in movement between the two sides. When the nerve has been badly damaged recovery of movement can be expected only after surgical restoration of the continuity of the nerve. In Bell's palsy some degree of spontaneous recovery is the rule in most cases, though in many recovery is incomplete and there may be considerable spasm. When, however, the nerve appears to be electrically dead decompression often encourages a better and quicker recovery of function. Sometimes decompression is followed within a few days by some return of function. This cannot be because of new nerve fibres growing down, and can only be accounted for by the restoration of activity as the result of decompression to some of the nerve fibres which did not formerly give any sign of life. This reversibility of function brought about by relief of pressure on the nerve is the principal reason for advising decompression in Bell's palsy (Cawthorne, 1951).

The results of these procedures are given in Table VII, in which they are classified as "good," "fair," or "poor," depending upon the recovery of facial movement. A good

TABLE VII.—*Results of Facial Nerve Operations*

	Good	Fair	Poor
Decompression for Bell's palsy ..	86	21	3
Exploration for injury or infection ..	31	17	6
Nerve graft	19	8	5

result means a normal appearance of the face at rest, with ability to close the eye and to move the nose and the lips. A fair result means either a normal appearance at rest or slight spasm on the affected side, with, however, fair movements of eye, nose, and mouth. A poor result means either no movement or only slight return of movement, or pronounced spasm.

Comment

In this short survey of facial palsy it will have been noted that as regards treatment surgical exposure of the nerve trunk at the site of the lesion is the only alternative to leaving things to nature. Support to the paralysed muscles, electrical stimulation of the paralysed muscles, and massage of the paralysed muscles contribute nothing at all towards the recovery of conductivity in the nerve, and so they should be regarded as supportive measures that may maintain tone in the muscles and are undoubtedly good for morale. What other benefits they confer is not certain.

In a case of facial palsy which is slow to recover and for which surgery is either not indicated or desired, an intra-oral splint will reduce the deformity from a drooping mouth. Vigorous manipulation of the face with grease-covered fingers three or four times a day is sufficient exercise for paralysed facial muscles. As soon as there is some recovery, active movements in front of a glass should be practised at least twice a day.

If all treatment, including local surgery, fails to bring about any return of function at all, the choice will then be between facio-hypoglossal anastomosis and a fascial sling procedure; the respective merits of these procedures were discussed by Sir Charles Ballance (1934) and Sir Harold Gillies (1934). We have no personal experience of either of these forms of treatment, though we have seen good results from each. We still feel, however, that in a case of facial palsy where the nerve is electrically dead an attempt should be made whenever it is surgically possible to expose the nerve at the site of the lesion. In this way it is possible to see what is wrong, and this is the first stage in providing the right remedy.

Summary

The frequency of facial palsy is attributed to the facial nerve's long and tortuous course through a small bony canal in the temporal bone.

Anything which causes a segment of the nerve or of its covering to swell in the bony canal is apt, by squeezing the blood out of the vessels, to result in ischaemia. This is thought to be the cause in Bell's palsy, which in this series of 557 cases of facial palsy accounted for 62%. Injury was responsible for 15% and a lesion at the geniculate ganglion for 7%.

By testing the ability to taste and to lacrimate in a patient with facial palsy it is often possible to differentiate between a lesion near the stylomastoid foramen, at the geniculate ganglion, and in the nucleus.

The results of exposing the facial nerve trunk at the site of the lesion in 196 cases are given; and it is suggested that in a case of facial palsy when the nerve is electrically dead an attempt should be made, whenever it is surgically practicable, to explore the nerve at the site of the lesion.

We record our indebtedness to Dr. E. A. Carmichael for all the help he has given us in the investigation of patients with facial palsy.

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INFLUENZA VACCINATION IN A RESIDENTIAL BOYS' SCHOOL

REPORT TO THE MEDICAL RESEARCH COUNCIL COMMITTEE ON CLINICAL TRIALS OF INFLUENZA VACCINE

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Cases of clinical influenza began to occur in Wellington College, Berks, soon after the school returned from the Christmas holidays on January 21, 1956. The outbreak reached its peak in February and was later shown to be due to influenza virus A. Of 664 boys present at the beginning of the term, 200 had been inoculated on December 9 or 11, 1955, with a saline vaccine prepared from the A/Eng/19/55 strain—one of three vaccines used during the same winter in a Medical Research Council field trial, the results of which are to be reported elsewhere. The A/Eng/19/55 strain, isolated in Carmarthen in April, 1955, is one of a group referred to by Isaacs (1956) as Eire '55 viruses. These viruses differ antigenically from those of the Scandinavian group. In addition, 100 of the 200 inoculated in 1955 had been inoculated in December, 1954, with a saline vaccine which contained a Scandinavian A strain (A/Eng/1/54),

a recent American A strain (A/Missouri/303/52), and the Lee virus B strain, in equal proportions. A further 120 boys had been inoculated with this vaccine in 1954 but were not reinoculated in 1955. Both vaccines were prepared by adsorption of virus on to aluminium phosphate; they were made by Dr. F. Himmelweit, of the Wright–Fleming Institute. Each dose (1 ml.) contained 20,000 haemagglutinating units of influenza virus as determined by the "plastic plate" method described in World Health Organization Technical Report Series No. 64 (1953), and 10 mg. of aluminium phosphate. The inoculations were made subcutaneously.

In both 1954 and 1955 the parents of all boys present in the school were asked in writing whether they would like their sons inoculated against influenza, and all those for whom permission was given were vaccinated. It is possible, though unlikely, that the susceptibility to influenza of those inoculated differed from that of boys of the same age whose parents did not give permission. All the inoculations were performed by the school medical officer (G. F. C. H.), who was also responsible for the subsequent clinical supervision of the boys. When the outbreak of influenza occurred he did not remember which boys had been inoculated and he did not refresh his memory from the records. It seems probable that in deciding to admit a boy to the sanatorium or in diagnosing an illness as influenza he was not biased for or against the vaccinated. For these reasons it was thought that study of this outbreak might give some indication of the value of the vaccines used, though permitting less certain conclusions than a properly controlled trial.

Records and Specimens

Records were analysed for all boys admitted to the sanatorium or seen at the medical officer's surgery but not admitted, for a two-month period from January 21 to March 20, 1956. Only first attendances at the surgery for a given complaint were included. The records were grouped according to type of illness—the admissions into clinical influenza, other respiratory illness, and non-respiratory illness, and the surgery attendances into respiratory illness and non-respiratory illness. Between February 8, when it became apparent that an outbreak was developing, and March 1, when the outbreak seemed almost at an end, an attempt was made to take throat swabs for virus isolation from all cases admitted and diagnosed as influenza. This was done for all but four cases. Swabs were also taken from two boys admitted with influenza on February 7; altogether swabs from 73 patients were available for examination. Wooden-shafted throat swabs were used and the ends broken off into small screw-capped bottles containing 3 ml. of broth. The bottles were kept at −70° C. in transit to the laboratory, where they were stored at −30° C. until they were tested.

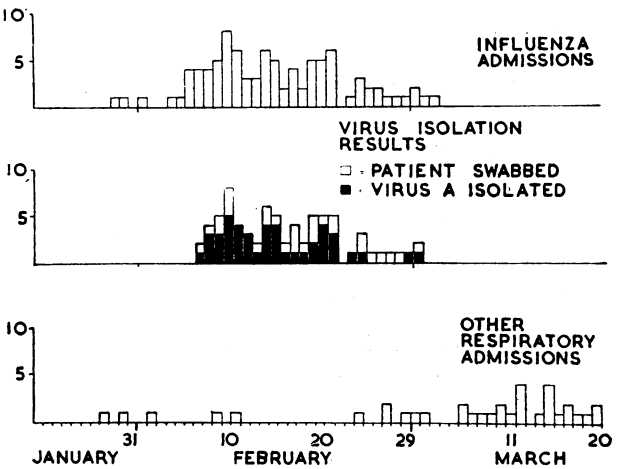
Laboratory Methods

Each specimen, with added penicillin and streptomycin, was inoculated in the amniotic cavities of 10-day-old fertile hen eggs, using six eggs for the first passage and four eggs for any subsequent passage. The fluids from negative eggs were subjected to second amniotic passage. The first amniotic passage was repeated, using the original material of those specimens which were negative after two amniotic passages. Inoculated eggs were incubated for 72 hours; all eggs which died during the first 48 hours of incubation were discarded. After chilling to 4° C. individual amniotic fluids were harvested and tested against human and fowl erythrocytes at 4° C. and at room temperature for the presence of haemagglutinating agents. The embryos and egg membranes of all eggs showing haemagglutination were tested for the presence of complement-fixing antigens against sera known to contain antibody to influenza viruses A, B, or C. Influenza strains were analysed by haemagglutination-inhibition tests, using specific sera prepared in ferrets. The technique closely resembled that described in World Health Organization Technical Report Series No. 64 (1953) with minor modifications.

Findings

Influenza virus A was isolated from 44 (66%) of the 73 swabs. The 29 swabs from which influenza virus was not isolated were tested in HeLa cell tissue culture for the presence of viruses of the adenovirus group and found negative. Serological evidence of influenza virus A infection was obtained from two out of three boys from whom paired blood specimens were taken. Virus was isolated from the two boys with positive serological findings but not from the boy with negative serological findings. Of the 44 strains isolated, 42 were found to resemble viruses of the Scandinavian group and were similar to the virus A strains in the 1954 vaccine; they differed from the strain used in the 1955 vaccine. The remaining two strains did not give clear-cut results and are being investigated further.

The daily number of admissions to the sanatorium with influenza and other respiratory illness during the period studied is shown in the Figure. The patients swabbed and those from whom virus was isolated are also shown. There



Daily number of admissions to sanatorium with influenza and other respiratory illness, and laboratory findings.

seems little doubt that the majority of cases diagnosed as influenza clinically were virus A infections. The "other respiratory illness" admissions in March were not tested in the laboratory but were mostly diagnosed as "septic throat." They were clinically unlike the illnesses seen in February.

The age distribution of boys inoculated in either 1954 or 1955 or in both years was very similar to that for those not inoculated, but the group inoculated in 1955 necessarily included a higher proportion of young boys than did the group inoculated in 1954. This would make comparison of the sickness experience of the groups difficult if influenza had shown any tendency to attack one age group more than another. In the uninoculated, however, the clinical influenza attack rates were similar at all ages. The rate for those born in 1937–8 was 15%, in 1939 25%, in 1940 19%, in 1941 21%, and in 1942–3 18%.

TABLE I.—*Admissions to Sanatorium With Influenza*

	No. at Risk	No. Admitted	Admission Rate		No. of Isolations / No. Swabbed	
Uninoculated ..	344	68	20%	18%	40/55	44/67
Inoculated 1954 only	120	14	12%		4/12	
,, 1955 ,,	100	8	8%	5%	0/4	0/6
,, 1954 and 1955	100	2	2%		0/2	

In Table I are set out the influenza admission rates in the uninoculated and in the various vaccine groups. Those inoculated in 1954 only had significantly less influenza than the uninoculated $\left\{\frac{\text{Difference}}{\text{Standard error}}=\frac{8}{3.7}=2.2\right\}$, and those inoculated in 1955 or in both years fared even better. Though the figures are small they suggest that those inoculated twice gained more protection than those inoculated in 1955 only. The virological results supported the clinical findings, all 44 strains being isolated from boys either uninoculated (40) or inoculated in 1954 only (4).

In Table II are shown the admission rates from causes other than influenza and surgery attendance rates of illnesses not requiring admission in the same groups of boys as in

TABLE II.—*Sickness Experience From Causes Other than Influenza*

	Admission Rate		Surgery Attendance Rate	
	Other Respiratory Illness	Non-respiratory Illness	Respiratory Illness	Non-respiratory Illness
Uninoculated	6%	4%	11%	34%
Inoculated 1954 only ..	3%	10%	9%	35%
,, 1955 ,, ..	4%	10%	10%	50%
,, 1954 and 1955	7%	6%	7%	35%

Table I. The figures for all four groups are fairly similar apart from a high rate for non-respiratory illness attendance in those inoculated in 1955 only. The reason for this high rate is not known, but there is no evidence that it was related to vaccination. There is nothing in Table II to suggest that the lack of influenza in the inoculated was due to cases being diagnosed as something else.

Summary and Conclusions

An outbreak of influenza A occurred in Wellington College in February, 1956. Of the 664 boys present, 120 had been inoculated in December, 1954, with a vaccine containing influenza virus A and B strains, 100 had been inoculated in November, 1955, with a vaccine containing the A/Eng/19/55 strain, and a further 100 had been inoculated on both occasions. The admission rate to the sanatorium with influenza in these three groups was 12%, 8%, and 2% respectively, compared with 20% in the 344 boys who had not been inoculated. Throat swabs were taken from 73 of the 92 patients with clinical influenza, and influenza virus A was isolated from 44 of them. Forty of the strains were from uninoculated boys and the remaining four from boys inoculated in 1954 only. No strains of virus were isolated from boys who had received the 1955 vaccine.

Though the investigation did not constitute a controlled trial reasons are given for believing that the above rates are comparable.

It is concluded that some protection still remained from the 1954 inoculations and that a high degree of protection was obtained from the 1955 inoculations, particularly in boys who had also been inoculated a year earlier. The virus A strains isolated during the epidemic resembled the strains (A/Eng/1/54 and A/Missouri/303/52) used in the 1954 vaccine, but differed from the strain (A/Eng/19/55) used in the 1955 vaccine.

We are indebted to Dr. G. B. Bruce White, of the Virus Reference Laboratory, Colindale, for testing specimens for viruses of the adenovirus group, and to Dr. N. Wood, of the Reading Public Health Laboratory, for his help.

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MERCURY ABSORPTION AND PSORIASIS

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Mercury compounds in ointments and pastes have been used for many years in skin diseases. Mercury ointments are still widely used in the treatment of psoriasis, and such applications often contain varying proportions of salicylic acid and tar. Absorption of mercury through the skin is known to occur, but is not commonly stressed in textbooks. Thus Goldsmith and Hellier (1954) state that "mercurials . . . are especially easily absorbed." They state that the literature is "very confused" and distinguish between "penetration" and "absorption."

Toxicity due to mercury absorption in the treatment of psoriasis must be rare, and has not been recognized in this clinic. Psoriasis is uncommon before the age of 4 years. This investigation resulted from the appearance of a child of 2 years with widespread psoriasis. As mercury is the probable cause of most cases of pink disease, the question arose of how much was absorbed. With ordinary applications it was important to obtain this information, since the local action of mercury in the treatment of psoriasis is imperfectly understood—in contrast to that of dithranol, tar, and ultra-violet light, which probably involve a process of oxidation or burning of the lesion.

That poisoning can occur from external use of mercury is well known. Not so long ago it was considered necessary in the treatment of syphilis to produce by mercury inunction gingivitis, loosening of the teeth, and excessive salivation. Such inunctions were performed with friction on areas where the skin was thin, the anterior forearm being a favourite site. However, applying mercury compounds to diseased skin without friction is a different matter. It might be expected that mercury would be more easily absorbed through damaged skin, as in eczematous or ecthymatous lesions, than through intact skin. In psoriasis, absorption might yet be different for two opposing reasons. On the one hand, thick psoriatic scaling could be expected to retard absorption of ointment. On the other hand, the histology of psoriasis shows the skin papillae to be greatly thinned and elongated. So there is only a much-thinned epidermis between the summit of any individual papilla and the horny layer; this explains why grattage of a psoriatic plaque causes small bleeding-points—due to damage to the superficially placed papillary loops. This feature of the psoriatic structure might well facilitate absorption of ointment.

Literature

Some findings of the more important recent papers on cutaneous absorption of mercury in humans not suffering from psoriasis, and in experimental animals, are summarized below.