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A New Look at Infectious Diseases

Herpes Simplex and Zoster

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British Medical Journal, 1973, 1, 406–410

Among agents that infect man the herpesviruses have many characteristics that merit close attention, not least because they are among the few viruses that at present are amenable to antiviral chemotherapy. Some manifestations of the commonest, herpes simplex and varicella-zoster virus, are described.

Herpes Simplex

The first invasion of the non-immune host is known as the primary infection, an event which may or may not go unnoticed. The virus establishes itself, mostly comes to terms with, and stays in a latent form in its host—usually during the remainder of the life of the latter. The primary infection provokes an immune response, both humoral and cellular, which as a rule protects the host against reinfection with the same agent, but not against reactivation of the latent virus—which in some, though by no means all, patients may later cause recurrence and illness.

Many epidemiological surveys[1–3] have now made it clear that the incidence of herpes simplex infection during the first two decades of life is closely correlated with the degree of "development" of any one group of people. In primitive communities infection during early childhood is common, and usually goes unnoticed. In more highly developed countries, particularly in the higher social classes, the primary infection occurs later and later. In older age groups at present most people will have been infected. During the first quarter of the first year of life most children are protected against infection because of the presence of antibodies passively transferred from the mother. The primary infection often goes unrecognized in small children,[4] who develop a transient mild illness, usually a stomatitis which is often misdiagnosed as "teething" or "tonsillitis." The virus is usually transferred from the mother, or some other person with cold sores who is in intimate contact with and has kissed the child. Only about one in three of those who get a primary infection will later have clinical recurrences,[3] usually in the form of the well-known cold sores on or around the lips. Various stimuli may provoke the recurrence: fever from whatever cause—for example, in pneumonia; menstruation; ultraviolet light (strong sunlight, the seaside, or high altitude); emotional upsets (possibly due to release of adrenaline); trauma (shaving, etc.); and even cannabis smoking.[5]

The eruptions usually occur in the same sites in any individual. After tingling and itching, often for only an hour or two, vesicles erupt which take from three days to three weeks to heal with a median of about ten days. Recurrent herpes is easy to recognize when it occurs round the mouth. Impetigo may be mistaken for it, and sometimes the herpetic lesion may get secondarily infected with staphylococci. When recurrent herpes occurs in other sites, the true diagnosis may be missed. The area covered may be extensive, and may look like zoster—for example, on the forehead or the buttocks. If the lesion does not respect dermatomal distribution herpes simplex should be suspected. Recurrent herpes simplex may be associated with pain, possibly because the latent virus is in or near ganglia and nerve fibres.

LATE PRIMARY INFECTION

If the primary infection occurs in the second decade or later—an event particularly common in nurses and medical students (who on the whole are recruited from sections of the population where primary infection occurs late)—the disease caused may be dramatic. The commonest clinical manifestation is stomatitis,

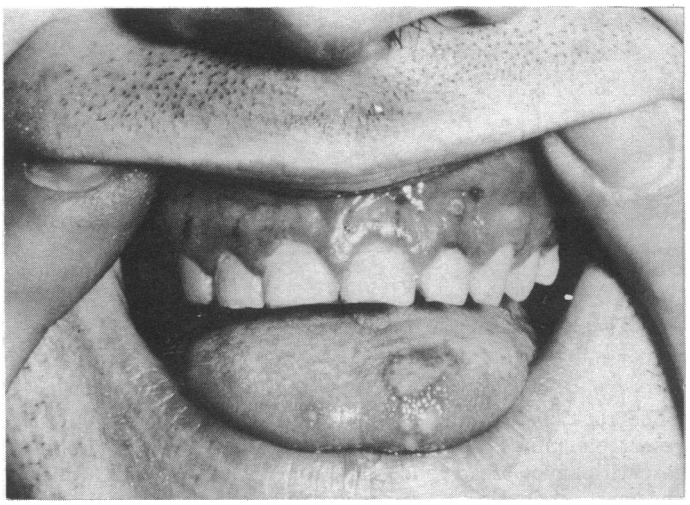

FIG. 1—Herpetic stomatitis.

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which may be severe (fig. 1). The oral cavity, tongue, and fauces show herpetic lesions, which clinically may be indistinguishable from those seen in certain coxsackie virus infections and sometimes in infectious mononucleosis. There is usually considerable cervical adenopathy and systemic influenzal symptoms reflecting the viraemia. Occasionally the disease becomes generalized with splenomegaly, hepatomegaly, generalized adenopathy, and occasionally evidence of disease of the central nervous system with clouded sensorium. Another fairly common manifestation of the primary infection is the herpetic whitlow, which may affect one or several fingers (fig. 2). Characteristically the patient has an extremely painful finger pulp with vesicles, initially with clear fluid. Untreated, the lesion takes three to four weeks to heal[6] and it may later recur in the same finger. Herpetic whitlow of the toes is rare but does occur.

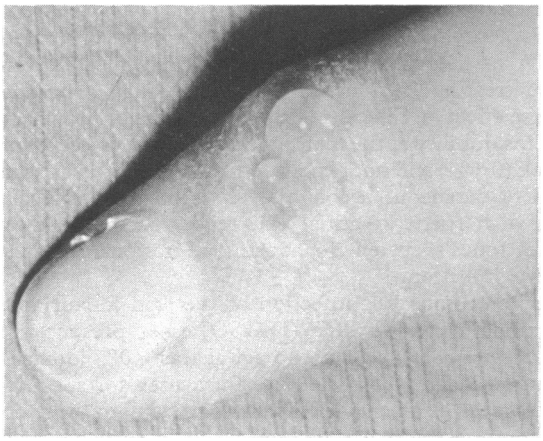

FIG. 2—Herpetic whitlow.

EYE INFECTION

Infection of the eye with herpes simplex virus may be a serious hazard to the patient's sight. Unilateral conjunctivitis with regional adenopathy is characteristic of the primary infection (fig. 3), and in some patients there are coarse punctate epithelial opacities. Recurrent keratitis is a major problem. Dendritic ulcers are caused by replication of the virus in the corneal epithelium, associated with only minimal anterior opacification. Dendritic ulcers may be associated with stromal keratitis, and the severest lesion is the amoeboid ulcer which occupies an extensive area of the surface of the cornea. The latter is usually caused by topical application of steroids.[7]

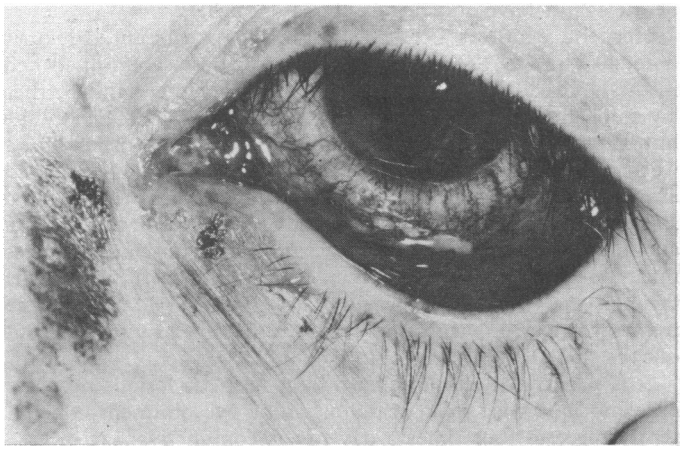

FIG. 3—Unilateral herpes simplex conjunctivitis.

SKIN LESIONS

Eczema herpeticum (Kaposi's varicelliform eruption) occurs in patients with extensive skin disease, notably in those with atopic eczema. A vesicular eruption may cover large areas of the surface of the skin (Fig. 4) and systemic symptoms are common. The often inevitable use of steroid preparations in patients with eczema facilitates the spread of the virus.

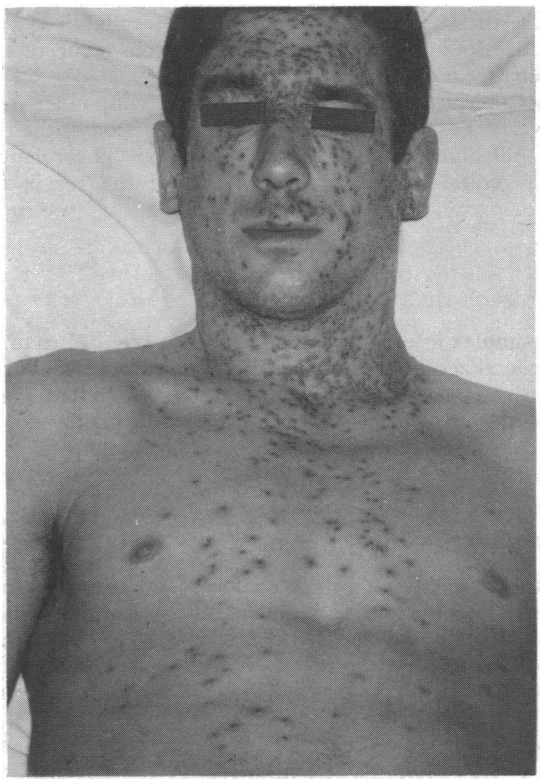

FIG. 4—Eczema herpeticum.

INFECTION OF NERVOUS SYSTEM

The most dreaded herpes simplex infection is that of the central nervous system. Pure meningitis is uncommon[8] and is relatively harmless. Encephalitis would seem to be one of nature's accidents: it is not in the best interest of the virus to kill its host, and this happens in about half of those who contract the disease. The remainder have varying degrees of neurological defects, frequently so severe that only about half of the survivors return to some sort of useful life. Possibly active chemotherapy offers these patients an advantage, but speed of diagnosis is of paramount importance. Any patient who unaccountably shows features such as personality change, or has fits, localizing signs in the central nervous system, and often a fever should without delay be referred to a centre that is equipped to undertake appropriate investigations (such as E.E.G., carotid angiogram, brain biopsy, and virological investigations—including fluorescent antibody staining of the brain biopsy).

GENITAL HERPES

Recently it has become obvious that genital herpes is usually caused by a strain (or strains) of herpes simplex virus which serologically and in other respects differ from the ordinary strain (Type 1). The so-called Type 2 herpes simplex virus (there may be intermediate forms[9]) is ordinarily, but not invariably, sexually transmitted. The primary infection occurs from the time sexual activity begins[10]; the rise in antibody to Type 2 virus takes place later in a population than that to the

Type 1 strain. In men lesions appear as clusters of vesicles on various parts of the penis or in the urethra, and sometimes in the perineum and on the thighs. These may be associated with local adenopathy and generalized malaise and influenzal symptoms. In women there may be extensive involvement of the vagina, vulva, and perineum, and occasionally generalized infection with lesions elsewhere (on the hands and in the pharynx). Sometimes the primary lesion is associated with disease of the central nervous system—usually myelitis with sensory and motor symptoms affecting the legs, such as isolated muscle weakness and paraesthesiae, but occasionally true encephalitic symptoms may occur. Both in men and women the lesions recur, often with distressing frequency.

Genital herpes appears to have become commoner in more recent years, possibly because of the introduction of the contraceptive pill and decreased use of condoms. The relationship between genital herpes and carcinoma of the cervix is still uncertain.

INFECTIONS OF THE NEWBORN

Herpes simplex infection of the newborn is rare in Britain. This often generalized infection has a high mortality. The source of infection is usually the mother's genital tract[11] and most infections are due to Type 2 virus. In the survivors defects of the central nervous system, eyes, and skin are common.[12]

IMMUNE DEFECTS AND HSV

In the immune individual reactivation of latent virus is usually contained. Patients whose immune mechanisms have become deranged may be in the same position as the non-immune individual with the additional disadvantages of a defective immune mechanism. Severe and sometimes lethal infection may occur in patients with malignant disease, such as Hodgkin's disease, multiple myeloma, and lymphatic leukaemia. Patients on high doses of steroids or immunosuppressive drugs, or both, are in a similar position (fig. 5). Generalized herpes has been a major cause of death in a few patients who have undergone organ transplantation.

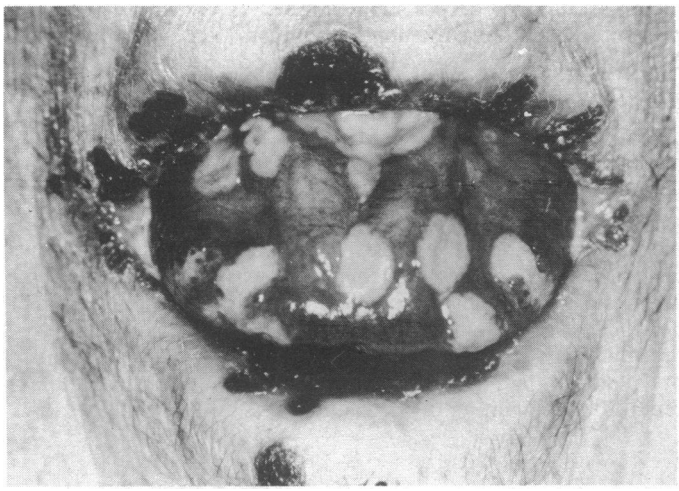

FIG. 5—Severe herpetic stomatitis in patients on high doses of steroids.

TREATMENT

If there is the slightest doubt about the diagnosis, virological confirmation, and at least a specimen for virus culture, must be obtained before treatment is begun. Most areas have a virus laboratory that will undertake simple tests. With a throat swab material is taken from lesions on the skin or from the throat. The swab is broken off into Hanks's transport medium, and the container sent to the laboratory without delay. A dry swab is useless. A sample of serum should also be sent. If the lesion is recurrent, antibody will be present, but if the specimen is taken early enough in the primary infection antibodies may not yet have appeared and a second specimen about ten days later will show a rise in antibody. If there is doubt about which specimens are appropriate the local virus laboratory should be consulted.

The treatment of choice of infection with herpes simplex virus is with specific chemotherapy. During the last ten years it has become apparent that at least two chemotherapeutic agents are of proved value in its treatment: idoxuridine and cytarabine (cytosine arabinoside). Others which show promise—for example, vidarabine[13]—may become available.

Idoxuridine

Idoxuridine is a thymidine analogue which gets incorporated into the DNA of the virus and arrests its multiplication. In doses used in treating local herpes simplex lesions it has no obvious adverse effect on the cells of the host. A major drawback is the relative insolubility of the material. Idoxuridine in cream or watery solution (0·1%) will not penetrate the skin and is useless in the treatment of cutaneous lesions.[14–16] It is of great value in the treatment of herpetic keratitis. The reader should refer to Patterson and Jones's review[7] for details of rational chemotherapy of eye infection.

For primary and recurrent infection of the skin idoxuridine dissolved in dimethyl sulphoxide (DMSO) is at present the treatment of choice.[17] It is possible to get at least 40% idoxuridine into solution in DMSO. The pure form used for spectroscopy is preferable as a solvent; it penetrates the skin to the deep fascia within minutes, and has not been shown to have toxic effects in man.[17] For recurrent cutaneous herpes simplex lesions the application of a 5 to 20% solution with a brush to the lesions four times a day for three days nearly always causes rapid disappearance of vesicles, and recurrences in the same site become rare; on the other hand, patients so treated tend to get recurrences later in other, new sites.[18] It is wasteful and often directly harmful to continue application after the first few days. The skin will become macerated and possibly secondarily infected with bacteria. This solution must not be put into the eye.

In genital herpes simplex a similar solution—preferably of a 40% strength—is often useful. Type 2 H.S.V. is more resistant to idoxuridine than is Type 1. More frequent applications may be needed but it is particularly important not to continue for more than about three days on the genitals, where maceration of the skin otherwise causes an apparent continuation of the lesions. In all chemotherapy of herpes simplex infections it is essential to start treatment as soon as possible in an attack while the virus is replicating actively. It is quite useless to use idoxuridine or any other substance in between attacks, for the chemotherapeutic agents have no effect on the latent virus.

Herpetic whitlows need more intensive treatment. Idoxuridine, 40% in DMSO should be applied on a piece of lint soaked in the material to the lesion; the whole is covered with further layers of dry lint, the finger splinted, and the hand raised. The lint is rewetted daily and the treatment continued—usually for a week as it has been found that virus no longer can be isolated after that period. Pain usually disappears within 24 to 48 hours. In the untreated patient virus is usually recovered for three weeks and pain is present for over a week. Under no circumstances must surgical incision be done. This will spread the virus through the tissues and prolong the disease. The skin of the treated finger will wrinkle, as grease is dissolved by the DMSO but the skin soon returns to normal once treatment has stopped.

It is particularly important to treat herpetic lesions near the eye, and it is wise to insert idoxuridine eye ointment concurrently three to four times a day to the conjunctival sac in case herpes virus gets into the eye.

Systemic idoxuridine has been used in the treatment of herpes simplex encephalitis. Though some patients may have benefited, the value of this drug in trials designed to yield comparable results still has to be properly assessed, not least because of the potential toxicity (particularly to the bone marrow) of large systemic doses.

Cytarabine

Cytarabine is a cytidine analogue which probably acts on herpes simplex virus by incorporating into it DNA, arresting virus replication. It is easily soluble in water and in doses of 3 mg/kg/day or less given by rapid intravenous injection in a single daily dose has proved to have minimal or no effect on the normal bone marrow, and to be free of other side effects.[19] It is useful in the treatment of severe herpes simplex infections, and is at present the drug of choice in these. So far the drug has only been available for clinical trial in hospitals. In nine adult patients with primary *gingivostomatitis* treated with 3 mg/kg/day for one day and 2 mg/kg for another three, the clinical response was dramatic in all, and all visible lesions cleared within three to four days. In eight cases virus could be isolated for 4-6 days (median 5), and in only one was virus present for 20 days (the average for an untreated patient). There has been no recurrence so far; the first patient was treated three years ago. Four patients aged 47-64 with immune deficiencies with severe recurrent stomatitis responded equally dramatically, and virus could be isolated for only 2-4 days; none of these had had previous antiviral chemotherapy. Eczema herpeticum responds with equal promptness, both clinically and as measured by virus isolation.

Severe herpes simplex infections—such as pneumonitis, hepatitis, and encephalitis—are at present probably best treated with systemic cytarabine. These patients should be treated in hospital by people familiar with the drug, and in the case of encephalitis in centres with special diagnostic and treatment facilities.[20]

Other Forms of Treatment

Old remedies such as surgical spirit probably at best help to prevent secondary infection with bacteria of cutaneous lesions. Antibiotics have no effect on the viruses and should not be used unless there is evidence of secondary bacterial infection. As DMSO is bacteriostatic this problem hardly ever arises if idoxuridine in DMSO is used. Photosensitization destroys the infectivity of herpes simplex virus in vitro and recently treatment of cutaneous infections with a 0·1% solution of neutral red to the base of lesions and exposure to visible light has been used, particularly in the U.S.A., with some apparent success.[21] Convincing double-blind trials still need to be done. Vaccination with killed antigen has been attempted, particularly by French workers, but so far the results in man have been unconvincing. Interferon or interferon inducers have not yet been shown to have a place in treatment of herpes simplex infection, with the possible exception of the eye.

It cannot be stressed often enough that topical and systemic use of steroids in herpes simplex infections, often used with remarkable recklessness, must be condemned. More often than not the lesions are made much worse, and occasionally what started as a trivial infection becomes distressing or even life-threatening. There is a small place for steroids in the few patients who are hypersensitive to the herpes simplex virus, but then only under cover of antiviral agents, and only in the hands of somebody thoroughly familiar with the manifestations of these infections.

Herpes Zoster

Chickenpox is caused by another herpesvirus, the varicella-zoster. Although most of the population contracts the infection in childhood, it often goes unrecognized because the manifestations are so trivial that the rash is missed. The behaviour of the virus has much in common with that of herpes simplex. Although the patient recovers, the virus remains in a latent form, probably in the nervous system as in the case of herpes simplex. Immunity against reinfection is the rule, but sooner or later the latent virus becomes reactivated. The stimuli required are very similar to those causing recurrent herpes simplex. Because the patient's immune mechanism already has been exposed to the virus by the primary infection the recurrence is usually localized, and usually to the distribution of one or more sensory nerves, often due to local trauma.[22] This produces the characteristic belt-like picture of "shingles" or "zoster." A vesicular eruption, more or less extensive, is the rule, often preceded by pain or paraesthesia for up to three weeks. Occasionally no vesicular eruption follows, and this "herpes sine herpes"—probably first recognized by Loeb in the 1920s—may masquerade as myocardial infarction, gall stones, renal colic, and appendicitis, to mention a few differential diagnoses. The true nature of the illness will not be arrived at unless the possibility of zoster is borne in mind. Occasionally varicella-zoster virus is reactivated in the spinal cord giving rise to anterior poliomyelitis with muscle palsies, or in the cranial motor nerves or their nuclei giving rise, for example, to facial nerve or oculomotor palsies. Very occasionally zoster encephalitis, often with bizarre focal signs, may develop. This should always be suspected if a patient with segmental zoster develops symptoms and signs referable to the central nervous system.

Zoster increases in frequency and severity with advancing age. The gradual disappearance of detectable antibody to the virus after the age of 50[23] is probably a measurable reflection of the failure of the immune mechanism of the host to keep the latent virus in check. Contrary to common belief there is no good evidence that the appearance of zoster suggests the presence of hidden malignant disease. In some 800 patients with zoster we have not found an incidence of malignancy higher than that in the population at large from the same area. It is an entirely different matter that zoster may be more severe, recur frequently, and become generalized in patients who have malignant disease, particularly of the lymphoid tissues, or in those whose immune mechanisms are defective for whatever other reason.

In about a third of all patients with zoster infections outlying lesions will be found. This probably reflects temporary viraemia. Within 4-5 days of the onset of the disease the level of antibody to varicella-zoster virus normally rises and new scattered lesions are rare. If the generalized rash becomes dense, the term "generalized zoster" is used, though clearly this is a matter of degree. If it is severe—as often in patients with, for example, lymphatic leukaemia, when the rash may be confluent and haemorrhagic and internal organs such as lungs, liver, and gut may be affected—the condition may threaten life. Patients may get zoster repeatedly; as a rough guide any patient who has had zoster once has a 5% chance of getting it again. Bilateral zoster is very uncommon, but there is no foundation for the popular belief that "if zoster meets in the middle the patient will surely die." Untreated zoster, and in particular bilateral zoster of the sacral segments below S2 often causes bladder paralysis (and even zoster lesions in the bladder).[24]

The dreaded and most distressing feature of zoster is "post-herpetic neuralgia," perhaps better named "zoster neuralgia." This is caused by destruction of and dense fibrosis of the sensory nerves and ganglia. In acute zoster virus has been found in the skin, neurilemma, and the ganglion cells of the affected area,[25] and it can hardly surprise that the damage may be extensive, particularly in the elderly.

TREATMENT

Zoster should be treated with specific chemotherapy, and at an early stage, preferably within the first few days of the appearance of a rash. Once the virus has done permanent damage to the

nerves, and the virus has ceased to replicate, no amount of drug, however active against varicella-zoster virus, is of any avail. Zoster responds to idoxuridine in DMSO, but a strong solution must be used (35-40%) to get consistently good results.[26] The material must be applied continuously for three to four days; a piece of lint cut to the shape of the lesion is wetted with the solution and rewetted daily, and covered with further layers of lint, the dressing being kept in place with some bandage such as netelast. In the vast majority of patients pain has gone within seven days, and late sequelae are rare. Success depends on early institution of treatment. A few patients are sensitive to DMSO, which may produce wheals and itching. This is usually controlled with antihistamines. The pain in the acute phase of the illness must obviously be relieved with analgesics.

Considerable experience with cytarabine in both Britain and the U.S.A. suggests that its administration may achieve results comparable to those achieved with idoxuridine.[27] When zoster affects the sacral segments or the maxillary division of the trigeminal nerve—or when generalized zoster or zoster encephalitis occur—cytarabine should certainly be used, though these latter patients should be treated in hospital. A modest dose (3 mg/kg) the first day, followed by 2 mg/kg in a single daily intravenous rapidly administered dose for another three days has, in our hands, been remarkably effective. Vidarabine has shown promise.

Compared with antibacterial antimicrobials the few active antiviral agents that can safely be used in man are still formidably expensive. The cost of treating common herpes simplex lesions with idoxuridine is relatively trivial, but the average cost of materials used for a course of treatment for a zoster patient is still £30-60, a fact I—who have treated well over 600 patients—am acutely aware of. This treatment could safely be used in general practice and one hopes that the cost will decrease. Double-blind trials to evaluate the relative merits of cytarabine in uncomplicated zoster are in progress.

The cost, however, appears to be more than justified by the prevention of the consequences of zoster. The patient with zoster neuralgia which may plague him or her for many years is in a pitiful plight, and the doctor has little to offer. If the pain is unbearable the effect of a local anaesthetic to the nerve(s) should be tried. If relief is obtained and the patient is prepared to put up with anaesthesia, permanent blockade of the affected nerve—for example, with alcohol—must be offered. In pain in the trigeminal area, the effect of Tegretol (carbamazepine), so effective in "tic doloreux," is worth trying. Large doses of vitamin B_{12} and other therapy with no scientific basis should be discouraged.

In acute zoster as in active herpes simplex infection the use of steroids must be discouraged. More often than not severe exacerbation of the zoster lesions is the result of topical or systemic administration of corticosteroid preparations.

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Medical Education

Applying for Junior Hospital Posts

E. D. SEVER, P. C. ROBERTSON, B. J. SYKES

British Medical Journal, 1973, 1, 410-411

Two senior house officer appointments at Northampton General Hospital (610 beds) were advertised in the *British Medical Journal* in July 1972. These appointments were in general medicine and general surgery and attracted many applicants.

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The standard of the applications submitted varied greatly and it seemed that many of the candidates had done themselves less than justice. We felt, therefore, that it would be useful to analyse these applications and to suggest how they might have been improved.

Results

Altogether, 129 applications were received—89 for the appointment in general medicine (one year) and 40 for that in general