Carotid Blood Velocity During Cough
Studies in Man

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SUMMARY Utilizing a Doppler ultrasonic flowmeter catheter, right carotid artery blood velocity was measured during 91 coughing episodes in 16 patients. Such coughing reduced carotid blood velocity by 40 ± 22% (control = 34 ± 8 cm per second, cough = 20 ± 9 cm per second, p < 0.001). There was an insignificant low degree of correlation between the level of simultaneously recorded mean right pressure and the percent decline of peak carotid blood velocity, suggesting that impaired venous return was not the only factor responsible for the observed changes. It is concluded that (1) coughing diminishes phasic carotid blood velocity and (2) reduced cerebral perfusion may play a role in the pathogenesis of cough syncope.

Methods
Sixteen patients comprised the study group; there were 12 men and four women whose ages ranged from 23 to 58 with a mean of 44 years. Eight subjects had normal cardiovascular function and seven had coronary artery disease. One patient had mitral stenosis. No subject had symptoms suggestive of cough syncope. All diagnoses were established on the basis of right and left heart catheterization, indicator dilution curves and selective coronary cineangiography. Normal subjects were studied because of the presence of chest pain or systolic murmurs originally thought to repre-
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Discussion

Lightheadedness or syncope attendant to coughing has been ascribed to: (1) reduction of cerebral perfusion consequent to reduced venous return and diminished stroke output (Valsalva simulation), (2) a "squeezing" of cerebral venous and arterial blood to extracavitary sites, and (3) cerebral concussion. All three mechanisms appear related to the marked elevations of intrathoracic pressure generated by tussive episodes and specifically the transmission of this rise in pressure to the intra-abdominal and intracranial spaces.

In patients with cough syncope, it has been observed that fainting episodes occur with marked rapidity, and occasionally within five seconds after onset of the tussive effort. Furthermore, the unconsciousness may precede a decline in systemic blood pressure, thereby suggesting that arterial hypotension, reflex or mechanical, cannot be the sole cause of this syndrome. Our results indicate that coughing usually produces an abrupt reduction of the velocity of blood flowing within the carotid artery of conscious supine man. Insofar as the magnitude of cough is related to a rise in mean right atrial pressure, it is apparent that this diminution of phasic instantaneous carotid blood velocity is not due to a decline in peak aortic blood velocity. Such reduced carotid arterial blood velocity arising as a result of tussive effort may be based on an impedance to blood flow induced by the sudden rise in intracranial pressure.

Since the Doppler ultrasonic catheter utilized in this study measured blood velocity and not volumetric flow, it is possible that the hemodynamic alterations associated with coughing produced either increased cerebral arterial resistance or a distention of the carotid arteries, effectively increasing lumen area and reducing the velocity of flow. The development of a catheter which can simultaneously measure lumen area and instantaneous blood flow is feasible, but has not
yet been accomplished. Despite this drawback, it can be surmised that the velocity of oxygen-bearing red blood cells within the carotid artery and effective cerebral perfusion are diminished during coughing.

The data described herein were obtained from patients not subject to cough syncope, and confirmation of these findings in such affected individuals requires future investigation. Furthermore, cough syncope may represent a clinical state based on varied etiologies such as herniated cerebellar tonsils or reflex cardiac arrest. In conclusion, the syndrome of cough syncope, usually encountered in patients with respiratory disorders, may be related to an instantaneous reduction of carotid artery blood velocity.

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