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drink and developed a violent coughing spell. The ECG monitor strip then showed a variable second- and third-degree A-V block with a ventricular rate of ±35/min. (Fig. 2). Because of the potential danger of this arrhythmia, atropine was given subcutaneously, with prompt disappearance of the inspiration-induced heart block. After 12 days of uninterrupted recovery the patient was discharged.

The clinical impression was that the

The clinical impression was that the patient had coronary insufficiency. The radiological finding of a pulmonary infiltrate with a normal temperature, normal leukocyte count and sudden dyspnea in the absence of congestive heart failure would suggest the diagnosis of pulmonary embolism or pneumonia.

Discussion

There have been some reports in the English literature of transient seconddegree heart block associated with respiratory movement.4-7 It appears that the stretch receptor reflexes are concerned in the pathophysiology of this abnormality.1,8 Schwartz and Schwartz⁶ described a patient who developed second-degree atrioventricular block during deep inspiration resulting in syncopal attacks. Unlike our patient he had received digitalis, and the authors suggested that the vagotonic effect of this drug could be potentiated by the pulmonary stretch receptor reflex. This explanation has not been fully accepted, and there is a second hypothesis9 based on the increased venous return during deep inspiration and consequent distension of the right atrium which might increase vagal tone. Furthermore, intermittent complete A-V block triggered by swallowing and attributed to vagotonia has been described in a patient with myocardial infarction.12 In the patient whose case we describe coronary insufficiency most likely existed, but the role of deep inspiration and the Valsalva maneuver in the production of heart block cannot be disregarded. Stimulation of the pulmonary stretch receptor reflex may explain this event and, moreover, it can be argued that the pathological change in the left lower lobe, with decreased compliance, would potentiate such a stretch receptor reflex. This view is supported somewhat by the fact that it was not possible to elicit the Wenckebach phenomenon after the clinical improvement and radiological resolution of the lesion even though atropine had already been discontinued.

Several factors have been implicated in the pathogenesis of cough syncope, 10 namely, (1) decreased venous

Inspiration- and cough- induced atrioventricular block

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It is well known that the autonomic nervous system plays an important role in the control of conduction through the atrioventricular (A-V) node. Stretch receptor response in blood vessels, heart and lungs is mediated by the vagus nerve,1 and the cardiac effects of vagal stimulation² include depression of rhythmicity of both the sinus and A-V nodes, prolongation of the refractory period of the junctional tissues and depression of sino-atrial and atrioventricular conductivity. It is also known that the right vagus is distributed mainly to the sino-atrial node and junctional tissues, while the left vagus ramifies in the atrioventricular conducting tissue and upper portion of the bundle of His.3

In the following presentation a patient is described who developed transient second-degree heart block on deep inspiration, Valsalva maneuver and coughing. The role of the vagus nerve and respiratory movements along with the clinical importance of this unusual condition is discussed.

Case history

A 77-year-old Caucasian male complain-

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ing of retrosternal pain, shortness of breath and diaphoresis was admitted to the Coronary Monitoring Unit a few hours after the sudden onset of these symptoms. His history revealed that he suffered from a chronic peptic ulcer and long-standing diabetes mellitus; a transurethral prostatectomy with bilateral orchidectomy had been performed for carcinoma of the prostate one year prior to this admission and he was being treated with stilbestrol. He had never received digitalis or any other drug known to depress atrioventricular conduction.

Pertinent physical findings were a blood pressure of 170/90, normal temperature, râles in the left infrascapular region and a soft systolic ejection murmur at the apex and base of the heart. A chest radiograph showed an infiltrate 3 cm. in diameter at the left base which almost cleared within seven days. Repeated creatinophosphokinase, lactic dehydrogenase, serum glutamic oxalacetic and serum glutamic pyruvic transaminase estimations over the first three days were normal.

An ECG taken on admission was normal and subsequent tracings showed slight RS-T segment elevation in leads 3 and aVF. A few hours after admission a continuous ECG tracing demonstrated second-degree A-V block with Wenckebach periods during deep inspiration (Fig. 1). It is also of interest that the P-R interval progressively shortened during the subsequent expiration and returned to normal. The A-V block was reproducible with the Valsalva maneuver.

During the second hospital day the patient aspirated milk while trying to return, (2) dilatation of peripheral arterioles, (3) compression of intracranial capillaries and veins due to great increase in CSF pressure, (4) increase in cerebral vascular resistance induced by hypocapnia of coughing and (5) "concussive" effect caused by the sudden rise of intracranial pressure transmitted from thorax and abdomen via CSF.11 Our case suggests yet another possibility, that coughing may cause a syncopal attack by a sudden decrease in cardiac output resulting from heart block with a slow ventricular rate. Besides its pathophysiological interest, this phenomenon has clinical significance in that it may respond to vagolytic agents.

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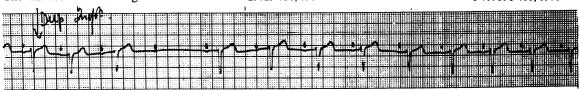


FIG. 1—Electrocardiogram showing a Wenckebach phenomenon during deep inspiration and gradual shortening of P-R interval during subsequent

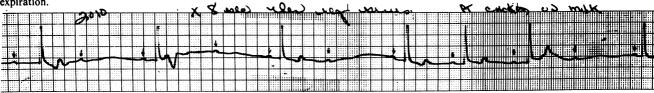


FIG. 2—Electrocardiogram taken during a coughing spell and showing second-degree heart block. The first complex could represent a third-degree A-V block.

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