Pruritus.

Results, despite fantastic claims to the contrary, are most disappointing.

Psoriasis. Stimulative. (15 per cent.)

Results, if observed over long periods, are usually unsatisfactory, however good the initial response.

Rickets. (a) Sedative, (b) Stimulative. (90 per cent.)

The efficient formation of vitamin D can best be started by small doses, especially in cases with extensive changes.

Synovitis.

No general guide can be laid down, as the nature and present condition of the synovitis are of prime importance in selecting treatment.

Ulcers. (a) Sedative, (b) Stimulative. (30 per cent.)

Moderately small doses should be tried first; if no response, chronic ulcers are well worth treating with counter-irritant doses.

The foregoing list, which even after many "comb-outs" still contains twenty-seven conditions, includes only seven in which I have estimated ultra-violet to have a value of 75 per cent. or over. That the valuation is approximately correct I have little doubt, just as I have complete faith that ultra-violet light treatment constitutes a valuable advance in modern therapy if a sober view is taken of its field of activity.

Conclusion.

Before drawing this review to an end I would like to refer briefly to a small investigation that has just been completed, thanks to the facilities given me by Professor McLroy, and to the unsparing work of Dr. Bunbury, my late clinical assistant. In this investigation the mothers of every alternate baby born in the maternity wards at the Royal Free Hospital from January to June were given a series of doses of light, and record was kept of the weights of their babies at birth, at 11 days, at 6 weeks, and at 12 weeks. The remaining babies constituted the controls. In this way approximately 1 ton weight of babies have been observed. Though the final figures have not yet been collated it is possible to state that the increase of weight of sixty-five babies whose mothers had had irradiation was *plus* 28.5 per cent. at the end of six weeks, and of a similar non-irradiated group *plus* 29.3 per cent. All other figures are on the same scale, and indicate how careful one must be before claiming that "light treatment" is "wonderful" for nursing mothers or for any other purpose.

In the near future, however, I hope to see such genuine knowledge of this subject that prescriptions with full instructions as to lamp, distance, dose, area of skin exposed, etc., will be the rule rather than the rare exception. I hope also that when these prescriptions are not dispensed by the doctor they will be given in writing, as an ordinary prescription is given to a chemist, to workers on a national roll compiled as recommended in Cardiff by the Representative Body.

ABDOMINAL ANEURYSM CAUSING THROMBOSIS OF THE INFERIOR VENA CAVA.

BY

KENNETH P. FOOKS, M.B., CH.B.,
BIRMINGHAM.

THE following case is worthy of being recorded by virtue of the rarity of the condition, the very unusual course of the disease, and the difficulty experienced in reaching an accurate diagnosis until a catastrophe preceding a fatal termination made it obvious.

An engineer, aged 35, was admitted to the General Hospital, Birmingham, on August 21st, 1928, giving the following history. In 1912, at the age of 18, having previously been in good health, he was treated for iritis, which, he stated, followed a blow on the eye. The iritis cleared up rapidly under treatment, the nature of which is not known. Two years later he was admitted to this hospital, under the late Dr. Saundby, complaining of swelling of both legs, the onset being sudden and accompanied by pain in the legs. A diagnosis of bilateral femoral thrombosis was made, but no local or general cause was discovered. There was no clinical evidence of syphilis, but the Wassermann reaction was not determined. After some weeks the legs ulcerated and the oedema diminished. During the ensuing six years he had several transient recurrences of oedema, but no pain occurred with these

attacks. In 1918 he began to have difficulty in walking of the type associated with intermittent claudication. This symptom persisted to a variable extent for the rest of his life. In 1920 large veins appeared over the abdomen and the ulcers healed. A year later he was readmitted, under Dr. Stanley Barnes, complaining of severe aching pains in the lower part of the back, and he was found to have the large superficial venous collateral circulation typical of obstruction of the inferior vena cava. He was tender over the lower lumbar spines, and had a bilateral extensor plantar response. The Wassermann reaction was negative. Thrombosis of the inferior vena cava, with a light degree of myelitis, was diagnosed, the myelitis being thought to be due to a local impoverishment of the blood supply secondary to the thrombosis. His pain disappeared under electrical treatment, and he returned to work. He remained free from pain until 1927,

when he had a recurrence of symptoms, of a short duration, similar in every respect to those of 1921.

Four weeks before admission he had a similar recurrence of pain of very great severity, especially located in the left side of the abdomen. The pain caused complete insomnia, and after two weeks of acute distress a swelling appeared in the left groin, followed by numbness and uselessness of the left leg. Violent nocturnal stabbing pain arose, shooting down the left thigh.

On examination (August 21st, 1928) extensive symmetrical pigmented papery scars were found on both legs. Large tortuous veins coursed over the abdomen from the groins to the substernal notch and the axillae, the blood flow in every case being upwards. A diffuse, solid, tender swelling occupied the left groin, and the left leg was flexed at the hip, any attempt at extension causing violent pain. No pulsation could be detected in the arteries of the lower extremities except a feeble intermittent pulse in the left posterior tibial. There was no oedema of the legs. The radial pulses were regular, rapid (120), and of high tension, the blood pressure being 160 systolic and 106 diastolic. The abdomen was

uniformly distended and tympanitic, prohibiting palpation of its contents. There were now no signs of myelitis.

Thirty-six hours after admission he had an attack of agonizing pain shooting down the left leg, the abdominal distension increased, and vomiting and collapse occurred. From that time until death the pain recurred at intervals, necessitating the frequent use of morphine, and the radial pulses became very thin and rapid, being imperceptible at times. The swelling in the groin gradually diminished, and the left common femoral pulse was felt for a few hours on the third day. On the fourth day a large mass became visible and palpable in the left side of the abdomen, extending from the costal margin to the iliac crest, and from the left kidney region to within three inches of the umbilicus, and exhibiting pulsation of an expansile character. A soft systolic murmur was audible over the swelling. An x-ray examination at this time showed no sign of abdominal aneurysm, but a lateral view was impossible. The swelling increased steadily in size until it filled the greater part of the left side of the abdomen and the meteorism subsided. A spreading discoloration of the skin of the left loin and groin appeared. The day before death, which occurred a week after admission, a collection of fluid was detected in the left pleural cavity, but it was not tapped, as the patient was dying.

At the necropsy he was found to have, at the bifurcation of the aorta, a large sacular aneurysm, which had eroded the bodies of the fourth and fifth lumbar vertebrae to within half an inch of the spinal canal. The posterior wall of the aneurysmal cavity was formed by this eroded surface, and on the left side the junction of the sac with the lip of the vertebral hollow had broken down, allowing the escape of blood into the retroperitoneal tissues, where a huge haematoma was formed. The swelling in the left groin was due to this extravasation of blood tracking down the psoas muscle. The inferior vena cava was indistinguishable two inches below the renal veins, where it became stretched out over the sac of the aneurysm as a fibrous band. The femoral and iliac veins were represented by thick fibrous cords partly recanalized by small vessels running outwards near the bifurcation of the aorta, apparently into the lumbar veins. The arteries of the legs were patent, but a loose mass of laminated clot was partly blocking the openings from the aneurysm into the common iliac arteries. The heart was normal, and the aorta showed no aortitis or atheroma. The other organs were examined and found to be healthy. No signs of syphilitic infection were found. In the left pleural cavity a quantity of blood was discovered that had tracked up through the crura of the diaphragm.

Comment.

The condition revealed at the necropsy explains the course of this distinctly puzzling case.

There can be little doubt that, fourteen years before death, a developing aneurysm of the abdominal aorta caused thrombosis of the inferior vena cava, with its subsequent train of symptoms, and, slowly eroding the vertebrae, gave rise to the attacks of lumbar pain. The myelitis and intermittent claudication must have been due to a deficient arterial supply from the vessels partly obstructed at their origins by the mass of clot in the sac. The sac then broke away from the vertebrae posteriorly, allowing leakage into the left psoas sheath. More extensive tearing preceding death caused the formation of a large false aneurysm, rendering the condition obvious for the first time.

Apart from the rarity of this condition several points of interest arise. In the first place the etiology of the aneurysm is difficult to determine. With the possible exception of the iritis no evidence, clinical or pathological, of syphilitic infection was present. The aneurysm itself, however, was of the sacular variety, and, in the absence of any possibility of occupational trauma, must be considered to be syphilitic. The onset before the age of 20 implies that the infection was congenital or neo-natal in origin, although no stigmata of congenital syphilis were present. It was unfortunate that the grave condition of the patient prohibited the taking of a second Wassermann reaction.

The slow progress, fourteen years, and the absence of lumbar pain, except for two short periods, are remarkable when the degree of bone involvement is considered.

Lastly the absence of any sign of erosion on an antero-posterior radiograph is noteworthy, and suggests the desirability of a lateral view in cases of "backache" of obscure causation.

I have to thank Dr. Stanley Barnes for permission to publish these notes.

OCCUPATIONAL PRESSURE NEURITIS OF THE DEEP PALMAR BRANCH OF THE ULNAR NERVE.

BY

WILFRED HARRIS, M.D., F.R.C.P.,

SENIOR PHYSICIAN TO ST. MARY'S HOSPITAL AND TO THE HOSPITAL FOR
NERVOUS DISEASES, MAIDA VALE.

PRESSURE paralysis of the whole ulnar nerve is not uncommon, either as the result of pressure of the head of the humerus in the axilla following dislocation of the shoulder, or as the result of pressure upon the nerve during sleep, or under prolonged anaesthesia, from the forearm being kept acutely flexed at the elbow, or resting against the sharp edge or back of a chair. It is rare for the nerve to suffer from the results of pressure in the forearm itself, though a certain degree of neuritis may accompany Volkmann's ischaemic myositis as the result of too tight bandaging of the limb. As the result of prolonged pressure in the palm of the hand upon the median nerve branches I have seen numbness and partial anaesthesia of the three middle digits; and prolonged repeated pressure, as in floor scrubbers, or in gardeners, from the use of the trowel, may cause atrophy of the thenar eminence from an occupation neuritis of the branches of the median to the opponens and abductor pollicis.

Until recently I have never seen a pressure neuritis of the deep palmar branch of the ulnar, nor do I know of any description of such condition, but two cases which I saw within a week were so clearly limited to this nerve branch that possibly its apparent rarity may be due to want of recognition. Both cases were identical in the type of paralysis and in the complete absence of complaint of pain or of anaesthesia. The first patient was a bootmaker, whose constant use of one weapon, a knife, in cutting out the leather, had raised horny callosities of the skin of the palm on the inner side of the hypothenar eminence. The other was a motor-cyclist, whose hobby was long week-end rides on a high-powered machine, and whose right hand had suffered from his trick of gripping the handle-bar and controls too tightly. In each case the paralysis was limited to abduction and adduction of the three outer fingers, and adduction of the thumb. There was no wasting of either the thenar or hypothenar eminence, and no weakness of abduction or opposition of the thumb and little finger, but great difficulty was experienced in writing and in picking up a small coin or pin. Neither the first, second, nor third fingers could be moved laterally in abduction or adduction, nor could the little finger be adducted towards the ring finger, nor could the thumb be adducted towards the index, though abduction of the little finger could be performed normally.

It will be remembered that the deep palmar branch of the ulnar nerve supplies the adductor pollicis, the inner head of the flexor brevis pollicis, all the interossei, and the two inner lumbricals. The four dorsal interossei abduct the index and ring fingers away from the middle finger, and move the middle finger laterally, in both directions, while the outer two palmar interossei adduct the first and ring fingers towards the middle finger, and the third palmar interosseus adducts the little finger towards the ring finger. The deep palmar branch of the ulnar, after supplying the hypothenar muscles, passes deeply to follow the course of the deep palmar arch, crossing the metacarpal bones just below their heads. It is here that prolonged and repeated pressure against the inner side of the palm damages the deep palmar ulnar branch, and the recognition of this condition depends on the loss of the lateral movements of the three middle fingers, and of adduction of the thumb and of the little finger, while abduction of the little finger remains normal, and there is no pain or sensory paralysis. A rare abnormality in the nerve supply of the two outer dorsal interossei is sometimes met with, as I have seen on three occasions, when these are supplied by the median nerve, and consequently in such a case these two muscles would escape paralysis from pressure on the deep palmar ulnar branch.