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 decreased 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 atrial 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.

COUGH SYNCOPE is a well-recognized clinical entity which has intrigued clinicians for approximately 100 years. Previous studies have documented the influence of tussive episodes on intrathoracic, cardiovascular or cerebrospinal fluid pressures and changes in fluoroscopic images of the right atrium, vena cava and pulmonary vein. In addition, EEG monitoring has been performed during the act of coughing. We report here the influence of cough on phasic carotid arterial blood velocity.

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 represent heart disease. Patients were studied in a supine position.

Phasic instantaneous right carotid artery blood velocity was measured with a Doppler ultrasonic flowmeter catheter as previously described. Under local anesthesia (1% lidocaine), the crystal-tipped catheter was introduced into a brachial arterial incision and advanced to the origin of the right carotid artery. In addition, a standard fluid-filled catheter connected to a Statham P23Db strain gauge was advanced from a medial antecubital vein to the right heart for the purpose of obtaining right atrial pressures. All subjects were instructed to cough in varying degrees while phasic carotid artery blood velocity, mean right atrial pressure and lead II of the ECG were recorded. Catheter tip position and stability were monitored by means of constant fluoroscopic image intensification. In six subjects, phasic aortic blood velocity also was recorded during cough.

The Doppler catheter utilized in this study measured the velocity of blood cells flowing past its tip and reflected volumetric flow only as a function of stable arterial lumen diameter.

Results

Ninety-one coughing episodes in the study group reduced peak carotid artery blood velocity by an average of 40 ± 22%. Mean (±1 SD) values for carotid blood velocity were 34 ± 8 cm per second in the control period and 20 ± 9 cm per second during the tussive efforts (fig. 1). There is a significant (p < 0.001) difference between the mean values. The cough-induced decline of peak carotid blood velocity was immediate in onset and uniformly occurred within one second of each tussive effort (fig. 2). When the diminution of peak carotid blood velocity was plotted as a function of the rise of mean right atrial pressure, there was a low degree of correlation which was not statistically significant. The greatest reduction of phasic carotid blood velocity, however, was generally observed at the peak rise of mean right atrial pressure in the individual case.

In the six subjects studied, there were minimal or no significant tussive-related alterations of peak aortic blood velocity and during traumatic edema. Physiol Bohemoslov 10: 8-14, 1961

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velocity, while cough of a magnitude to effect identical or similar augmentation of mean right atrial pressure resulted in precipitous reductions of carotid blood velocity (fig. 3).

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 (2) a “squeezing” of cerebral venous and arterial blood to extracavitary sites,1 and (3) cerebral concussion.8 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.8

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.1,3 Furthermore, the unconsciousness may precede a decline in systemic blood pressure,4 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,4 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.1

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 velocity is feasible, but has not

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**FiguRe 1.** Distribution of values for peak right carotid blood velocity in the control period and during cough for 91 tussive episodes in the study group.

**FIGURE 2.** Lead II (LII) of the ECG, mean right atrial (RA) pressure, and right carotid blood flow velocity in a normal 54-year-old woman. Two tussive episodes consisting of six voluntary coughs resulted in a rise in mean right atrial pressure and immediate decline of carotid blood velocity.
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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References

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