

Coughs cause systemic blood flow

JEFFREY M CARY, RICHARD KRUGMEIER, BILL NEWMAN, BRIAN ROSS, JOHN BUTLER

From the Departments of Medicine and Surgery, University of Washington, Seattle, USA

ABSTRACT Although it is known that rhythmic coughing can preserve consciousness during ventricular fibrillation, the arterial pressure transients which result have not yet been shown to reflect forward blood flow in man. The effectiveness of cough in causing forward flow in eight normal volunteers with bradycardia was studied. They coughed between cardiac cycles, using an audio and visual display of the electrocardiogram. The force of the cough was varied and measured with an oesophageal balloon. Blood flow was recorded with a Doppler velocity probe over the radial artery and a finger or ear photoplethysmograph. Motion artefact on the Doppler record due to coughing was excluded by transiently obstructing the brachial artery. We compared the areas under 5-10 consecutive Doppler and photoplethysmograph pulse flow tracings due to the cough and heart beat with those due to the immediately preceding heart beat alone. They were significantly increased. This augmentation was greater in those flow pulses accompanied by a more vigorous cough. It is concluded that coughing is associated with an effort dependent forward flow pulse in the arterial circulation.

Kowenhoven *et al*¹ were the first to report that blood could be made to flow out of the closed chest by externally applied pressure to the chest wall. They proposed that this blood flow was brought about by squeezing the heart between the sternum and the spine. This "external cardiac massage" was accepted as the basis of cardiopulmonary resuscitation by the American Heart Association in 1974² and the mortality rate of sudden cardiac arrest appears to have been dramatically reduced at the same time that rapidly administered cardiopulmonary resuscitation by trained bystanders became available. There are even indications that the early efforts at external chest compression by the lay population may be more successful than the cardiopulmonary resuscitation administered later by trained personnel.³ The implication of this observation is that rapid re-establishment of vascular perfusion is essential for successful resuscitation. It also suggests that it may not be the specific method of chest compression but rather the development of positive intrathoracic pressure that produces the blood flow. Criley *et al* have shown that patients developing ventricular

fibrillation during cardiac catheterisation may carry out their own positive intrathoracic pressure resuscitation and maintain consciousness by coughing in a forceful, rhythmic fashion.⁴ In his study, patients developed peak arterial systolic pressures of about 140 mm Hg during coughing. This and the prolongation of consciousness suggested that flow to the brain was preserved. It has been known, however, since the studies of Weale and Rothwell-Jackson⁵ that there is an equal intrathoracic increase in venous and arterial pressures during chest compression. Thus the presence of an arterial pulse during coughing does not necessarily reflect a forward blood flow, although the preservation of consciousness suggests it. We designed a study to evaluate the effectiveness of coughing in man using non-invasive methods for assessing blood flow at different anatomical sites and correlating the intrathoracic pressure with the measured blood flow.

Methods

Eight men aged 21-45 years were selected because they had considerable bradycardia secondary to athletic training. The subject observed the electrocardiogram displayed on an oscilloscope and listened to a sound triggered by the QRS complex. An oesophageal balloon was passed to the distal oesophagus.⁶ Blood flow was measured by two

Address for reprint requests: Professor John Butler, Division of Respiratory Diseases RM-12, University of Washington, Seattle, Washington 98195, USA.

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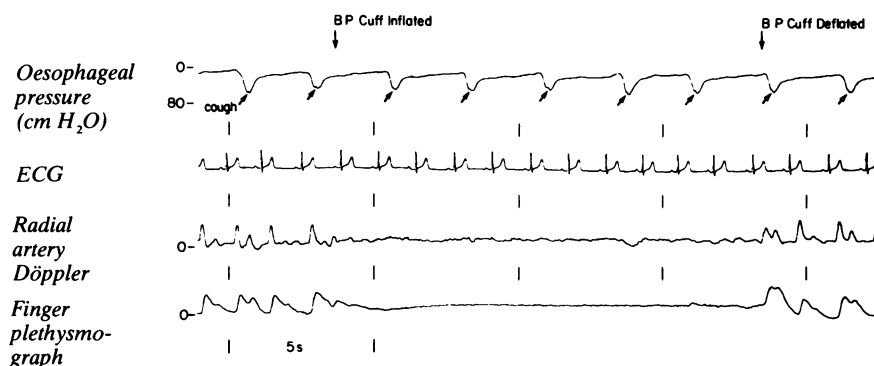


Fig 1 Effect of brachial artery occlusion on pulse signal. To exclude motion artefacts induced by coughing, a blood pressure cuff around the upper arm was inflated above the arterial pressure at the first vertical arrow and deflated at the second arrow. The flow pulses disappeared during the period of occlusion.

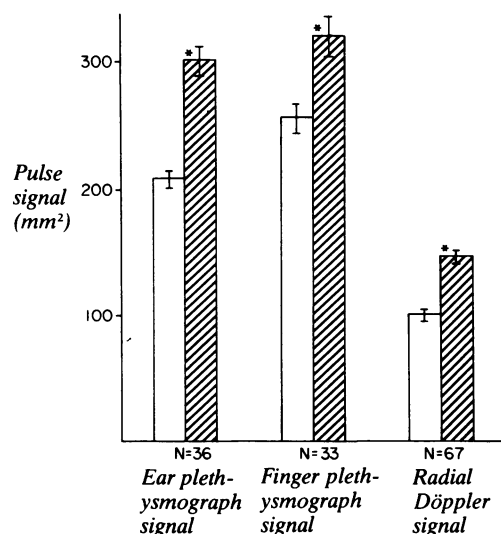


Fig 2 Flow due to cardiac pulse alone contrasted with that due to the succeeding pulse augmented by cough (means and standard errors). A radial artery Doppler display was always used; in about half the studies the photoplethysmograph was used on the ear and in the rest on a finger. □ cardiac pulse; ▨ cardiac pulse plus cough. * $p < 0.001$.

devices: a Doppler velocity probe over the radial artery and a finger or ear photoplethysmograph.⁷ The Doppler velocity meter transmits a continuous beam of ultrasound through the skin, subcutaneous tissue, and vessel wall into the passing blood stream. The ultrasonic signal reflected from the moving particles in the blood is shifted in frequency in proportion to the flow velocity. The instrument produces a signal proportional to instantaneous blood flow. Volume flows at the same position may be compared if it is assumed that velocity profiles and vessel

calibre do not change. In the photoplethysmograph light is transmitted through a capillary bed and detected by a photosensor. Changes in the volume of the capillary bed between the light source and the photosensor modify absorption of the light. The rate of change of volume of the capillary bed between the light source and the photosensor reflects arterial inflow. These methods allow comparison of pulse volumes with the normal (control) pulse but were not calibrated in flow units.

The subjects lay in the supine or, to minimise possible oesophageal pressure artefacts, the lateral position. They were taught to cough by executing a brief Valsalva manoeuvre against a closed glottis after a tidal inspiration and practised increasing their effort using an oscilloscopic display of the oesophageal pressure. Then they coughed immediately after QRS complexes of random cardiac cycles, with normal effort initially and then with increasing or decreasing vigour in a random fashion.

To assess motion artefact in the Doppler and plethysmographic recordings during the coughing manoeuvres the brachial artery was occluded by inflating a blood pressure cuff around the upper arm to above systolic pressure. The subjects continued to cough and the radial artery Doppler and finger plethysmographic signals were followed.

The effect of five to 10 consecutive coughs on the Doppler and plethysmographic signals was analysed by comparing the area under the pulse signal generated by the sum of the cough and the cardiac action with the area under the immediately preceding pulse signal generated by the cardiac action alone. Timing and technical artefact prevented some measurements, but about the same number of coughs were analysed for each subject.

Coughs during systole were excluded. The flow signal areas obtained by planimetry reflected pulse volumes and were compared with a paired t test.

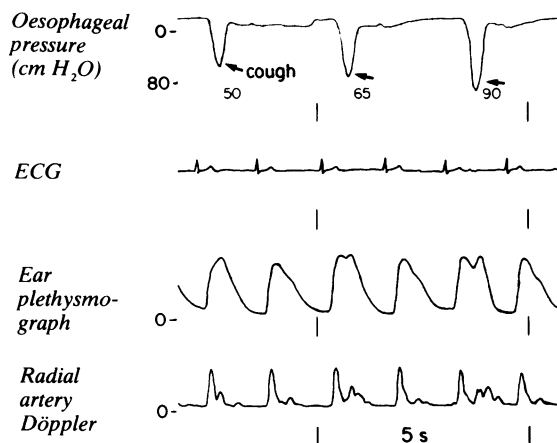


Fig 3 Effect of cough effort on pulse signal. Coughs of increased vigour with oesophageal pressures of 50, 65, and 90 cm H_2O caused progressively greater flows.

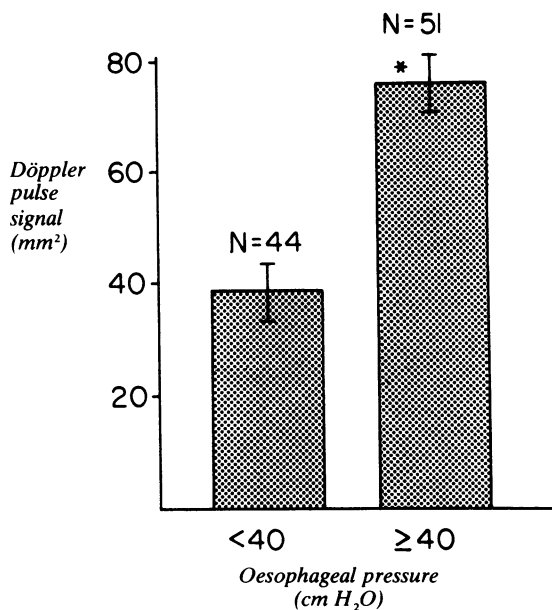


Fig 4 Blood flow (means and standard errors) assessed by Doppler pulse signal for coughs producing pressures greater than and less than 40 cm H_2O . * $p < 0.001$.

Results

There was little motion artefact in the radial artery Doppler or finger plethysmograph tracing when

blood flow to the arm was prevented by inflation of the blood pressure cuff (fig 1). When the cuff was released the expected flow signals from the cardiac cycles and the cardiac cycles plus cough returned,

The ear photoplethysmograph, the finger photoplethysmograph, and the radial artery Doppler data showed that the pulse volumes generated by cough plus cardiac action were significantly greater than those due to cardiac action alone (fig 2). Any position artefact was minimised by the comparison with the pulse due to cardiac action alone which immediately preceded that due to cardiac action plus cough. We did not find an effect of the diastolic timing of the cough on its flow augmentation or on the succeeding pulse.

The augmentation of blood flow was related to the effort and positive pressure developed during the cough (fig 3). Since it proved impossible to standardise the oesophageal pressures reached during coughing, the pulse volumes generated by coughs with oesophageal pressures greater than 40 cm of water were compared with those when pressures were less than 40 cm of water. There were significantly greater pulse volumes produced by the more vigorous coughing (fig 4).

Discussion

The time and vigour of coughing were analysed on the basis of oesophageal pressure. When this is measured in the supine position, changes during breathing may not reflect intrathoracic pressure changes. These errors seem to be related to changes of lung volume, which may displace the organs and alter the weight of the mediastinum on the oesophagus. In our studies lung volume during the cough (functional residual capacity plus tidal volume) was not altered, so such artefacts are unlikely, particularly since the results of studies of the relationship between pressure and pulse signal in subjects in the lateral decubitus position were not different from those in the supine position. Motion due to cough did not affect the Doppler arterial velocity trace. Although sustained changes in intrathoracic pressure are reflected in the veins, venous backflow transients which might have affected the photoplethysmograph are probably prevented by venous valves.⁸

Our normal subjects with sinus bradycardia coughed during diastole when the aortic valve was closed. This is the time when cardiac assist devices such as the aortic balloon pump are used. It must be emphasised that their cardiovascular state was very different from that of patients with ventricular fibrillation. Yet our study indicates that the systemic

pressure pulse, known to be produced by coughing in these patients and associated with the preservation of consciousness, is associated with a forward flow pulse in the periphery. As would be expected if intrathoracic pressure was the cause of the forward pulse, the flow pulses were greater when the oesophageal pressures were higher. This confirms in man the direct relationship between intrathoracic pressure and flow reported by Rosborough *et al* in anaesthetised dogs with simulated coughs produced with the aid of an "external glottis."⁹

The mechanism of this forward flow is thought to be the difference between the arterial and venous pressures outside the chest during the positive intrathoracic pressure phase. This pressure, unable to squeeze air out of the chest because of the closed glottis, squeezes out blood from the lungs, left heart, and aorta. Venous backflow regurgitation appears to be limited both by venous valves and by collapse of the veins at the thoracic outlet caused by the positive intrathoracic pressure transients.⁸ Consequently, a driving pressure exists between the arteries and veins outside the chest during the cough and forward flow occurs.

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