GLOSSOPHARYNGEAL NEURALGIA ACCOMPANIED BY UNCONSCIOUSNESS*

JAMES L. THOMSON, M.D.†
Norfolk, Virginia

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The purpose of this presentation is to discuss the diagnostic criteria of glossopharyngeal neuralgia and to emphasize the role of the syndrome that resembles that of the carotid sinus reflex, as seen in an occasional case.

The syndrome of glossopharyngeal neuralgia was first described by Weisenburg in 1910 in a case of angle tumor pressing on the 9th cranial nerve. Ten years later Sicard and Robineau reported 3 cases of glossopharyngeal neuralgia. During the following 7 years until 1927, 2 cases had been reported by Harris, from the Mayo Clinic, and 2 by Dandy. Adson in 1924 presented a technique for peripheral nerve section in the neck for this nerve. He said that it was a difficult surgical procedure because of the anatomical position of the nerve. At the time he had explored the possibility of cutting the nerve intracranially, by cadaver dissection, illustrating the unilateral cerebellar approach. However in 1927 Dandy reported the first intracranial section of the nerve, when he operated upon 2 patients within 6 weeks' time through a unilateral cerebellar craniectomy wound. Since then there have been many case reports illustrating the diagnostic and operative features of the disease.

Glossopharyngeal neuralgia differs from trigeminal neuralgia (tic douloureux) only in the location of the pain. Both have characteristic locations of the sudden, severe, paroxysmal, lightning-like pain, frequently set off by external stimuli. Each generally occurs in middle to late life. It remains only to distinguish the location of the pain by taking a careful history to establish the correct diagnosis. However, occasionally the patient is unable to give an exact account of the pain either because of its severity or because of mental deficits, such as senility, low mentality, etc. As we all know, this pain is located in the glossopharyngeal region, back of the tongue or in the ear. Confusion in diagnosis should occur only when the most severe pain is located in front of the ear or seemingly within it. Then the differential diagnosis of neuralgia of the nervus intermedius (nerve of Wrisberg), or 3rd division tic must be made. Cocainization of the throat usually, but not always, abolishes the pain for some minutes and thus aids the diagnosis.

Intracranial section of the 9th nerve alone generally relieves the pain entirely but if the ear pain is especially bad, section of the upper two filaments of the vagus should be done, which has been stressed by both Dandy and Spurling.

The resultant anesthesia is confined to the pharynx, nasopharynx, back of the tongue and tonsillar pillars. No motor deficit has been shown resulting from the paralyzed stylopharyngeus muscle.

A much less frequent involvement in the disease process is the resemblance to the syndrome of the hyperirritable carotid sinus. The carotid sinus receives its nerve supply from branches of the glossopharyngeal (nerve of Hering—an afferent branch),

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† 405 Wainwright Bldg., Norfolk 10, Virginia.
vagus and sympathetic nerves. The nerve of Hering has been shown to be the depressor nerve from the sinus and the vagal branches respond to chemical stimuli, such as atropinization. Weiss\textsuperscript{11} and associates worked out the carotid sinus syncope syndrome, describing three types: (1) vagal type, in which syncope results from cerebral anoxia caused by reflex cardiac asystole; (2) depressor type, in which syncope results from fall in blood pressure alone; and (3) cerebral type, in which syncope occurs without change in cardiac rate or blood pressure. When the neuralgic pain is unusually severe, bradycardia to asystole, hypotension, syncope and convulsive movements may occur. This group of symptoms resembling those of the carotid sinus reflex is mediated through the afferent glossopharyngeal branches in the throat rather than from within the sinus (nerve of Hering).

Following this work of Weiss, reports of intracranial section of the 9th nerve for hyperirritable carotid sinus reflex have appeared and the results are quite satisfactory.

Syncope and cardiac arrest associated with glossopharyngeal neuralgia were first referred to by Riley and associates\textsuperscript{6} in 1942, but no operation was performed. In 1948 Ray and Stewart\textsuperscript{4} reported a similar case and referred to another of Browder's which had been seen but not reported. Ray's patient was completely relieved by intracranial section of the 9th nerve.

In 1950 Roulhac and Levy\textsuperscript{7} were the first to note convulsive seizures associated with glossopharyngeal neuralgia. The patient was a 72-year-old white woman with a 20-month history of typical pain in the left side of her throat. Three weeks previous to admission, she began having convulsive movements shortly after the onset of a paroxysm of throat pain. There was twitching of the mouth, with clonic movements of the arms and upward deviation of the eyes. These would last only a few seconds. She was put on Phenobarbital and Dilantin, and improved to such a degree that the carotid sinus reflex element of the neuralgia was almost missed. She was discharged and 10 days later was readmitted, having many more paroxysms of pain with an increased number of convulsions. Then an electrocardiogram was done demonstrating asystole. The left glossopharyngeal and upper two filaments of the vagus nerve were sectioned intracranially with prompt and complete relief of the pain and convulsions. Roulhac and Levy believed that the syncope and convulsions occurred from afferent stimuli arising in the glossopharyngeal nerve rather than the carotid sinus, the mechanism being bradycardia with a fall in systemic blood pressure and cerebral anoxemia (Weiss—vagal group classification).

Richburg and Kern\textsuperscript{8} in 1953 reported a similar case. The patient was a Texas farmer, who had severe throat pain with frequent attacks of asystole, syncope and convulsions. The sinus syndrome was relieved by administration of atropine. Intracranial section of the 9th nerve and upper two filaments of the vagus nerve gave permanent relief of the entire syndrome.

The present report concerns another case of glossopharyngeal neuralgia associated with convulsions but without asystole or fall in blood pressure, which was initially mistaken for tic douloureux of the 3rd division of the left 5th cranial nerve. Temporal preganglionic subtotal sensory neurectomy was done without relief. The true nature of the pain was later recognized, and the 9th nerve was cut intracranially with prompt relief of all symptoms.

\textbf{CASE REPORT}

Case \#11914-52. A 67-year-old colored woman was referred stating that for 3 months she had had intermittent, excruciating pain in the left side of the face. It would sometimes appear
by talking, eating, coughing, or drinking cold water. It seemed to be intense in front of the ear and involve the lower portion of the cheek and jaw. There was a second-degree burn on the left cheek from using heat. Also she had lost a great deal of weight, being afraid to eat because of fear of onset of pain. No further history could be obtained from the patient on repeated questioning and this was fragmentary, as the attacks of pain were so close together and she cooperated very poorly. When attempting to talk she would stop quickly, having a severe paroxysm of pain lasting 10 to 30 seconds. At one time she would point to the angle of the jaw, and again would hold the chin region. At other times she complained of the ear hurting. Similarly stroking the jaw region would give the impression of producing a pain, but the paroxysms were so frequent that it was uncertain that a trigger zone existed. The tonsillar and posterior tongue regions were stimulated without positive information.

Examination. The patient was of average size. B.P. was 130/88. On the left check at the angle of the jaw was a burn, 3 cm. in diameter, which was healing. The nasopharynx and ears showed no pathology. Both lung fields were clear and no enlargement of the heart was found. The heart rate and sounds were normal. Abdominal and pelvic examinations were negative. Neurological findings were normal excepting for the appearance of severe pain.

The blood showed 11.4 gm. of hgb., 6,100 WBC, sedimentation rate of 24, negative Wassermann, fasting blood sugar of 125 mg. per cent, N.P.N. of 27, and alkaline phosphatase of 1.7 units. Urine was normal. Roentgenograms of skull, cervical spine and temporomandibular joint were normal.

1st Operation. Temporal preganglionic sensory neurlectomy of the lower two thirds of the left 5th cranial nerve was done under intratracheal anesthesia 3 days after admission.

Course. When the patient had reacted well from the anesthesia she complained of the same pain and exhibited the same type of behavior toward it. During the next 3 days the paroxysms were more frequent, and severe. She now refused to eat because of precipitating an attack.

On the 4th postoperative day, while observing an attack of very severe pain, vision suddenly became fixed, her head dropped forward, and unconsciousness ensued with strong tonic-clonic movements of the head, upper extremities and, to lesser degree, of the legs. These movements lasted 10 to 15 seconds, stopped, and she quickly regained consciousness being momentarily free of pain and well oriented. During several similar episodes of unconsciousness the pulse remained strong with the rate not being reduced more than 20 beats per minute (not less than 60 per minute), and the blood pressure remained above 120/70. The nasopharynx was cocainized thoroughly but the paroxysms of pain would continue with and without external stimulation.

A nose and throat consultation was obtained but examination failed to demonstrate nasopharyngeal pathology or a trigger zone in the pharyngeal region. A diagnostic pneumoencephalogram was done 7 days postoperatively and appeared normal.

2nd Operation. Five days later the left glossopharyngeal and upper two filaments of the vagus nerve were sectioned under intratracheal anesthesia through a unilateral cerebellar cranietomy approach.

Course. On awakening from this procedure she was free of pain and no further convulsive movements occurred. B.P. remained at the preoperative level of 180/80, convalescence was satisfactory, and she was discharged symptom-free 8 days later. When last seen the following month, she had had no return of the pain.

COMMENT

This case is unusual in that there was no appreciable change in heart rate or blood pressure during the carotid sinus syndrome period of the pain. It would correspond to the cerebral type of Weiss. Pressure on each carotid sinus before and after operation had no effect on the carotid sinus reflex syndrome. Certainly this woman's pain reflex was mediated in the throat, and by excessive stimuli through the afferent glossopharyngeal nerve caused the convulsive phenomenon, similar to that which would be initiated through a hyperirritable carotid sinus.
A CLAMP FOR TEMPORARILY OCCLUDING SMALL BLOOD VESSELS

SAMUEL P. W. BLACK, M.D., AND WILLIAM J. GERMAN, M.D.

Department of Surgery, Yale University School of Medicine, New Haven, Connecticut

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A small, light, and effective artery clamp may be adapted from an inexpensive electrical test clip* (Fig. 1). Historically, this type of artery clamp, a small spring forceps also known as a “bulldog” or “serre fine,” dates from about the middle of the nineteenth century and has changed but little in form since that time. It was not until the advent of aseptic surgery at a somewhat later date, however, that it became possible to occlude a blood vessel temporarily while a procedure was performed upon it with routine success in re-establishing its continuity and patency. Concomitant with this achievement the artery clamp, of which the spring forceps is but one type, came to assume its present importance. There are now many forms of artery clamps available commercially, and DiPalma3 has devised a method of making a small artery clip from a safety pin.

The clamp presented herein is made of phosphor-bronze and weighs 2 gm. It takes a high polish and may easily be filed free of sharp edges. The instrument is small enough to be applied to a blood vessel by means of a silver-clip applicator, which, when placed on the “catch,” allows the operator to position the clamp about the vessel with accuracy and relative ease before allowing it to close (Fig. 2). It can occlude any artery caught completely between its jaws, which are smooth enough to be applied directly to the vessel if so desired. Should too strong a pressure be exerted the spring can be forced together; or, if the jaws exert too weak a pressure, this may be altered by forcibly separating the spring. Removal of the small screw at one end of the clamp allows the position of the instrument to be controlled by means of a thread placed through the opening thus made.

* Termed “Wee-Pee-Wee” clip by the manufacturers, The Mueller Electric Company, Cleveland, Ohio.