on a quick answer for the cause of death, thus not allowing adequate investigation in cases of this nature? Why does the coroner have to pronounce his verdict at all in these cases, when there exists a panel of experts more qualified to decide—that is, the P.M.P. or the U.I.C.C. Consultative Committee?

Dr. Thurston points out that "recording of causes of death is important for the national statistics on which so many calculations and decisions depend." The asbestos industry is fully aware of the health hazards associated with it, but inaccurate death certification can only serve to give the wrong impression and harm an enlightened industry producing a product for which in many instances there is no known substitute.—I am, etc.,

H. C. LEWISOHN.

Turner Brothers Asbestos Co. Ltd.,
Rochdale, Lancs.

REFERENCE

- ¹ Report of Working Group on Asbestos and Cancer, *Brit. J. Industr. Med.*, 1965, 22, 165.

Avoidable Hazards

SIR,—Your leading article "Asbestosis" (8 July, p. 62) was most timely. It is probably not realized that asbestos is used by some dental surgeons in postoperative packing materials following the operation of gingivectomy. It is very likely that a considerable number of dental nurses are exposed to quite heavy concentrations of powdered asbestos in the preparation of these dressings. I have noticed that some nurses shake the bottle before opening. A white cloud of fine asbestos powder appears immediately the lid is removed.

As there are other dressing materials available which do not contain asbestos and which are just as good there is no justification in continuing to use these preparations.—I am, etc.,

Leeds 16.

JOHN E. DEB. NORMAN.

Chloasma and the "Pill"

SIR,—Dr. D. B. E. Quamina observed (3 June, p. 638) that chloasma is becoming more common as a side-effect of oral contraceptives, especially in Caucasians living in sunny countries. He says that very little reference has been made to this contraceptive chloasma and believes that analysis of a large series may help to determine whether it is the oestrogen or the progestogen in the pill which causes this complication. This presupposes that only one of these hormones is culpable. Oestrogen alone can cause chloasma, even in the male,¹ but I have not come across chloasma caused by progestogen in the absence of oestrogen. It is my opinion that the progestogen acts on the oestrogen-primed melanocytes,² and that chloasma is most likely to be precipitated by sunlight in chloasma-prone patients who have been taking anovulants for a long time, or in high dosage. Thus marked chloasma has been reported from Europe after as little as three months' medication with the large doses of anovulants sometimes used to control gynaecological troubles³; and several surveys have shown that the incidence of chloasma increases with the length of time for which the anovulant has been taken.

From various series already published it

can be concluded that, for a given oestrogen dosage in a "combined pill," the greater the dosage of progestogen the greater the incidence of chloasma. Changing from one brand of anovulant to another would thus be of no help unless there was a reduction of hormone dosage. Chloasma can also be caused by "sequential therapy."⁴

Dr. Quamina, in Trinidad, is in an excellent position to help to unravel the chloasma mystery. The incidence of chloasma among the anovulant-taking women of another Caribbean island (Puerto Rico) is reported to be as high as 34%.⁴ Is there a similar percentage among the natives of Trinidad? I had imagined that the most deeply pigmented races would be chloasma-free, but it has been stated⁵ that chloasma can be seen frequently in the African negro woman if a careful examination is made.—I am, etc.,

RONALD CARRUTHERS.

Department of Dermatology,
General Hospital,
Launceston, Tasmania.

REFERENCES

- ¹ Jadassohn, W., *Arch. Derm.*, 1958, 78, 427.
² Carruthers, R., *Med. J. Aust.*, 1966, 2, 17.
³ Thiers, H., Villedieu, P., and Moulin, G., *Giorn. Ital. Derm.*, 1966, 107, 1335.
⁴ Resnik, S., *J. Amer. Med. Ass.*, 1967, 199, 601.
⁵ Rice-Wray, E., and de Ferrer, S. A., *Sem. Méd. Méx.*, 1966, 50, No. 647.

Antinuclear Factors

SIR,—The comment in your leading article on "Antinuclear Factors" (10 June, p. 654) that "despite the absence of any certain evidence it may be safely concluded that they have some role in the pathogenesis of [connective tissue] disease" is in our view incorrect.

Only 84% of patients suffering from systemic lupus erythematosus, 81% of patients with systemic sclerosis, and 35% of patients with discoid lupus erythematosus have antinuclear antibodies, even in the active phase of the disease.¹ The titres of antinuclear antibodies are not necessarily proportional to the activity, progress, or treatment of disease.² These antibodies are found in several diseases and no antibody is characteristic of any particular disease. They are found in symptomless relatives of patients suffering from connective tissue diseases^{3,4} as well as 4% of control subjects not suffering from connective tissue diseases.¹ Antinuclear antibodies cross the placenta without harming the foetus or newborn child,⁵⁻⁷ and have been transfused into human volunteers without causing disease.⁸⁻¹⁰ Transfusion of serum containing L.E. cell factor does not cause any damage to rats made tolerant to human serum proteins¹¹ nor does it inhibit the growth of cells in tissue culture.¹²⁻¹³ Thus there is no evidence that antinuclear antibodies can initiate tissue damage.

Nevertheless, tests for antinuclear antibodies are valuable in clinical practice. In a recent assessment of the value of one test (the rat liver immunofluorescence test) we¹⁴ have found that the connective tissue diseases can be divided into two groups. The first group includes disorders like systemic and discoid lupus erythematosus and systemic sclerosis in which high titres of antinuclear factor are frequently found, and the second those diseases like polyarteritis nodosa, various types of vasculitis, and dermatomyositis in which antinuclear factor is usually absent. We concluded that (a) the presence

of antinuclear factor in high titre (more than 1 in 64) usually indicates a connective tissue disease such as systemic or discoid lupus erythematosus, systemic sclerosis, Sjögren's syndrome or rheumatoid arthritis, and almost certainly excludes polyarteritis nodosa or dermatomyositis; (b) patients with a titre of 1 in 64 or higher and showing no or few abnormal symptoms or signs should be followed for a long time because they are at risk to develop a connective tissue disease of the first group; and (c) in the absence of any other evidence of connective tissue disorder low titre antinuclear antibodies (1 in 16 or less) can be ignored.

The role of antinuclear antibodies in human disease has still to be determined.—We are, etc.,

N. R. ROWELL.

Skin Department,
The General Infirmary,
Leeds.

J. SWANSON BECK.

University of Aberdeen,
Aberdeen.

REFERENCES

- ¹ Beck, J. S., *Scot. med. J.*, 1963, 8, 373.
² — and Rowell, N. R., *Quart. J. Med.*, 1966, 35, 119.
³ Holborow, J., and Johnson, G. D., *Arthr. and Rheum.*, 1964, 7, 119.
⁴ Fennell, R. H., MacLachlan, M. J., and Rodnan, G. P., *ibid.*, 1962, 5, 296.
⁵ Beck, J. S., and Rowell, N. R., *Lancet*, 1963, 1, 134.
⁶ — Oakley, C. L., and Rowell, N. R., *Arch. Derm.*, 1966, 93, 656.
⁷ Moghtader, R., Kahn, F., Moussy, P., Roussellet, F., and de Séze, S., *Presse méd.*, 1967, 75, 381.
⁸ Benze, G., Cserhati, S., Kovacs, J., and Tiboldi, T., *Ann. rheum. Dis.*, 1958, 17, 426.
⁹ — Kovacs, J., and Cserhati, J., *Brit. med. J.*, 1959, 2, 864.
¹⁰ Marmont, A., Negrini, A., and Damasio, E., *C. R. Soc. Biol. (Paris)*, 1962, 156, 1012.
¹¹ Clark, R. F., Burkhardt, C. R., and Bates, H. R., *Arthr. and Rheum.*, 1963, 6, 573.
¹² Scheffer, E., *Proc. Kon. ned. Akad. Wet.*, 1961, 64, 501.
¹³ Williams, A. M., and Schilling, R. F., *Proc. Soc. exp. Biol. (N.Y.)*, 1961, 107, 302.
¹⁴ Rowell, N. R., and Beck, J. S., *Arch. Derm.*, 1967, in press.

Combined Antifungal and Antibiotic Therapy

SIR,—Professor P. C. Elmes's assertion in *Prescriber's Journal*¹ that "one in five strains of *Monilia* are resistant to nystatin" has been refuted effectively in the pages of the *B.M.J.* by such distinguished and authoritative writers as Professor E. Drouhet (18 March, p. 699), R. Holt and R. L. Newman, G. T. Stewart, and H. I. Winner (1 April, p. 51). We are in full agreement with these previous correspondents, having in 11 years of extensive use of nystatin encountered many therapy-resistant patients, but no resistant strains of *Candida albicans*. Permit us, however, to comment on another aspect of P. C. Elmes's article which has received rather less attention.

Professor Elmes considers that "antifungal agents are only necessary in serious infections or if local lesions persist after the tetracycline is stopped. . . . Combination tablets such as Mystecilin or Lederstatin are therefore unnecessary. . . ." Fleeting reference is made to systemic candidiasis, but the practitioner is left without guidance about the prevention of such dissemination in patients receiving prolonged antibiotic therapy. Combined anticandidal and antibacterial therapy is strongly indicated in patients in whom oral and gastrointestinal candidal reservoirs may become a focus for systemic invasion or widespread mucosal and