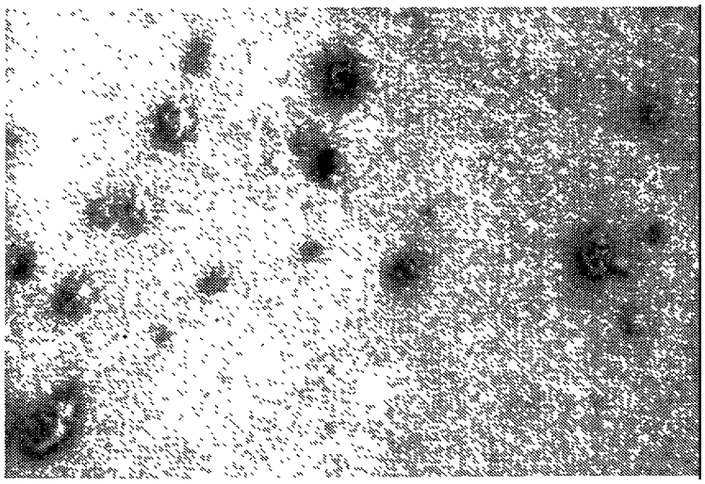

fig. 2-Rash on back of thorax.

Jan. 11, 1955: The left eye appreciates only light and dark.

Jan. 28 : The scalp area heals to a certain point and then breaks down into shallow ulcers.

Feb. 13 : Perforation of left corneal ulcer, soon followed by panophthalmitis.

March 1 : Considerable herpetic neuralgia.

April 2 : Large areas of the scalp are still raw, though the underlying tissue seems to have a good blood-supply.

In view of the reported coincidence of generalised herpes zoster and leukIBmia, blood-·counts were done on Dec. 8, 1954, and Feb. 3 and Dec. 30, 1955, with normal findings. In the right eye there were senile degenerative changes around the macula.

Description of Rash

The rash was widely scattered over the trunk and limbs with a maximal concentration on t-he back of the trunk ; the hands and feet were unaffected. There were no successive crops of spots. The spots ·were small vesicles, of different sizes, each surrounded by a small ring of erythema. Some were hIBmorrha gic ; others contained clear fluid. There was no irritation.

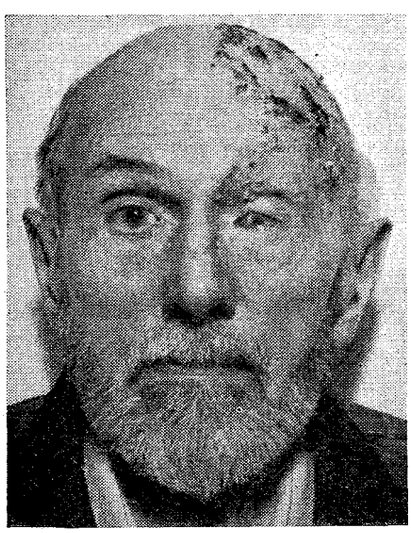

Fig. 3-Twelve months later, showing **scarring with distortion of eyelids, and residual scabs in affected area**

Treatment

Local and systemic administration of peni cillin, aureomycin, neo mycin, and cortisone hadnoeffect.

Present Condition

The scarred scalp is now (April, 1956) well demarcated bv some contraction of the scar tissue. The scars are brown and deeply depressed. The left eye lids are deformed. The scalp skin is thin and atrophic and readily breaks down into shal low ulcers. Stabbing pains are experienced over the scalp with a con stant sensation of " stinging ants " beneath the skin of the left orbit. There is residual brown pigmentation on the trunk and limbs, corresponding to the distribution of the rash. The left eye is completely blind.

Discussion

In this case the generalised rash was either herpes zoster or varicella. The subsequent deeply depressed brown scars were unlike the usual slightly depressed white scars of varicella. Varicella developed in neither of two susceptible contacts who nursed the patient.

The generalised rash was probably caused by virremia. Brain (1933) suggested that the virus of herpes zoster may have both neurotropic and dermatotropic properties. Hewitt (1954) described "herpes zoster generalisatus" or " herpes zoster varicellosus " in four chronically ill patients ; and this generalised form may develop in patients with leukremia. Age and lowered resistance to infection may be factors in such a spread. Aberrant herpetic vesicles may be scattered widely over the body, and these may be so numerous as to be indistinguishable from varicella ; profound toxremia is common.

Hremorrhagic necrosis occurs particularly with herpetic lesions of the ophthalmic division of the trigeminal nerve. Involvement of the nasociliary branch of this nerve leads, as in the present case, to ocular complications.

Summary

A case of severe herpes ophthalmicus with concurrent generalised herpetiform rash is described.

After seventeen months the scalp skin is thin and atrophic and still breaks down into shallow ulcers. The left eye is blind.

It gi;ves me great pleasure to thank Dr. J. R. Simpson, of Exeter, and Mr. G. Flint, of Lympstone, for their help and advice. Mr. H Fish, of Sidmouth, produced the excellent photographs.

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Preliminary Communication

AUTO-ANTIBODIES IN HASHIMOTO'S DISEASE (LYMPHADENOID GOITRE)

PATIENTS with Hashimoto's disease (lymphadenoid goitre) have high serum-y-globulin levels [1-3] which give rise to abnormalities in the flocculation tests of liver function-e:g., zinc-sulphate and thymol turbidities and colloidal-gold flocculation. The highest values are -found in untreated patients with large goitres. In a post operative survey of patients who had a thyroidectomy for histologically proved Hashimoto goitre 6 months to 23 years previously, the y-globulin levels and flocculation tests were normal in the great majority of cases. In two patients tested before and at intervals after thyroidec tomy, the y-globulin levels slowly returned to normal during the 6 months after operation.[4]

The raised y-globulin levels, their delayed return to normal after thyroidectomy, and the infiltration of the thyroid with lymphoi4 tissue, lymphocytes, and numerous plasma cells suggested that an immune response might be involved in this disease. To test this hypothesis, pre cipitin reactions between the sera of patients with Hashimoto goitres and saline extracts of normal and

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PRECIPI'rIN REACTIONS OF SERA WITH THYROID EX·rRACTS*

Serum	No. of cases
Hashimotot:	
Untreated ..	2
Thyroxine-treated ..	4
4 months postoperative ..	1
Small goitre, treated	2
Normal	2
Myxcedema ..	2
Thyrotoxicosis ..	5
Non-toxic nodular goitre	**6**
Multiple myeloma ..	**1**

* No difference was observed between extracts of thyrotoxic and normal thyroid.

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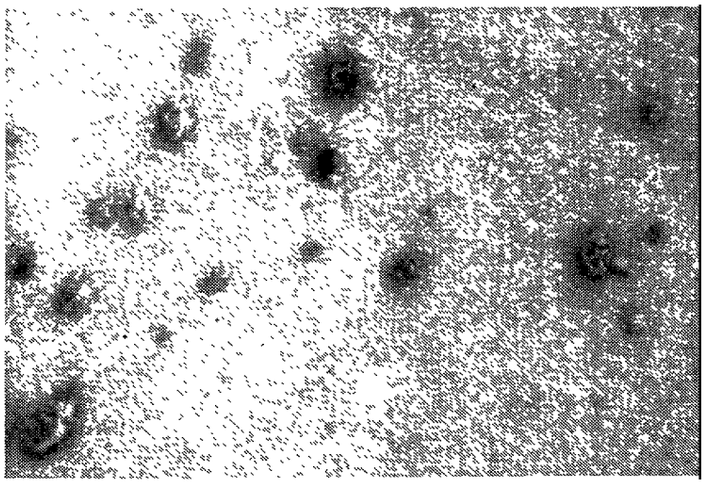

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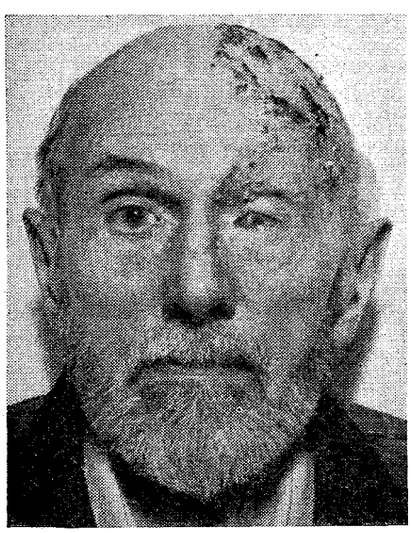

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thyrotoxic human thyroid glands were carried out. The results are shown in the accompanying table. Patients with Hashimoto goitre gave precipitins, but normal subjects and patients with other thyroid diseases showed negative results.

To establish whether the .antibodies were organ specific to thyroid gland, saline extracts of the thyroid and other organs from the same individual were tested against the serum of a Hashimoto patient. No pre cipitins were obtained against brain, liver, kidney, spleen, lymph-node, or parotid gland. Cross-reactions between a Hashimoto serum and saline extracts of rabbit and rat thyroid glands could not be demonstrated by precipitin reactions. In an attempt to identify the antigen(s) in the thyroid extract, human thyroglobulin was purified.[5] Comparable precipitins were obtained, suggesting that thyroglobulin is implicated.

Recent experiments by Rose and Witebsky [6] on the immunisation of rabbits with extracts of their own thyroid appear to support our findings. Precipitins were obtained in the serum of these animals, and were shown to be organ-specific against rabbit thyroid. Histological examination of the remaining lobe of the thyroid gland

5. Derrien, Y., Michel, R., Roche, J. *Biochim. biophys. Acta*, 1948, 2, 454.
6. Rose, N. R., Witebsky, E. *J. Immunol.* 1956, 76,417.

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Medical Societies

ROYAL SOCIETY OF MEDICINE

Hypothermia

AT a meeting of the section of experimental medicine on Oct. 9, with Prof. C. A. KEELE, the president, in the chair, Hypothermia was discussed.

Sequelw of Circulatory Arrest

Dr. E. NEIL, after describing a series of experiments indicating that reduction in temperature does not of itself affect the acid-base equilibrium of the blood, showed that during hypothermia acidrnmia occurs which is not explicable on the basis of a respiratory effect. This acidiemia is mild unless circulatory arrest is superimposed upon cooling, when it may become more severe.

·Dr. Neil showed that the liver is not seriously damaged during hypothermia, and any cellular change is evanes cent. But circulatory arrest and the resulting venous congestion produces alarming histological appearances in the hypothermic liver. These changes depend on the time that the circulation is obstructed, and he suggested that as short a period as was possible should be insisted upon. To support the histological findings a series of experiments on the clearance of galactose was carried out, and the result of this liver-function test supported the histological evidence for the view that venous congestion rather than cold alone accounted for the damage to the liver. Addi tional evidence was provided by an experiment designed to relieve this congestion by removing venous blood from the inferior vena cava. When as much as a third of the blood volume was removed in this way during the period of circulatory arrest the effects on the liver resembled the picture in hypothermia alone. Dr. Neil drew attention to the very large amount of venous blood that had to be withdrawn, and suggested that the technique is unlikely to be practicable in man.

Dr. SHEILA SHERLOCK criticised the use in this context of the word "congestion" and suggested that "impair ment of venous outflow " might be more appropriate. She asked whether ascites accompanied the liver damage and whether centrizonal necrosis had been demonstrated in patients who had failed to survive circulatory arrest _during hypothermia. Dr. NEIL reiterated his view that both congestion and anoxia were responsible for these changes and that ascites, did in fact accompany the liver damage, but he could not report liver changes in a small series of cases in which the patients had not survived operation. He drew attention to other centres of damage; similar changes had been found in the kidneys, the adrenals, and the gut.

Practical Problems

Dr. B. G. B. LucAs discussed the two most usual methods of inducing hypothermia-namely, reducing temperature by surface-cooling, and cooling the blood stream itself.

He made clear that there was no funda-. mental difference between these two methods, but sug gested that, while surface-cooling was wholly satisfactory for children ·below the age of three or four, blood stream cooling could be more easily carried out in patients not easily moved from bath to operating-table and back. He described briefly his method for blood-stream cooling whereby a coil of plastic tubing is immersed in saline solution containing alcohol IO% and solid carbon dioxide. This mixture provided a constant temperature of -2°C. The msophagus gave a most accurate indication of the central body temperature, but the skin of the forehead could be a fair guide once the temperature had fallen below 30°C.

As regards the safe duration of circulatory arrest at various levels of hypothermia, there was still no clear-cut evidence, but arrest should not be longer than five minutes at 28°C. Longer than that t me required lower temper atures. He denied that ventricular fibrillat on was a barrier to lower temperatures, and was of the opm10n that no drugs were of any prophylactic value against this complication.

Hypothermia in Clinical Surgery

Mr. D. N. Ross based his remarks on experience of 70 patients all of whom had withstood circulatory arrest for five to ten minutes. He suggested that the most important hazard was anoxic brain damage, particularly that associated with ventricular fibrillation. There was no real difficulty in electrical defibrillation provided that this technique was used in the proper circumstances. Defibrillation should not be attempted unless the myo cardium had been oxygenated by cardiac massage. He agreed with Dr. Lucas that drugs were of little avail· and stressed the need to avoid trauma. Returning to the problem of duration of circulatory arrest, Mr. Ross agreed that a safe maximum was ten minutes at 28°C.