When he was thoroughly under its influence, which occupied twenty minutes, the growth was further examined by those present and myself. I proposed to drill into the centre of the exostosis, fix into it the screw-tap, undermine it by drilling through its base, and then to snap it off by using the screw-tap as a lever. The periosteum was first cut through and torn away as far as was practicable; the blood, flowing very freely, was allowed to be unchecked for a few minutes, as the vessels of the head were much congested. The meatus was now rinsed with cold water, and a large speculum introduced well down upon the growth and plugged with cotton-wool. The boring apparatus was brought alongside the patient. Drill A was made to play

FIG. 3.

upon the centre of the growth, and kept in the axis of the osseous meatus, entering the bone to the extent of two millimetres. Drill B was now mounted and used to a depth of about six millimetres. Into this hole the tap was now firmly fixed, and the speculum removed. Having well cleansed the meatus, I made three separate drill-holes at the base of the exostosis, entering the bone to the extent of some seven or eight millimetres, and, in addition, tried to get drill C to work laterally, without, however, much success, the density of the bone and the clogging of the teeth with debris rendering it extremely tedious.¹ Thus weakened at its base by the drill-holes, and not suspecting the large area of its attachment internally, I thought to break off the growth by using the screw-tap as a lever. The tap unfortunately broke off close up to the bone without accomplishing this purpose. The pair of stump forceps were now used, but only enabled me to bring away a part of the growth containing the broken end of the screw. (See this line of fracture in Fig. 4.) The part of the exostosis now remaining, being more deeply seated, offered considerable difficulties in the way of removal. Strong traction directly outwards was made upon the auricle, with slight rotation to the *left*; and the bicuspid forceps, touched with oil externally, were introduced (closed), and rotated about a quarter turn to the *right*, accompanied by such a pressure into the meatus as to bring them down upon the remainder of the growth, but without pushing the tubular portion of the *auricle* into the osseous meatus, and thus encroaching upon available space. Opening the forceps as much as possible under the circumstances, taking advantage of the vertical diameter of the meatus, I obtained by a little manœuvring what was undoubtedly a firm grip of my opponent, and brought away the rest of the bony mass, not by traction, as I should thereby have lost my hold, but by a very delicate, yet firm, rotatory movement. The membrana tympani was now seen to be partly hidden by a flap of periosteum, which peeled off the inner surface of the growth during its extraction, remaining loose in the meatus. The greater portion of this was carefully taken away, its attached border being too close to the drum to warrant its entire removal, otherwise I should have been careful to get rid of the whole covering of the growth.

¹ During the boring process I kept my right or operating hand very firmly pressed against the side of the patient's head, so as to have proper control over the drills.

A front view of the entire exostosis is very faithfully portrayed of its natural size in Fig. 4. It measured eleven millimetres from apex to the extreme edge of base, which reached the annulus tympanicus; eight millimetres from

FIG. 4.

above downward, and seven millimetres in thickness. Smooth and irregular on its free surface. The shaded part in Fig. 4 represents an injury by the forceps. It is quite flat on its attached surface, except where channelled by drill-holes. It weighs .237 of a gramme.

Soon after coming to the patient vomited several times, and complained very much of pain in the lower frontal region. Did not feel more than a little discomfiture in the meatus, and could hear very fairly. Towards the evening he became very deaf, which lasted five days, much to his alarm. I had previously warned him of this effect, so that he was not so much discomfited as he might otherwise have been. The patient, who was of a plethoric habit, had taken saline aperients just before the operation, and was put on spare diet for a few days afterwards. Very little inflammatory action appeared in the meatus, which was cleansed four times daily with warm carbolic lotion (1 in 75). In a few days granulations appeared. These were first controlled by perchloride of iron, and afterwards by nitric acid. Not seeing the patient frequently after the first week, two of the granulations became sufficiently large to remove with a snare. The hearing was perfectly restored at the end of ten days, and on my last inspection, five months after the operation, scarcely a trace of the site originally occupied by the exostosis could be discovered.

There being no history of syphilis (acquired or otherwise) or hereditary predisposition, the growths are, no doubt, attributable to persistent irritation due to measles at an early age.

I believe from the cases of exostosis and periostosis of the external auditory meatus which have come under my notice, that when they are not of syphilitic origin the patient frequently possesses a more or less marked tendency to general hyperostosis or osteo-sclerosis.

The time occupied by the above-described operation, including the administration of the anæsthetic, was thirty-eight minutes. My thanks are due to Drs. Yeates and Wright, and Mr. Lloyd, for their very able assistance on the occasion.

A

CONTRIBUTION TO CLINICAL HÆMOMETRY.

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(Continued from p. 362.)

OUR observations may conveniently be grouped under three heads; first, those made in acute febrile diseases; secondly, in chronic diseases; thirdly, in those pathological conditions in which the blood is either the primary seat of the morbid process, or, at any rate, the theatre of its most conspicuous manifestations.

A. ACUTE FEBRILE DISEASES.

Scarlet Fever.—I. Ernest C—, aged four, mild but typical case. Sore throat and rash. No complications.

Obs. 1. Fifth day of illness, two days after appearance of eruption, which is fully developed all over the body. Pulse 130; temperature 102°. N=4,670,000; G=0.61.

Obs. 2. Nineteenth day; temperature 98°. Free perspiration ever since. Rash faded twelve days ago. Constipation. No albuminuria. N=7,510,000; G=0.38.

II.—Samuel B—, aged sixteen. Typical rash and bad sore throat. No complications.

Obs. 3. Third day of illness. Rash universal. No sweating or diarrhoea. Pulse 128; respiration 30; temperature 104.4°. N=4,460,000; G=0.66.

Obs. 4. Nineteenth day, eleventh after complete defervescence, since when the temperature has been normal or subnormal. Peeling. Skin moist. No purging or albuminuria. $N=6,400,000$; $G=0.41$.

In the single case of scarlet fever, with high temperature, observed by Leichtenstern, he failed to detect any diminution in the hæmoglobin of the blood, either during or after the attack. This agrees very well with our results; for, although a very considerable increase in the number of corpuscles was observed to take place during convalescence, this increase was almost exactly balanced by a corresponding fall in their individual value, the absolute richness of the blood in hæmoglobin thus remaining nearly constant. The complete nature of the compensation is well shown by multiplying the figures representing the number into those representing the functional value of the corpuscles.

I. (1) $4,670,000 \times 0.61 = 2,848,700$.

(2) $7,510,000 \times 0.38 = 2,853,800$.

II. (3) $4,460,000 \times 0.66 = 2,943,600$.

(4) $6,400,000 \times 0.41 = 2,624,000$.

Measles.—Henry S—, aged twenty-five; typical case. Catarrhal symptoms with profuse sweating, which ceased when the rash came out; no complications; ran a mild course, ending in recovery.

Obs. 5. Third day of eruption, which is universal. Pulse 104; respiration 24; temperature 101.6° .

$N=6,980,000$; $G=0.53$.

Obs. 6. One week later: Defervescence complete; bowels confined; moderate sweating. $N=6,160,000$; $G=0.59$.

Acute Rheumatism.—John L—, aged sixteen. First attack; polyarticular inflammation with profuse sweating; loud mitral murmur; no pericarditis; pulmonary congestion; constipation; no albumen in urine.

Obs. 7. Fifth day of illness: Temperature 103° ; pulse 104; respiration 36. $N=5,970,000$; $G=0.73$.

Obs. 8. A week later: Temperature 97° .

$N=6,330,000$; $G=0.72$.

Acute Lobar Pneumonia.—I. Lewis B—, aged twenty-eight. Consolidation of left lower lobe; sudden onset; high fever and delirium; no albuminuria; chlorides absent from urine; rusty sputa.

Obs. 9. Fifth day of illness: Delirious. Pulse 130; respiration 44; temperature 103.4° . $N=5,310,000$; $G=0.66$.

Obs. 10. Nineteenth day (eleventh since defervescence): Lungs have quite cleared up. $N=5,720,000$; $G=0.57$.

II. Fanny J—, aged twenty-one, a well-nourished and healthy girl. Consolidation of left lower lobe. Temperature 103° to 104° ; rusty sputa; onset abrupt.

Obs. 11. Fifth day of illness: Sweating freely; bowels confined; pulse 112; respiration 30; temperature 101° .

$N=5,840,000$; $G=0.51$.

Obs. 12. Twentieth day (two weeks since crisis): Lungs clear; bowels confined; frequent sweating.

$N=5,720,000$; $G=0.57$.

In the first of these two cases there was a slight increase in the number of corpuscles, with a corresponding reduction in their individual value. In the second no material variation was observed. The result agrees substantially with that obtained by Leichtenstern in two cases of simple acute pneumonia. He noticed no decided diminution in the proportion of hæmoglobin in either of them.

Typhus.—I. Hannah L—, aged thirty; typical rash; high fever (103.2° to 105°); delirium; tremors; dry tongue; prostration; free sweating throughout. On the evening of the fourteenth day the temperature rose to 107° , and the urine was found to contain one-third albumen; ice pack. By the next morning the temperature had fallen to 97.2° , rising again in the course of the day to 99.8° . Death on the evening of the fifteenth day.

Obs. 13. Thirty hours before death: Skin dry and hot; pulse 150; respiration 48; temperature 105° .

$N=4,190,000$; $G=0.71$.

II. Mary F—, aged twenty-two; typical eruption; temperature 104° to 105° ; pulmonary congestion; delirium; deafness; tremors; sordes on tongue. Recovery.

Obs. 14. Sixth day: Rash well out; skin dry and harsh; bowels relaxed; pulse 124; respiration 64; temperature 103° .

$N=4,430,000$; $G=0.79$.

Obs. 15. Fifteenth day: Bowels still rather relaxed; pulse 88; respiration 20; temperature 98.6° .

$N=4,460,000$; $G=0.69$.

In both of the above cases the proportion of corpuscles, both during and after the attack, was somewhat lower than normal, though the value of the individual corpuscles was

not much impaired. It is as curious as it was unexpected to find how little the composition of the blood—as regards its red corpuscles and hæmoglobin—was altered in a most severe case of the disease, on the fourteenth day of its course, and only thirty hours before its fatal issue. And this in a malady which is commonly supposed—and probably with justice—profoundly to affect the constitution of the circulating fluid. We were unable to find any recorded observation on a case of typhus fever to compare with our own.

Enteric Fever.—I. Mary H—, aged seventeen; typical example of the disease, running its course in four weeks in a strong and healthy girl; delirium; rose spots; abdominal tenderness; high fever (104.6°). Recovery.

Obs. 16. Nineteenth day (?): Pulse 128; temperature 102° . Flushed and sweating; diarrhoea very moderate.

$N=6,590,000$; $G=0.44$.

Obs. 17. Five weeks after defervescence; out of bed for ten days. $N=6,410,000$; $G=0.53$.

II.—Elizabeth A—, aged sixteen. On admission rose spots, diarrhoea, abdomen swollen and tender. Fever very high; pulmonary congestion. On fourth day after admission the temperature rose from 105° to 106° . Cold baths; death; ulcers in ileum.

Obs. 18. Two days after admission (second week of disease?): Respiration 24; pulse 130; temperature 104.8° . Diarrhoea moderate; no sweating. $N=6,420,000$; $G=0.53$.

III.—John V—, aged eighteen. Typical case, with delirium, rose spots, slight epistaxis. Temperature 104° to 105° . Much pulmonary congestion. Died a week after admission. Ulcers in ileum, &c.

Obs. 19. Two days after admission (third week of illness): Severe diarrhoea. Respiration 32; pulse 112; temperature 104.2° . $N=6,160,000$; $G=0.57$.

IV.—Annie M—, aged twenty-six. Moderately severe case, with profuse diarrhoea.

Obs. 20. Fourteenth (?) day of illness:

$N=5,060,000$; $G=0.69$.

V.—Emily G—, aged twenty-one. Febrile disturbance with profuse diarrhoea for a week before admission. Enteric fever suspected; doubtful.

Obs. 21. Four days after admission: Eight loose motions during previous twenty-four hours. No sweating. Temperature 102.5° . $N=6,600,000$; $G=0.45$.

The general conclusion to be drawn from a consideration of the above observations is, to us at any rate, of an unexpectedly negative kind. Enteric fever, even when it immediately threatens life, does not appear to affect either the number or the quality of the red corpuscles to any marked extent. Hæmorrhage from the bowel would no doubt do so; but this is accidental, not of the essence of the disease. Substantially the same results were arrived at by Andral and Gavarret as regards the proportion of red corpuscles, and, more recently, by Leichtenstern, as regards the amount of hæmoglobin in the blood. In six cases of enteric fever, the latter observer examined the blood repeatedly, and found that “during the first, second, and third week, so long, in fact, as the fever lasts, the proportion of hæmoglobin undergoes no change.” He adds, however, that in a majority of cases the subsidence of pyrexia is attended by a decided and rapid decrease in the relative amount of this constituent. Whether this “post-febrile anæmia,” which did not show itself in the only one of our cases (Obs. 17) in which it might have been detected, depends on a hypocytosis, or a hypochrosis, or a combination of both, must for the present remain uncertain.

What strikes one most in looking over the series of observations comprised in the present group, is the very small influence exerted on the red corpuscles and the hæmoglobin by even the most severe febrile disorders, such as might naturally be supposed to modify profoundly the whole constitution of the blood. That the nutrition of the body generally is much interfered with by all fevers, especially by such as last for any time, is a commonplace. It may be, as Leichtenstern suggests, that the total mass of the blood, like that of the solid tissues, is uniformly diminished by the febrile process, without any material change in the relative proportion of its constituents. This hypothesis may serve, at all events, as a provisional explanation of what would seem, at first sight, to be altogether anomalous.

In marked contrast to the immunity of the red corpuscles in all the pyrexial disorders which have come under our

notice, is their liability to destruction in malarial fever. Kelsch's¹ observations on this subject, though published several years ago, do not appear to have attracted all the attention they deserve. They were made at the military hospital of Philippeville, in Algeria, where every variety of paludal disease is met with in great profusion. In his examinations of the blood he employed the numerator of Malassez; unfortunately, no parallel series of chromometric observations was carried out, so that it must remain doubtful for the present whether the hypocytosis was or was not accompanied by a corresponding degree of hypochrosis. The visibly profound anæmia displayed by the victims of ague, however, renders it highly probable that no sufficient increase in the value of the corpuscles occurs to counterbalance the effect of the great reduction in their number.

Numeration was employed in about 70 cases of paludal fever, and in many of them it was repeated daily throughout the course of the disease. The cases are grouped under the three heads of simple fever, malignant fever, and malarial cachexia. The most striking effect of the miasma on the corpuscles is manifested when a previously robust and healthy man is attacked for the first time. Under such circumstances the daily loss of red corpuscles may amount to several hundred thousand, occasionally to one million per cubic millimetre of blood in every twenty-four hours. This is during the initial period of the malady, when the symptoms are of a continued or remittent type. The initial period is usually of short duration, and is followed by a longer one, during which the fever is definitely intermittent (quotidian or tertian), and the number of corpuscles continues to decrease uninterruptedly, though less rapidly than in the first period. A succession of paroxysms will sometimes bring down the number of discs from the normal proportion of five or six millions to one million, or even to five hundred thousand, per cubic millimetre. As the blood is more and more impoverished, the process of destruction grows slower and slower, until at last, when the disease has entered on its third period, characterised by irregular febrile exacerbations, with more or less prolonged intervals of apyrexia, the number of corpuscles simply oscillates to and fro about an average minimum. At this stage a tendency may show itself to a very gradual restoration of the corpuscular elements; or their number may continue to diminish, though very slowly; or it may remain quite stationary for weeks together, notwithstanding the energetic use of quinine and iron, and the comparative infrequency of febrile paroxysms. This last condition is met with more especially in profoundly cachectic patients, with great enlargement of the spleen.

In the malignant form of paludal fever, the rapidity of corpuscular disintegration is proportionate to the previous good health of the patient. When he is a robust and well-nourished man, the corpuscles disappear at the rate of from half a million to one million per cubic millimetre in twenty-four hours; when his health has been broken by previous attacks, and he is already suffering from malarial anæmia, the daily loss varies from one to two hundred thousand per cubic millimetre. It is a noteworthy fact that the loss may go on, and may even amount to several hundred thousand corpuscles per cubic millimetre every day, while the axillary temperature continues normal; bearing no proportion, as it does in fever of the simple type, to the intensity of the febrile movement. On recovery from an attack of the malignant form, the restoration of the corpuscles is usually pretty rapid.

It is clear that in diseases of this class we have to do with some specific action of the paludal miasm upon the formed elements of the blood. Nothing at all resembling the effects just described has ever been noticed by ourselves or others in even the most acute febrile maladies which we have had an opportunity of studying. The destruction of the red corpuscles is not dependent on the pyrexia as such, though it probably stands in close relation to the melanæmia which is so frequent a consequence of the endemic fevers of marshy districts. It is to be hoped that those who enjoy suitable opportunities for the purpose will not fail to repeat Kelsch's observations and amplify them by the simultaneous use of the chromometer. We have not ourselves had a chance of examining the blood in even a case of ordinary ague, and are therefore unable to say how far, if at all, the conclusions stated above may be applicable to the very mild forms of malarial intoxication which now and then present

themselves in our wards. Should those conclusions be substantiated, their practical value as regards both diagnosis and prognosis is too obvious to require detailed exposition.

(To be continued.)

CASE OF BILATERAL PARALYSIS OF THE MUSCLES SUPPLIED BY THE RECURRENT LARYNGEAL NERVE.

By J. A. ORMEROD, M.B. (Oxon.), M.R.C.P.

CASES of unilateral paralysis of the laryngeal muscles are, I suppose, tolerably common. Cases of bilateral paralysis of individual muscles, as, for instance, the posterior crico-arytenoids, are being recorded in increasing numbers; but bilateral paralysis of all the laryngeal muscles has hitherto, I believe, been but rarely observed. I therefore venture to publish an account of a case at present under my observation, albeit I have been unable to watch it from the commencement, and the termination of it still remains to be seen.

Von Ziemssen, in his "Cyclopædia of Medicine," mentions eleven cases, six observed by himself, the remainder by Türck, Traube, Münck, and Bäumlér. In three of these the paralysis was complete on both sides; in seven complete on one side, incomplete on the other; in one (a remarkable case, supposed to be caused by the pressure of a distended pericardium) the paralysis was incomplete on both sides. The cause was in four cases œsophageal cancer; in one cancer of the thyroid gland, in five aneurism. I find records of three other cases: one¹ by Ludwig Brieger in a phthisical patient; the paralysis complete on both sides; the cause (determined post mortem), pressure upon the recurrent nerves by enlarged lymphatic glands. And two² by Karl Kittler, in the first of which there was complete paralysis on the right side, with paresis upon the left, ascribed to aneurism; in the second, paralysis not quite complete on the left side, and paresis on the right; the cause undetermined, but supposed to be catarrh. It appears, therefore, that in the majority of these fourteen cases the paralysis was incomplete on one side, and that the cause was usually a tumour or aneurism.

The symptoms are so well described by Von Ziemssen that I need only recapitulate them. On laryngoscopic examination, the vocal cords are seen to occupy the cadaveric position, lying midway between the positions of phonation and deep inspiration, and not altering their position on phonation or inspiration. If the paralysis is incomplete on one side the cord of that side moves, but sluggishly and insufficiently, and it does not, as in cases of purely unilateral paralysis, compensate for the inactivity of its fellow, by crossing the middle line to meet it. There is absolute aphonia (at least in cases of complete paralysis), inability to cough with force, waste of breath on coughing or talking, so that prolonged talking puts the patient out of breath.

In the observation of the following case I have to acknowledge the help of Dr. Felix Sémon in pointing out to me some essential symptoms, and of Dr. de Havilland Hall, who also saw the case and agreed in the diagnosis.

Esther C—, aged thirty-eight, marked with small-pox and looking rather out of health, came to me at the Metropolitan Free Hospital in September last, complaining of cough and loss of voice, and of feeling generally out of sorts. The larynx was seen to be deeply seated, the ventricular bands were swollen and overhanging, the arytenoid cartilages a little larger than normal, and the right standing on a higher level than the left. The vocal cords were slightly congested, the right was quite immovable; the left moved but did not reach the right. The pharynx was slightly granular. The chest and neck were perfectly normal. She was treated with benzoin inhalation and iodide of potassium in doses of five to ten grains three times a day. Her appearance and general health improved, but the aphonia remained.

By November the symptoms of chronic laryngitis had subsided, and I was able to examine the patient under more favourable conditions. The following facts were then made

¹ Contribution à l'Anatomie Pathologique des Maladies Palustres. Par A. Kelsch. Arch. de Physiologie, vol. ii., 1875.

¹ Virchow, Hirsch, Jahresbericht, 1878.

² Schmidt, Jahrbuch., vol. clxxx., p. 167.