deserves special comment. This disease is a primary degeneration of the neuro-epithelium of the retina on a hereditary basis, the importance of blood relationship among parents for the manifestation of the disease having been established (Nettleship, 1907–8).

Cataracts often complicate retinitis pigmentosa and are always present in cases of Werner's syndrome. If we accept the view that retinitis pigmentosa belongs to a group of hereditary diseases which have this in common, that the cells and tissues affected become prematurely senile (Verhoeff, 1931), then retinitis pigmentosa and the cataracts, common to the two diseases, are further evidence that Werner's syndrome is a heredo-degenerative disease of unknown origin. (It will be noted that our patient's parents were first cousins.)

From the foregoing it would appear that premature senile changes, rather than endocrinopathies, offer the best explanation for the functional and structural changes described in Werner's syndrome.

Summary

The clinical and post-mortem findings in a case of Werner's syndrome with uraemia and hypertension are described.

The renal failure was found to be due to vascular contracted kidneys and arteriosclerotic occlusion of the right renal artery.

The presence of retinitis pigmentosa in Werner's syndrome is an interesting finding which, together with other evidence, points to a heredo-degenerative disease in widely distributed body cells and tissues, affected by inherent characteristics which result in their degeneration.

REFERENCES

Agatston, S. A., and Gartner, S. (1939). *Arch. Ophthal. (Chicago)*, **21**, 492.
Ellison, D. J., and Pugh, D. W. (1955). *Brit. med. J.*, **2**, 237.
Irwin, G. W., and Ward, P. B. (1953). *Amer. J. Med.*, **15**, 266.
Kleeberg, J. (1949). *Acta med. orient. (Tel-Aviv)*, **8**, 145.
Müller, L., and Andersson, B. (1953). *Acta med. scand.*, Suppl. 283.
Nettleship, E. (1907–8). *Roy. Lond. ophthal. Hosp. Rep.*, **17**, 1, 151, 333.
Oppenheimer, B. S., and Kugel, V. H. (1934). *Trans. Ass. Amer. Phycns*, **49**, 358.
—— —— (1941). *Amer. J. med. Sci.*, **202**, 629.
Perloff, J. K., and Phelps, E. T. (1958). *Ann. intern. Med.*, **48**, 1205.
Rothmund, A. (1868). Albrecht v. Graefes *Arch. Ophthal.*, **14**, Pt. 1, p. 159.
Schott, J., and Dann, S. (1949). *New Engl. J. Med.*, **240**, 641.
Thannhauser, S. J. (1945). *Ann. intern. Med.*, **23**, 559.
Verhoeff, F. H. (1931). *Arch. Ophthal. (Chicago)*, **5**, 392.
Werner, C. W. O. (1904). Dissertation, Kiel.

"Unlike Russian doctors, who are merely members of a branch of the Medical Workers Trade Union, Chinese doctors still retain their association. But though its journals continue to print some work of an international standard—which the *Chinese Medical Journal* presents in excellent English—the incessant political exhortations of some of the association's officers force one to conclude that they see themselves as primarily the representatives of government, not of the doctors who may elect them."—From a lecture by Dr. T. F. Fox, editor of the *Lancet*, at the 87th Annual Meeting of the American Public Health Association (*Amer. J. publ. Hlth*, 1960, **50**, suppl., 28).

TREATMENT OF CHRONIC FURUNCULOSIS

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Recurrent furunculosis is often caused by reinfection with staphylococci from the patient's carrier sites. Hobbs *et al.* (1947), Valentine and Hall-Smith (1952), and Tulloch (1954) have shown that the causative staphylococci often come from the patient's own nose. Gould and Cruikshank (1957) and Copeman (1958) have had considerable success in preventing recurrences of furunculosis by the use of antiseptic nasal creams.

Staphylococci from sites other than the nose may also cause furunculosis. Superficial lesions of the skin, such as eczema, otitis externa, and blepharitis, are sometimes colonized by staphylococci and may be important sources of reinfection. Areas of normal skin also are sometimes colonized by staphylococci, especially the perineum (Hare and Ridley, 1958; Ridley, 1959). We observed cases of recurrent furunculosis which were caused by staphylococci from the perineum.

We report here the results of treating chronic furunculosis by disinfecting the patients' anterior nares and other carrier sites and by eradicating staphylococcal carriage from members of their families.

Methods

The patients investigated were 58 adults who came to the dermatological out-patient department between January, 1957, and June, 1959, with histories of recurrent furunculosis of at least six months' duration. Several had suffered from boils and styes for years. Alternate patients were selected for the control and the test groups. 15 patients defaulted. After spending six months as controls, 13 patients were transferred to the test group. There were altogether 33 test cases and 23 controls, most of whom were followed up for at least six months from the beginning of treatment. A few were observed for only four months. Freedom from furuncles during this period was regarded as a cure.

At the first attendance swabs were taken from a furuncle and from the anterior nares, and also from the ears and eyelids when indicated by the history or clinical findings. During the later part of the study perineal swabs were also taken. Further swabs were taken during and after treatment, usually at monthly intervals; and also from the relatives of some patients, as described below.

The bacteriological methods were described by Alder *et al.* (1955). Staphylococci were phage-typed by the method of Williams and Rippon (1952).

Treatment.—All the patients were instructed to swab the boil-bearing areas twice a day with a 1 in 3,000 aqueous solution of mercuric chloride. No other treatment was used for the control cases. The test cases were

given an antiseptic cream to apply to the anterior nares two or three times daily, for at least three months. Except for the first few patients (who were treated with "graneodin" ointment), the preparation used was "neobacrin" (neomycin + bacitracin) made up by Glaxo Ltd. in a water-miscible base instead of the standard greasy base. The same cream was applied to the eyelids when eye swabs were positive in the absence of inflammation. Blepharitis was treated with "neocortef" cream (which contained hydrocortisone in addition to the antibiotics). Otitis externa was treated with neocortef drops. During the later part of the investigation hexachlorophane baths (Ayliffe *et al.*, 1959) were prescribed for perineal carriers. The baths were taken daily for one week, then every second day for two more weeks, and then twice weekly for six to eight weeks. Those who could not carry out the bath routine were given "zac" talcum powder (containing 0.3% hexachlorophane) to apply daily to the perineum, buttocks, and trunk.

Results

The results of treatment are summarized in Table I. Of the 23 control cases, 3 were cured and 20 continued to have boils at frequent intervals. Of the 33 test cases, 22 remained free from lesions for at least six months from the beginning of treatment or soon afterwards; five were cured when family sources of reinfection were eradicated; one patient was improved but continued to have occasional boils; and five showed no improvement. One of these failures was a patient with boils on the buttocks who may have been a perineal carrier; but his perineum was not swabbed and hexachlorophane baths were not given.

TABLE I.—*Frequency of Furuncles in the Control and Test Groups of Patients*

	Total Patients	Average Duration of Observation (Months)	Patient-months	Total Furuncles	Furuncles per Patient-month
Controls ..	23	5·4	125	154	1·2
Treated patients:					
During treatment	33	4·7	156	45	0·3
After ,,	19	3·6	68	2	0·03

Clinical improvement was usually apparent soon after the treatment began, even when swabs from the carrier sites remained positive. But some of the patients who were eventually cured continued to have a few boils during the first few weeks of treatment. Recent experience suggests that they might have been cured more rapidly by more intensive treatment of nasal carriage and by the simultaneous use of hexachlorophane baths or powder.

The effect of treatment on the nasal carriage of staphylococci was less impressive than the clinical results. After three months of treatment 5 of 24 patients were still carrying their original strains in their noses, compared with 17 of 20 untreated controls. However, the nasal swabs which stayed positive during treatment usually yielded only scanty growths of staphylococci, and it is evident that the reduction in the load of infection was generally sufficient to prevent a recurrence of furunculosis.

Of 11 nasal carriers whose swabs became negative and whose noses were reswabbed one month or more after stopping treatment, three had again become carriers of their original strains.

Distribution of Furuncles in Relation to Sites of Carriage and Properties of Staphylococci

Of 58 patients, 51 (88%) were nasal carriers of the strains which caused their boils. This level of nasal carriage was similar to that found by Tulloch (1954) in a previous study of furunculosis, and was much higher than the carrier rate of 47% found in 258 healthy adults outside hospital whose noses were swabbed in 1957 and 1958.

Eye swabs from 11 of 21 patients yielded *Staph. aureus*, but all had blepharitis or had previously suffered from it. Of seven patients with present or recent otitis externa, five gave positive ear swabs. Perineal swabs were taken from 27 patients (19 men and 8 women), and 15 (56%) were positive (9 men and 6 women). This figure is probably higher than in the general population, because perineal swabs were at first taken only from patients with boils on the lower part of the body. However, most of the perineal swabs were taken after this examination had been adopted routinely. The perineum was undoubtedly an important carrier site.

Staphylococci of different phage-patterns differed in their distribution among the carrier sites (Table II) and there was a corresponding difference in the areas chiefly affected by boils (Table III). The commonest causes of boils were phage-group II strains and the group I strains 52A/79 and 80, which between them were responsible for 39 of the 58 cases. The 52A/79 strains showed a tendency to cause styes and boils on the upper part of the body, with a similar predilection for the nose as a carrier site to that previously observed in nurses (Alder *et al.*, 1955; Thompson and Gillespie, 1958). Type 80 staphylococci, on the other hand, were more widely distributed in carrier sites, and boils caused by them were seldom confined to the upper part of the body. Group II strains also caused widely distributed lesions, but were carried in the nose more often than on the perineum.

Of the 58 staphylococci which caused furunculosis, 25 (43%) were penicillin-resistant. Thirteen of these were phage-type 80 strains, eight of which were resistant also to streptomycin and tetracycline. A few of the antibiotic-resistant infections were probably attributable to recent contact with hospitals.

Type 80 staphylococci have been responsible for a good deal of serious sepsis in maternity hospitals and general hospitals in Bristol during recent years. Maternity hospitals have been particularly important sources from which these strains have spread into the general population. Recently, after the prophylactic use

TABLE II.—*Nasal and Perineal Carriage of the Staphylococci which Most Often Caused Furuncles*

Phage Pattern	Nose	Perineum	Nose and Perineum
80	6	3	2
52A/79	7	0	0
Group II	17	1	3

TABLE III.—*Distribution of Lesions Caused by Staphylococci of the Types Shown in Table II*

Distribution of Lesions	Phage Patterns		
	80	52A/79	Group II
General	5	1	9
Upper parts of body ..	1	5	8
Lower ,, ,, ,, ..	7	1	2
Total patients ..	13	7	19

of hexachlorophane powder in the maternity hospitals (Simpson *et al.*, 1960; Beryl D. Corner, personal communication), there has been a reduction in the incidence of type-80 staphylococcal infections outside hospital.

Sources of Infection in the Family

Other members of the families of 30 patients had recently suffered from boils. Nasal swabs were taken from 22 families, from 14 of which strains similar to those that had afflicted the patients were isolated from one or more relatives. Five cases of recurrent furunculosis were cured only when family carriage was discovered and treated. The following case was instructive.

Swabs from a boil and from the nose, eyelids, and perineum of a man with a 10-year history of furunculosis yielded a phage-type 42E staphylococcus. His carrier sites were twice rendered free from staphylococci, and his furunculosis cleared up, only to recur within two months. It was then discovered that his wife and two sons were nasal carriers of the same strain. All four were treated with neobacrin nasal cream and hexachlorophane baths. No more boils occurred during the treatment or during a subsequent follow-up period of five months.

Discussion

The detection and eradication of sources of reinfection is of primary importance in the treatment of recurrent boils and styes, and should be carried out before resorting to other methods of treatment. A thorough clinical and bacteriological search for carrier sites on the patient's own body should first be undertaken. The nose and superficial lesions such as blepharitis and otitis externa are often important sources of staphylococci, and so is the perineum, especially when boils are affecting the trunk, buttocks, and thighs. The search for sources of reinfection should extend to other members of the family when necessary.

The clinical success of our treatment was remarkable, in view of the fact that complete suppression of nasal carriage was often slowly and sometimes only temporarily achieved. Subsequent experience has shown that a more intensive treatment, with nasal disinfectant cream applied five or six times instead of twice a day, usually suppresses the carrier state within a few days; and that persistence with the treatment for a further two weeks often cures it permanently, provided that other sites are treated simultaneously. Nevertheless, it may be wise to minimize the risk of recolonization by continuing the treatment for longer than this minimum period. A recent study of the treatment of nasal carriage among in-patients suggests that a nasal cream containing neomycin and chlorhexidine (" naseptin ") may be the agent of choice. Sensitization to neomycin has been reported, but appears to be very rare (Jawetz, 1956; Calnan and Sarkany, 1958). It did not occur in the present investigation, nor in some thousands of in-patients who used nasal cream containing neomycin (Gillespie *et al.*, 1959).

Since staphylococci may be carried on areas of normal skin other than the perineum, and therefore might not be detected by swabbing, our present practice is to prescribe hexachlorophane baths or powder for all patients.

Successful treatment of the staphylococcal carrier state depends largely on the patient understanding the purpose of the treatment and his diligence in carrying it out. He should be given a clear and simple explanation of the nature of staphylococcal carriage and infection. Time spent thus is never wasted. The uninformed patient is almost certain to weary of the treatment and to give it up too soon.

Summary

The treatment of chronic furunculosis by disinfecting sites of staphylococcal carriage was investigated in a controlled trial. Most patients were cured by disinfecting their own carrier sites, but it was sometimes also necessary to eradicate staphylococcal carriage in members of their families.

The nasal carrier site was the most important source of reinfection, but staphylococci from the perineum and from superficial lesions of the skin were also important. Perineal carriage was sometimes associated with boils on the trunk, buttocks, and thighs.

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REFERENCES

Alder, V. G., Gillespie, W. A., and Thompson, Margaret E. M. (1955). *J. Path. Bact.*, **70**, 503.
Ayliffe, G. A. J., Alder, V. G., and Gillespie, W. A. (1959). *Lancet*, **2**, 456.
Calnan, C. D., and Sarkany, I. (1958). *Brit. J. Derm.*, **70**, 435.
Copeman, P. W. M. (1958). *Lancet*, **2**, 728.
Gillespie, W. A., Alder, V. G., Ayliffe, G. A. J., Bradbeer, J. W., and Wypkema, W. (1959). Ibid., **2**, 781.
Gould, J. C., and Cruikshank, J. D. (1957). Ibid., **2**, 1157.
Hare, R., and Ridley, M. (1958). *Brit. med. J.*, **1**, 69.
Hobbs, Betty C., Carruthers, H. L., and Gough, J. (1947). *Lancet*, **2**, 572.
Jawetz, E. (1956). *Polymixin, Neomycin, Bacitracin*. Antibiotics Monographs, No. 5. Medical Encyclopedia Inc., New York.
Ridley, M. (1959). *Brit. med. J.*, **1**, 270.
Simpson, K., Tozer, Rosemary C., and Gillespie, W. A. (1960). Ibid., **1**, 315.
Thompson, Margaret E. M., and Gillespie, W. A. (1958). *J. Path. Bact.*, **75**, 351.
Tulloch, L. G. (1954). *Brit. med. J.*, **2**, 912.
Valentine, F. C. O., and Hall-Smith, S. P. (1952). *Lancet*, **2**, 351.
Williams, R. E. O., and Rippon, Joan E. (1952). *J. Hyg. (Camb.)*, **50**, 320.

HAEMOPERITONEUM IN VON WILLEBRAND'S DISEASE

CASE OF OVARIAN FOLLICULAR RUPTURE

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The term " von Willebrand's disease " is used to describe a somewhat ill-defined haemorrhagic diathesis in which spontaneous bleeding takes place from mucosal surfaces, such as nose, gums, gastro-intestinal tract, urinary tract, and uterus. Only seldom do haemorrhages of deep tissue or joints occur. In most cases there is a definite familial history in which the inheritance is dominant and usually non-sex-linked.

The exact nature of the defect or defects in the haemostatic mechanism in this syndrome is obscure, but laboratory tests show a prolonged bleeding-time in association with normal numbers of circulating platelets. Platelet function is usually normal, while capillary morphology function may be abnormal (Macfarlane, 1941). It is now recognized that in this syndrome low