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Near-drowning, scuba diving:
An unusual late sequela of bulbar polio

by

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Abstract

This case report illustrates an unusual hazard of underwater sports: vagal neuropathology secondary to early poliomyelitis which resulted in residual palato-pharyngeal paresis. Gag and swallowing reflexes appeared to function adequately but in fact were not normal. When stressed, during water aspiration, they were inadequate, resulting in great risk to the underwater enthusiast. A history of early viral myelitis must be considered as a potential hazard in underwater sports.

Introduction

It has been estimated that there are almost 3 million underwater divers in the United States and 200,000 more are trained and certified each year (Becker and Parell, 1979). In Canada scuba diving is a rapidly growing underwater sport, which is undertaken by 40–50,000 certified scuba divers (Crook, 1977).

Although the dangers of diving have been popularly associated with great depths, it must be recognized that both snorkeling and scuba diving in shallow water—even in a swimming pool—present risks to the diver (Strauss, 1979).

It is recognized that conditions such as claustrophobia, mental instability, epilepsy, cardiovascular insufficiency, diabetes, chronic pulmonary disease, and dental cavities exacerbate the risks of scuba diving (Strauss, 1979).

The most common injuries suffered by divers are sinus and otitic barotrauma (Becker and Parell, 1979). As the otolaryngologist is likely to be consulted regarding these injuries, he or she should also not only be familiar with their diagnosis and treatment but should be able to identify persons especially 'at risk' for these injuries. The otolaryngologist should be particularly cognisant of nasal airway impairment, chronic sinusitis, tympanic membrane perforations, badly scarred tympanic membranes and radical mastoid cavities (Becker and Parell, 1979; Farmer and Thomas, 1976; Caruso *et al,* 1977; Wilson, 1972; Fagan *et al,* 1976).

An unusual cause of diving (or swimming) injury which has not previously been described results from residual vagal pathology secondary to poliomyelitis. Swallowing abnormalities have not previously been considered as a hazard of diving but, as the case below illustrates, they should be considered. The individual with a swallowing and/or gagging reflex abnormality is at risk when involved in underwater sports.

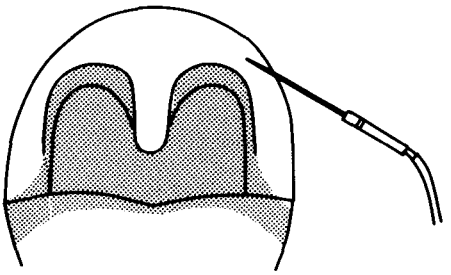
Case Report

Mr. H. T. is a healthy 54-year-old lawyer who on two occasions almost drowned when swimming. He had been learning to snorkel and was taking scuba classes.

On the first occasion, while snorkeling, he unexplainedly choked on some water. Fortunately, he was in sufficiently shallow water to walk ashore. The episode led to a bout of pneumonia from which he completely recovered.

The second episode was during his first free-dive of the season. He had been underwater when he began to experience trouble with his mask. He surfaced and attempted to blow up his personal floatation device. However, he began to choke and lost consciousness. Fortunately, a fellow swimmer was nearby and he was rescued. He was apparently unconscious for fifteen minutes. Hospitalization was only required overnight and he showed complete recovery. Subsequent questioning suggested that he was aspirating water inappropriately. On reviewing his early history, it was noted that he had polio at age 28

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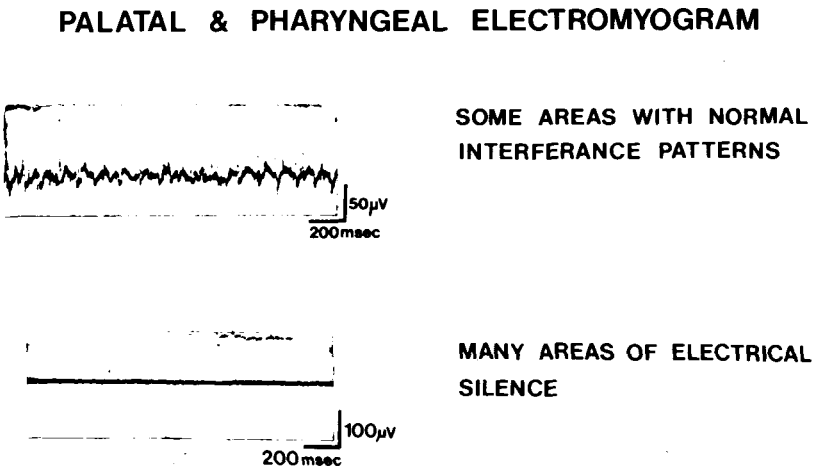


FIG. 1

Electromygraphic examination of the palato-pharyngeal muscles shows absent electrical activity on the right side.

with palatal paresis and some difficulty in swallowing. However, he and his physicians believed that he had recovered completely and apparently had no sequelae. The rest of his history was unremarkable.

Physical examination showed that his left vocal card was normal while the right one was a little erythematous. Both cords moved normally. The right side of his palate was insensitive to touch and there was evident poor gag reflex. The rest of the otolaryngologic examination was normal.

The patient underwent electromyographic examination of the laryngeal/pharyngeal musculature and this was found to be normal. There was, however, marked reduction in activity of the muscles of the right side of the palate. Many areas were entirely silent electrically (Fig. 1). Similar findings were encountered in the constrictor muscles of the pharynx.

It was concluded that the patient suffered from a residual minor right-sided palato-pharyngeal paresis. The only evident etiology of this disability was a neuro-muscular dysfunction secondary to his earlier poliomyelitis. His gag and swallowing reflexes had been functioning adequately but not normally. When stressed by scuba diving they

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were inadequate, exposing him to the risk of drowning.

Discussion

To the best of our knowledge this is the first reported case of near-drowning secondary to the sequelae of early poliomyelitis. Residual pharyngeal and palatal pareses were responsible for two incidents which were nearly fatal in an underwater swimmer. Reviews of otolaryngologic pathology in diving and swimming (Wilson, 1972; Macfie, 1964; Taylor, 1959; Adolfson and Berghage, 1974) discuss many common and rare conditions, but apparently no paper has dealt with viral myelitis as an etiologic agent in drowning.

Damage to the vagus nerve trunk or to its pharyngeal branches results in paralysis of the levator veli palatini muscles and of the pharyngeal constrictors. Unilateral palatal paralysis gives rise to defective elevation of the palate and movement of the uvula toward the affected side. Unilateral pharyngeal paralysis produces drooping of the pharyngeal wall on the affected side. This may be accompanied by difficulty, usually slight, in clearing secretions from the throat and some interference with swallowing (Davis *et al.*, 1975).

The polio virus can be widely disseminated or very localized in the body. Once within the gray matter of the nervous system, it shows considerable selectivity in its damage. Paralysis of swallowing is the most frequent abnormality in cranial nerve function. Many patients with transient paralysis of swallowing later show little or no residual pharyngeal weakness. This suggests that the acute dysfunction is due to involvement of an integrating center for swallowing rather than the result of direct invasion of the cells of the nucleus ambiguus. However, where there is residual weakness, the nucleus has been shown to be partially invaded (Webb and Kuhlenbeck, 1981).

In our own case it is probable that early polio resulted in permanent damage to the vagal supply of the right palato-pharyngeal musculature. It is probable that the nucleus ambiguus was involved although this is impossible to ascertain absolutely. The patient was able to cope with this pathology and in fact was unaware of it until the system was severely stressed by aspiration of water during diving. Thus, though the pathology did not affect his day-to-day pharyngo-palatal activities, when stressed by the demands of scuba diving, the system broke down.

Prior to the introduction of the polio vaccine (1955–inactivated; 1959–live), polio was an epidemic disease. The last major paralytic polio outbreak was in 1953, affecting over 31,000 people in the U.S., Canada, Australia and New Zealand (Melnick, 1978) However, sporadic outbreaks have continued up until the present. Of those afflicted with paralytic polio, approximately 1–2 per cent have been left with gross residual sequalae, (Melnick, 1978). The number of individuals with unrecognized sequalae can only be guessed. The present case is an example of the latter and stresses the importance of considering childhood and early adulthood viral myelitis in the etiology of unexplained drownings.

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