have been used. An additional coat of solution is then applied to enhance the final appearance.

The splint is now ready for removal from the cast. This is done by cutting it in two halves down the sides in such a situation as is deemed suitable, bearing in mind that the splint will be worn in these two halves buckled together. The splint is pulled off the cast, the adherent plaster inside scraped off and a further layer or two of solution placed inside to give a smooth finish. Whilst this is drying the halves of the splint are placed on the cast to counteract a tendency for this layer to contract and narrow the splint.

The next stage is fitting the splint on the patient. The halves are placed in position and the upper and lower ends marked and later trimmed with strong scissors or a knife. Any other places which require trimming are then dealt with.

A number of quarter-inch holes are drilled in the splint for ventilation purposes (plate V, figure 10) and it is handed to the shoe-maker for completion. The edges of both halves of the splint are bound with leather which is stitched on, and straps and buckles are placed on the splint in suitable positions (plate V, figure 6). It is convenient to have one side of the splint fastened with straps and buckles and the other permanently attached by leather straps acting as hinges (plate V, figure 9). When a splint for the leg and foot is made in this way a strip must be cut off its medial side on the dorsum of the foot to allow ease in applying and removing the splint (plate V, figure 8). In splints for the hand and forearm a separate opening is necessary for the thumb which should not pass out between the halves. When making a shell for a sacro-iliac belt or a spinal jacket it should be heavily moulded into the soft parts above the iliac crests (plate V, figure 5).

Summary

(1) Brief mention is made of the materials commonly employed in the manufacture of splints, and attention is directed to certain disadvantages in the use of plaster of paris.

(2) The advantages of celluloid for making splints are discussed, and the type of case in which this medium is particularly useful is indicated.

(3) A method of constructing celluloid splints is described in some detail.

I have pleasure in acknowledging my indebtedness to Dr. W. B. Roantree, Chief Medical Officer, Kolar Gold Field, for his kind help in preparing this article, and my thanks are due to Messrs. John Taylor & Sons, the Managers, for permission to publish it.

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SIMMONDS' DISEASE

By R. VISWANATHAN, B.A., M.D., M.R.C.P. (Lond.), T.D.D. (Wales)

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IN 1907 Paulesco demonstrated that removal of that pituitary gland caused weakness, loss of weight and death. In 1914 Simmonds reported the first case of the disease called after his name. A woman aged 38 years, after recovering from puerperal sepsis, developed amenorrhœa, increasing weakness, progressive loss of weight, anæmia, and premature senility. He put it down to atrophic degeneration of the anterior pituitary, due to septic emboli. Adequate reviews and compilations of Simmonds' disease have appeared in the medical literature during recent years, notably by Silver (1933), Calder (1932) and Graubner (1925). Farber (1940) reported a case in which he found *post mortem* marked microsplanchnia of all organs, and a pituitary stalk tumour (cystic epithelioma) which was evidently causing pressure atrophy of the anterior pituitary. Though previous reports were mainly based on post-mortem diagnosis, during recent years cases have been reported which successfully responded to replacement therapy with anterior pituitary extract. Dunn (1936) and Moehlig (1936) have reported cases of cure effected by anterior pituitary hormone therapy. Bergmann (1934) described a number of cases of emaciation and leanness which responded to pituitary.

Ætiology.—Post-mortem finding of anterior pituitary degeneration or atrophy is proof positive of the ætiological factor operating in Simmonds' disease. The atrophy is caused not only by septic emboli, as in Simmonds' own case, but also by syphilis, tuberculosis, pressure by tumour, etc.

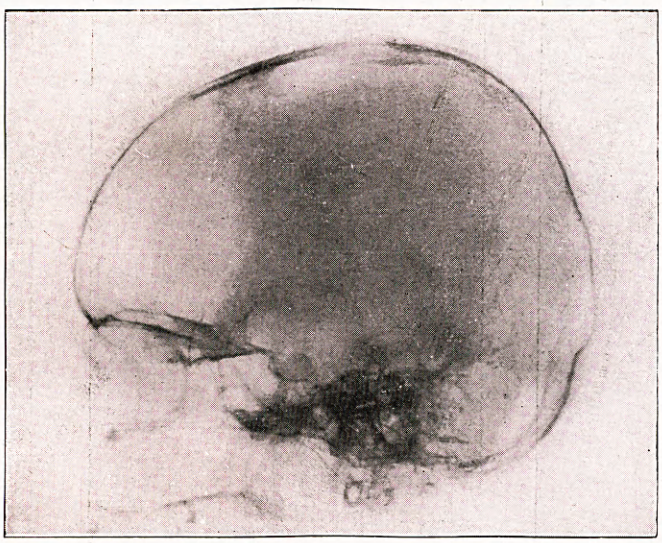
Symptomatology.—The disease starts insidiously usually during maturity, characterized by cachexia of a remarkable degree. Wasting is so great that the patient is reduced to practically skin and bone. Premature senility sets in with loss of, or grey, hair, falling out of teeth, and dry, wrinkled, toneless skin. There is subnormal temperature. Pulse is slow and feeble. Blood pressure is very much below normal. Sugar tolerance and basal metabolism are diminished. The patient usually does not exhibit any sensation of thirst or hunger.

DESCRIPTION OF PLATE V

Fig. 1.—Strips of lead bandaged in position for making a shell for a spinal jacket.
Fig. 2.—Cutting over the strips of lead.
Fig. 3.—The shell before removal.
Fig. 4.—The shell after removal.
Fig. 5.—Completed preliminary shell.
Fig. 6.—Celluloid jacket for high dorsal tuberculosis of spine.
Fig. 7.—Cast for a leg splint.
Fig. 8.—Completed splint for quiescent tuberculosis of knee.
Fig. 9.—Leg splint showing hinge-straps and buckles.
Fig. 10.—Leg splint in use.

PLATE VI

SIMMONDS' DISEASE : R. VISWANATHAN



Case of Simmonds' disease. Note destruction of clenoid processes, and the round shadow in the sella turcica.

Excretion of water is reduced. Finally, the patient becomes comatose and dies.

Treatment is as a rule unsatisfactory. But successful results with substitution therapy have been reported during recent years.

The case reported below is in all probability one of Simmonds' disease though post-mortem confirmation could not be obtained unfortunately. *X*-ray findings and the clinical course of the disease however are definitely in favour of the diagnosis.

Case report

Hindu female, aged 50 years, was admitted into the King George's Hospital on 13th August, 1941, for weakness and loss of weight of two months' duration.

Three years back she suffered for three months from continuous fever of undetermined origin. Administration of a dose of snake venom is supposed to have cured her.

Three months back she developed pain in the right hand spreading gradually to the whole of the right upper extremity. This was followed by slowly developing monoplegia. A month later she developed hiccough and vomiting. Her periods had stopped for five years.

Condition on admission.—The patient was in an extremely emaciated condition, lethargic, apathetic, and slow in answering questions. There was no evidence however of any psychic disturbance. Muscular power was weak in the right lower limb and lost almost completely in the right upper limb. There was also slight facial paresis on the right side. Deep reflexes were brisk on the right side. Babinski's sign was positive on the same side. The precordial area of dullness and the area of liver dullness were markedly diminished. Pulse was of low volume and tension, rate being 58 per minute. Blood pressure was 90 systolic and 70 diastolic. There was no sugar or albumin in the urine. Hæmoglobin was 70 per cent. Blood: Wassermann was strongly positive.

C.S.F.—Wassermann positive, moderate; proteins 35 mgm. per 100 c.cm.; globulin present; Langes's test 002221000.

Fundus examination report.—Both discs show signs of optic neuritis. Margins blurred—vessels dilated, tortuous and dark in colour. They are buried here and there by the exudates on the discs. Lamina cribrosa is filled up by exudates.

X-ray examination.—Chest: heart narrow and vertical. Liver shadow smaller than normal. Skull: bones thinner than normal. Pituitary fossa larger than normal. Anterior glenoids destroyed. A dense oval shadow seen in the region of the sella turcica confirmed by stereoscopic pictures.

B.M. rate.—30 per cent.

Glucose tolerance test.—

Before giving glucose	..	91.7 mg. per cent.
½ hour after 30 gm. glucose	..	184.4 „ „
1 „ „ „ „	..	212.8 „ „
1½ hours „ „ „	..	232.6 „ „
2 „ „ „ „	..	213.8 „ „

Progress.—Vomiting stopped within a few days after putting her on 5 units of insulin and intravenous glucose, but, in spite of feeding, cachexia became progressively worse. A state of complete prostration developed; urine and stools were passed in the bed though there was no incontinence. The patient was taken home in a moribund condition. Information was received that she died a week later.

Discussion.—Progressive extreme emaciation, amenorrhœa, increasing lethargy leading to semi-coma and ending in death, the presence of a shadow in the region of the sella turcica as

(*Concluded at foot of next column*)

SULPHONAMIDES IN TOPICAL APPLICATION

By H. J. HAMBURGER, M.D.
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THE topical application of sulphonamide compounds is a clinical method of immediate practical importance. A short survey of data from the available literature seems desirable. I am in a position to supplement such survey from my studies on sulphonamides in local use, begun in 1937.

Literature.—As compared with the scores of papers on sulphonamides and related compounds as systemic remedies, the topical employment of the drugs occupied only few workers up to 1940. That is understandable, if we recall that originally sulphonamide was designed as treatment against systemic affections, such as, for instance, septicæmia of streptococcal origin. Here, an even distribution of the chemical throughout the infected organism was required. Wherever the bloodstream is carrying the infection, there sulphonamide action is necessary. But there are circumscribed conditions, in which the full concentration of the therapeutic agent is desired on the spot. If the blood supply thereto is insufficient, as indeed the physiology of the diseased body endeavours to arrange around big purulent cavities, in order to protect itself against invasion by the germ, then topical application offers advantages. At any rate, from the outset of clinical employment of sulphonamides their usefulness in this particular way of administration was revealed.

Small sports injuries, cuts, abrasions were treated prophylactically with prontosil by Jaeger (1936). Other earlier papers on the subject have been reviewed by Hamburger

(*Continued from previous column*)

shown by radiographic examination—low basal metabolic rate and diminished sugar tolerance—are points in favour of a diagnosis of pituitary cachexia which goes by the name of Simmonds' disease. The possibility of Addison's disease is ruled out by the absence of pigmentation.

In this case the atrophy of the anterior pituitary is due to pressure by the tumour as evidenced by the *x*-ray picture. Owing to the positive Wassermann reaction of the blood and of the C.S.F. and the presence of an upper motor neurone lesion, it is possible that the mass causing the pressure was a gumma.

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