intact tympanic membrane but with fistula into the mastoid process, on the posterior or superior wall of the osseous meatus. A mastoid operation will be necessary for this class.

TREATMENT OF THE MARGINAL PERFORATION.

The tympanic membrane has a perforation, which extends to and involves the annulus tympanicus and is associated with granulations, which are sessile, pedunculated, or may be simulated by pouting mucous membrane. The posterior superior quadrant is the common site. After thorough cleansing of the meatus, it is often possible to aspirate secretions from the attic. The meatus is thoroughly cleansed by syringing and mopping. All adherent sloughs are removed and the pent-up secretion aspirated with the Siegle speculum. Any large granulation is removed after the application of cocaine and adrenalin. The meatus is filled with absolute alcohol for ten minutes and then thoroughly dried by mopping. A 1 per cent. iodine in boric acid powder is insufflated into the meatus, filling it completely. The patient is instructed not to touch the ear, except to mop out any discharge with a cotton-wool applicator. He is seen at intervals of four days at first, but later at intervals of seven days, when the ear is again insufflated with the iodine powder without any further preliminary treatment with alcohol. This is repeated until the ear heals—usually within two months.

The treatment is painless and the iodine does not produce any effect on the skin of the meatus. If the powder is still to be seen at the weekly visit, the discharge has diminished to a minimum or has ceased completely. To obtain a view of the tympanic membrane it will then be necessary to remove the powder by syringing. If the lesion is present (in the early stages it is often present but quiescent, presenting a pale picture, compared with its congested red appearance at the commencement of treatment) the powder is again insufflated. If there is any doubt whether there is any discharge or not, the powder test should be applied. This consists of insufflating boric powder and seeing the ear next week. The presence of the powder, undisturbed and snow-white, is proof positive of the absence of any discharge in the interim.

Effects of the Iodine Powder.—The profuse discharge rapidly abates, often after the first application, the ear remaining free from any overt discharge. Thus, as far as the patient is concerned, a "cure" of the otorrhœa has been wrought. He is freed from a troublesome discharge and is rid of the ritual of a daily aural toilet. Though at this stage otoscopy would reveal the absence of any powder and the focus is still active, the patient is delighted with the results. Parents are pleased that no active treatment is required from them at home. The fœtor of the discharge will disappear, and within a week or two the otorrhœa becomes inodorous, gradually muco-purulent, mucoid, and, in successful cases, finally ceases completely. The sessile granulations shrink up, as does the œdematous prolapsed mucous membrane pouting down from the attic region and from the periphery of the perforation. A rapid epithelisation occurs, and within a few weeks will be complete.

I have repeatedly observed a cure of the otorrhœa in cases with a marginal perforation with granulations. Many of these cases had failed to react to all other forms of active treatment and it appeared that only operation was left, yet the iodine powder cleared up the condition. It is interesting that these cases heal by migration inwards of the periphery of the perforation, so that the latter assumes a central position, where it may remain patent or may completely close over.

I have also found the powder cure cases where there has been a central perforation, but not with the dramatic effect of zinc ionisation. Cases of central perforation with granulations on the periphery of the perforation are cured by the powder.

If there is any active discharge the powder never cakes or obstructs. It acts as a sponge, absorbing secretions. It only remains in situ if the discharge has ceased.

REPRESENTATIVE CASE REPORTS.

CASE 1.—A boy, aged 7, had had right and left ear discharge for two years, with occasional bleeding from the right ear but no pain. When seen on Nov. 24th, 1930, he had septic tonsils and adenoids. The right meatus was full of offensive pus; when this was removed a marginal perforation, situated in the upper posterior quadrant of the drum, was seen. Excessive granulations surrounded and occluded the perforation, which involved the annulus tympanicus. Secretions could be aspirated with the Siegle speculum.

The ear was cleaned and dried. Absolute alcohol was applied for ten minutes; the excess was mopped away and the meatus filled with 1 per cent. iodine in boric acid powder. On Dec. 1st no discharge was complained of, and the meatus was moist. It was filled again with iodine powder. On Dec. 8th there was no discharge, but there was no trace of the powder in the meatus. Silver nitrate was applied to the granulations, and iodine powder again applied. On Dec. 15th the ear was dry and iodine powder was reapplied. On Jan. 12th, 1931, the ear was dry, and on Jan. 26th the perforation was healed, and the child heard normally.

He had also had pus in the left meatus, the condition being the same as in the right ear, with attic granulations. The treatment was identical with that of the right ear, and within the same period the discharge had dried up completely and the perforation had healed.

No treatment is carried out in the interval between the attendances, but the frequency of insufflations must depend on the rapidity with which the discharge appears.

CASE 2.—A girl, aged 9, had had purulent, offensive discharge from the right ear, but no pain, for 12 months. She had had her tonsils and adenoids removed six months previously. When seen on Nov. 4th, 1930, she had a marginal perforation occupying the postero-superior quadrant of the tympanic membrane and annulus, surrounded by exuberant granulations, from which, after the ear had been thoroughly cleaned, offensive secretions, indistinguishable from cholesteatomatous debris, could be aspirated with the use of the Siegle speculum. Absolute alcohol was applied for ten minutes; the meatus was dried and filled with iodine powder. On Nov. 11th there was marked improvement: the pus was not so profuse in amount and not offensive. The meatus was again filled with iodine powder. On Nov. 18th there was no discharge, and the meatus was moist. Iodine powder was reapplied then, and again on Dec. 2nd and 9th. On Jan. 27th, 1931, the ear was dry, a dry perforation remaining.

CASE 3.—A girl, aged 11, had had purulent discharge from both ears continuously for seven years. There was no pain. She had been attending the out-patient department of a teaching hospital for months, but had never had tonsils or adenoids removed. When seen on May 9th, 1930, both ears had the meatus full of offensive pus. This was cleaned. Adherent offensive slough occupied the depth of the meatus, deep to which the entire meatus was occupied by a perforation which extended to the annulus tympanicus in all visible directions. Granulations pouted from the margins, and pus could be aspirated from the attic. For the three subsequent months she was treated with repeated zinc ionisations with little effect, the meatus being found to have a purulent slough in its depth each week. It was decided to advise operation. When the iodine powder treatment came under my notice this case was one of the earliest put on it. In the meanwhile the tonsils and adenoids were removed. On Sept. 12th the condition of both ears was the same as in May. Alcohol was applied for ten minutes, followed, after drying, by the iodine powder. This was repeated each week to both ears for six weeks, at the end of which period both ears were completely free from discharge. The patient was seen again in December, 1930, when the ears were dry and there was a complete *restitutio ad integrum* of both tympanic membranes. The mother commented on

the fact that this was the first time for seven years that the ears had ceased from discharging. Six months later the ear were still dry.

CASE 4.—A woman, aged 38, had had discharge from the left ear for many years, necessitating a daily aural toilet to keep the ear clean and free from odour. There was no pain. A perforation was observed through Shrapnel's membrane, marked by the usual sentinel granulations and extending on to the roof of the osseous meatus. It was covered by a yellow adherent slough, and a bent probe detected bone. The granulations could be sucked out of the perforation. Alcohol was applied, followed by iodine powder, and the treatment was repeated on Sept. 27th. One week later the ear had not run in the interval, though there was no trace of any powder left in the meatus. The powder was insufflated at weekly intervals, and within a month the ear ceased to discharge, and the patient was very impressed. The ear dried up completely.

I have treated over 300 cases with very gratifying results. The vast majority of these cases were between the ages of 5 and 15. Unfortunately, it is not possible to foretell which cases will respond to the iodine powder treatment or which will prove a failure. Many cases respond, much to the observer's surprise; while others, in which a good result should ensue, fail. But the treatment is well worth a trial before recourse to the radical operation.

CLINICAL AND LABORATORY NOTES

CASE OF ASBESTOS-LIKE BODIES IN THE LUNGS OF A COAL-MINER WHO HAD NEVER WORKED IN ASBESTOS.

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AND

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IN March of this year a man, aged 71 years, was seen in consultation with a doctor ten miles from Manchester, and was later admitted to the Manchester Royal Infirmary under the care of one of us (F. E. T.). The doctor who had attended him reported that he had had several attacks of hæmoptysis in the preceding month, but as several recent sputum tests had had negative results the case had not been accepted by the local tuberculosis authorities.

The man himself said he was a coal-miner, and had as a young man worked in Alabama in mines. On consulting books of reference it was found that no asbestos is found in Alabama, which is noted for coal and iron, "Alabama iron" being famous all the world over. In 1923 it is stated that 80 per cent. of the asbestos produced came from that part of Quebec which is south of the St. Lawrence. Alabama and Lancashire mines were the only mines he had ever worked in. These points later became of interest and importance owing to the curious findings at the post-mortem and in the histological examinations of portions of tissue removed thereat, and later examined by one of us (J. S. D.).

On examination on admission to the hospital the man was very thin, obviously much emaciated, and very weak. There was a history of typhoid fever in December, 1920, and of operation for a strangulated inguinal hernia in December, 1930, since which time his health had never fully recovered. On Feb. 19th, 1931, he had his first hæmoptysis, spitting up "about four pints" of blood, while a few hours later a second hæmoptysis occurred. A cough had been constant since then, whilst the sputum remained blood-tinged and night sweats occurred. A week before admission a rigor occurred, and delirium followed, and persisted up to the time of admission. Examination of the chest showed nothing abnormal on the left side beyond the presence of a few râles and rhonchi over the lower part of the left lung behind. On the right was massive dulness, more marked below. Well below the apex at the back and to the outer side were signs of cavitation. The area of dulness surrounding the area of cavitation gave no evidence of softening to auscultation, but there was rather an absence of breath sounds. Low down in the right axilla friction was heard. A tentative diagnosis of pulmonary tuberculosis was made, with possible new-growth supervening. In the next 48 hours a positive sputum test was obtained. A blood Wassermann taken about this time was negative. The Widal reaction was positive to *Bacillus typhosus* 1 in 160.

Radiographic examination of the chest reported: "As the patient was unable to sit up satisfactory examination was impossible. The film taken shows displacement of the heart and mediastinum towards the right side. There is a large irregular opacity in the right lower chest, with mottling in the upper and middle lobes. There is some mottling also visible in the left middle and lower lobes. The appearances are not typical of tuberculosis. ? neoplasm."

The urine contained a trace of albumin. The pulse-rate at first was 90–100, but later rose to 116–120. The range of temperature was morning, 98°; evening, 99°–100°. He gradually got weaker, and died on April 14th. The autopsy was held on the 15th.

A synopsis of the post-mortem findings follows, with a description of the histology and some notes on these findings by one of us (J. S. D.).

REPORT OF AUTOPSY.

Lymph glands: Thoracic and abdominal glands along spinal column are heavily anthracosed.

Pericardium: About 100 c.cm. excess fluid.

Heart (400 g.): Enlarged; myocardium, hypertrophy, more marked right side; tricus. V, three fingers; mitral V, two fingers; aorta, early atheroma.

Right and left pleuræ: Each 500 c.cm., straw-coloured fluid. Right lung (900 g.): Whole of lower and lower half of upper lobe are firm, almost black with white mottling, and at apex of lower lobe a cavity containing

FIG. 1.

Asbestos-like bodies in the lungs, showing forms, with rounded ends. In Fig. 3 the central core can be clearly distinguished. (× 500.)

a ruffled wall and evidence of tubercle. Left lung (1360 g.): Lungs showed some degree of softness, as though some neoplastic invasion were present. Both lungs heavily anthracosed. Left consolidated only in its lower lobe. No abnormalities evident in air passages. General appearances suggest a chronic fibroid tubercle, possibly associated with a neoplasm.

Stomach: Nil. Small intestine: Some small tuberculous ulcers in lower ileum. Large intestine: Tuberculous ulceration of cæcum.

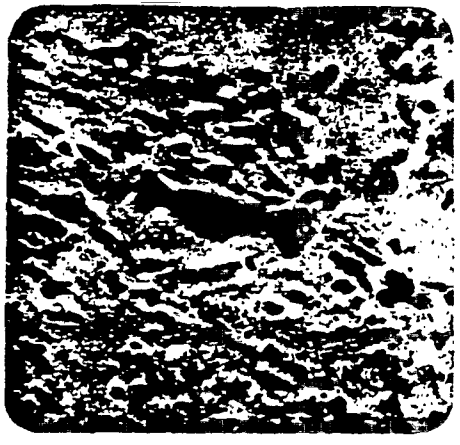
Liver (1600 g.): Moderate passive congestion. Pancreas: Nil. Spleen (340 g.): Enlarged and moderately firm. Hyperplasia and congestion. Thyroid: Nil. Adrenals: Nil. Right kidney (160 g.): Congested. Left kidney (160 g.): Congested. Urinary bladder: diverticula present. Genital glands: Middle lobe of prostate quite appreciably enlarged.

Anatomical diagnosis: Chronic fibroid tuberculosis of lungs. Pulmonary asbestosis.

HISTOLOGICAL EXAMINATION.

The extensive consolidation of the lower lobes of both lungs was found to be due to caseating tuberculosis with a very diffuse distribution; it appeared to represent a rapid lymphatic spread, and in large areas it completely obscured the lung-structure. Tubercle bacilli were present in moderate numbers.

FIG. 2.



There was fairly marked anthracosis, the pigment in fine granules, being mostly aggregated in the peribronchial connective tissue.

The upper parts of the upper lobes were almost free from tuberculosis, and so it was possible to recognise a moderate patchy overgrowth of fibrous tissue in these areas. This was mature in type, but not very dense, and did not form laminated nodules. Here and there in the fibrous tissue were scattered some minute brownish bodies singly or in groups of five to ten (Figs. 1 and 2). They were mostly slender rods with bulging ends, and they measured from 30 to 70 microns in length by 2 to 5 microns in thickness. On high-power examination, it could be seen that most of these bodies had a black opaque central core, while the brown material formed an outer lamina and was semi-transparent (Fig. 3). There was no appearance of the regular segmentation which commonly occurs in asbestos bodies. On treatment with potassium ferrocyanide and hydrochloric acid, the brown material took on an intense blue colour, evidencing an abundant content of free iron. The bodies were subsequently recognised in all parts of the lungs, though nowhere numerous.

COMMENT.

The main point of interest in the case is the occurrence in the lungs of bodies which somewhat resemble those that have been described in the lungs

FIG. 3.

of asbestos workers. They simulate these in size and general shape, especially in the rounded swellings at the ends; in colour; and in their abundant content of free iron. They differ from the familiar asbestos bodies in being usually coarser and less regular, in possessing a black core of opaque material, and in the absence of any appearance of segmentation. It is presumed that they are minute fragments of some other iron-containing mineral, which have been inhaled into the lungs and undergone some chemical change under the influence of the tissue fluids resulting in the formation of a peripheral lamina from the iron-containing part. They do not appear to have caused a very serious fibrous reaction in the lungs in this case.

GALLOP RHYTHM:

ITS CLINICAL SIGNIFICANCE IN CERTAIN CASES.

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GALLOP rhythm has long been known to be a serious cardiac sign. The name is best reserved for the true gallop rhythm where the first heart sound is reduplicated (∪∪—); for, in agreement with Gee's teaching, a doubling of the second sound (—∪∪) is properly termed canter rhythm. It is important to differentiate between the two, for canter rhythm is heard in many conditions none of which is as serious as those which yield gallop rhythm. These two rhythms are easily confused, for even a slight degree of tachycardia may appear to accentuate the middle component of the three (∪—∪); and during the varying phases of respiration this accentuation will seem to move sometimes to the third component (∪∪—), sometimes to the first (—∪∪).

It is here desired to draw attention to a special variety of gallop rhythm, and to emphasise the importance of its recognition. For according to the

age at which it appears it may be the only sign which either allows for the provision of adequate treatment or leads to a correct diagnosis and a more clear prognosis. This special variety is a reduplication of the first sound, clearly heard, over a small area close to the left border of the sternum at the second intercostal space or third cartilage. In many cases it will be heard at all the auscultatory areas; but if it is not audible at this localised spot, it is unlikely to be heard elsewhere. There are, of course, many cases where gallop rhythm is heard at the mitral area, yet not at the base; these belong to a different category.

ACUTE RHEUMATISM.

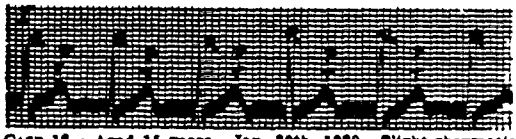
The significance in acute rheumatism varies with the age of the patient. In children and young persons it is almost always associated with delayed conduction from auricle to ventricle, the electrocardiogram showing a lengthened P–R interval. These two features, gallop rhythm and long P–R interval, are frequently the earliest evidence of rheumatic carditis. Tachycardia, about 120 per minute, is usually present, while the temperature may not be raised above 99° F. The discovery calls for immediate rest in bed; the earlier this is obtained, the more quickly does the P–R interval close. Administration of full doses of salicylates in addition appears to hasten the closure. If treatment is delayed or imperfectly carried out, the P–R interval may remain permanently longer than normal, and the gallop rhythm will then persist.

It is probable that the lengthened P–R interval is due to an infiltration of the auriculo-ventricular junction rather than to vagal effect, for atropine produces no alteration except in occasional cases. However, the effect of atropine on the P–R interval is a complex one, and as yet imperfectly understood. Sound-photographs seem to show that the earlier of the two short components is produced as a result of auricular contraction occurring outside its usual timing in the cardiac cycle.

Eighteen cases of this nature have recently been observed in patients under the age of 25 years. In each case a lengthened P–R interval was confidently predicted prior to electrocardiogram. Confirmation was clearly obtained in 17 cases; the eighteenth proved within normal limits. With early treatment, serial electrocardiograms on successive days revealed a rapid closure of the P–R interval; pari passu the gallop rhythm became less clear. Consequently, for purposes of confirmation it is necessary to take an electrocardiogram on the same day on which the gallop rhythm is clearly heard. Figs. 1 and 2 are electrocardiograms from a typical case.

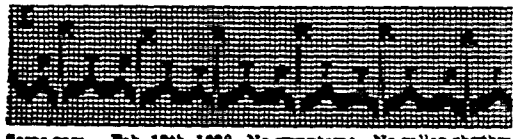
CASE 1.—Girl, aged 15, seen in out-patient clinic at St. George's Hospital, on Jan. 30th, 1929, complaining of mild rheumatic pains in legs and arms; headaches; easily fatigued. No breathlessness or cyanosis. No history of scarlet fever or definite rheumatic fever. On examination, tachycardia, 116, persisting with rest and not due to nervousness. Temperature normal. Heart normal in size, regular rhythm; sounds normal except for reduplication of the first sound at the second left interspace near the sternum; no murmurs. Clinical diagnosis: First degree A–V block, probably rheumatic. Confirmed by electrocardiogram (Fig. 1) on same day. Admitted to hospital for rest in bed, no special treatment. Heart-rate gradually slowed, gallop rhythm became less clear. On day after admission, atropine sulphate gr. 1/75, subcutaneously,

FIG. 1.



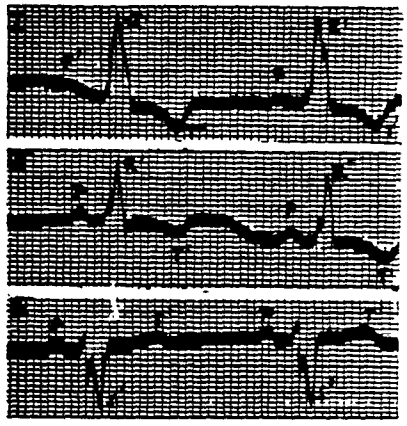
CASE 1.—Aged 15 years. Jan. 30th, 1929. Slight rheumatic pains. Gallop rhythm and tachycardia present; no other abnormal cardiac signs. Lead I. rate = 114. P. and T. fused. P–R = about 0·31 sec.

FIG. 2.



Same case.—Feb. 13th, 1929. No symptoms. No gallop rhythm. Rate = 96. P–R = 0·20 sec.

FIG. 3.



produced no effect on the P–R interval. Feb. 13th: Clinically normal, no gallop rhythm. Electrocardiogram normal (Fig. 2). November, 1930: Signs of early mitral stenosis. Electrocardiogram: P–R normal.

Effective treatment was denied to another girl in the first instance, owing to the true nature of her trouble being unrecognised. Now after several years' observation, gallop rhythm is still in evidence and the P–R interval remains abnormally long.

CASE 2.—Girl, aged 10, came to out-patient clinic at Victoria Hospital on May 6th, 1927, complaining of fainting attacks with loss of consciousness for 3–5 seconds. Scarlet fever at age of 7. No rheumatic history. On examination pulse 120, persistent, regular. Temperature normal. Heart normal in size. Apical first sound loud and slapping. First sound reduplicated at pulmonary area. Electrocardiogram confirmed clinical diagnosis of first degree rheumatic A–V block. P–R = 0·28 second for rate = 125 per minute. The parents refused admission to the Victoria Hospital and sought advice at another hospital, where apparently the nature of her trouble was not recognised, and no rest was ordered.

April, 1928: Returned to Victoria Hospital, still suffering from fainting attacks. Gallop rhythm still present. Electrocardiogram: P–R = 0·24 second for rate = 110 per minute.

No subsequent treatment, however, has been effective, and the clinical state and the electrocardiogram remain practically unchanged at the present time.

BUNDLE-BRANCH BLOCK.

In adults of middle age and older this form of gallop rhythm usually indicates a block in one branch of the bundle of His. It is more clearly heard when the right branch (old nomenclature) is affected. Fig. 3 is the electrocardiogram from a case of this