No. 4.—A. G—, female, æt. 32, married. Developed volvulus on 4.4.24, four years after admission; operated on by Mr. Gow. Mental condition: Delusional insanity, sullen, morose and uncommunicative; smiles childishly; says people have spread false rumours about her for years. At times becomes confused and depressed, becoming more simple-minded; laughs to herself and has auditory hallucinations. Threatened return of volvulus on 28.4.25, relieved by enemata. Transferred to Lancaster Mental Hospital on 23.9.25.

No. 5.—H. S—, male, æt. 47, single. Developed volvulus on 20.5.25, two years after admission. Mental condition: Delusional insanity—says he know show to cure all disease and that he has been appointed to the medical staff; he talks to himself and hears imaginary voices. Becomes very depressed at times and complains of his abdomen in a fantastical manner. After operation stitches of wound cut through owing to patient's continual coughing, and a fistula was formed. Patient lived two and a half years.

No. 6.—K. H—, female, æt. 57, married. Has been admitted to mental hospital eight times since 1914. Developed volvulus on 22.10.27, one year and two months after admission. Mental condition: Confusional insanity—very dull and confused, unable to answer simple questions, auditorily hallucinated, disorientated as to time and place; has to be fed, washed and dressed. Operated on by Mr. Gow. Still in Prestwich (1).

No. 7.—G. A. B—, male, æt. 58, single. Developed volvulus, but was not operated on, and died. Patient had been in Prestwich Mental Hospital 4 years and 9 months. Mental condition: He had been in a state of profound dementia since admission, and in poor physical condition.

No. 8.—L. D—, female, æt. 39, single. Developed volvulus five years and five months after admission, but not operated on and died. Mental condition: Delusional insanity with ideas of reference and persecution: accused someone of always interfering with her intestines.

We are much indebted to Dr. Blair, Medical Superintendent, for permission to publish the notes of these cases.

(1) Patient was discharged on February 18, 1928.

The Mongol: A New Explanation.

By R. M. Clark, M.B.Edin., Medical Superintendent, Whittingham Mental Hospital, Lancashire.

It is now nearly sixty years since Langdon-Down first distinguished the mongol, and it must be admitted that to-day the ætiology of this type of idiot is as obscure as ever.

Disease of the thyroid gland is known to cause three of the most distinctive pictures in medicine: (*a*) Myxœdema; (*b*) its congenital form cretinism, caused by hypothyroidism; and in contrast (*c*) exophthalmic goitre, caused by hyperthyroidism. In my view it is possible to add a fourth type, the mongol, which I believe to be a congenital form of exophthalmic goitre. If not, we may ask where is the congenital hyperthyroidic infant?

Very little is known of the effect of hyperthyroidism on the fœtus of the mammal except that it causes death. Striking differences are discernible between the mongol and the product of congenital hypothyroidism, *e.g.*, the cretin has not the abundance of hair, the physical agility, the bright vivacious manner and the lively emotions common to the mongol and to the

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hyperthyroidic individual. On the other hand, few similarities between exophthalmic goitre and mongolism are obvious.

Hyperthyroidism in the adult causes protrusion of the eyeballs, Moebius's sign, throbbing heart and blood-vessels, altered muscle tone, muscle tremor, sympathetico-vascular changes in the skin, hair changes and altered metabolism, and were such a condition to exist in a rapidly developing fœtus and cease at birth—even as a sudden and complete remission may occur in exophthalmic goitre—the resulting infant would, I believe, at birth present the features of mongolism. In support of such a theory, one must bear in mind the responsiveness to environment and altered conditions during growth, especially rapid growth, of all structures, including muscle, tendons, vessels, glands, skin, etc.

Thyroid has a definite action on the development of muscle, and on the tone of voluntary muscle and muscle of the hollow viscera, including stomach, intestines and blood-vessels, and it is conceivable that antenatal hyperthyroidism causes the general muscular hypotonus and explains the pot belly, hernia, over-flexibility of the limbs and low blood-pressure of the mongol.

On a closer examination of this problem on the above lines it seems remarkable and more than a coincidence that in both conditions so many similar points are attacked, even although the results of the attack differ. In exophthalmic goitre one finds eye, heart, blood-vessel, skin and hair changes and altered metabolism, and in the mongol one finds changes in the same structures with stunted growth, resulting from altered metabolism.

A more careful examination of one of these abnormalities, *viz.*, the condition of the eyes—a characteristic feature in both diseases—reveals the reason for the different results. If protrusion of the eyeballs existed during intra-uterine life prior to, during and after the formation of the palpebral fissures, there would result stretching of the coverings of the eyeballs and of the developing ocular muscles and pressure on the eyeballs. At the same time the skin and tissues are more elastic in the vertical than in the horizontal line—corresponding to the line of the less extensile palpebral margins. This would account for such features found in mongols as the thin eyelids, the high arched eyebrows, small eyes and liability to polar cataracts, and the defective and falling out eyelashes. Stretching of the tissues over the bridge of the nose would explain the absence or ill-development of the nasal bones with flattening of the bridge of the nose, and the stretch and pull from this region would account for the short squat nose, while the retraction of the point of the nose would explain why the nostrils look forward and are narrowed. The increased intra-orbital pressure would

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explain the thinning of the bones lining the orbital cavities, and if protrusion of the eyes ceased at birth—as it ceases in a remission in exophthalmic goitre—the resulting slackness would account for epicanthus, strabismus and rolling movements of the eyes. The stretched eyelids would ill fit the eyeballs, germs and dirt would be harboured, and cause the blepharitic and other inflammatory troubles so common in mongols. In later years growth would take up the slack; strabismus and epicanthus would gradually improve, as they do when the mongol grows up. It is possible, too, that the slanting eye so characteristic of the mongol may be explained by antenatal hyperthyroidism causing eyeball protrusion and contraction of Landstrom's muscle in the fœtus. It appears to me, then, that hyperthyroidism has been at work on the fœtus that becomes a mongol.

It is agreed that the mongol presents other characteristic features: it is undersized and of stunted growth, it shows resemblances to former ancestors, it is an " unfinished child," and its mental development shows a parallelism with its physical development. These characters are the result of changes in the eyes, the hands and the feet, the vertebral column, skull and brain—those parts, indeed, which are concerned respectively with stereoscopic vision, with skill, increasing tactile sensibility and discrimination in the hands, and the erect posture, all of which, Elliot Smith has shown, have played the recent and all-important part in the great increase in the growth of the neopallium and in the evolution of the brain. In the mongol ontogeny has stopped short at a late stage in the life-history of the human fœtus, and yet development has gone on to maturity and viability.

The cause of mongolism, then, should account for the features just mentioned, and the following experiments show that hyperthyroidism can account for them: (1) Thyroid fed to tadpoles causes protrusion of the eyes. It also causes metamorphosis to occur prematurely, so that it is possible to obtain diminutive frogs only one-tenth the length of normal frogs. (2) Thyroid fed to axolotls increases metabolism by 35%, body-weight decreases and skeletal changes occur. (3) Thyroid fed to pregnant bitches, rabbits and guinea-pigs causes injury and death of the fœtus. (4) Thyroid accelerates maturing of spermatoblasts, and in larger doses causes degeneration and death, with fatty changes and increase in cholesterin. In consideration of the above facts, I submit the theory that hyperthyroidism has a similar action on the human fœtus to that which it has on the tadpole. The effect may (as tissue culture experiments suggest) result from increased metabolism with more active cell division preventing differentiation—in this case,

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the latest parts to differentiate suffering. It brings the human fœtus to precocious maturity—light in weight, short in stature, stunted in growth and often premature—like the diminutive frogs. As with the tadpoles, the latter part of ontogeny has been cut out and the fœtus brought to maturity when it is recapitulating a late stage in its ancestral history—an "unfinished child" with its "likenesses," "retrogressive features" and anthropological explanations. In fact such an infant would be in accord with what is known of the mongol, including the frequency of congenital heart affections and of cryptorchidism.

Fœtal hyperthyroidism might account for the defective or deficient thyroid glands sometimes found in mongols, even as the thyroids of thyroid-fed tadpoles are smaller than in controls. So the mongol's neck may show no signs resulting from a swollen fœtal thyroid, and we have an explanation for the myxœdematous manifestations occasionally met with in mongol children and mongoloid adults. Hypothyroidic signs are more frequently found in mongoloid adults than in mongol adults, the reason probably being that, as in the tadpole, the relative sizes of the thyroid and the whole body determine thyroid balance, and so the mongol's thyroid is sufficient for its small and stunted body, but the mongoloid's thyroid is insufficient for its normal-sized body. My view is that hyperthyroidism, affecting the fœtus *in utero*, is the cause of mongolism. The hyperthyroidism may be maternal or fœtal in origin, and I take it to include over-susceptibility of the embryo to thyroid hormone and over-elaboration of thyroid hormone, owing to the capacity of other tissues of the body to produce it from iodine or other substances.

The two explanations given for the eye condition, (*a*) eye protrusion in the fœtus, and (*b*) the arrest of evolution—are conflicting only on the surface. In hyperthyroidism in the fœtus, protrusion of the eye occurs as it does in the tadpole, and this complicates the changes in that region in the mongol. A point in favour of this theory is the observation that many mothers, during the war, attributed the birth of mongols to fright during air-raids. A further factor present in some cases may be the deprivation of light, for a higher percentage of mongols is said to occur in the population of large fog-screened cities. Hence light treatment, which is beneficial in exophthalmic goitre, may possibly be of value in the prevention of mongolism.

The theory here postulated may apply to negroid and other types of imbeciles, for in Graves' disease exophthalmos does not always occur. It raises interesting questions, *e.g.*, evolution has already brought round the eyes from the side of the head, as in the fish,

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to the front position in man, thus attaining recently acquired stereoscopic vision. Has not this change been brought about by diminishing action of thyroid on the fœtus and weakening of the pull on Landstrom's muscle, which, in adult hyperthyroidism, gives rise to Mœbius's sign? Myopia is widely prevalent amongst the human species, and recently has been shown to be caused by the slight divergence of the eyes. The inner muscles of the eyes in trying to correct this are subjected to strain which produces myopia. Many cases of myopia are now being reduced and many others have been entirely cured by the use of prismatic glasses, which restore the parallelism, and allow the eyes to remain in their natural slightly divergent position.

May not this divergence of the eyes in myopia be due to an incompleteness of this most recent attainment of evolution, and result from antenatal hyperthyroidism causing contraction of Landstrom's muscle in the fœtus?

Other conditions as well as mongolism, *e.g.*, fœtal monstrosities, have by some authorities been attributed to endocrine disturbances, by others to "reversion." It is more likely that a theory which incorporates both views is the correct one, and if thyroid acts on the human fœtus, when ontogeny is at the gill stage, in a similar way to its action on the gill stage of the frog, monstrosities can easily be explained. In view of the fatty and cholesterin changes brought about in spermatoblasts by thyroid it is possible that hydatid mole is an analogous condition. The problem of the mongol and the thyroid is wrapt up with the problem of evolution and of gland balance. Positive proof of my view seems as yet impossible, and no excuse is required for the speculative nature of the views expressed in this paper, which, if not immediately acceptable, will be of use if they call attention to the application to general medicine of some of the findings of experimental embryology, tissue culture and thyroid feeding.

Bacterial Change in Mental Disorder: Preliminary Note on Morgan's Bacillus. By F. H. STEWART, M.A., D.Sc., M.D., D.P.M., Major, Indian Medical Service (retired), Cheddleton Mental Hospital.

IN a previous communication (*) it has been shown that among intestinal bacteria of the coliform group *Bacillus paracoli* and *Bacterium coli mutabile* are at least four times more common in the insane than in the general community, and that they are to be

* *Vide* "Bacterial Changes in Mental Disorder: Coliform Group," by the same author, *Journ. Ment. Sci.*, 1928, p. 59.

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