

*Candida albicans* in the sputum leads to suspicion of moniliasis, is to take every step to exclude other diseases. It should not be forgotten also that the use of antibiotics predisposes to invasion by this organism. The only reference I have been able to find to the use of intravenous gentian violet in broncho-pulmonary moniliasis dates from 1928,<sup>1</sup> and on general principles it would seem very unlikely that an effective dose of this agent could be brought by this route to the site of what is essentially a surface infection of the bronchial tree. It would seem more logical to attempt to treat the condition by inhalation of a nebulized solution. The strength of solution used in this way should be determined by the patient's tolerance. Probably 1:5,000 would be tolerated. If, as is likely, there are associated lesions of the upper respiratory tract, these could, of course, be treated as usual by local applications of gentian violet. In a case in which there is good reason to suspect a serious primary involvement of the lungs or in which secondary moniliasis is an important complicating factor, it would be worth trying the effect of nystatin, although this antibiotic is poorly absorbed when given by mouth. 500,000 units four times a day would be a suitable dosage.

## REFERENCE

<sup>1</sup> Stovall, W. D., and Greeley, H. P., *J. Amer. med. Ass.*, 1928, 21, 1346.

## Paper Disks for Phage Typing

**Q.**—Are paper disks for phage typing of staphylococci,<sup>1</sup> similar to paper disks used for antibiotic sensitivity, available? Are they satisfactory? If so, where can they be obtained?

**A.**—These disks are not available in this country, and they are not regarded with favour by those expert in this type of work. They have to be left on the surface of the medium, and lysis is seen only as a halo surrounding them. This deprives the test of its quantitative aspect: as usually performed it employs concentrated and dilute phage, which may produce either confluent lysis or isolated plaques: lesser degrees of susceptibility to phage would be undetected by the disk method.

## REFERENCE

<sup>1</sup> Mora, E. C., and Eisenstark, A., *J. Lab. clin. Med.*, 1958, 51, 802.

## NOTES AND COMMENTS

**Desensitization to Streptomycin.**—Dr. HAROLD WILSON (London, W.1) writes: I am interested in your reply in "Any Questions?" (*Journal*, February 27, p. 665) to a query about desensitization to streptomycin. Crofton<sup>1</sup> distinguishes clearly between patients sensitized to streptomycin as a result of treatment and nurses who become sensitive as a result of handling the drug. He finds that desensitization is comparatively easy in the case of the former but difficult and sometimes hazardous in that of the latter. Your expert suggests that nurses should be given an initial dose of 10 µg., followed, if there has been no reaction, by 100 µg. In these highly sensitive subjects reactions are apt to occur to extremely low doses of streptomycin. One of Crofton's cases developed a rash after doses of 50 µg. and 100 µg. given intramuscularly, while Cohen and Glinsky<sup>2</sup> reported a very alarming reaction with damage to the labyrinth after an initial dose of 100 µg. In my own experience<sup>3</sup> an extensive eruption occurred after doses of 20 and 40 µg. given intramuscularly. I would suggest a much more cautious scheme of desensitization for nurses, and advise an initial dose of 2 µg. followed by daily doses of 5, 10 and 20 µg. Steroid cover is likely to reduce the reaction but not to suppress it completely. It is doubtful if antihistamines have any influence on a purely eczematous condition. Desensitization is not always permanent.

## REFERENCES

- <sup>1</sup> Crofton, *Brit. med. J.*, 1953, 2, 1015.
- <sup>2</sup> Cohen and Glinsky, *J. Allergy*, 1951, 22, 63.
- <sup>3</sup> Wilson, H., *Brit. med. J.*, 1958, 1, 1378.

OUR EXPERT replies: The original question was, "Is it possible to desensitize a patient who had a reaction after being given streptomycin and dihydrostreptomycin systemically?" In the answer I gave details of the skin testing and desensitization of such a patient (sensitized by injection) with references to the literature. However, as is quite clear from the literature and clinical practice and as is again emphasized by Dr. Wilson, those sensitive by contact, such as nurses and dispensers, are in quite

a separate and more sensitive group than those sensitized by injection.

It was because of the risk of some readers not appreciating this and possibly applying the dosage advised for the questioner's case to a nurse or dispenser, that I called attention to the other more sensitive group. I wrote: "In patients very allergic to streptomycin, such as nurses or dispensers hypersensitive by contact, skin-testing should start with 10 µg. . . . In patients . . . who develop sensitivity during treatment . . . 10 mg. is advised. . . . Desensitization starts with a test dose just below that which gives a positive reaction, and, in the absence of any reaction, the dose is subsequently doubled each time. . . ."

I suggested 10 µg. as an initial test dose because I could find no record of a dangerous reaction having occurred with this dose. It must be emphasized, however, that the dose used in an initial skin-test must always be judged very carefully in relation to the history in that particular case. I would agree with Dr. Wilson that in the exceptional patient an even smaller dose might be used as a starting test dose. I would also like to emphasize that, if a marked local reaction occurs with a skin-test dose such as 10 µg., later skin-testing should be carried out with a smaller dose, 5 µg. or 2 µg., until the dose at which a positive reaction is just obtained is known. Desensitization should then begin with a dose just below that which only just gives a positive reaction. I would agree that a steroid cover would reduce any reaction but not suppress it completely, but cannot agree that antihistamines have no suppressive action.

I sincerely welcome Dr. Wilson's contribution on this important and difficult subject. Had the original question been on this specially sensitive group I would have certainly dealt with it in more detail and brought Dr. Wilson's important personal contribution to the notice of readers again.

**Ultrasonics in Physiotherapy.**—Dr. E. PHILLIP (Wellington, New Zealand) writes: Under "Any Questions?" (*Journal*, November 7, 1959, p. 968) you answer a question on ultrasonics in physiotherapy. Ultrasonic waves certainly produce heat through friction at the junction of various tissues with different densities. This, though, is only a side action. Sound waves produce a physical movement of molecules. A micro-massage with rapid intense movement of particles produces an intense intra- and peri-cellular movement of fluid which removes waste products and helps healing. The one danger is the possibility of cell disruption by too strong an intensity inaccurately used. Ultrasonics used by a skilled operator under the guidance of a competent physician for suitable disabilities are a valuable treatment and take their place among the tools of the physiotherapists. They have been used in Wellington increasingly over the last few years.

OUR EXPERT replies: This is an interesting comment. Certainly the increasing use in Wellington is the reverse of the situation in England, where ultrasound was tried and found to have few indications. In England ultrasonics have been used less and less over the last few years.

**Correction.**—The obituary of Dr. Barbara G. R. Crawford (March 26, p. 972) gave her maiden name as Rutherford instead of Rutherford. Dr. Crawford is survived by a daughter and two grandsons (and not by two sons as stated in the obituary).

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