displaced from favour by the far more effective procedure of myringotomy; and, secondly, "every general practitioner" does not possess the experience and nicety of judgment which alone can decide whether myringotomy still has a reasonable chance of averting an acute mastoid or whether things have already gone too far for anything short of immediate mastoidectomy to have a hope of relieving the condition and possibly preventing a catastrophe.

Case I

A gunner aged 22 was admitted to a military hospital on January 17, 1941, with a history of left otalgia followed by otorrhoea. Temperature 99.4°, pulse 80. "Exacerbation of an old C.O.M.S.(L)" was diagnosed. Next day temperature tended to swing between 100.6° and 97.4°. On the 19th it settled to normal, and he was given guttae glyc. and phenol, fomentations, s.v.r. drops, and mist. aspirin. He was discharged to a convalescent home on the 25th, with normal temperature but profuse otorrhoea. From January 28 to 31 sulphapyridine, 1 gramme four-hourly, was given. Temperature was very slightly irregular above and below normal. He was transferred to a convalescent depot on February 7.

On February 10, when referred to me, there was thin somewhat offensive left otorrhoea, slight deafness in the left ear, medium-sized postero-inferior perforation, but no vertigo or tinnitus. He was returned to the convalescent depot with the recommendation that he be kept under close observation and that dry mopping and insufflation of 1% iodoboric powder be carried out. On the 13th he was readmitted to hospital with general malaise and severe pain behind the left ear. Otorrhoea was profuse and the left ear was pushed somewhat forward; there was no mastoid tenderness, but the patient looked ill. Subacute left mastoiditis was diagnosed. The next day left epitympano-mastoidectomy was performed. Very extensive involvement of the mastoid region was found. The bone was of mixed cellular and sclerotic type. Aditus, attic, antrum, and cells contained pus under some pressure. Healing by first intention was secured, and recovery was uneventful; the otorrhoea soon dried up, and there was very little loss of hearing.

Case II

A lance-corporal aged 21 was referred to me at a military hospital on January 21, 1941, with a history of having first noticed left-sided otorrhoea while on leave. There was slight deafness, but no pain; trismus, said to date from the extraction of a left lower wisdom tooth, was present. He stated that he had been treated by a civilian doctor and given, among other things, tablets of sulphapyridine, but no details as to dosage, etc., were available. He looked ill. There was a moderate amount of thin non-offensive otorrhoea, left cervical adenitis, and medium-sized postero-inferior perforation, but no mastoid tenderness.

He was admitted and kept under close observation, with dry mopping of the otorrhoea, counter-irritation over the cervical glands, gargles, and mentholization. During the next few days the trismus improved, but the temperature showed a slight tendency to swing between 98° and 99°. In the afternoon of January 26 there was a sudden exacerbation, with malaise, swelling of the left parotid region, slight left facial paresis, and gradual filling up of a fluctuating swelling on the posterior aspect of the lobule of the left ear. On the 27th he had a definite toxic appearance; the above-mentioned swellings had increased and the left facial paresis had deepened. Temperature normal; pulse 98. Left epitympano-mastoidectomy was performed. There was generalized osteomyelitis in an acellular mastoid with a perforation near the tip, "tracking" into the lobule of the ear and into the parotid region. This was thoroughly cleared and the wound packed open, secondary suture being performed on February 5. The wound then healed by first intention, the facial paresis recovered completely, the otorrhoea dried up, and the perforation in the drum healed. The hearing is good.

Case III

A second-lieutenant aged 28 was admitted to a hospital on February 18, 1941, with severe otalgia in the left ear of sudden onset and a history of acute left otitis media two years previously. Slight pulsatile left otorrhoea was present, but no

mastoid tenderness. Sulphapyridine and a spirit-and-flavine wick were used, and on March 11 he was given a week's sick leave, the ear being then dry and all symptoms relieved. On March 15 diplopia developed, and on the 18th he consulted the eye specialist, who referred him to me. He gave the following history: For some days he had had fairly severe neuralgic pain centred somewhat vaguely between the left ear and the left eye, which had gradually changed to a dull ache behind the left eye; then on the 15th, as stated above, he had for the first time noticed the diplopia. On examination I found complete paralysis of the left external rectus; in other words, he presented the complete picture of the Gradenigo symptom-complex, indicating the presence of petrositis. A small perforation was found in the anterior portion of Shrapnell's membrane, from which a thin trickle of pus was escaping. Radiographs showed considerable relative radio-opacity of the left mastoid region and the petrous pyramid, with loss of the cellular outlines.

A left Schwartze mastoidectomy was performed on March 21. There was widespread involvement of the mastoid cells, which contained pus, while the mucosa was thickened. Two polypi were impacted in the aditus, and on their removal pus under considerable pressure escaped from the attic region and the middle ear. Healing took place by first intention, and convalescence was uneventful. All symptoms have been removed, while with the assistance of electrical stimulation of the paralysed muscle complete recovery from the paralysis has been secured and the diplopia has disappeared. The perforation in the drum has healed and the hearing seems to be normal.

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Medical Memoranda

Intestinal Haemorrhage as a Complication of Sulphonamide Therapy

The recent occurrence of melaena in two children under treatment with sulphonamide drugs stimulated an inquiry into the recorded incidence of this complication. The scanty references in the literature and the absence of records of its occurrence in this country make these notes and observations of value.

CASE REPORTS.

Case 1.—A boy aged 5 was admitted to the Southport Infirmary on January 23, 1941, with a history of severe vomiting of two days' duration. Lumbar puncture produced 4 c.cm. of turbid fluid from which meningococci were later grown on culture. Next day he was given 0.5 gramme of sulphapyridine soluble intramuscularly and transferred to the borough isolation hospital. On admission he was drowsy and irritable, and the temperature was 98.4° F. He did not appear to be very ill. Soluseptasine 0.5 gramme intramuscularly was ordered four-hourly. Next day after five injections his temperature had remained normal, but head retraction and opisthotonos were pronounced. lumbar puncture produced no fluid, and cisternal puncture only 2 c.cm. of opalescent fluid. Sulphapyridine soluble 0.5 gramme intramuscularly was ordered three-hourly. On January 27 the boy was much improved and took 0.5 gramme of sulphapyridine by mouth four times daily for four days. On January 31 he appeared normal, and received three 0.5-gramme doses of sulphapyridine, and on February 1 two doses of 0.5 gramme each. In the night of February 1 he passed a small tarry motion, and on February 2 two large melaena stools. He was very pale, but his condition did not give rise to anxiety. Parenteral salines were given. As we were unable to account for the melaena a blood film was made late at night and suggested a diagnosis of myeloid leukaemia. The temperature was 99° F., the spleen was not palpable or enlarged to percussion, the liver was not enlarged, no enlarged glands could be felt, no petechiae were seen, the urine was normal, and there was no evidence of jaundice. The blood film, however, showed a considerable increase of white cells, estimated at more than 100,000, of which 92% were polymorphs, 6% myelocytes, and 2% lymphocytes. The red cells were rather pale, and showed anisocytosis; 2% nucleated red

cells were found. Unfortunately the cell counts were deferred until next day and the child collapsed and died suddenly during the night.

Case 2.—A girl aged 8 was admitted to the Southport Infirmary on February 12, 1941, with a history of headache and vomiting of a few hours' duration. Lumbar puncture produced 6 c.cm. of opalescent fluid from which meningococci were later grown on culture. The child was admitted to the borough isolation hospital the next day, after receiving an intramuscular injection of 1 gramme of sulphapyridine soluble and 1 gramme of sulphathiazole by mouth. On admission she was very listless and semicomatose, temperature 100.4° F. Sulphathiazole 1.5 grammes was ordered four-hourly. The temperature became normal the next day and did not rise above 99° F. thereafter. On February 14, following difficulty in swallowing, 1 gramme of sulphapyridine soluble was given intramuscularly four-hourly, followed by sulphathiazole 0.75 gramme four-hourly for three days. On February 18 the child did not appear well and seemed more comatose. Seven grammes of sulphathiazole was given in the next twenty-four hours, when it was finally stopped. On February 20 she passed a small melaena stool, but remained well and did not complain. A blood film appeared normal and a white cell count totalled 14,200. On February 21 another final melaena stool was passed, and the child thereafter progressed satisfactorily until discharge on March 10.

DISCUSSION

In neither child was there a history of a tendency to bleed and the family history in each case was negative. In Case 1, apart from the abnormal findings in the blood film, there were no clinical signs to suggest that the child was suffering from myeloid leukaemia, but the melaena was originally attributed to this possibility in the absence of any more feasible explanation. The occurrence of melaena in Case 2 gave reason to the consideration that the bleeding in Case 1 had been due in large part at least to the sulphonamide drugs.

Long and his associates (1940) report the rare occurrence of hyperleucocytosis during sulphanilamide and sulphapyridine therapy, generally in the presence of acute haemolytic anaemia, of which there was no evidence in this case. They also report that unexplained bleeding from the gastro-intestinal tract has been seen in one or two cases of sulphanilamide and sulphapyridine therapy, and that one patient who received sulphapyridine had bleeding severe enough to cause death. They record no instance of gastro-intestinal bleeding following sulphathiazole. Blake recorded two instances of gross intestinal haemorrhage in patients receiving sulphanilamide, in neither of whom was there any other plausible explanation for the unward reaction.

These two records of melaena occurring among a comparatively small number of patients receiving sulphonamide drugs may stimulate the interest of others with greater experience and better facilities for investigation and lead to a better knowledge of its true incidence.

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H. D. Wright (*J. Path. Bact.*, 1941, **52**, 237), in a record of the three types of *C. diphtheriae* in 8,040 cases of diphtheria in Liverpool during 1937-40, states that this period was marked by a notable increase in the number and proportion of *gravis* cases. There was a slight decrease in the proportion of cases in the age period 5-9 years. The difference in age incidence between the different types was slight. The case fatality was highest in the *intermedius* infections, particularly among the older children, and lowest in the *mitis* group. The greatest number of deaths were due to the *gravis* type. Deaths from toxæmia predominated in the *intermedius* and *gravis* infections and from pneumonia or respiratory involvement in the *mitis* infection.

Reviews

HARDY ANNUAL

The Medical Annual, 1941. A Year Book of Treatment and Practitioner's Index. Fifty-ninth year. Editors: H. Letheby Tidy, M.D., F.R.C.P., and A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S. (Pp. 516; 39 plates, plain and coloured. 23s. net.) Bristol: John Wright and Sons, Ltd.; London: Simpkin Marshall, Ltd.

This valuable work, as its devotees know, is published by Messrs. John Wright at Bristol, a town that has borne the full brunt of enemy attacks from the air. The publishers and the editors have not, however, been deterred by what must have been considerable difficulties, and both are to be congratulated on maintaining the standard of the familiar green-covered volume. There is one small criticism that we would make. In a yearbook that is primarily meant for the general medical reader we doubt whether there is much point in including special plates illustrating, for example, the prevention of elephantiasis chirurgica, seminoma of the testis, an operation for persistent thyroglossal duct. The illustrations are, as usual, very well reproduced, and there are a number of excellent line drawings in the text.

It is impossible to refer in detail to the numerous items, arranged in encyclopaedic form from A to Z. The article on vaginal thrush is accompanied by two instructive half-tone illustrations; a long and helpful article on electroencephalography is contributed by Mr. Grey Walter and Prof. F. L. Golla; the two editors—Dr. Letheby Tidy and Prof. Rendle Short—contribute articles on the medical and surgical aspects respectively of abdominal disorders; Prof. R. M. F. Picken describes schistosome dermatitis, which so far has been found to occur in one place only in Great Britain—Roath Lake, Cardiff. The common and the uncommon all receive skilful treatment at the hands of practised commentators.

STATISTICAL STUDIES OF TUBERCULOSIS

Studies on Tuberculosis. Four Papers by Different Authors. The American Journal of Hygiene Monographic Series, No. 16, 1941. Baltimore: The Johns Hopkins Press, 1941. Publication financed by the Rockefeller Foundation, New York City.

This monograph is devoted to four papers describing two series of epidemiological investigations.

Eugene L. Opie, Persis Putnam, and E. Joyce Saward portray the spread of tuberculosis in negro families of Jamaica, B.W.I. The disease follows a much more rapid course than in the white people of European extraction, and is readily transferred from the first sufferer in a family to other members of the household, adults as well as children, so that many of them may die within two years after the disease has been introduced into the family. The proportion of those with tubercle bacilli in the sputum is much greater than among white persons of the United States, and greater than among American negroes. This factor and uncleanly habits, unhygienic housing conditions, and lack of facilities for segregation all promote the spread of disease in Jamaica. Though the tuberculin reaction in Jamaica, as elsewhere, has little value as an index of manifest disease, the frequency of lesions visible radiographically and of manifest disease increases with intensity of reaction. From what has already been said, it may be anticipated that asymptomatic and latent lesions were found much less often in Jamaican negroes than in white adults (and even less frequently than in American negroes). The authors conclude that the high level and rising incidence (with age), frequency, and mortality from tuber-