difference in this aspect of their behaviour in the body.

Studies by Owen[2] in puppies with all four tetracyclines in common use have shown that although oxytetracycline produces the least degree of discoloration visible in ordinary light the teeth of puppies treated with it show yellow fluorescence in ultra-violet light of similar degree to that produced by tetracycline, although less intense than that produced by demethylchlortetracycline. Tetracyclines are also deposited in the nails, and Douglas,[3] who gave 1 g. daily to volunteers, found that yellow fluorescence of the base of the nail in ultra-violet light was caused by all four within 21 days. He adds, "There appeared to be no difference between the capacity of oxy-, chlor-, and demethylchlor-derivatives and the parent drug to produce this phenomenon."

The implication in the question that the possibility of producing this effect is a contraindication to the use of tetracyclines is disputable. A sufficiently serious indication should surely outweigh this disadvantage.

REFERENCES

1 Wallman, I. S., and Hilton, H. B., *Lancet*, 1962, 1, 827.
2 Owen, L. N., in press.
3 Douglas, A. C., *Brit. J. Dis. Chest*, 1963, 57, 44.

Liver Damage and Myocardial Infiltration

Q.—*Does liver damage predispose to fatty infiltration of the myocardium? I have in mind a patient with a history of four attacks of hepatitis (diagnosed as infective) who died of coronary occlusion associated with fatty myocardium at the age of 47.*

A.—Liver damage does not predispose to fatty infiltration of the myocardium. In fact, patients with liver disease are less liable to atheroma generally. The incidence of myocardial infarction is about a quarter in patients with cirrhosis compared with total cases without this disease.[1]

REFERENCE

1 Howell, W. L., and Manion, W. C., *Amer. Heart J.*, 1960, 60, 341.

Inheritance of Manic-Depressive Insanity

Q.—*Is manic-depressive insanity a hereditary condition? If so, what are the chances of the children of a man who suffers from it inheriting the disease?*

A.—There is evidence to show that heredity plays an important part in manic-depressive insanity. It has been suggested that manic-depressive psychosis is determined by a single autosomal dominant gene of weak and variable expression.[1] This has not been proved conclusively, but it is a theory that fits many of the facts. As to genetical risk, the incidence of manic-depressive psychosis in the offspring of affected parents has been estimated as about 12%.[2] This figure agrees fairly closely with those of other workers. Although it indicates a relatively low incidence of manic-depressive psychosis among the offspring this estimated risk does not cover individuals of cyclothymic temperament, an excess of whom may often be seen in the families of patients with manic-depressive psychosis, and whose temperamental characteristics may be due to a weaker expression of the same gene.

REFERENCES

1 Slater, E., *Proc. roy. Soc. Med.*, 1936, 29, 981.
2 Stenstedt, A., *Acta psychiat. scand.*, 1952, suppl. 79.

Prednisolone and the Foetus

Q.—*Does prednisone taken during pregnancy affect the foetus? Does it cause adrenal hypoplasia?*

A.—Prednisone and other corticosteroids, given in large doses, cause interruption of pregnancy in laboratory rodents. There is no evidence that, given in therapeutic dosage to pregnant women, they have any adverse effects on the pregnancy or the foetus. When corticosteroids are used for the treatment of adrenal hyperplasia the dosage employed is entirely physiological, the drug making up the deficiency in the patient's own inadequate cortisol production. No question, therefore, can arise as to the propriety of administering corticosteroids to these patients during pregnancy. Anxiety is apt to be felt about the pharmacological use of corticosteroids in pregnant women. The indications in these cases are usually severe asthma or collagen disease, and are themselves likely to be a serious threat to the pregnancy. The use of corticosteroids in such circumstances, in minimal effective dosage and with due alertness for possible complications, seems to be quite justified.

Corticosteroid therapy inevitably causes adrenal hypoplasia, and it is therefore important to prevent the occurrence of acute adrenal insufficiency, such as might be precipitated by the stress of labour or some intervening pregnancy complication, by giving generous corticosteroid "cover" at the time of the stress.

Chicken-pox in the Foetus

Q.—*A woman is said to have developed a chicken-pox rash on the day she gave birth to a child, and the child developed a chicken-pox rash the next day. Is this possible? What is the earliest age at which chicken-pox can occur?*

A.—Babies have been born with a chicken-pox rash already present, infection taking place through the placenta. Usually the mother has her rash about a fortnight earlier, the viraemia which accompanies the rash being responsible for the infection of the foetus. The illness then develops in the foetus after an incubation period of around a fortnight. In the case quoted, infection of mother and foetus appears to have been simultaneous. At the time of infection in chicken-pox there probably is, as in smallpox, a transient initial viraemia during which the virus invades the cells of the reticulo-endothelial system, whence it escapes at the end of the incubation period to invade the bloodstream and cause the rash. It would appear that in the present instance infection of the foetus occurred during this initial viraemia.

Follow-up Polio Vaccination

Q.—*Is it advisable for children aged 5 to 12 who completed their poliomyelitis immunization course over two years ago to have a booster dose of oral vaccine now?*

A.—The Standing Medical Advisory Committee on Immunization advised[1] that young children who have had a full course of injections of Salk poliomyelitis vaccine should receive one further dose either of Salk or Sabin vaccine at school-entry age. No advice has been given about subsequent doses except that when poliomyelitis occurs in an area all children in that area should receive a dose of Sabin vaccine, and that those who have not previously been immunized should have at least two follow-up doses to complete their immunization course.

REFERENCE

1 *Brit. med. J.*, 1963, 1, 934.

Hard Water and the Skin

Q.—*My tap-water contains 16.46 parts per 100,000 of mineral matter and 5.59 parts of chlorine/chlorides. The total hardness (Clark's scale) is 2.0 and the temporary hardness 0.1. The pH is 5.6. Is this likely to be the cause of dryness and soreness of the skin of my hands, and what can be done about it?*

A.—The dryness and soreness of the skin described will almost certainly be due to the hardness of the water, the mineral content, and particularly calcium carbonate, acting as irritants. More soap has necessarily to be used and this is a further source of irritation. This can be counteracted by the installation of a domestic softener in which, commonly, sodium zeolite is employed to replace the calcium carbonate by sodium carbonate. A similar result will follow the addition of sodium carbonate crystals (bath salts or washing soda).

Correction.—In our report of the Plenary Session "Drugs in Mental Disorder" at Oxford (July 27, p. 233), Dr. Maurice A. Partridge was reported as expressing disapproval of isocarboxazid ("marplan") on the ground that it produced undue side-effects, and of having a preference for phenelzine ("nardil") in the treatment of depression. Dr. Partridge informs us that he actually said that he preferred isocarboxazid to the other monoamine oxidase inhibitors on the ground that it appeared to be as effective as the others while producing minimal side-effects.