chi^2 = 1{\cdot}52$, implying a lack of significant difference between the various treatments. Although these patients were from hospital practice, the results are certainly very similar to those of Dr. Porter, and of Hunter *et al.*[2], and would support the view that antidepressant drugs tend to become established in clinical practice on inadequate evidence.—I am, etc.,

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Blue Light and Jaundice

SIR,—Inasmuch as I am an "enthusiastic" supporter of phototherapy, I feel that I should comment on the ultra-conservative editorial comment, "Blue Light and Jaundice" (4 April, p. 5).

Workers in this field[1 2] now recognize that bilirubin can produce learning difficulties in humans at lower serum concentrations than were previously realized. Previous studies have not detected these defects because of inadequate testing. It is for this reason that a safe and simple method to replace exchange transfusion is needed. The statement that phototherapy is "unlikely to be adequate treatment" in preventing low bilirubin kernicterus is an opinion with which I certainly do not agree. Preliminary data suggest that this is the type of infant who will benefit. Phototherapy is also now widely being used to control mild haemolytic disease. There has been no convincing evidence published to support the original suggestion by Franklin[3] that the photochemical products might be toxic. There is considerable evidence that these products are not toxic either in vivo, in vitro, and in human infants.[4] I would ask that the author of this leading article and others read the Bilirubin Symposium[4] quoted, and not just the summary as has apparently been done, for more data on this important point. I would also refer the reader to two recent articles,[5] for more reassuring evidence in human infants. Published human clinical experience for several countries with phototherapy now numbers over 5,000 cases, and no serious toxic effects have been reported to date.[7] More recent studies in two human infants with Crigler-Najjar syndrome employing ^{14}C labelled bilirubin and phototherapy indicate that the breakdown products are rapidly and safely excreted.[8]

Early feeding may have limited the incidence of hyperbilirubinemia of prematurity in one nursery in England to less than 1%, but this has certainly not been the experience in the U.S.A. Where this problem has been studied carefully, one usually finds that from 10-25% of premature infants can be expected to develop serum bilirubin concentrations of over 15 mg./100 ml.[7]

The leader writer also cites a reference[9] referring to a hypothesis that light might cause retrolental fibroplasia. This curious theory is certainly not widely held by many paediatricians.

I, and others, have not used "blue" light, but have employed broad spectrum light. One should not of course use "blue light," especially if you want to judge skin colour. Phototherapy units are now designed so that they not only allow better observation of the infant but also free access to the infant. The leader writer's quaint reluctance to allow even a minor change in the present lighting conditions in the nursery suggests that he knows that these conditions are indeed optimal for newborn infants. I challenge this idea. I am afraid that few doctors actually have any idea about the present lighting conditions in their nursery. Outdoor light on a bright day supplies 10,000 foot candles of broad spectrum light. I have found nurseries with 25 foot candles to 2,000 foot candles in the same nursery. Phototherapy units supply only about 300-500 foot candles of light. "Phototherapy" is,