given every four hours. He was kept on boiled water with the occasional addition of a little brandy.

The two following days saw him steadily going downhill; there were no physical signs in chest or abdomen, no cough, no vomiting; the bowels were opened two or three times daily, and the motion, although not offensive, looked like cooked spinach. On chest and abdomen were a few patches of erythema, which I took to be "sweat rash," and there was a ring of erythema round the umbilicus. He was passing urine normally, and it showed nothing unusual on examination. His temperature ranged from 102° to 103°, pulse rate 140, respiration rate 30 to 40. There were photophobia and conjunctival injection, and his tongue and lips were dry and sore. He was completely hypotonic and the tendon reflexes were absent. The tips of his fingers and toes were now slightly reddened, swollen, and painful to touch, and owing to general weakness and the condition of the mouth he began to refuse fluids, which up to this time he had taken in large quantities. These had consisted of secway, boiled water, brandy and water, and a little orange juice.

On July 25th he was semi-comatose with a temperature of 103°. His fingers and toes and the greater part of his hands and feet were bright red, swollen, and acutely tender. They looked as if the boy had been playing with a wet coloured toy and the dye had come off on to his hands. His motions were still like cooked spinach, not too frequent and not offensive, and, more with the idea of increasing his fluid intake than washing away any poisonous bowel contents, I commenced giving him regular large bowel irrigations with normal saline, leaving a few ounces in the colon each time. He was also given an ounce or two of normal saline with a little brandy by the rectum at regular intervals, and as much fluid by the mouth as possible. All other medication was stopped.

On July 26th and 27th this treatment was continued, and a slight but definite improvement was noted. He was having up to 2 oz. of brandy daily and as much fluid as possible. He had wasted rapidly in spite of fluid intake, and was and looked very ill, taking no interest in his surroundings and occasionally making a feeble cry and holding up his hands for treatment. They were being wiped with spirit and dusted with powder. His temperature still ranged between 102° and 103° and his mouth required constant attention.

On July 28th he was much better; his temperature was 101°, his hands and feet were less red and painful, and the motions a better colour. He began to take more by the mouth again, and Valentine's meat juice and albumin water were added to the diet. The next day his temperature was 99.5°, his motions were formed and a greenish-yellow colour, and as he was taking plenty of fluid by the mouth the rectal washouts and instillations were stopped, and a dilute Nestlé's milk mixture was given in addition.

From this time he improved daily, his temperature fell to normal, his diet was gradually increased, and his hands and feet became less red, swollen, and painful, and commenced to peel in large patches, complete casts of some of the fingers being shed in one piece.

In another week he was up in a chair, and a few days later out in a perambulator. His appetite was enormous; he constantly called for more food, and as his tongue was clean and his bowels normal he was indulged within reason. When last seen, three weeks after the commencement of the acute stage of the illness, he was seemingly quite well except for the wasted and flabby condition of the leg muscles. He had no motor or sensory paralysis, but the tendon reflexes were still absent, and although he was pronounced fit to return home his parents were warned against a too rapid convalescence, with special reference to the use of his legs.

The features of the case which impressed me most were, first, the prodromal history, which made me suspect meningitis; then the symptoms suggestive of early pneumonia which did not develop, and later the complete hypotonic state, and the striking condition of the extremities, which is a marked feature of the clinical picture.

Except for the rise in temperature and the acuteness of the illness this case seems to have been fairly typical, and leads me to suggest that the origin of the toxæmia causing the "polyneuritis" is intestinal.

REFERENCE.

¹ *Medical Annual*, 1924.

A SERIES OF CONSECUTIVE OPERATIONS ON THE MASTOID.

BY

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DURING the past eight years I have performed close on 400 mastoid operations; those recorded here have been performed during the last three years and nine months, and comprise both private and hospital patients. I am greatly indebted to Mr. Bolton, the house-surgeon, and Mr. Lynch, the acting house-surgeon, for extracting and tabulating the cases in the hospital record—a somewhat difficult task. Any case of otorrhoea of over two months' duration has been considered chronic.

The operations may be classified as follows: (1) for primary acute mastoiditis, in which the mastoid and the otorrhoea are simultaneous, or the otorrhoea has lasted for a few days only; (2) for acute secondary mastoiditis, an acute condition grafted on to chronic otorrhoea; (3) for chronic otorrhoea, performed for the cure of the discharge and to improve hearing and relieve vertigo. The type of operation adopted for the acute uncomplicated cases was a modification of the Schwartz operation.

In my early cases after the ordinary Schwartz operation there was a sagging inward of the posterior conchal portion of the membranous meatus. This caused a blurring in hearing power; to overcome this I remove a fair amount of the posterior bony wall and place a suture in such a position as to make traction. Unless the tissues are very much damaged in the acute cases the wound is partially sutured and the antrum drained by gauze for twenty-four to forty-eight hours. Opinion at the Annual Meeting of the British Medical Association at Nottingham was opposed to this and favoured the open granulation method. I am unrepentant. It sometimes happens that an acute case is so severe that a more radical operation than the modified Schwartz has to be done. In such a case a flap is made in the membranous meatus and the whole of the posterior bony wall is removed. In the chronic cases the modified radical operation was invariably done unless there was much destruction of the tympanic membrane; the complete radical operation was then performed. In the radical operation the posterior wall of the tympanum was never touched.

The modified radical operation, which is based on Bondy's operation, consists in the complete removal of the posterior wall and roof of the external meatus and attic. It is tedious but well worth while. The aim of the conservative operation is not only to cure the discharge but to improve hearing. This is invariably attained; with a large resonance chamber and ossicles and membrane untouched the hearing power should be improved. After the acute operation healing was usually complete in a fortnight; after the modified acute operation healing usually took a month; after the modified radical operation epidermization in my recent cases, blessed by good nursing, has been extraordinarily rapid—frequently a month, sometimes six or eight weeks. Any underlying constitutional disability, such as syphilis or tuberculosis, delays healing, as does eczema occasionally.

Among the acute primary cases there were one cerebral abscess, two lateral sinus infections with thrombosis, and two without. In the acute secondary mastoid cases there were three instances of cerebral abscesses, eight of lateral sinus infection with thrombosis, and three without. Among the acute cases was one septic pneumonia and one diagnosed as pulmonary embolism. The age varied from 1 year and 2 months to 67 years.

No case of labyrinthitis occurred in this series. Some of the cases had labyrinthine symptoms, which cleared up after the mastoid operations. All cases of serous meningitis recovered.

There was one case of secondary acute mastoiditis in a patient aged 17, admitted with definite signs of cerebral pressure—that is, a high temperature (103°), pulse 56, and marked mental hebetude and vomiting. Mastoid signs were not typical, but the mastoid operation was decided upon. On opening up the antrum pus came away under pressure. There was a considerable destruction of bone, and the dura was exposed. There was a large cholesteatoma occupying the antrum and middle ear. I trephined over the temporo-sphenoidal lobe. There was no pulsation of the membrane; the dura was carefully opened, and pus sought for in vain. Next day the symptoms were better, the mind was clearer, and the temperature was down, but the pulse was slower. There was no papilloedema, and no homolateral hemianopia. I asked him how he was. He said, "Better, nurse." "Have you got any headache?" He answered, "No, nurse." I said, "I'm not the nurse, I'm the doctor." He replied, "Yes, nurse." This I took to be due to my having explored the anterior part of his temporo-sphenoidal lobe (Ballance). The following day his pulse was 46. His answers were normal. There was induced nystagmus in the left eye—the operated side—of the cerebellar type. I therefore decided to trephine over the posterior fossa, and an inch and a half of bone was removed above the lateral sinus. There was no bulging and no pulsation. The dura was incised, the brain exposed, and the venules were seen to be engorged but silent. Pus was sought for in vain.

After this second operation he made a complete recovery, and left the hospital with quite good hearing

power. This seems to me to be one of those cases of encephalitis so well described by Symons.

Furuncle is mentioned, not as being a complication of acute mastoid disease, but for the opportunity of making this assertion—that in all cases of long-standing otorrhoea in which a furuncle large enough to close or partially close the meatus occurs a radical mastoid operation should be done, especially if the furuncle be on the posterior wall of the meatus. In each of three recent cases I was fully justified in this line of action. In many of these cases there was a cholesteatoma, and this might mean erosion of bone; in one case the pus tracked backwards between the skull and the dura to make its exit by a hole in the skull in the sub-occipital region, burrowing its way beneath the pericranium into the soft tissues of the neck, where I ran it to ground, after freely removing the bone from the mastoid to the secondary hole in the skull. The dura was covered with granulations, as was the lateral sinus, and at the completion of the operation the lateral sinus burst. This man has so far made slow progress. His age is 47, and he has well marked emphysema of both lungs. The lateral sinus was never opened and the jugular never tied unless there were definite signs of phlebitis, when the jugular was tied high up in the neck and the lateral sinus exposed; if thrombosis was present it was dealt with in the usual way. If there had been signs of sinus infection, such as rigors without definite signs of phlebitis, the sinus was not opened, but if there were definite signs of phlebitis with or without thrombosis the sinus was opened, allowed to bleed, plugged, and saline given. In a boy with marked lateral sinus thrombosis, necessitating wide excision of bone for removal of a septic clot which extended very near to the torcular, a complete recovery resulted with good hearing power. In all the acute cases, primary or secondary uncomplicated, there were no deaths. Skin grafting was never done.

THE ETIOLOGY OF LEUKAEMIA.

BY

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THE key to the etiology and nature of leukaemia is presumably to be found in the study of those factors which control the leucocyte content of the blood. While the line of investigation necessary is at present rather obscure, the constant recording of cases, especially those which do not strictly fall into any standard classification, may throw some light on the main causative factors. For this reason I have analysed the histories of twenty-five cases of leukaemia observed during the last six years.

The series consisted of 11 patients with myeloid leukaemia, of whom 7 were males and 4 females, and 8 cases of acute and chronic lymphatic leukaemia, 5 in males and 3 in females. The remaining 6 cases were atypical. The red blood count was below 1,000,000 in 37 per cent. of the series.

Myelogenous Series.

In the myelogenous series the total number of leucocytes ranged from 32,000 to 954,000 per c.mm. and the lymphocyte proportion from 0.8 to 40 per cent. In the case with the most marked lymphocytosis the polymorphonuclears and myelocytes were both 29.6 per cent. and the lymphocytes later fell to 1 per cent. The polymorphonuclear count was never lower than 29 per cent. and was usually above 40 per cent. This latter point may have something to do with the better prognosis of myelogenous leukaemia as compared with the non-granular form, where the polymorphonuclear count was never higher than 18 per cent. and ranged from 3.7 to 18 per cent.

Bad teeth or pyorrhoea occurred in 7 cases (64 per cent.), night sweats in 3 (27 per cent.), and a chronic infective septic focus of some kind was present in 82 per cent. of the cases.

There was nothing in any of the family histories to support the possibility of contagion from other patients. The duration of the disease varied from two to eight years.

The spleen was enlarged in all cases, and other lymphatic glands in 2 cases. The temperature usually ranged between the normal and 100°; in 3 cases there was no pyrexia and in one case the temperature reached 102°. Out of the 11 cases 7 were discharged as "better."

Non-Granular Series.

In the non-granular series, out of 9 cases 4 were chronic, 5 acute. The total leucocytes ranged from 4,800 to 928,000, but in one atypical case the figure was as low as 600. The lymphocytic percentage varied from 74 to 96. In all cases except one it was 80 per cent. or over.

Pyorrhoea or some chronic primary focus of infection was present in the histories of 6 cases (66 per cent.). Night sweats were recorded only in one case. The length of history was from one to seven months in the acute and five to eighteen months in the chronic forms. There was nothing in any family history to support contagion with any other case. The spleen and other lymphoid glands were enlarged in all cases except two; in one of these there was no splenic enlargement and in the other the spleen only was enlarged. There was usually no pyrexia in the chronic cases; the temperature rose as high as 105° in the acute. Two patients were discharged—these were of the chronic type; the remainder died.

Etiological Theories.

Of theories as to the causation of this disease only two need be considered in the light of present knowledge.

1. *The Tumour Theory.*

This theory is the most popular one at the present time. It is based mainly on certain changes which appear to be analogous to tumour formation, such as the excessive and apparently purposeless proliferation of leucocytes, and their tendency to metastasize. Several factors, however, indicate that if the nature of the agency instigating the condition is similar to that of tumour formation, it behaves quite differently from any other kind of malignant lesion. So far as is known, in a necropsy of a case of leukaemia, no site can be indicated as the possible primary focus.

The changes in the bone marrow and reticulo-endothelial system are more or less uniform, but certain areas may tend to be more dense than others. Patches of leucocytic deposit or gross infiltration will be relatively more marked in places, but these differ microscopically from centres of malignant proliferation. "The newly formed cells are not in possession of resolving power (of fermentative nature?) which proper tumour cells possess, and by which they entirely destroy the organs attached."¹ This is a very essential difference; it practically amounts to the difference between a benign leucocytic infiltration and an invasion by malignant cells.

Patients, moreover, have been known to survive for ten years or longer, and the condition may become entirely arrested for as long as seven years (Price). These are points hardly compatible with our knowledge of carcinomatosis and sarcomatosis.

2. *The Infective Theory.*

In the discussion on leukaemia and allied conditions at the Annual Meeting of the British Medical Association at Nottingham, Dr. R. Donaldson, after surveying some of the arguments against the sarcomatosis theory, concluded that there was more to be said for a possible virus or irritant, using the latter in its broad sense.² This certainly appears to be the most plausible etiological theory, and a survey of the present series of cases would seem to bring out points in its favour. Early investigators such as Virchow held this view and maintained that leukaemia was the result of specific infection. Others believe that it is non-specific in origin. In the histories of the above cases, 17 showed varying degrees of irregular temperature; in all the acute and atypical varieties the temperature was 103° or higher, but the chronic lymphatic patients had no rise at all.

There was a history of pyorrhoea, tonsillitis, sore throat, bronchitis, or some chronic infective focus in 82 per cent. of the classified cases and in all the atypical ones. The