applied to the hole in the end of the halometer. The halo diameter and the date are recorded on the label of the slide, and kept so that the progress on liver diet may be noted. Most red cells preserve their size indefinitely, but I think that where the plasma is relatively abundant (as in severe pernicious anaemia) the red cells must be very slowly stretched in the course of weeks by shrinking of the plasma film; so it is well to measure (or post) the film without many days' delay. I do not heat the films.

PRE-PERNICIOUS ANAEMIA.

Our former conceptions of pernicious anaemia apply only to the late stages; the early stages are quite different. Enlargement of the red cells is the first reliable sign; anaemia is then slight, and is only shown by a small deficiency of haemoglobin. The pathologist may report "blood film normal, slight secondary anaemia, colour index well below unity." The usual overfilling of the red cell with haemoglobin has often not begun; nor has haemolysis, for the van den Bergh test is negative. In this early stage the patients are usually florid and well nourished, so that everyone is incredulous when anaemia of any sort is diagnosed. In fact, the term "pernicious anaemia" is inappropriate in the early stage revealed by enlargement of the red cells, and a special name is needed, such as "pre-pernicious anaemia." This condition would progress to the familiar pernicious stage if left untreated, for achlorhydria is almost invariably found, sore tongue is usual, and the size of the red cells soon becomes normal under liver treatment, which is known scarcely to influence other anaemias.

Similarly, subacute combined degeneration of the cord can usually be recognized by the enlarged red cells (because the neurotoxic and haemolytic toxins generally act in concert) at a far earlier stage than has hitherto been clinically possible. In the space of a year I have thus identified three cases, in which the patients complained solely of tingling of the hands and feet. This is a great advance, because if nervous tissue is once destroyed it cannot be regenerated. The streptococcicidal effect of hydrochloric acid should prevent the nervous disease from progressing further, while liver cures the early pernicious anaemia, as shown by the restoration of the red cells to their normal size.

SUMMARY.

Pernicious anaemia has not yet been defined; it is a syndrome of about a dozen symptoms, none of which is present of necessity, unless nowadays we except enlargement of the red cells. In the appended table I have set down these symptoms in double columns, and it is evident that those of the early stage of pernicious anaemia are

Table showing the Distinguishing Symptoms of Pernicious and Pre-pernicious Anaemia (not Absolute).

Symptoms.	Pernicious Anaemia.	Pre-pernicious Anaemia.
Nine surface differential symptoms:		
Complexion	Pale or lemon	Commonly florid
Haemolysis	Present	Usually absent
Poikilocytosis	Present	Absent
Anisocytosis	Conspicuous	Inconspicuous
Polychromasia	Present	Absent
Nucleated red cells	Often present	Absent
Colour index	High	May be normal or low
Blood platelets	Absent	Present
Body weight	Well retained	Reduced initially
Six basic symptoms in common:		
Enlarged red cells	Present	Present
Achlorhydria	Present	Present
Tiredness	Present	Present
Glossitis and oral sepsis	Often present	Often present
Spinal cord changes	Often present	Often present
Response to liver	Marked	Marked

so different from those of the later stages that it is absurd and misleading to include them in the same syndrome, and a separate name is required. It is pernicious anaemia before it has become pernicious; hence the name "pre-pernicious anaemia."

If, as I believe, it can be accepted that enlargement of the red cells is an invariable feature peculiar to pernicious anaemia in all its active stages, then the two dissimilar syndromes (pernicious and pre-pernicious anaemia) can be included under a single definable disease called megalocytic anaemia. But this definition can hardly be adopted until a ready means of measuring the average size of red cells is available to the clinician. I believe the halometer supplies this want.

Abstracts of ten cases which bore out the opinions expressed above were submitted with this paper, but space does not permit of the publication of more than the two case reports appended.

CASE I (from Dr. Sproule).

A florid, well-nourished unmarried woman, aged 25, complained of loss of strength, appetite, and weight for one year. She had a sore tongue last year. The gums tasted nasty when sucked; her teeth were good. Examination was negative. The red cells numbered 3,200,000 per c.mm.; haemoglobin 90 per cent.; colour index 1.2. The red cells were irregular in size, but not in shape; there were no platelets. The blood halo was bright and sharp, but too small (4.1 degrees). Her weight was 8 st. 3 lb. At her first visit, in June, 1928, a diagnosis of early pernicious anaemia was made, and she was given liver and hydrochloric acid. In two months she felt quite well and resumed her work and tennis. She stopped treatment and did not report herself again, though solemnly warned that she must do so. After six months (December, 1928) I found her address and insisted on a fractional test meal, which duly showed achlorhydria. She had started to lose weight—6 lb. in three weeks—presumably the beginning of a relapse. The van den Bergh test was normal. The red cells numbered 4,500,000 per c.mm.; haemoglobin 80 per cent.; colour index 0.8. Blood halo 4.4 degrees; hence the red cells were improved in size, but not yet normal (4.6 degrees), owing to stopped treatment. She hated eating liver, so she minced it raw, rubbed it through a fine sieve, and mixed it with a cup of weak warm bovril seasoned with pepper and salt. This she drank three times a day with her eyes shut.

On April 23rd, 1929, she was very well and had gained 24 lb. in the nine months' treatment. Haemoglobin 100 per cent.; a few platelets. She can walk five miles. Blood halo 4.78 degrees (originally 4.1 degrees).

CASE II (from Dr. Cameron).

This patient, seen in November, 1928, was a well-nourished well-coloured man of 45. He complained only of tingling in the feet and finger-tips for a month. The legs felt a little numb and stiff; there was no anaesthesia. The reflexes were normal. Blood halo 4.2 degrees, sharp and bright. A diagnosis of subacute combined degeneration and pernicious anaemia (early) was made. This was confirmed by finding achlorhydria, and colour index 1.1, red cells 3,000,000 per c.mm., haemoglobin 65 per cent., white cells 9,600 per c.mm., a few poikilocytes and microcytes (Dr. Adamson). When seen again, after six months on liver and hydrochloric acid, he felt very well, and his colour was better than ever before. The tingling was much less; none in the hands. Walks a mile. Sexual power had returned after nine months' absence. Haemoglobin 90 per cent. Blood halo 4.78 degrees (high normal).

LIGATURE OF INNOMINATE AND COMMON CAROTID ARTERIES FOR SUB-CLAVIAN ANEURYSM.

BY

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THE case reported below is of interest as a sequel to a fractured clavicle and because of its comparative rarity.

A fisherman, aged 33, on August 19th, 1928, was struck on the right side of the chest at sea with a messenger hook, and reached hospital next day. He was much bruised over the whole of the right chest, and x-ray examination showed a very oblique fracture of the right clavicle at the junction of the inner and middle thirds. He developed pneumonia, and was discharged on September 7th. Three weeks after leaving hospital he noticed a swelling above the right clavicle; in seven to ten days the swelling rapidly increased in size, and he was readmitted to hospital on October 5th.

Previous History.—He had trench fever in 1914; but in 1919 was discharged as A1.

On readmission he was very pale and looked ill; the temperature was intermittent. The mouth was extremely foul, full of septic roots, and pyorrhoea was well marked. He had a large pulsating tumour extending from above the clavicle as high up in the neck

as the cricoid and also below the clavicle; the sterno-mastoid muscle was displaced outwards. The radial pulse could not be felt. The left pupil was slightly dilated, and there was some proptosis. Pain was felt at the site of the tumour. An x-ray photograph showed a tumour occupying a big area in the upper chest, but on attempting to screen him he collapsed. The Wassermann reaction was negative.

As the tumour was growing so rapidly and appeared likely to burst, in spite of his septic condition it was decided to do an immediate operation on October 16th.

Operation.—The whole of the right arm was carefully bandaged with a thick layer of wool. An incision was made along the sterno-mastoid to join a transverse incision along the clavicle. The sac was extremely thin, but an attempt was made to dissect it down, when it was found to extend to the left side and under the left sterno-mastoid. The middle third of the clavicle was resected subperiosteally, and it was then seen that the tumour arose from the subclavian artery. The sac ruptured, and the bleeding was very severe; this was stopped by the finger used as a plug to close the opening. The common carotid was exposed and tied, and the vagus nerve demonstrated. There was no part of the subclavian artery which could be tied. Traction was now made upon the common carotid, the pleura pushed downwards, when the innominate artery was exposed. No great difficulty was met in passing the aneurysm needle, and the artery was tied securely with two ligatures of kangaroo tendon, side by side. This immediately stopped all haemorrhage. The sac was then removed as far as possible, and the adherent portion sewn over. The clavicle was replaced, the periosteum sewn over it and joined by kangaroo tendons.

The patient left the theatre in good condition. During the night and the next two days the right side of the head was obviously colder than the left; capillary circulation in the hand was present the next day, and the arm kept warm and comfortable.

He ran an intermittent temperature and developed bedsores and some suppuration in the wound; the centre piece of the clavicle separated. His mouth gave a good deal of trouble, and the roots and all teeth were extracted. Later he developed an empyema on the right side; a rib was resected, and the condition healed in three weeks.

He was discharged from hospital on April 10th, rapidly gaining weight and looking well.

I have to thank my colleague Mr. Cecil Burnett for his able assistance, and Dr. A. Thomson, the house-surgeon, for skilful anaesthesia in a poor subject for a long operation.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

RENAL ABNORMALITY.

(With Special Plate.)

APART from the pathological condition found, the following case appears to be interesting as an example of a physical stigma of degeneration in a congenital mental defective.

The patient was a mentally deficient lad, aged 20. He was operated on for acute appendicitis, and died, leaving a faecal fistula at the site of operation. A post-mortem examination was made, and the following are notes having bearing on the condition found.

Having failed to find the kidneys in their normal situation, it was decided to explore a pelvic tumour occupying a position in the mid-line below the fifth lumbar vertebra. This tumour was retroperitoneal and gave a doughy resistance on palpation. When the peritoneum was incised there was no difficulty in enucleating the tumour, but owing to a single attachment it could not yet be removed from the body. This attachment was traced downwards, and turned out to be a ureter. About two inches from the bladder this ureter gave off a branch from its left side, which, when it was traced upwards, was found to enter a scarcely developed left kidney which was lying against the much larger right kidney (see Special Plate, Fig. 1). The ureters therefore joined and had a common entry into the bladder. Both kidneys were now removed and examined macroscopically, microscopically, and bacteriologically.

Right Kidney (weight 238 grams).—Nodular elevations filled with pus were present on the surface. On being cut open (Fig. 2) the kidney was found to consist mainly of abscess cavities with very little normal kidney substance, and no macroscopic differentiation between cortex and medulla. The pus contained *B. coli communior* and *Strep. faecalis*. Sections showed some normal kidney substance,

enormous leucocytic infiltration of the kidney, and a streptococcus; bacillary forms did not show up on section.

Left Kidney.—Except for its size and its weight, which was 17 grams, the condition of the left kidney was similar to that of the right.

The faecal fistula communicated with copious pus in the appendicular region. There did not appear to be any direct connexion between this and the kidneys.

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INTRAVENOUS ANTITOXIN IN AN OUTBREAK OF SCARLET FEVER IN A PUBLIC SCHOOL.

SCARLET fever of a severe semi-septic type broke out among the boys of a public school in February, 1929. The first nine cases were treated by ordinary expectant methods and without antitoxin. The course of the disease in these gave rise to much anxiety, and complications were frequent, as noted in the table below.

Then occurred the worst case of all, in a boy aged 13, who, when seen by me about the seventh day of the disease, had already a severely ulcerated throat, enlarged and tender cervical glands, double acute suppurative otitis media, and acute mastoid suppuration on one side. He was given 10 c.cm. of scarlatinal antitoxin intravenously, unfortunately without effect at this stage. The sepsis developed into pyaemia with multiple purulent arthritis, involving incisions into the hip-joint, the elbow-joint, and both ankle-joints, and the double mastoid operation. The serum was given too late in this instance, and the dose was too low for such a severe case.

The subsequent patients, sixteen in number, were treated early in the acute stage with antitoxin intravenously, according to the technique which I have previously described.¹ In these sixteen cases the acute stage, though apparently of the same sharp type as the others, was arrested within twelve to twenty-four hours of the injection. Further, no desquamation occurred; the complications were limited to mild adenitis in one patient, who received antitoxin on the fourth day of disease, and one simple hordeolum. All the sixteen patients were allowed up within a week, and were discharged from isolation in about a fortnight's time. All were boys between the ages of 12 and 18, except for one master aged 49. All received the injections within the first four days of their disease, generally on the second or third day.

No ill effects were noted from the serum administration, although one boy had a moderately severe rigor, and two had hyperpyrexia for a few hours after the injection. In one case intravenous serum appeared to be contraindicated owing to a history of hay fever. In this case an intradermal test injection of 1 in 10 dilution of the serum was performed, and it did not produce any reaction. Accordingly the usual dose, 10 c.cm. of serum, was injected intravenously, and no ill effect was noted.

The following table gives particulars of the complications noted in this interesting series.

SCARLET FEVER COMPLICATIONS.

Nine Cases not treated with Antitoxin.

Suppurative otitis media with persistent otorrhoea...	2
Non-suppurative otitis media	7
Albuminuria or nephritis about third week	1
Empyema of maxillary antrum and frontal sinus	1
Dacryocystitis	1
Pneumonia	1
Jaundice	1
Adenitis	7
Nasal discharge	6
Desquamation	9

Sixteen Cases treated with Intravenous Antitoxin.

Adenitis	1
Hordeolum	1

The only case omitted from the table is the one previously noted of severe pyaemia, in which the serum was given too late to be effective.

The injections, with the exception of the first two, were made by Dr. Hubert L. Ellison, to whom I am indebted for detailed information as to the course of the outbreak.

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¹ Banks and MacKenzie: *Lancet*, 1929, i, 381.