COUGHING AND UNCONSCIOUSNESS

The So-Called Laryngeal Vertigo

J. P. PROUDFIT, M.D.* and LOUIS J. KARNOSH, M.D.
Department of Neuropsychiatry

LARYNGEAL VERTIGO is a dramatic syndrome, first described by Charcot[1] in 1876, in which coughing produces unconsciousness with or without convulsions. At times only giddiness follows the cough which is attributed to spasm of the larynx with closure of the glottis. The cough is reported to be distinctive in character and is usually preceded by a tickling sensation in the throat. In some cases the cough is mild. The syndrome occurs most commonly in middle aged men. They are described as being plethoric, emphysematous, or having a tendency to hypertension. There is a frequent association with laryngitis or bronchitis. Many physicians are unaware of the existence of such a condition.

The syndrome is not rare. Whitty[2] reviewed the literature in 1943 and reported approximately 85 cases. Many cases have not been reported because as Rook[3] says, "They do not get past the general practitioner who adopts a very reasonable 'wait and see' attitude," the attack often not being repeated. In the past year one of us, on a general diagnostic service, has seen two examples of this syndrome. Adams[4] reports that 3 such cases were referred to him by a single physician within a period of a few months.

Case Reports

Case 1. A 43-year-old coal miner was seen at the Cleveland Clinic February 19, 1948. He complained that he had "blacked out" after coughing on 8 occasions during the past four years. He coughed violently until he "got blue in the face" and fell unconscious to the floor. He hurt himself in several of these falls. As he coughed he bent forward slightly and awakened as he hit the floor. There was never any convulsive movement or incontinence. After the spell he felt weak and nervous. He believed that he could abort an attack by sitting down and "supporting" himself. Six or 7 times a day he would think he was about to have an attack which did not materialize; for the past few weeks he had been slightly dizzy all the time. There had never been any true vertigo, tinnitus, or deafness.

Frequently small furuncles had developed in his ear canals off and on over the past six or seven years. He had a dry throat at times but no hoarseness and a non-productive cough. There was a mild constant ache in the mid-chest anteriorly, unrelated to exertion. He said that he wheezed in cold, damp weather and there were times at night when he had to sit up to get his breath. He denied exertional dyspnea. He had had considerable gaseous distress with a smothery feeling in the epigastrium occurring about fifteen minutes after meals but obtained some relief with soda. He drank about 1 quart of whiskey per month. He smoked a package of cigarettes a day.

Thirteen siblings were living and well. There was no history of epilepsy or allergic disease in the family. Pleurisy followed a herniorrhaphy in 1927. A urethral discharge was present in 1930, 1939, and 1943. Pneumonia occurred in 1904 and 1918.

**Former Fellow in Medicine, Cleveland Clinic. Now practicing in Washington, Pennsylvania.*

The patient was a large, obese white man of good color. He was 69½ inches tall and weighed 194 pounds. His pulse rate was 92 per minute. The blood pressure was 140/92. The ear canals were normal, as were the drums. The larynx appeared normal except for slight asymmetry of the epiglottis. The chest auscultation revealed persistent sibilant expiratory rhonchi in the right lower lobe. The heart was normal. Carotid pressure produced no symptoms. The finger nails were beaked but there was no clubbing. The reflexes were physiologic.

The vital capacity measured 3000 cc. (normal, 5200 cc.). The chest x-ray showed no evidence of active pulmonary disease. The electrocardiogram, urinalysis, and hemogram were normal. The sedimentation rate was 0.4 (normal up to 0.45) by the Ernstene-Rourke method. The fasting blood sugar was 108 mg. per cent. The Wassermann and Kahn tests were negative.

Case 2. A 57-year-old retail clothier of German-Jewish extraction was seen in the Clinic October 22, 1948. He gave a history of having become unconscious on 3 occasions following violent coughing. These attacks had occurred in April, 1947 and on October 8 and October 19, 1948. On the latter occasion he had fallen unconscious through a plate-glass window. There was no warning. The cough had a peculiar quality. It occurred in a severe paroxysm, "shutting off his wind." He then fell unconscious and remained so for two or three minutes without any jerking or incontinence. For five or ten minutes after recovering consciousness he felt slightly dizzy and confused. At times he had a sensation that there was something in his throat. He coughed a great deal in the mornings without dizziness or loss of consciousness. At times he even coughed so that he would vomit mucus.

He denied any ear symptoms. He had mild leg cramping at night only. He admitted drinking 1 to 4 shots of whisky daily. He smoked a pipe only. He denied hoarseness. He had influenza in 1918 and gonorrhea in 1922. There was no history of epilepsy or convulsions, nor was there a history of epilepsy or mental disease in his family.

The patient was an obese white man with a sallow complexion. The height was 68 inches. He weighed 212 pounds. The pulse rate was 82 per minute. The blood pressure was 138/92. The ears were normal with the canals almost insensitive to manipulation. The vocal cords were thick and injected. The heart and lungs were normal. The reflexes were physiologic. Hyperventilation produced no symptoms. Carotid sinus pressure on one side and then the other produced no symptoms. Simultaneous pressure over both carotid sinuses precipitated unconsciousness for five seconds or so, accompanied by generalized clonic convulsive movements.

X-ray examination of the chest showed a haziness at the right base but was interpreted by the radiologist as "probably negative." The blood count was normal as was the Kahn and Wassermann tests. The electroencephalogram was normal. The report stated that there were a few irregular waves from the left parietal leads which appeared to be artifacts.

Case 3. A 48-year-old salesman first visited the clinic on February 16, 1949. He gave a history of having coughed since 1937. Since 1943, he had experienced asthmatic attacks. The cough and the asthma had become more severe since 1947. In the year before coming to the clinic he had increasingly frequent blackouts with severe coughing paroxysms. Following coughing he suddenly slumped to the floor unconscious and in a matter of seconds regained consciousness. There were no convulsions nor incontinence. The attacks had varied in frequency from 3 or 4 a day to only 1 or 2 per week.

He claimed moderate dyspnea on exertion with some tightness in his chest. He drank little alcohol but smoked a package of cigarettes per day.

There was no family history of epilepsy or mental disease. His father died of tuberculosis and diabetes as did an aunt and uncle. He had malaria as a child. In 1918 he had influenzal pneumonia and in 1921 smallpox. He acquired gonorrhea in 1921. He had painless jaundice for three days in 1925, and in 1938 a kidney stone had been passed.

His height was 68 inches he weighed 220 pounds and his blood pressure was 126/74. There was vitiligo of the hands and penis. The right ear canal contained a large wax plug. The chest was somewhat barrel-shaped. Auscultation disclosed an occasional expiratory wheeze. The heart and reflexes were normal. There was a recurrent left inguinal hernia.

The urinalysis was normal, and the hemoglobin 14 Gm. The leukocytes numbered 10,500 per cu. mm. with 69 neutrophils, 6 eosinophils, 6 monocytes, and 19 lymphocytes. A blood sugar count was normal while the Wassermann and Kahn tests were negative. Chest x-ray showed prominent lung markings but was interpreted as being within normal limits

Comment

Obesity characterized each of the 3 reported cases. The patients were 20, 38, and 42 per cent above their standard weights. There was little tendency to hypertension. In case 1 the blood pressure measured 140/92, the highest recorded in the 3 cases. None gave any personal or family history of epilepsy. All said that violent coughing precipitated the attacks. True convulsions occurred in none nor did any give a history of laryngitis. There were minor laryngeal abnormalities noted in cases 1 and 2. Case 1 showed slight asymmetry of the epiglottis, and case 2 showed thickening and injection of the vocal cords. The larynx of case 3 was not examined. An attempt to cause unconsciousness by encouraging voluntary coughing in cases 1 and 2 produced no symptoms. The chest x-ray in each case was normal. Only case 2 had an electroencephalogram; it was normal. Unilateral carotid sinus pressure produced no symptoms in the 2 cases tested. In one of these, case 2, simultaneous pressure over both carotid sinuses caused unconsciousness with generalized convulsive movements. Attacks produced in this case by coughing were unaccompanied by convulsions according to the history. The significance of symptoms brought about by bilateral carotid sinus pressure is open to question. Authorities state that bilateral pressure should never be employed.[5,6]

Etiology

The etiology of laryngeal vertigo is subject to considerable debate. Laryngeal vertigo, the name most commonly used in the literature, is a misnomer because practically none of the patients had any true vertigo. That the syndrome is laryngeal in origin is indicated by the tickling in the throat which precedes the coughing. Jackson and Jackson[7] state that the appearance of the larynx during the attack is that of glottis and sphincter closure. The etiology is generally discussed under 3 headings: (1) a reflex phenomenon, (2) a laryngeal epilepsy, and (3) cardiovascular causes.

Among the reflex causes mentioned is the carotid sinus reflex. It seems unlikely that this plays much part. Whitty[2] points out that Ferris[8] makes no mention of cough in their detailed analysis of carotid sinus syncope. Sigler[6] in a recent report on 970 cases tested and found to have hypersensitive carotid sinus reflex mentions cough and unconsciousness in none of his cases. He lists 101 symptom complexes resulting from carotid pressure. Cough occurred in 6.25 per cent of the cases. In practically all of the reported cases of laryngeal vertigo pressure over the carotid sinuses has failed to produce symptoms. In one of our cases simultaneous pressure over both carotid sinuses did produce unconsciousness but those who have written most on the carotid response state that bilateral pressure should never be employed. In certain individuals such

a procedure will produce unconsciousness simply by shutting off enough of the brain's blood supply. Thus in our case it seems unlikely that the unconsciousness produced was a result of carotid sinus reflex.

The syndrome could be due to a direct reflex from the larynx via the laryngeal nerves. Movement of the larynx, as in talking, has been reported to precipitate syncope in sensitive subjects.[9] This would coincide with the increased association with laryngitis or bronchitis. Ferris[8] mentions briefly that he has seen cardiac syncope in which the reflex originated in the larynx.

Syncope could be caused by irritation of the bronchi with cardiac arrest. Weiss[6] points out that Sunder-Plassman has demonstrated the existence of sensory nerve endings, similar to those in the carotid sinus, in the intra-pulmonary portions of the bronchi and the muscularis mucosa. Stimulation of these results in cardiac inhibition. Reflex syncope could likewise be mediated via the aortic or cardiac depressor nerve from stimulation of its receptors in the pulmonary vessels.

Lilly[10] reports a case of Arnold's nerve reflex cough syndrome in which stimulation of the posterior and inferior portions of the auditory canal reproduced the symptoms. The patient had experienced unconsciousness after coughing.

Wilson [11] raised the question of an unconsidered laryngeal or respiratory reflex associated with coughing, laughing, excitement, and in his case, swallowing. He regarded laryngeal vertigo and cataplexy as related clinical entities. He presupposed a definite individual susceptibility.

Whitty[2] presents the evidence in favor of epilepsy as the basis for this syndrome. He concludes that the majority are primarily neurogenic: either a true reflex laryngeal epilepsy, an epilepsy with a laryngeal aura, or venous congestion from violent coughing in a patient with a cerebral cortex predisposed to epilepsy. In the first, the cough would be the trigger mechanism setting off the attack. Epilepsy seems unlikely in view of the normal electroencephalographs in these cases. In only one reported case (Whitty's third case) was there a record suggestive of epilepsy. Whitty believed that he ruled out cerebral congestion as a cause when he failed to reproduce the symptoms by inflating a sphygmomanometer cuff placed about the patient's neck.

The cough reflex is most commonly initiated by stimulation of the afferent nerve endings in the region of the tracheal bifurcation or in the laryngeal mucosa. During forced expiratory effort with the glottis closed considerable pressure is developed within the lung. This results in decreased venous return and poor oxygenation as indicated by the familiar expression, "coughed till he got blue in the face." Cerebral congestion results. Rook[3] points out that blowing through a tube in an effort to maintain a column of mercury at a height of 40 mm. will result in unconsciousness in certain individuals. Negative acceleration of human subjects on a centrifuge, with the blood rushing to the head, has not produced unconsciousness. However, there is no noticeable increase in intrathoracic pressure in such experiments as there is with coughing. Pertussis coughing may produce cerebral hemorrhage. Rook[3] believed that this evidence favored cerebral congestion with anoxia and increased intracranial

pressure as the cause of laryngeal vertigo. It may well be that two or more factors must be present to produce an attack of unconsciousness. It is not surprising, therefore, that attempts to reproduce the symptoms by voluntary coughing or by such methods as attempting to obstruct the venous return from the head do not meet with success.

Weiss,[5] in discussing syncope, says that it can result from increased intrapulmonary pressure, as in the Valsalva experiment. The act of coughing, consisting of a short inspiration followed by closure of the glottis and a forcible expiration with the production of considerable increase in intra-pulmonary pressure, is not unlike the Valsalva effort.

Increased intraspinal pressure and engorgement of the intracranial vessels is a cause of syncope particularly in stooping or lowering the head.[5] Coughing is generally performed in just such a position. Coughing causes increased cerebrospinal pressure and cerebral congestion.

Treatment of laryngeal vertigo is entirely symptomatic. Phenobarbital is probably of value but only as a sedative and not as an anticonvulsant.

It seems well to conclude that the etiology of laryngeal syncope is complex. Factors of importance in its production include a direct reflex originating in the larynx, reflexes arising in sensory end-organs within the bronchi, increased intrapulmonary pressure with anoxia (Valsalva), and increased intracranial pressure and cerebral engorgement. There seems little reason to implicate epilepsy, cataplexy, or a carotid sinus reflex for which there must be an individual susceptibility.

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