prolonged cooling. There was further opportunity for bacterial multiplication on the following day, when the sliced pork was warmed with hot gravy.

The disproportion between the high number of persons apparently at risk and the number of people affected may be due to chances of distribution of contaminated and non-contaminated joints, or to variations of individual susceptibility as observed by McClung (1945).

The evidence presented emphasizes the danger of consumption of meat dishes prepared the previous day. Ideally, meat should be eaten on the day it is cooked. When this is not practicable provision should be made for rapid cooling after cooking, adequate refrigeration, and speedy serving of the meat after removal from the refrigerator.

Summary

An outbreak of food-poisoning due to heat-resistant *Cl. welchii* is described.

The organism was recovered from roast pork which had been consumed and from the faeces of 18 out of 40 persons affected.

Cultures isolated from the roast pork and from 6 out of 17 stools were of identical serological type. Other evidence, however, suggested that the outbreak was due to a mixed infection with at least two serological types.

The risk involved in the serving of meat dishes prepared the day before their consumption is emphasized.

We are indebted to Dr. Betty Hobbs for carrying out the typing of the cultures isolated from this outbreak. Our thanks are also due to Dr. M. W. Hutchings for help in following up these cases and to Mrs. E. Arnold for valuable technical assistance.

REFERENCES

- Hobbs, B. C., Smith, M. E., Oakley, C. L., Warrack, G. H., and Cruickshank, J. C. (1953). *J. Hyg., Lond.*, **51**, 75.
McClung, L. S. (1945). *J. Bact.*, **50**, 229.
Oesterling, S. (1952). *Nord. hyg. T.*, **33**, 173.

Medical Memoranda

Multiple Mongolism in a Family

Mongolism as a rule occurs only sporadically in family pedigrees, and it was formerly believed that there was never more than one mongol born in a family. This theory usually holds good, as Warner (1949) could collect from the world literature only 42 families in which two siblings were affected, excluding instances when twins were involved.

The birth of three mongoloids in one generation of a family is rarer still, and we have failed to find a previous instance in British literature. The first description of such an occurrence was that of Herrman (1917). Reports by van der Scheer (1919), Fantham (1925), Malkin (1928), Bixby (1940), and Benda (1947) followed. Recently the following came to our notice.

FAMILY HISTORY

The father, a farm cowman, was born in 1905, and the mother, formerly a domestic servant, was born in 1909. They married in 1925, and are known to belong to the dull group of a rural population, the maternal family being of poorer stock than that of the father. There is no consanguinity or history of mental disease in the ascendants. Both parents are virtually feeble-minded and are unable to remember the year of their marriage, the dates of birth of their children, or the years in which three children died. They could recall the sequence of the children's birth only with mutual prompting and with the assistance of one of their daughters. The mother has had five miscarriages,

but is uncertain when two of them occurred. Known miscarriages took place in 1939, 1942, and 1946.

The birth order of the children is as follows: girl, D, 1926; girl, B, 1930; girl, K, 1931; girl, E, 1932; boy, H, 1934; girl, M, 1937; girl, F, 1941; girl, P, 1943; boy, R, 1945; girl, C, 1948. Of these, the girl, P, died at the age of 14 months from tuberculous peritonitis and the boy, R, at 6 months from pneumonia. Apparently no unusual symptoms were experienced during any of the pregnancies.

H is now employed as a cowman, K and B as factory hands in semi-skilled work, and F attends school, having an intelligence quotient of 101. D, who was admitted to a certified institution in 1933, is a mongol imbecile with an intelligence quotient below 30. M was admitted to the same institution in 1941, and is likewise a mongol imbecile, with an intelligence quotient of 30. E was recognized as a typical mongol by the family practitioner shortly after birth and died aged 4½ months, the cause of death being certified as mongolism and bronchitis.

COMMENT

Such families have aroused interest from two aspects. Firstly, it was hoped that their study might lead to an eventual elucidation of the condition, and, secondly, they should be borne in mind when the mother of a mongol seeks advice about whether future pregnancies may result in the birth of further mongol children.

This case well illustrates the contention that hereditary factors indicative of germinal inferiority are evident in the majority of instances of multiple mongolism. It further supports the thesis that in such cases there is an increased tendency to miscarriages; indeed, some of these may be abortions of mongols. It is suggested that if advice is sought by the mother of a mongol child and the family background and medical examination of both parents prove satisfactory, then the "mother can be encouraged to have more children without risk" (Benda, 1947).

Our thanks are due to Dr. T. W. E. Royden, the family practitioner, for his help in the study of this family.

J. V. MORRIS, M.D.

R. C. MACGILLIVRAY, M.B., F.R.F.P.S., D.P.M.

Little Plumstead Hospital, Norwich.

REFERENCES

- Benda, C. E. (1947). *Mongolism and Cretinism*, Heinemann, London.
Bixby, E. M. (1940). *Amer. J. ment. Defic.*, **45**, 201.
Fantham, H. B. (1925). Cited by A. F. Tredgold, *Textbook of Mental Deficiency*, Baillière, Tindall and Cox, 1952. London.
Herrman, C. (1917). *Arch. Pediat.*, **34**, 494.
Malkin, B. (1928). Abstracted in *Bibliog. Eugen.*, **1**, 148.
van der Scheer, W. M. (1919). *Ned. T. Geneesk.*, **1**, 328, cited by T. Halbertsma, *Amer. J. Dis. Child.*, **1923**, **25**, 350.
Warner, R. (1949). *Amer. J. Dis. Child.*, **78**, 573.

Chlorcyclizine Hydrochloride Poisoning in an Infant

Toxic effects with antihistamine drugs are common in adults, but are seldom severe enough to require hospital treatment. Children have frequently been the subject of acute accidental poisoning by these drugs, probably because of the attractive presentation of the pills, and the effects have often been extremely serious. A number of fatal cases have been described. The main clinical feature of them all has been convulsions. The increased liability to fits in epileptics taking diphenhydramine hydrochloride ("benadryl") and tripeleennamine hydrochloride ("pyribenzamine") was shown in E.E.G. studies by Churchill and Gammon (1949). In large doses the antihistamines appear generally to act as central convulsants.

Rives *et al.* (1949), in their fatal case due to methapyrilene hydrochloride, reported the occurrence of cerebral oedema with petechial haemorrhages, which might account for this clinical picture. Their case also showed an acute nephrosis. Tobias (1949) reported the first fatal case in this country. It showed similar clinical features to the present case, but the patient died in three hours. There was no necropsy report. In two cases the necropsy findings were confined

to lung congestion, acute emphysema, and early bronchopneumonia (*British Medical Journal*, 1951). The latter three cases were all due to mepyramine maleate ("anthisan") poisoning, as were the cases reported by Miller and Pedley (1950) and by Prain (1950), in which the findings were essentially the same, with the addition of cerebral congestion. The mepyramine cases all showed a massive catarrhal discharge from the upper respiratory tract. This does not seem to have been seen with other antihistamines.

Davis and Hunt (1949) reported a fatal case due to diphenhydramine poisoning in a child aged 2 years. The clinical picture was again similar, except that respiratory depression occurred and was the eventual cause of death. The necropsy findings were generalized congestion of the lungs, brain, and other organs, with some petechial haemorrhages into the thymus and myocardium.

Various lines of treatment have been suggested, one of them being the use of histamine itself (*Lancet*, 1950). The value of this would seem doubtful, since direct antagonism between the substances in question has never been demonstrated, although it is possible that free antihistamine might be rendered harmless in this way. Gaddum (1948) suggests that antihistamines may act by blocking the histamine receptors, and this is also the view held by White (1950), who states: "Antihistamines do not antagonize histamine by combining with it chemically or by inducing opposite pharmacological responses, as does adrenaline . . . they block histamine receptors in the tissues. It therefore seems doubtful whether histamine can be effective. . . . In some people, notably asthmatics, even though saturated with an antihistamine, it is possible that injections of histamine would produce severe reactions."

The use of various histamine-releasing drugs has also been advocated, but such a view can gain little support from the generally accepted theory of antihistamine action as briefly stated above. Geiringer (1952) states that morphine might well be of value on account of its histamine-releasing property. The danger of morphine in children on account of their peculiar sensitivity to its respiratory depressant action is well known, and would make one extremely reluctant to use it.

Both atropine and adrenaline hasten death from mepyramine poisoning in experimental animals. Overdosage with histamine also hastens death, although small doses appear to be beneficial; the ratio of histamine to antihistamine required for this beneficial effect varies greatly from species to species (Geiringer, 1952). The amount likely to be of value in man is at present unknown. It would therefore seem unjustifiable to administer histamine in cases of antihistamine poisoning, at least on present knowledge.

The following case would seem to be the first serious toxic effect reported due to chlorcyclizine hydrochloride ("histanin"). The treatment adopted was similar to that used in the case reported by Snyderman (1949), who described poisoning by methapyrilene hydrochloride ("thenylene hydrochloride") in a child of 16 months. There was a convulsive stage lasting four hours, controlled by soluble phenobarbitone and chloral. The child had completely recovered after 24 hours. A short period of cardio-respiratory depression was successfully overcome with rectal caffeine.

CASE REPORT

At noon on January 14, 1952, a boy aged 16 months took 18 tablets of chlorcyclizine hydrochloride (50 mg. each, total 900 mg.) which had been prescribed for his mother for nausea of pregnancy. He was almost at once discovered to have done so. At 12.45 p.m. he began to look vacant and was taken to the doctor's surgery, where he vomited. He was sent to the casualty department, where he arrived at 2 p.m. He was then having incessant generalized convulsions and was cyanosed.

His stomach was washed out with sodium bicarbonate solution, and intramuscular paraldehyde, 0.25 ml., was given every ten minutes. At 3 p.m. soluble phenobarbitone, 1 gr. (65 mg.) every 20 minutes, was substituted for the paraldehyde, and atropine, 1/200 gr. (0.32 mg.), was given, as his

secretions were becoming excessive. His condition had improved sufficiently at 4.30 p.m. for him to be transferred to the children's ward. On arrival there at 5.15 p.m. he was slightly cyanosed. There were continuous generalized convulsive movements. Respiration was rapid (55 a minute) but full.

Treatment was continued on similar lines, with both paraldehyde, 1 ml. intramuscularly every 30 minutes, and sodium phenobarbitone, 1 gr. intramuscularly hourly. Prophylactic penicillin, 100,000 units six-hourly, was given. Convulsions ceased at 8.30 p.m. and did not recur. Anti-convulsants were discontinued.

The next day he remained extremely sleepy. His eyes were open and he appeared to watch events without much interest. His blood picture showed: Hb, 110%; white cells 22,000 (polymorphs 58%, eosinophils 2%, lymphocytes 32%, monocytes 8%); red cells appeared normal. His progress over the next three days was slow: the level of consciousness gradually improved, but he appeared very ataxic. There were no localizing signs in the central nervous system. On January 18 he remained still drowsy and his actions were rather automatic. Dexamphetamine sulphate, 2.5 mg. twice daily, was started. This resulted in some speeding up in the process of recovery. On January 23 the patient was sitting up, but very unsteadily. There was no nystagmus. His expression was vacant at times; his intelligence did not appear to be great. Blood picture showed: Hb, 82%; red cells, 4,500,000; C.I., 0.9; white cells, 7,000 (polymorphs 52%, eosinophils 1%, lymphocytes 43%, monocytes 4%).

On January 26 dexamphetamine sulphate was omitted and he was discharged from hospital. He was due to be seen at the out-patient clinic on February 14, but was unable to attend, as he had pulled a pot full of nearly boiling tea over himself a week before and had suffered severe scalds. When seen on February 21 he was perfectly fit and appeared normal in all respects, having completely lost his vacant expression. A further blood picture showed: Hb, 75%; white cells, 22,000 (polymorphs 41%, eosinophils 4%, lymphocytes 51%, monocytes 4%). The raised white blood count was of doubtful significance in view of his recent burns, but no abnormal cells were seen.

There was no albuminuria or other evidence of renal damage at any time.

COMMENT

This was a severe case of poisoning by an antihistamine drug, the main features of which were the prolonged convulsive stage of nearly seven hours, unassociated with any respiratory depression, and the slow recovery, with lethargy and ataxia.

When the child was first seen the most pressing problem seemed to be to stop the convulsions, for it was felt that their continuance would inevitably lead to cerebral damage. Treatment was therefore confined to anticonvulsant drugs.

The child made an uninterrupted recovery despite the use of several drugs which have an adverse effect in experimental animals, and it seems doubtful whether laboratory tests in these animals are applicable to man.

I wish to thank Dr. S. D. V. Weller for permission to publish this report on his patient; Dr. W. E. Budd for assistance in the treatment; and Dr. D. Finlay, of Burroughs Wellcome and Co., for supplying much valuable information.

MICHAEL C. GILL-CAREY, B.M., B.Ch., D.Obst.R.C.O.G.
Late House-physician, Children's Department,
Dartford Hospital Group.

REFERENCES

- British Medical Journal*, 1951, 2, 1530.
Churchill, J. A., and Gammon, G. D. (1949). *J. Amer. med. Ass.*, 141, 18.
Davis, J. H., and Hunt, H. H. (1949). *J. Pediat.*, 34, 358.
Gaddum, J. H. (1948). *British Medical Journal*, 1, 867.
Geiringer, E. (1952). *Ibid.*, 1, 163.
Lancet, 1950, 1, 28.
Miller, A. A., and Pedley, E. (1950). *British Medical Journal*, 1, 1115.
Prain, J. H. (1950). *Ibid.*, 1, 1375.
Rives, H. F., Ward, B. B., and Hicks, M. L. (1949). *J. Amer. med. Ass.*, 140, 1022.
Snyderman, H. S. (1949). *J. Pediat.*, 35, 376.
Tobias, M. (1949). *British Medical Journal*, 1, 1098.
White, J. S. (1950). *Lancet*, 1, 95.