Cough Syncope Due to Atrio-Ventricular Conduction Block

Daiji SAITO, M.D., Shigeru MATSUNO, M.D.,
Kiminori MATSUSHITA, M.D., Hikaru TAKEDA, M.D.,
Tatsuo HYODO, M.D., Shoichi HARAOKA, M.D.,
Akiharu WATANABE, M.D., and Hideo NAGASHIMA, M.D.

SUMMARY

A case of cough syncope due to A-V conduction block of the heart is reported in this paper. A-V conduction block and light-headedness were induced by coughing but not by an Aschner test, carotid sinus massage, Valsalva maneuver, pharyngeal stimulation, and stimulation of systemic baroreceptors. Permanent right-ventricular pacing completely abolished the patient's symptoms. The results suggest that hypersensitivity of a broncho-pulmonary reflex to coughing was responsible for the A-V conduction block and resulting syncopes.

Additional Indexing Words:

Atrial fibrillation A-V block Tussive syncope

COUGH syncope is a conspicuous and well-defined syndrome. Clinically, the fainting fit is always associated with a paroxysm of coughing, often occurring after a few vigorous coughs. A very sudden onset of giddiness is followed by loss of consciousness. While many theories have been advanced concerning the mechanism of this syndrome since the first description of Charcot¹⁾ in 1876, recent investigations have paid particular attention to the hypotheses that cough syncope can be attributed to transmission of markedly elevated intrathoracic pressure to the cerebrospinal fluid²⁾ or to reduction in venous return to the heart,³⁾ resulting in cerebral anoxia and syncope. This report describes a patient who presented repeated episodes of loss of consciousness after coughing. Investigations revealed that the mechanism for this patient's syncope is probably a hypersensitive bronchopulmonary reflex which induces a complete A-V conduction block of the heart. Permanent right-

From the First Department of Internal Medicine, Okayama University Medical School, Okayama.

Address for reprint: Daiji Saito, M.D., First Department of Internal Medicine, Okayama University Medical School, 2-5-1 Shikata-cho, Okayama 700, Japan.

Received for publication November 21, 1981.

Manuscript revised January 10, 1982.

ventricular pacing completely relieved the patient's symptoms.

CASE REPORT

A 77-year-old housewife was admitted to our department of Okayama University Hospital for the evaluation of frequent episodes of clouding of consciousness associated with coughing. Her spells of lightheadedness occurred whenever she coughed, even after moderately vigorous coughs, and persisted during paroxysms of coughing. In one instance, when she caught a cold and the coughing continued for about 10 sec, the patient fell to the floor and exhibited clonic spasms. The onset of the spells was not related to body or head position, and she was asymptomatic between the episodes. She first noted similar spells about 5 years ago, although, until December 1979, they had been less frequent, milder, and shorter than the more recent spells. She neither smokes nor consumes alcohol. Her father died at age 45 of cerebral bleeding, one sister died in her 40s from peritoneal disease, and another in her 40s from mammary cancer.

Upon physical examination the patient showed no distress. Her blood pressure was 118/70 mmHg and her pulse was 70–80 beats/min with absolute arrhythmia. Her jugular venous pulsations and her thyroid gland were normal. She had no carotid bruits. Her left ventricular apical impulse was in

Fig. 1. A 12-lead electrocardiogram on admission. Mild ischemic changes in the ST-segment were observed in leads II, III, aVF, and in the left precordial leads. The rSr' pattern in lead V₁ was also noted.

the fifth intercostal space and 1.5 cm left of the left mid-clavicular line. The heart sounds were normal; no cardiac murmurs or other abnormal sounds were present despite absolute arrhythmia. Her lungs and abdomen were normal. The lower extremities showed superficial varicosities and no peripheral edema. The neurological findings were within normal limits.

Complete blood counts, urinalysis, serum electrolytes, urea nitrogen, uric acid, fasting blood sugar, serum enzymes, and erythrocyte sedimentation rate were all within normal limits. The serum cholesterol was 184 mg/100 ml, HDL-cholesterol 45 mg/100 ml, and the triglyceride 87 mg/100 ml. The electroencephalogram and a CT-scan of the brain revealed no abnormalities. Thallium myocardial imaging was negative and a chest X-ray was normal. A 12-lead ECG on admission revealed atrial fibrillation with rSr' pattern in lead V₁. An ischemic ST-segment depression of less than 1.0 mm was observed in leads II, III, aV_F, and in the left anterior precordial leads (Fig. 1). An exercise stress test achieved a heart rate of 136 beats/min and showed no significant ST-T changes. Several days after admission, coughing was induced by massaging the trachea at a mid-cervical level, while recording the lead II ECG continuously. Coughing induced a marked prolongation of R-R intervals without significant changes in atrial activation (f wave). Fol-

Fig. 2. Lead II ECGs during coughing in the control period (upper two rows), and after subcutaneous (third row) and intravenous (bottom row) injection of atropine.

Fig. 3. Lead II ECGs during stimulation to reproduce A-V block.

lowing three consecutive coughs, there was a maximum ventricular pause of 7,060 msec (Fig. 3) which induced presyncopal symptoms. Immediately after the cessation of coughing, the ventricular responses returned to the pre-coughing level. However, neither the Aschner test, Czermak-Hering tests (carotid sinus massage), Valsalva maneuver with the oral pressure 40 mmHg nor mechanical stimulation of pharynx could reproduce an A-V block of the heart (Fig. 2). Stimulating systemic baroreceptors with intravenous infusion of methoxamine, in a dose that elevated arterial blood pressure from control levels of 130/78 mmHg to 176/100 mmHg, exhibited no significant effects on the electrocardiographic R-R intervals. A subcutaneous injection of 0.25 mg of atropine sulfate increased the heart rate to 100 beats/min on the average. The inhibition of ventricular responses caused by coughing was partly relieved by the dose of atropine. Ten min later, an additional dose of atropine (0.25 mg) was infused intravenously, resulting in a complete cessation of the cough-induced suppression of A-V conduction (Fig. 3). Two weeks after admission, a His-electrogram was recorded to assess the site of the A-V block. While the H-V interval at rest was within a normal range (40 msec), we unfortunately could not induce coughing during the cardiac catheterization. The right ventricular pressure was 33/4 mmHg and the pulmonary capillary wedge pressure 9 mmHg.

One month later a permanent right ventricular pacemaker was set at a rate of 70 beats/min in the demand mode, and, thereafter, the patient remained asymptomatic.

DISCUSSION

The mechanism of the syncopal episode after coughing has been only partly explained. McIntosh et al³⁾ measured intrathoracic, intracranial and intravascular pressures during coughing and suggested that a "squeezing" of blood from the intracranial vasculature, in combination with a decrease in cardiac output, may have induced cerebral anoxia that resulted in syncope. On the other hand, effects of a Valsalva maneuver during coughing was emphasized as a cause of syncopal episodes by Pedersen et al.²⁾ An increase in the intrathoracic pressure may completely arrest the circulation of caval veins at the opening of the thorax, such that the venous blood flow to the heart ceases. Kerr and Eich⁴⁾ proposed that another mechanism may explain the underlying pathophysiology leading to the syncopal episode. In a study of patients who had loss of consciousness following voluntary coughing, they postulated that severe coughing resulted in a cerebral concussion, with a sudden depolarization of cerebral cells.

The actual mechanism of the syncope in our patient does not meet any of explanations described above. In our patient, coughing was always associated with an atrio-ventricular conduction block (A-V block), leading to presyncopal fits. Atropine eliminated the A-V block and syncopal episodes during coughing with accompanied increase in the basal heart rate. Moreover, right ventricular pacing completely abolished the symptoms even during paroxysms of severe coughing. In his first description, Charcot¹⁾ proposed that the syncope resulted from a cardio-inhibitory response to stimulation of the vagal nerve. However, Charcot retracted this hypothesis afterwards. Thus, there are no reports attributing a syncope during coughing to an A-V block. However, we believe that this report shows that an A-V block during paroxysms of coughing is at least one causal factor for the syncope, although it is unlikely that an A-V block is the sole mechanism for cough syncope. Syncope following coughing may be induced by several factors and, in general, an increase in intracranial pressure or a decrease in the venous return to the heart, due to a marked rise in intrathoracic pressure during coughing may be major factors to cause cough syncope.

Previous studies have demonstrated a rich cholinergic innervation of the atrio-ventricular node.^{5),6)} Manicia et al⁷⁾ demonstrated that even physiological ranges of baroreceptor activation could have a marked influence on the atrio-ventricular node. The pre-His conduction time (A-H time) was increased markedly by stimulation of baroreceptors during atrial pacing. Only coughing produced an A-V block in our patient; the Valsalva maneuver, Czermak-Hering test, pharyngeal stimulation, and stimulation of systemic

baroreceptors with methoxamine-induced hypertension failed to produce an A-V block. Although the findings do not explain why an A-V block was induced only by coughing, our data suggest that hypersensitivity of broncho-pulmonary receptors is responsible for the atrioventricular conduction disturbance. Carotid sinus receptors, pharyngeal vagal receptors, systemic baroreceptors and efferent vagal nerves did not play a direct role in the syncopal episode in our patient. The Valsalva maneuver also stimulates the broncho-pulmonary receptors, but the pressure in the oral cavity during the Valsalva maneuver is approximately 40 mmHg, while alveolar pressure during a cough exceeds 100 mmHg.⁸⁾ Therefore, the Valsalva maneuver may not be able to elevate pulmonary alveolar pressure enough to activate broncho-pulmonary receptors and to cause a reflectional A-V conduction disturbance. It was demonstrated in awake dogs that both a baroreflex, and a broncho-pulmonary reflex (Hering-Breuer reflex) could cause A-V block when the sinus node was unable to respond with its usual slowing.⁹⁾ Since the patient in this report here had atrial fibrillation, the sinus node was unable to slow the atrial activation in response to vagal stimulation. This may explain why the vagal stimulation during coughing resulted in the suppression in A-V conduction. Thus, it is most likely that hypersensitivity of broncho-pulmonary receptors to coughing caused excessive stimulation of the vagal nerve, resulting in A-V block and fainting in our patient.

REFERENCES

1. Charcot JM: Séance du 19 November 1876, Comptes rendus des seances et memoires de la société de biologie, 336
2. Pedersen A, Sandoe E, Hvidberg E, Schwartz M: Studies on the mechanism of tussive syncope. *Acta Med Scand* **179**: 653, 1966
3. McIntosh HD, Estes EH, Warren JV: The mechanism of cough syncope. *Am Heart J* **52**: 70, 1956
4. Kerr A, Eich RH: Cerebral concussion as a cause of cough syncope. *Arch Intern Med* **108**: 248, 1961
5. Kent KM, Epstein SE, Cooper T, Jacobowitz DM: Cholinergic innervation of the canine and human ventricular conduction system: anatomic and electrophysiologic correlations. *Circulation* **50**: 948, 1974
6. Il'Ina-Kakueva EI: Morphology and pathopharmacology: a histochemical study of the cholinesterase activity of the nerve cells and conducting system of the heart. *Bull Exp Biol Med* **46**: 1270, 1958
7. Mancia G, Bonazzi O, Pozzoni L, Ferrari A, Gardumi M, Greforini L, Perondi R: Baroreceptor control of atrioventricular conduction in man. *Circulat Res* **44**: 752, 1979
8. McCann WS, Bruce RA, Lovejoy FW Jr, Yu PNG, Pearson R, Emerson EB, Engel G, Kelly JJ: Tussive syncope, observations on the disease formerly called laryngeal epilepsy. *Arch Intern Med* **84**: 845, 1949
9. James TN, Urthaler F, Hageman GR: Reflex heart block; baroreflex, chemoreflex and bronchopulmonary reflex causes. *Am J Cardiol* **45**: 1182, 1980