oedema, with its characteristic profuse, watery, blood-stained sputum. This is the picture of a pure left ventricular failure. As already explained, it is due to the pulmonary congestion and to the diminished capacity of the respiratory reserve.

Such an attack may often occur during the day, following an attempt at coughing, but the alarm experienced is never as great. The fact of its occurrence at night is almost certainly due to the sudden stimulation of the depressed cough reflex by an accumulated secretion into the bronchial tree.

In right ventricular failure, on the other hand, the symptoms and signs are due to the increased back pressure in the systemic circulation. When the patient is ambulatory the first sign of this increased pressure is seen in the legs and ankles, where the patient may notice himself that on taking off his shoe at night an impression remains. If he is in bed, this oedema will be noticed first over the sacrum (but not by the patient!). Normally, when a patient is lying flat, the jugular veins stand out as cords which are easily compressible, and which may show some transmitted systolic pulsation from the auricle, but on sitting up, the veins collapse and are not seen, except, perhaps, in the root of the neck. When the systemic venous blood pressure is increased, the jugular veins stand out even in the sitting-up position, showing marked congestion, and are no longer so easily compressed. The height to which the jugular veins rise when the patient is sitting up can be taken as an indication of the degree of venous congestion. This venous congestion shows itself in other places. The small venules on the face may become distented, as will the veins coursing over the forearm. The liver takes a good deal of the initial shock of this increased pressure, becoming enlarged. This increased pressure may interfere with the normal functioning of that organ, and so lead to jaundice. If the pressure is great enough, or maintained for long enough, there will be a rise in pressure in the hepatic vein, which is then transmitted to the venous system of the abdomen. This will lead to a transudation of fluid within the abdomen, and ascites will result. In the kidney the back pressure manifests itself by the presence of albumin in greater or lesser degree. Casts containing red blood corpuscles may be found on microscopy of the urine, and if in some cases the circulation is so slowed that infarction occurs, a frank haematuria may result. (The urine is usually brick red in colour, from the presence of urates.) An examination of the fundus oculi will reveal how generally is this venous obstruction, for besides showing distended veins there may be small haemorrhages where those small veins have ruptured.

In left ventricular failure, because of the pulmonary congestion and the shallowness of respiration, cyanosis is often present, although never as marked as in right ventricular failure. In the latter, the cyanosis is due, at first, not to anoxia, but to the stagnation of blood flow which occurs on the venous side of the circulation. This is proved by the fact that the administration of oxygen does not improve the patient's colour, whereas venesection will do so immediately. Later, when pulmonary congestion becomes marked, oxygen administration will be beneficial.

(To be concluded.)

Part III.—Hypertensive disease; the diagnosis of valvular disease and a discussion on the significance of the systolic murmur.

Clinical Page

By STELLA INSTONE, M.D., M.R.C.P.

Case I

A CASE OF POLYRADICULONEURITIS

Case Note

Mrs. C., aged 32, had never had any serious illness until July 1942, when she developed gastro-enteritis. No other member of her household was affected, nor was there any local epidemic of food-poisoning at this time. Full investigations proved bacteriologically negative, and her serum failed to agglutinate any organism of the Coli-Typhoid-Dysentery-Abortus group. Her blood-count showed a leucopenia with diminution of the granular cells:
This was compatible with infection of bacterial or exotoxic nature, although no pathogen could be isolated from the urine, faeces or blood. Clinically, the illness was of a mild type, and after ten days' symptomatic treatment she became afebrile and appeared well. Three weeks from the onset of her diarrhoea and vomiting she was allowed up without ill effect until the fourth day of convalescence. On that occasion, she was walking about the room when she suddenly lost the use of all four limbs, and had to be helped back to bed. There had been no premonitory symptoms apart from a complaint the previous day of pain in the muscles of both shoulders. On examination, her appearance was seen to have changed from that of convalescence to a state of considerable weakness and distress. She lay motionless in bed with a curiously expressionless face, answered questions in a weak and toneless voice, and complained of discomfort in the throat and of difficulty in swallowing.

Her temperature was 99·6, pulse rate 108, blood pressure 110/60, and respiration rate 24 per minute. Breathing was shallow, laboured and entirely thoracic, with no appreciable movement of the abdominal muscles. The throat and nasopharynx were dry and slightly injected, the tongue moist and furred.

Examination of the nervous system revealed normal function of all the cranial nerves, with loss of all the tendon-jerks and of the abdominal reflexes. Voluntary movement was grossly impaired in all four limbs, dorsiflexion of the hands and feet being especially weak. The appreciation of light touch, pain and temperature was absent in the hands and feet and diminished elsewhere. Proprioceptive sensibility was intact, and the ocular fundi appeared normal. A slight degree of neck rigidity was present. The cerebrospinal fluid was under slightly increased pressure and showed no abnormality other than a protein content of 100 mgm. per 100 c.c. It was sterile on culture. Examination of other systems proved negative, except to show slight cardiac dilatation with heart sounds of poor quality, and a generalised impairment of air entry in the chest.

**Progress and Treatment.**—On the above findings, a diagnosis was made of acute infective polyradiculoneuritis, some involvement of the upper motor neurone being assumed from the disappearance of the abdominal reflexes.

Once established, the paralysis did not progress, and the neurological signs remained unchanged for several days, after which there was a gradual recovery of muscular power in the limbs and trunk, followed by a return of function of the nuchal muscles, which previously had been unable to raise the head from the pillow.

During the first week of this illness the temperature ranged between 98·8 and 100° F., and there was a constant tachycardia of 100–112 per minute, with a respiration rate of 22–28. The patient also complained of a painful burning rash on the palms of both hands; this consisted of bright pink circinate macules, most marked on the thenar and hypothenar eminences and over the palmar aspects of the fingers, and accompanied by increased sweating and tenderness of the affected parts. The appearance was not unlike that of the rash of pink disease, and was considered compatible with an affection of the vasomotor nerves. There was no obvious focal source of a toxic erythema, and an X-ray of the paranasal sinuses showed no infection there.

At the end of the first week the paralysed muscles began to respond to faradic stimulation, and this was considered to be a good prognostic sign.

Treatment consisted in supporting the paralysed muscles, taking the usual precautions against wrist-drop and foot-drop, and keeping the patient recumbent and upon as liberal a diet as she would take. Vitamin B\textsubscript{1} was given orally as Aneurin, 2 mgm. thrice daily. Faradism, massage and passive and active movements were given to the paralysed muscles daily from the end of the first week.

Convalescence was gradual, and signs of general and myocardial weakness were apparent for some weeks. Six weeks from the onset of paralysis the patient's general condition appeared normal, with practically complete recovery of her lost muscular power and sensation, but the abdominal and tendon reflexes were still absent and the palmar erythema had not quite faded.
Comment.—The relationship between gastro-intestinal infection and the subsequent polyneuritis in this case is open to doubt, but the infective nature of the latter illness is confirmed by the raised protein content of the cerebro-spinal fluid.

In the absence of any demonstrable septic focus, and from the clinical course of the case, it seems reasonable to assume a virus infection of the nervous system as the most likely aetiological factor.

I am indebted to Dr. A. M. C. Macpherson for permission to record this case.

CASE II
A NOTE ON THE TREATMENT OF NEPHROTIC OEDEMA

The following case is of some interest on account of the therapeutic problems presented.

Case Note
Mrs. P., aged 60, developed symptoms of diabetes mellitus six months ago, for which she was given a diet but no insulin. Four months later she noticed increasing breathlessness on exertion, which was followed by swelling of the legs, arms and abdomen to such an extent that she became unable to get about. Her sight had been failing for six months.

There was a history of 'kidney trouble' during pregnancy some thirty years ago, but there had been no recurrence of urinary symptoms until the onset of her present illness. Diet had been largely vegetarian of late; no history of alcoholism. On admission to hospital she presented a picture of extreme anasarca, with pallor, orthopnoea, ascites, and bilateral basal hydrothorax. Liver and spleen were moderately enlarged. Temperature, pulse and respiration rate were normal. The blood pressure was 190/100 mm., and there were signs of left and right ventricular hypertrophy. There was marked finger-clubbing. The ocular fundi showed silver-wire arteries, old bilateral retinal haemorrhages and advanced diabetic retinitis, and visual acuity was grossly impaired.

The urine contained sugar and albumin in considerable amount, and a trace of acetone. Microscopy showed red cells and a fair number of hyaline and epithelial casts.

Pathological Investigations
The blood showed a moderate degree of hypochromic microcytic anaemia (Hb 70 per cent), with a normal leucocyte count.

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood urea</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>100 mgm. per 100 c.c.</td>
</tr>
<tr>
<td>Blood cholesterol</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>250 mgm. per 100 c.c.</td>
</tr>
<tr>
<td>Total plasma protein</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4·5 grams per 100 c.c.</td>
</tr>
<tr>
<td>Fasting blood sugar</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>210 mgm. per 100 c.c.</td>
</tr>
<tr>
<td>Wassermann and Kahn reactions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Negative</td>
</tr>
</tbody>
</table>

Case Management
From clinical and biochemical observations it appeared probable that the gross generalised oedema in this case had a combined renal, cardiac and dietetic basis, but that the most important factor in its perpetuation was a hypo-proteinemia.

The diabetes was easily stabilised on a diet containing 150 gm. of carbohydrate, 100 gm. of protein, minimal fats and restricted fluids and salt, together with protamine zinc insulin,
Because of the rapidity with which mustard gas penetrates the tissues, ordinary chlorine preparations such as chloramine-T or bleach ointment must be applied immediately after contact to give any chance of preventing resulting blistering or ulceration.

Azochloramid in Triacetin 1:500, on the other hand, has been shown by carefully controlled experiments to penetrate some distance into the affected tissues, and to render innocuous the mustard gas which has preceded it. Azochloramid in Triacetin even if applied some time after contamination with the gas (up to several hours later) may be expected to reduce ulcer formation, if it does not actually prevent it.

With regard to treatment of mustard gas blisters, one authority has reported that, whereas the use of an agent such as chloramine-T will often effect healing in 4 to 6 weeks, the application of Azochloramid in Triacetin 1:500 appears to cure in as short a period as 2 to 3 weeks. It also limits the severity of the lesion and the amount of resulting scar tissue.

Azochloramid is a powerful yet innocuous surgical germicide distinguished from ordinary chlorine-liberating antisepsics by its remarkable stability, even in the presence of pus, serum, etc.

Azochloramid solutions are an essential item in the equipment of Decontamination Centres and First-Aid Posts.

Full information available on request to:
The Medical Department
WALLACE & TIERNAN, LTD.
Power Road, London, W.4
Telephone: CHIswick 6640

20 units, and soluble insulin, 15 units, daily. Ferri. ammon. cit., gr. xxx, was given thrice daily to improve anaemia.

During the first month of treatment an attempt was made to reduce the anaasarca by giving large doses of alkalies (potassium citrate, gr. xc, four times daily), with saline purgatives and vitamin B1 (Aneurin, 2 mgm. tds.). These measures produced no appreciable improvement, and it was therefore considered justifiable, despite the presence of diabetes and of chronic nephritis, to resort to local treatment. Multiple incisions were therefore made, not without trepidation and not without strict asepsis, into the skin of both legs and dorsal aspects of the feet. During the ensuing week the patient drained 50 pints of fluid through the incisions, which healed aseptically in due course (possibly helped by ascorbic acid, 50 mgm. daily).

In order to consolidate this improvement it was decided to give 50 c.c. of quadruple-strength plasma by slow intravenous infusion. Following this procedure, the plasma protein which had fallen to 2·5 grams percent after the above-drainage fluid, rose to 5·5 grams percent. The blood urea was now 70 mgm. per cent, and it was thought reasonably safe to commence weekly intramuscular injections of Salyrgan, 2 c.c.

It was noted that the diuretic effect of these injections was markedly increased if vitamin B1 was given for three days before and after the Salyrgan. A high protein diet of adequate vitamin content was continued throughout the treatment of this case. No ill effects were noted from therapy with mercury and after six weeks after making the skin incisions there was no significant return of the oedema, and the patient felt very much more comfortable, with no obvious dyspnoea. The blood pressure remained unchanged, as did the finger-clubbing; the latter would appear to be the result of chronic local anoxaemia following upon tissue oedema.

Comment.—This case is considered to be an example of the Kimmelstiehl-Wilson syndrome of diabetes accompanied by the haemolytic type of nephritis. There is, however, some nitrogen retention, as shown by the raised blood area, and there seems little doubt that the renal damage will prove to be progressive. Despite the bad prognosis, the symptomatic relief afforded by the above treatment seems to justify the measures employed.

My thanks are due to Dr. Ursula James for permission to record this case.