I have seen many cases of neuritis and lumbago cured by the use of ultra-violet light. I believe such cases are due to a specific anaphylaxis excited by proteins absorbed from the bowel while it is in a state of catarrhal inflammation. The sudden incidence is probably due to exercise creating a local acidosis from the lactic acid produced in the muscles, which tends to make the cells more sensitive to the proteins.

At least one of the three cases I have cited was a pure example of anaphylaxis, and, in her, peptone failed completely, whilst ultra-violet irradiation was a complete success, as it was in the other cases. Now it has been demonstrated that exposure to the sun's rays causes a general hyperleucocytosis with a relative neutropenia and a strong relative lymphocytosis. I frankly admit that this is comparable with the leucocytosis produced by the injection of peptone, and it is quite possible that this helps to account for the anti-anaphylactic action of ultra-violet light rays. But it is only one of the remedial results of ultra-violet light, and probably the least effective; this doubtless accounts for the frequent failure of peptone, and in any case for the short period of protection it affords. Another valuable therapeutic effect of ultra-violet rays is their directly germicidal action on the body, increasing the bactericidal powers of the blood, thus reducing the formation of bacterial toxins.

Probably the most useful function of ultra-violet rays is their power to increase the absorption of calcium and phosphorus, which, with the formation of vitamin D from the cholesterol in the skin, is doubtless the main factor in the cure of rickets. This also leads not only to the better nutrition of the bones and nervous system, but also to the alleviation of spasm and the induction of a calmative effect on the nervous system. Nor must it be forgotten that there is an increase of iron in the blood and a decided rise of haemoglobin in the erythrocytes, diminishing the risk of dyspnoea and asphyxia, so potent a factor in asthmatic conditions.

Finally, the internal secretions of the thyroid and adrenal glands are increased, and they are both valuable in the treatment of asthma and allied conditions.

It will be observed that the ultra-violet light treatment of asthma counters the varied factors in its causation, and is valuable, not only for cure, but also for prophylaxis.

A METHOD OF ESTIMATING CARDIAC OUTPUT.

BY

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THE problem of the correct treatment of heart disease reduces itself largely to cardiac output. The common type of heart failure means diminished cardiac output, and we shall never be able to tackle the problem of heart failure properly unless we have a reliable method for determining the output of the heart.

The following is a description of a method for estimating cardiac output. The underlying principle is simple. The venous pressure is measured by inserting a needle, preferably in the cephalic vein of the arm. A given number of heart beats are admitted. Then the pressure is taken again, and from the increased venous pressure we calculate the output per beat, or, if desired, per minute. It will be seen that the principle underlying this method is the determination of the single output by variation of the venous pressure. Particulars are as follows.

The pneumatic tourniquet of a sphygmomanometer is placed on the arm; a needle with as wide a bore as possible is inserted in the cephalic vein and connected with a manometer containing a solution of 0.7 per cent. sodium chloride and 0.5 per cent. citric acid. The venous pressure is now taken. Then the artery is compressed very rapidly by means of the airbag so as to obliterate completely the brachial artery. The venous pressure is taken again, after which the arterial blood is allowed to enter the limb for the

space of 5, 10, or 15 beats by relaxing the airbag. At the same time the pressure must remain high enough to prevent the venous circulation from being re-established.

A record of the number of beats which were allowed to pass into the lower part of the upper extremity. It will be seen from this chart that one is able to recognize those beats which do not completely pass through and to account for them in calculation.

The pressure in the veins is now taken again. It will be seen that it is necessary to take the venous pressure three times: first, when the circulation in the veins as well as in the arteries is free; secondly, when the circulation is cut off in the veins as well as in the arteries; thirdly, after a known number of beats have been admitted, the venous circulation still being cut off. The first and the third measurements are essential for our purpose, the second serving as control only.

The following readings are typical:

Venous Pressure Experiment.

V.P.=4 inches.

After compression=9 inches.

After eight beats (see chart)=23 inches.

∴ Rise=14 inches.

=1.75 inches per beat rise.

Case of specific carditis. Wassermann reaction ++; blood pressure 145/105; pulse rate at time of experiment=120 per minute.

It remains to say a word in regard to the counting of the beats, which is not so simple as might be imagined. The only satisfactory method of getting a reliable count is to place the pad of a Mackenzie's polygraph on the radial artery and record the beat by means of the needle on a photographic slide, such as is available in the electrocardiograph.

I am indebted to Dr. Crawford, assistant physician, Liverpool Heart Hospital, for valuable assistance in carrying out the experiment.

THE PROGNOSIS IN FRACTURES OF THE OS CALCIS.

BY

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IN any case of fracture two questions must be answered: How long will the patient be incapacitated for work and require compensation? and To what extent will he be permanently crippled?

The investigation here reported was undertaken to determine the duration of the disability, and the degree of final impairment of function in cases of fracture of the os calcis. Twenty-five such cases were treated at the Northern Hospital, Liverpool, in the period 1921 to 1924. Of these, only fourteen reported for re-examination after completion of treatment; they were all men, and their average age was 45 years. In every case the fracture was caused by a direct fall on the heel.

For comparative purposes the period of disability was taken as the time between the date of the accident and of the man's resumption of a permanent occupation; in most cases this was determined by proceedings under the Workmen's Compensation Act. The average was eleven months. In seven cases the period was between eight months and a year; the shortest period was three months, and the longest twenty months.

The final results were difficult to assess. All the patients were ultimately able to resume moderately heavy work. None became industrial cripples. All complained of pain in the heel when running, or walking fast on uneven surfaces, such as cobble-stones, or in damp weather. In

one patient these symptoms had persisted for seven years after the fracture.

To elucidate the factors determining the disability, I compared the clinical and radiological appearances in those patients who did well and returned to work within six months, with those of the patients in whom the disability persisted for more than twelve months. Certain factors seem to have no bearing on the prognosis; all cases showed varying degrees of callus excess below the malleoli; a slight valgoid deformity was present in two cases only. The presence of a plantar spur was common, but in one case only did it seem to cause part of the pain and require the use of an excavated heel, which partially relieved symptoms.

Comparison, however, of the range of movements of the foot and the radiological appearances led to certain definite conclusions.

In the group of cases with a short period of disability the movements of inversion and eversion of the foot were, with one exception, grossly limited, but free from pain. Radiological examination showed extensive comminution of the os calcis and osseous ankylosis of the astragalo-calcaneal joints. In the one exception the joint surfaces were intact, and only the lower part of the bone had suffered compression.

In the second group, of inveterate cases, the movements of the foot had a freer range, but were attended with pain on full eversion. The radiological appearances showed relatively slight amount of bony damage. In one case, indeed, the lamellar structure of the bone was completely restored five months after the accident, though with no corresponding clinical improvement. A constant feature of all the inveterate cases was the irregularity of the articular surfaces for the astragalus. In no case was bony ankylosis present.

The radiological findings and the pain on movement point

to the condition of the subastragaloid joints as being the determining factor in prognosis. At these joints occurs the second half of the movement from full inversion to full eversion of the foot, a movement elicited only by exertion, and effected by a vertical rotation of leg and astragalus on the os calcis. An osseous ankylosis of these joints produces a permanent but painless limitation of this movement, a loss which is by no means crippling; one man, indeed, returned to his work as a window cleaner in four months. On the other hand, a traumatic arthritis of these joints is perpetuated by any movements requiring eversion of the foot. As is always the case, fibrous ankylosis is more painful than osseous.

Conclusions.

From this confessedly small series the following conclusions arise:

The average period of disability after fractures of the os calcis is eleven months. The length of the period depends on the condition of the subastragaloid joints, and not directly on the amount of bone damage.

When these joints are ankylosed as a result of the fracture the foot improves in six months to an optimum stationary condition, with a limited but painless range of eversion of the foot.

Should the joints escape entirely the prognosis is so much the better, but when an incomplete fibrous ankylosis results from the fracture, improvement is slow and eversion is often permanently attended by pain.

The principle attributing most of the residual disabilities of fractures to implication of neighbouring joints is well exemplified by this series of cases.

My thanks are due to Mr. McMurray, honorary orthopaedic surgeon of the Northern Hospital, for his help, and permission to examine many of his cases, and to Dr. Oram for the use of the radiograms.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

A FATAL CASE OF ACUTE PULMONARY OEDEMA.

A NUMBER of cases of acute pulmonary oedema, most of which have fortunately responded to the recognized treatment, have recently been reported. The following fatal case seems worthy of note.

A factory girl, aged 16, was brought to the East London Hospital for Children in the early morning of January 14th, 1926, in a collapsed and breathless condition. She had done an ordinary day's work, had been out for a walk after supper, and went to bed at 11 p.m. perfectly well. At midnight her sister, who was sleeping in the same room, was awakened by her vomiting and gasping for breath. She was hurried to the hospital, and I saw her immediately on her arrival.

She was a well-nourished girl; she was very restless, rolling from side to side, and clutching at her throat. She was cyanosed, and her expression very anxious. Frothy, straw-coloured fluid was streaming from her mouth and nose. The heart was somewhat enlarged, and a systolic and presystolic murmur was heard at the apex; the heart rhythm was regular, and the pulse 80. The bases of the lungs were full of bubbling râles. The liver was not enlarged, and the spleen not palpable. There was no oedema of the ankles. A hypodermic injection of atropine 1/100 grain and morphine 1/4 grain allayed her restlessness, but she complained of intense thirst, although frothy fluid was streaming from her mouth. Two hours after the onset of her symptoms she died.

I ascertained from her mother that three years previously she had been in an infirmary for "heart trouble," but had led a normal and healthy life since then without any symptoms at all.

At the *post-mortem* examination, conducted the next day by Dr. Temple Gray, pathologist to the hospital, the heart was found to be enlarged and fatty, and there was definite mitral stenosis. The lungs were sodden, and large quantities of fluid exuded from them. Frothy fluid was also found in the bronchi.

The point worthy of particular note is that the rhythm of the heart was regular throughout the attack. The question arises as to what was the cause of the sudden onset of the alarming and fatal symptoms.

I am obliged to the resident medical officer, Dr. A. G. Ogilvie, for permission to publish this case.

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EXTENSIVE NECROSIS IN CHICKEN-POX.

THE following case of necrosis complicating varicella presents some unusual features which make it worthy of record.

On December 24th last I saw a female child, aged 2 years and 9 months, who had contracted chicken-pox from her sister, aged 8. The temperature was 103°, the pulse 132, and the tongue was coated, but the throat was normal. She objected to being moved. There was a slightly reddened, tender patch over the lower part of the right shoulder-blade alongside a vesicle. There were not more than twenty vesicles in all on the body and head. The heart and respiratory sounds were normal. Ichthyol ointment (20 per cent.) was applied on lint covering a wide area; the bowels were moved, and antipyrin prescribed in small doses every four hours. The following day the temperature had fallen a degree, but the general condition had not improved. The area of redness over the right shoulder-blade had increased and there was a wide margin of tenderness beyond.

During the succeeding forty-eight hours the condition grew worse. The affected area became discoloured and the patient more toxic. No further vesicles appeared. The temperature varied from 100.5° to 102°. The child was very fretful, restless, and rambling in her talk, and slept badly. On the fourth day she did not appear to see distinctly. She was, however, taking Benger's food and fluids in abundance. On this day I asked Dr. A. Whyte Cassie to see her in consultation. A small necrosed patch, rather smaller than a shilling, was then present at the original site of the inflammation, and below this the skin was discoloured. Tenderness and muscular spasm extended to the vertebral column posteriorly and to the middle line of the chest and abdomen anteriorly. There was tympanites and constipation. Dr. Cassie advised that the treatment should be continued, and small doses of brandy given.

The necrosed patch increased in size daily. There was no suppuration, but the body became dusky and mottled. Wet boric dressings, changed twice daily, were applied. By the morning of December 30th the patch, roughly oval in outline, measured about 1½ by 3 inches. On December 31st I asked Dr. Seiller, the medical officer of health of the district, to see her. The necrosed patch was then double the size of the preceding day and was beginning to separate at its upper margin. He advised continuance of the boric soaks, but felt, with me, that the prognosis was very bad. As a last resort I injected 7½ c.c.m. of antistreptococcal serum about 10 p.m., but the child died between 2 and 3 a.m. on January 1st.

The points of interest manifest in the case are: (1) The virulent, though not widespread, infection conveyed to the patient by her sister, a mild case seen sixteen days previously, who was never a day in bed. (2) The house and children were spotlessly clean; necrosis complicating