Conclusions

A survey of 100 employed diabetics shows that 77 per cent. lost no time from work because of their diabetes after the initial stabilization of treatment, and the figure is 85 per cent. if the ultimate stabilization on insulin of another 8 per cent. originally treated by diet alone is included; 55 per cent. lost some time from illnesses unconnected with their diabetes. Our survey has no statistical accuracy; even if it had there seem to be no figures available to compare the working time lost by our patients with that lost by a mixed non-diabetic population. We think, however, the survey shows that most treated diabetics are good employees from the health point of view, and hope that the widespread prejudice against their employment may be removed. In addition it should be pointed out that we have surveyed only the hospital section of the working community and that we know that the work loss of a higher stratum of diabetics, whose incentive to work is usually greater, is still less.

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ACUTE BULBAR PARALYSIS

REPORT OF TWO CASES

BY

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The cases described below present features of sufficiently unusual interest to justify their publication. First, the syndrome was identical in both instances, the bulbar condition being the only paralytic manifestation; and, secondly, both patients died twelve days after tonsillectomy.

Case I

This patient was a female child, aged 6 years, of a small and somewhat frail physique. On July 25 she had her tonsils removed by guillotine. She was seen again on the 29th, when she complained of frontal headache, earache, and slight pyrexia. There was a history of a previously restless night with sudden starting up in fright. In view of the local prevalence of poliomyelitis a routine examination of the nervous system was made, but no evidence of any nervous lesion could be found. The tonsil beds were healthy and granulating. There was no sign of congestion of the drumheads, nor was there any tenderness over the mastoid cells.

The following morning the temperature was normal and the headache was gone, but there were still shooting pains in the ears, with no deafness or signs of otitis. Two days later she was well and wanted to get up. She remained well until August 5, when there was a return of the headache with pyrexia of 99.5° F. The next morning the headache had gone, but the temperature had risen to 103° F. Thick mucus was present in the throat that was difficult to remove; she could not be persuaded to cough it up or to swallow it. She was able to swallow fluids in small quantities, but in a way that suggested the presence of some obstruction in the throat. On examination the throat appeared healthy, but the palate on the right side seemed to be lower. There was no sign of pharyngeal congestion, and the tonsil beds had quite healed. There was a copious amount of thick mucus which made examination difficult, but its removal by a swab was not unduly resented. The child did not look ill, nor did she complain of any pain or discomfort. Constipation was stubborn. During the course of the day the amount of

secretion in the throat increased to a degree which became alarming, and it seemed to threaten to choke her. At this stage a very transient drooping of the right side of the face was noticed on momentary occasions. She developed a wild look, and tried to strike out at anyone who was beside her; she was continually turning from one side to the other and occasionally leapt up in bed; she gave the impression of being enraged at her inability to remove the mucus, for she was able to speak and was quite rational. At this stage the temperature was still 103° F., and the pulse was 110. The only signs in the lungs were a few moist sounds at the bases. The respiratory rate was 22. She was given 1/100 grain of atropine to diminish the secretion, after which she appeared to quiet down and was able to snatch sleep at ten-minute intervals, when the mucus woke her again. The pulse rate did not rise in spite of the atropine.

During the early hours of the morning her condition became worse; the lungs became congested, the pulse rate rose to be uncountable, and the temperature began to fall. There was no attempt at coughing, and the secretion in the throat disappeared while the oedema of the lungs progressed with increasing cyanosis. The soft palate of the right side was much lower than that of the other side. The pupils were small and did not react to light. Towards the end there was a persistent pouting appearance of the lips which was quite symmetrical; she was able to speak, but in a muffled manner. Towards the end she repeatedly asked for drinks, which were mostly regurgitated through the nose.

The congestion and the cyanosis increased and some head retraction was evident. She died about fifteen hours after the first appearance of the acute dysphagia. At no time during the course of the disease was there any disturbance of the nervous reflexes or the muscle tone, with the exception of the terminal head retraction. She was conscious until she died, and was able to understand questions and to reply, although indistinctly.

Post-mortem Findings.—The lungs were oedematous, with several small haemorrhagic infarctions present, probably terminal. The heart muscle appeared healthy, but there was some dilatation of the right side. An excess of free fluid was present in the pericardium. Examination of the brain showed that the pia mater was markedly hyperaemic without any exudate. On section through the medulla the grey matter appeared hyperaemic and bulged. The spinal cord showed no macroscopic change.

Case II

A robust female child, aged 14 years, had her tonsils removed on July 25 on account of a tuberculous adenitis. She made an uneventful recovery from the operation, and was out in the fields a week later. The patient was seen again on the evening of August 6; she gave a history of having been out of sorts for the past two days, which was attributed to the strong sunshine. On examination she was found to have a temperature of 101° F., and, when asked, admitted that she had a slight headache. There was no neck rigidity, and Kernig's sign was negative; abdominal reflexes were active. The bowels had not acted for three days. On the following morning the temperature had risen to 103° F., and the headache was still present though not severe. The pulse rate had dropped to 80 and the respiratory rate was 20. There was a complaint of a thickness in the throat, and a difficulty in raising the phlegm. The throat appeared perfectly healthy and symmetrical, and the tonsil beds had healed up. A throat swab was taken, and the report later stated that no streptococci, haemolytic or non-haemolytic, were present. Examination of the chest failed to reveal any abnormal signs. Later in the day there did not appear to be much change except that the secretion was more copious. The pulse rate was still slow in proportion to the temperature. An hour and a half later the child was seen again. She was then leaping up in bed and flinging her arms about, with a wild look in her eyes. Her temperature was subnormal and the pulse was uncountable; the pupils were dilated. During examination she projected a copious quantity of stomach contents all over the bed. Following this a transient drooping of the

right side of the mouth was noticed, which disappeared as she moved. Shortly after a large quantity of strawberry-coloured froth welled up from the mouth; the child was still fully conscious, but, although making attempts, she was unable to speak. The restlessness was severe, but she settled slightly after morphine and atropine, but died half an hour later. It appeared that half an hour before she was last seen she had asked for something to eat and was given some milk mixture; she had taken this quite well and asked for the back rest to be lowered so that she could go to sleep. Apparently this was done, and was followed a few minutes later by the alarming symptoms described above. In short, she had appeared reasonably comfortable at 9 p.m. and was dead at 11 p.m.

Post-mortem Findings.—The lungs were markedly oedematous; the heart was somewhat dilated, with no free fluid in the pericardium, while the musculature appeared healthy. The brain and spinal cord gave no macroscopic evidence of congestion. Apart from slight enlargement and softening of the spleen there were no visible changes suggestive of any toxic process.

Both cases were given prontosil from the onset.

Discussion

There is a remarkable similarity in the course of the above two cases, and a discussion of their features appears to resolve itself into three definite stages: (1) the stage of invasion; (2) a stage of irritation; and (3) a stage of rapidly progressive paralysis.

1. *The Stage of Invasion.*—In the first case the symptoms were headache and possibly earache, accompanied by slight pyrexia that settled down in twenty-four hours and was followed by a period of comparative well-being. In the second case there was no headache, but the patient complained of feeling out of sorts.

2. *The Stage of Irritation.*—This stage is considered to begin with the return of the pyrexia and the headache. The first unusual symptom was the appearance of sticky mucus in the throat that was difficult to swallow. This mucus probably originated from the submaxillary and sublingual glands, and might be considered to be due to increased activity of the secretomotor fibres of the vagus which arise in one of the dorsal nuclei and are referred to by Samson Wright as the superior salivary nucleus. A pulse rate slow in proportion to the temperature was also found in both cases at this stage, due in all probability to stimulation of the depressor fibres of the vagus. A further common feature was obstinate constipation. It is apparent that all the symptoms of this stage can be attributed to vagal stimulation, which is considered to have been produced by toxic irritation of the dorsal nuclei in the medulla before the actual changes that later produced paralysis. No actual paralysis was demonstrable, and the difficulty in swallowing was attributable to the presence of the thick mucus, which could be swallowed only with an effort; fluids could be taken without much difficulty, and there was no regurgitation until later.

3. *The Stage of Progressive Medullary Involvement.*—The onset of this stage was fairly sudden in both instances. It was ushered in by episodes of explosive muscular activity in association with a wild appearance of the eyes. The temperature dropped and the pulse rate rose rapidly. Signs of pulmonary congestion became increasingly prominent, although there was no evidence of respiratory failure at any time. The mucus disappeared from the throat, and a right-sided paralysis of the soft palate was seen in one case. Transient right-sided facial paralysis was present in both instances. Ability to swallow was also impaired; later it was complete, with regurgitation

of food through the nose. Speaking was muffled, yet consciousness remained. The terminal event was pulmonary oedema, more acute in the second case. At this stage the prominent symptoms were associated with progressive impairment and ultimate destruction of vagal control, both motor and sensory. Here a short description of the relations in the medulla seems pertinent. The ascending fibres of the seventh, ninth, and tenth cranial nerves end in a column of dorsal nuclei from which the involuntary fibres arise. Descending fibres end in the grey matter just lateral to that column in the fasciculus solitarius. The voluntary fibres of the seventh, ninth, tenth, and eleventh cranial nerves arise in one column just anterior and lateral to the dorsal column and extend from the lower border of the pons through the medulla to the spinal cord. The former column may be taken to correspond to the posterior horn cells and the latter to the anterior horn cells of the spinal cord.

The rapid pulse, the disappearance of the mucus from the throat, the progressive paralysis of the soft palate and the muscles of swallowing, and the signs of cardiac failure are clinical evidence of damage in the region of the dorsal columns. The pulmonary oedema was probably secondary to ventricular failure from loss of vagal control. The presence of transient facial paralysis at the end suggests that the lesion was spreading upwards. Furthermore, the terminal wild appearance and choreic movements might have been produced from irritation in the region of the red nucleus and thalamus.

Only in the first case was there any sign of meningeal irritation as indicated by the terminal head retraction, and post-mortem evidence of meningeal congestion was found only in this case. Here also there was constriction of the pupils with loss of reaction to light, suggesting third nerve involvement. Extreme dilatation was apparent in the other case, possibly from sympathetic over-excitation by loss of vagal inhibition. Therefore, clinically, it appears that the seventh, ninth, and tenth cranial nerves were affected, while the close anatomical relationship of their nuclei in the medulla can leave little room for doubt as to the site of the major lesion. First the dorsal column corresponding to the posterior horn cells was affected and later the motor column, corresponding to the anterior horn cells. This is the usual sequence of events when the spinal cord is affected by the virus of acute anterior poliomyelitis.

The most striking feature of the above two cases is the fact that both patients died twelve days after tonsillectomy. It is perhaps permissible to suggest that they died from the virus which causes acute anterior poliomyelitis, especially since the disease occurs in the district in epidemic form. At present the disease is supposed to be due to a filterable virus which gains entry via the nasal mucous membrane. The path of invasion is apparently through the peripheral nerve terminals in that mucous membrane. It is highly probable that the virus entered through the raw tonsil bed to produce a specialized form of the disease. The tonsil area of the throat is innervated by the glosso-pharyngeal, and the infection travelled up the peripheral nerve to produce the damage in the region of the dorsal nuclei from which the glosso-pharyngeal arises. These children made contact with one another only for a short time after the operation; they lived a considerable distance apart, and had not been in contact with known cases of the disease. Two other children who were operated on at the same time are at present well.

Portions of the brain and spinal cord are being cultured, and diagnosis has yet to be confirmed by injection of brain

substance into monkeys. Since this will take some time it was considered advisable to report these cases while the disease was still prevalent.

Summary and Conclusions

1. That these are cases of polio-encephalitis.
2. That there is a definite association between the course of the disease and the presence of a raw tonsil bed, even although it was completely healed up when the symptoms of the disease arrived.
3. That the incubation period in these cases was about four days, presuming that the infection took place at the time of tonsillectomy.
4. That the route of the infection was from the raw tonsil bed to the dorsal nuclei via the peripheral fibres of the glosso-pharyngeal nerve.

HAEMORRHAGE FOLLOWING TONSILLECTOMY

BY

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The purpose of this paper is to discuss a subject which is of the utmost importance not only to all oto-rhino-laryngologists but to all surgeons, house-surgeons, and general practitioners who may be faced with the problem of haemorrhage following tonsillectomy. It is a very real problem and one, moreover, to which only scanty reference is made in even the largest and most authoritative of textbooks. Such haemorrhage is terrifying enough for the patient, and may be even more alarming for the medical man unaccustomed to dealing with such cases.

For the efficient treatment of haemorrhage following tonsillectomy a thorough knowledge of the factors concerned in its production is necessary, and careful consideration of these factors will do much to minimize the occurrence of this distressing complication. I propose to discuss this subject under the following headings:

I. *Factors influencing the occurrence of haemorrhage following tonsillectomy:* (a) pre-operative; (b) operative; (c) post-operative.

II. *Types of post-operative haemorrhage.*

III. *The management of a case of haemorrhage following tonsillectomy.*

FACTORS INFLUENCING ITS OCCURRENCE

Pre-operative

Although tonsillectomy may be indicated in any particular patient, certain precautions must be taken before operation is decided upon, and many factors remain to be considered before the actual time of operation is arranged. In taking a history of the case and during examination of the patient particular attention should be paid to the following points.

1. *Severe Tonsillitis or Peritonsillar Abscess.*—Recent acute local infections increase the risk of haemorrhage both during and after operation, and for this reason tonsillectomy should not be performed within a period of a few weeks following either of the above conditions. No hard-and-fast rule can be laid down as to the time which should be allowed to elapse before operation is undertaken, but I am of the opinion that, following such acute local infections, tonsillectomy should be

deferred for a period of not less than four weeks in the case of children and not less than six weeks in the case of adults.

2. *Infections of the Nose or Nasal Accessory Sinuses.*—Acute sinusitis, whether associated with a sore throat or not, is a contraindication to the immediate performance of the operation. A common cold comes under the same category. The presence of chronic sinusitis should not be overlooked. Adequate measures for its efficient treatment should be instituted before tonsillectomy is considered.

3. *Bleeding.*—Inquiry should be made as to whether there is any susceptibility to bleeding on the part of either the patient or any member of his family. The occurrence of prolonged haemorrhage following cuts or abrasions, or after dental extraction, demands the investigation of the patient's coagulation time and bleeding time. Normal limits for these are: coagulation time (Lee and White), 4 to 7 minutes; bleeding time (Ivy), 1 to 4 minutes. If these times are found to be prolonged it is possible that they may be brought within normal limits by the administration of calcium and alkalis or by blood transfusion. If these limits cannot be attained, then the question of operation, the risks of which should be explained to the patient, must be reconsidered. Unless it is absolutely necessary tonsillectomy should be deferred.

4. *Menstruation.*—On account of the psychological upset and the delayed coagulation time of the blood, operation should be avoided just before or during the first few days of a menstrual period.

5. *Jaundice.*—Tonsillectomy is contraindicated in the presence of jaundice due to any cause.

6. *Anaemia.*—In any severe anaemia operation should not be undertaken without preliminary treatment or even a blood transfusion.

7. *The Type of Patient.*—It is interesting to note the physical characteristics of any patient, for it is in those of the plethoric type, with short thick necks, that haemorrhage is most prone to occur. Heavy smokers, chronic alcoholics, and patients with highly excitable and emotionally unstable temperaments also suffer in this same respect. (All sandy-haired children are suspect.) Again, red-haired females and the subjects of thyrotoxicosis show a tendency to bleed excessively following operation.

As a routine, adult patients should be given 10 grains of calcium lactate thrice daily for three days before tonsillectomy, and be asked to abstain from alcohol and tobacco for a similar period.

Operative

Consideration of the various methods of tonsillectomy does not come within the scope of this paper, but the type of operation performed, however, does influence the incidence of severe post-operative haemorrhage. In a large series severe bleeding occurred in 2.6 per cent. of cases after guillotine enucleation, as compared with 0.7 per cent. of cases following removal by dissection (McNally, 1927). The figures will depend to some extent, of course, on the care and discrimination exercised by the individual surgeon in his selection of cases for operation.

It is a significant fact that patients who have been subjected to guillotine enucleation of tonsils nearly always vomit after operation, and the vomitus always contains blood. Douching the face and neck with ice-cold water at the end of the operation induces considerable reflex vasoconstriction and is usually sufficient to cause cessation of the haemorrhage, but it must be remembered that although bleeding may *apparently* have ceased, in that blood is not trickling from the mouth, a lightly anaesthetized patient may be swallowing the blood. It is important, therefore, that the actual tonsillar fossae should be inspected at the end of the operation and that the patient should not be sent back to bed until all bleeding has been controlled. In cases in which the adenoids have