Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Familial Incidence of Mongolism

Q.—*What is known about the mechanism of the inheritance of mongolism? The history of the family with whom I am concerned is as follows:*

Grandmother.—Normal, but with a bad family history of mental and nervous troubles.

First generation.—Two sons and four daughters, the youngest a mongol, the other five normal.

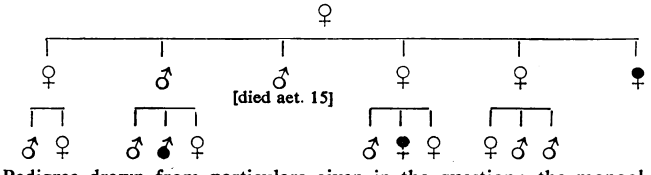
Second generation.—Eldest son: three children, one a mongol. Eldest daughter: two normal children. Second daughter: three children, two normal, one a mongol. Third daughter: three normal children.

The diagnosis in all three cases has been confirmed by experts.

The mothers of the mongol children were respectively aged 44, 40, and 39 when their mongol children were born. The maternal ages at which the unaffected children of the second generation were born were, in order, 35, 37; 38, 43; 34, 42; 31, 33, 38.

What are the probabilities of the normal children of the second generation, when they marry, producing a mongol child? Two mental specialists whom the parents of these children consulted before their marriage told them that mongolism was not inherited—an opinion which does not seem to be borne out by the above facts.

A.—The role of heredity in the production of mongolism is still obscure. A few writers deny that hereditary constitution is involved at all, but this seems too extreme a view. Large collections of data show that amongst the sibs of mongols the occurrence of similar cases is slightly but definitely higher than would be expected by chance, given the incidence of the condition in the general population. Familial incidence, however, is not necessarily a proof of the existence of a genetic mechanism; it might conceivably be due to some non-genetic abnormality in the mother. Much more convincing evidence is the raised incidence amongst more distant relatives of mongols. The practical importance of genetic constitution must be relatively small compared with that of other factors. One could imagine a dominant gene whose possession is necessary before the subject suffers from mongolism, but of the possessors of the gene only a small proportion would actually be mongols.



Pedigree drawn from particulars given in the question: the mongol children are shown in black.

The only other aetiological factor identified with certainty is the age of the mother; the incidence of mongolism rather more than doubles with each advance of five years after the age of 25, and in the oldest age groups may reach quite a high figure.

The pedigree described in the question is highly unusual. The specialists who were consulted were perfectly correct in advising that the risk was negligible that the brothers and sisters of a mongol might themselves have mongol children. The experience in this family does emphasize, however, that it is wise to guard oneself by explaining that the risk, though very small indeed, is nevertheless somewhat greater than for the ordinary person with no close mongol relative. In spite of what has happened the chances for the children of the normal persons of the last generation are probably still good and the risk, though definitely increased as against the chance of 1 in 700 or so that any random pregnancy will end with a mongol child, is likely to be quite small. In all probability it is less likely that one of these persons having a child when the mother is 25 will produce a mongol than that any random woman of 40 or over will do so.

Fibrocystic Disease of the Pancreas

Q.—*The first and third children of a healthy woman have fibrocystic disease of the pancreas. What are the chances of further children being affected?*

A.—The evidence has now become very strong that fibrocystic disease of the pancreas is due to a recessive gene. At all events once the condition has appeared in a sibship the empirical chance of any subsequent child being affected is close to the theoretical figure of one-quarter. Should this mother become pregnant again, the chance is one in four that the child will suffer from the disease.

Prefrontal Leucotomy

Q.—*(a) What are the indications for prefrontal leucotomy, and what is the prognosis? (b) Has the operation been used to alleviate craving in drug addiction?*

A.—*(a)* Prefrontal leucotomy is a symptomatic treatment used in a wide variety of conditions ranging from major forms of insanity, through chronic neurotic illnesses involving tension and distress of mind, to the states of demoralization that can be induced even in normal personalities by intractable pain. It is therefore impossible to state the operative indications succinctly, but (1) the patient should be severely incapacitated before the operation is contemplated, (2) other appropriate treatments, if any, should have been tried, and (3) the best results occur in conditions specially characterized by emotional excess in the form of agitation with tension and distress. The outcome depends on the nature, severity, and duration of the underlying condition, and on the personality of the patient. The better the patient's personality the better is the outcome likely to be. At best after the operation the patient may be a normal person without appreciable handicaps. But where the pre-operative condition has become so fixedly established as to show variation neither spontaneously nor in relation to circumstances, it will persist after operation, though usually in a modified and less distressing form so that the symptoms will be more easily tolerated. It is advisable to seek a second, and preferably expert, opinion before recommending the operation, and in the present state of our knowledge the prognosis should be guarded rather than confident.

(b) Yes, with variable results. The prevailing opinion is that there is some hope of success where the addiction has been more rather than less symptomatic in the sense that it has arisen from attempts to meet undue stress (whether psychic, as in drinking to alleviate depression, or somatic for intractable pain). The less obvious the cause and the less excuse for the addiction, the less good are the operative results. Indeed, where a personality is essentially an unsatisfactory one and the patient has taken to drugs apparently for little reason, the post-operative state is, if anything, liable to be worse, since the post-operative reduction of worry and of restraint is liable to encourage the search for even more immediate gratification of the desires. On the other hand, where drug addiction has arisen more excusably from intolerable pain or even in the hope of quieting a genuine psychic turbulence, the outlook is more hopeful. Quite a number of addicts subject to intolerable pain have been enabled to dispense with drugs altogether after this operation, and so have a few who drifted into the habit through genuine psychic stress.

The Hymen

Q.—*What is the origin of the human hymen?*

A.—The hymen is functional only in female mammals possessing a urogenital sinus (such as the sow), and in these it constitutes a barrier to the flow of urine into the vagina. The human female has no urogenital sinus, but the hymen persists, just as the male nipple persists, because it is part of the hereditary make-up of the mammalian body.

Anoxia and Liver Damage

Q.—*Is it true that the anoxia accompanying severe anaemia can cause liver damage?*

A.—It is important not to confuse anoxic anoxia—i.e., anoxia associated with low oxygen tension—with that due to anaemia, in which the oxygen tension is usually normal. The former cannot be compensated by an increased blood flow, whereas in the latter increased cardiac output may largely make good the deficiency. That anoxic anoxia may lead to liver damage has been undoubtedly established both in men and animals (Buchner, H., *Klin. Wschr.*, 1942, **21**, 721), severe cases resulting in extensive centrilobular necrosis. Uncomplicated anaemia, however, does not generally lead to necrosis: the more usual finding is some degree of fatty infiltration, as, for example, in inadequately treated cases of pernicious anaemia. On the other hand, a degree of circulatory impairment which might be innocuous in a subject with a normal haemoglobin level may well lead to anoxic liver damage in a severely anaemic subject whose liver cells have been dependent for adequate oxygenation upon an increased blood flow. Any increased metabolic activity of the liver in these circumstances may also upset the balance between supply and demand. Such an increased demand may well result from the metabolic disturbance engendered by major surgery and its accompanying anaesthetizing agents.

Vitamin D and Bronchitis

Q.—*An otherwise healthy boy of 9 years living under first-class conditions suffers from recurrent attacks of bronchitis during the winter. Would vitamin D be beneficial and, if so, has an ultra-violet lamp any advantages over direct administration? What, besides burns, are the dangers of such a lamp?*

A.—Scientifically conducted experiments with groups of children in Great Britain have so far failed to show that ultra-violet light externally or vitamin administration has any beneficial effect in reducing the incidence of respiratory tract infections. This does not exclude the possibility of an individual child receiving some benefit, but it makes it unlikely. The main danger of ultra-violet light, apart from burns, is the well-recognized risk of lighting up a tuberculous infection in the lungs—an unlikely event, however, in a boy of 9. There is also the point that the warming of the skin which occurs, if followed by exposure to colder conditions, may be succeeded by the sort of "chilling" which seems to precede some of the respiratory tract infections.

It may be permitted to follow this largely negative answer with some comments on the condition described. There are three possibilities which might be usefully considered: First, where is the infection coming from? If the child is "otherwise healthy" the question of infection from some other member of the household should be investigated. A sinus infection, a throat infection in mother or father with whom the children are living so to speak in symbiosis may spread to a child. Does the term "otherwise healthy" exclude the chance that the child has infection of an antrum, of the tonsils, or even a small area of collapsed and infected lung only to be recognized on *x*-ray examination? Secondly, has the possibility of allergy been excluded? Some cases of recurrent bronchitis in the young are in effect really asthma. Thirdly, it is fair to ask what the "first-class conditions" really mean. Overheated rooms, lack of fresh air, too many clothes, lack of exercise, or absence of precautions to keep warm after exercise may all occur at the best economic levels. Over-protection may result in lack of immunity.

Bleeding Gums

Q.—*An otherwise healthy man suffers from bleeding gums. Every morning his teeth and gums are covered with a brown nasty smelling deposit. What is the best treatment?*

A.—There are numerous forms of gingivitis due to many causes, and any treatment prescribed without investigation of possible causes is bound to be empirical. In general, local scaling and cleaning are indicated to keep the teeth clean and polished, thereby preventing food sticking to them, with finger-massage to restore tone in the gums. If the pockets round the teeth are deep, gingivectomy—i.e., trimming away superfluous and swollen gums—may be undertaken. Local penicillin in the mouth is of use in acute cases, but it is not much good in long-standing cases.

Treatment of Leucopenia

Q.—*A woman aged 55 has been taking "tridione" for several months for petit mal. In four months her total leucocyte count has fallen from 5,000 per c.mm. to 3,500 per c.mm. The tridione has now been stopped. What other measures should be adopted to increase the number of leucocytes to normal?*

A.—It is questionable whether a fall of the total leucocytes from 5,000 per c.mm. to 3,500 per c.mm. is of any significance, unless repeated counts showed the second figure to be constant. The range for 95% of normal individuals is 4,000–11,000 per c.mm.; thus it is possible that the count of 3,500 is a solitary low reading. If it is established that there is persistent leucopenia, tridione should be stopped and the patient kept under observation. The count will almost certainly rise spontaneously after withdrawal of the toxic agent. Folic acid, pyridoxine, and crude liver extract have all been thought at one time or another to stimulate leucopoiesis; it is more than doubtful whether they exert any such action, but they are worth a trial. There is, in fact, no drug known which increases the formation of leucocytes. The most important part of treatment is the immediate control of any infection should evidence of such occur.

NOTES AND COMMENTS

Splenomegaly in Thrombocytopenic Purpura.—Dr. Bernard Myers (London) writes: In your reply ("Any Questions?" September 30, p. 793) you state that "palpable enlargement of the spleen occurs in about one-third of patients with primary or idiopathic thrombocytopenic purpura." In a series of cases which Mr. Rodney Maingot and I had together which from the tests and clinical observation were essential thrombocytopenic purpura, there was not a single case, clinically or at the operation of splenectomy, in which the spleen was in any degree enlarged. I found when I made this statement to medical societies in the United States that the same was the experience of the profession there.

"Hospital Improvements."—This booklet by Miss Olive Matthews, which was referred to in an annotation last week (p. 878), is obtainable only from the author at 22, Harrington Gardens, London, **S.W.7** (1s. 6d., post free).

Correction.—Dr. Byron E. Hall (Rochester, Minnesota, U.S.A.) writes: In the *British Medical Journal* of September 9 (p. 585) a paper bearing the title "Studies on the Nature of the Intrinsic Factor of Castle" was published with my name as sole author. When the manuscript was submitted to Professor Justin Besançon, secretary-general for the First International Congress of Internal Medicine, last July, I was not cognizant of the fact that it would be published, especially prior to the meeting of the Congress. The manuscript was incomplete to the extent that the names of associates who had worked with me on this project and acknowledgments to certain pharmaceutical firms had been omitted. Dr. E. H. Morgan, formerly a fellow in medicine, Mayo Foundation, and Dr. D. C. Campbell, consultant in medicine, Mayo Clinic, should have been co-authors. Moreover, acknowledgments to Merck and Company, Rahway, New Jersey, for supplying crystalline vitamin B_{12}; to the Abbott Laboratories, North Chicago, Illinois, for extracts of hog gastric mucosa; and to the Upjohn Company, Kalamazoo, Michigan, for vitamin B_{12} concentrate and extracts of hog duodenum should have been included.

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