Relationship between expired lung volume, peak flow rate and peak velocity time during a voluntary cough manoeuvre

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SUMMARY

Tussometry is a new non-invasive method of assessing laryngeal function by analysing the airflow waveform produced by a voluntary cough. Ten healthy male volunteers performed five (n = 6) or six (n = 4) voluntary cough manoeuvres at varying lung volumes from total lung capacity to functional residual capacity. During each manoeuvre, airflow (litre min⁻¹) was plotted against time (ms) to record the peak flow generated by the cough (CPFR) and the time taken to achieve this (PVT). In addition, expired volume (CEV) was measured during each manoeuvre. Highly significant (P < 0.001) correlations existed between CPFR and PVT (r = 0.81), CPFR and CEV (r = 0.78) and PVT and CEV (r = 0.71). We conclude that PVT may vary with CPFR which in turn bears a direct relationship to expired lung volume during a cough manoeuvre. These relationships among different variables should be considered when interpreting the results of tussometry. (Br. J. Anaesth. 1994; 72: 298–301)

KEY WORDS

Cough. Measurement techniques: tussometry.

Cough is a complex centrally organized event that may be initiated reflexly or voluntarily. Normally it consists of inspiratory, compressive and expiratory phases [1]. The inspiratory phase, when present, is associated with glottic opening and the inhalation of variable amounts of air. The compressive phase consists of closure of the glottis and contraction of the expiratory muscles resulting in raised intrathoracic pressure. In the expiratory phase the glottis opens suddenly, causing explosive release of the trapped intrathoracic air. The term "laryngeal competence" is used often to imply a well coordinated upper airway and an adequate cough response [2]. It may be affected by sedation, anaesthesia and tracheal instrumentation [3–6].

Tussometry, described recently by one of the authors [7, 8], allows analysis of the airflow waveform produced by a voluntary cough (fig. 1). A normal wave is displayed as cough flow rate (litre min⁻¹) *vs* time (ms) and shows a rapid increase to peak—the cough peak flow rate (CPFR). It is suggested that the time taken to reach CPFR, the peak velocity time (PVT), is determined by laryngeal

opening at the onset of cough [9]; it correlates with vocal cord function in patients with cord palsy [7] and is potentially a useful indicator of laryngeal function in postoperative patients [10]. However, it is not clear if PVT depends entirely on vocal cord function or if other physiological variables, such as expired flow rate, volume, or both, influence its value. This study was conducted to investigate the relationships between PVT, CPFR and cough expired volume (CEV).

SUBJECTS AND METHODS

We studied 10 healthy male volunteers. The subjects were non-smokers and were not obese (obesity defined as a body mass index of > 29). All were staff members of the University Department of Anaesthesia and were familiar with the equipment. Informed consent was obtained from all subjects and they were studied in the morning, at least 2 h after consuming a light breakfast. Throughout the study the subjects were distracted by music of their choice.

Each subject was seated and the procedure explained. Two practice attempts were allowed. The subject was asked to breathe normally into an appropriately sized face mask connected to the measurement equipment. This consisted of a pneumotachograph to measure airflow ("tussometer") connected in series to a Morgan transfer test machine (interfaced with Elonex PC using Morgan software) to measure air volume (fig. 2). The Morgan transfer test machine, originally designed to measure lung volumes using the helium dilution technique and dry spirometry, allows a continuous breath-to-
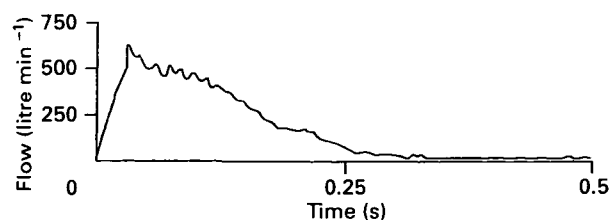

FIG. 1. A typical air flow–time wave during a voluntary cough.

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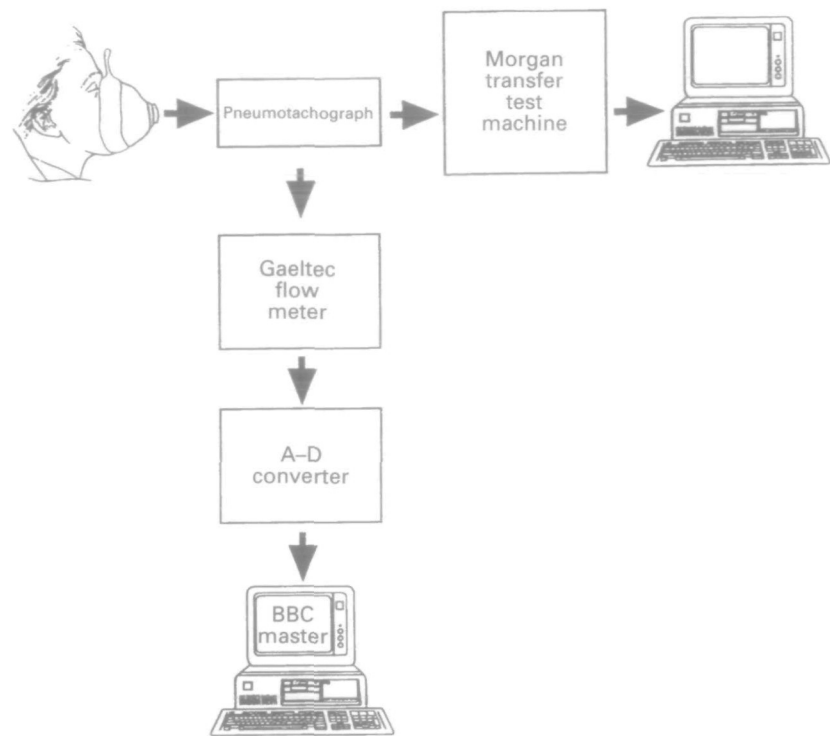
FIG. 2. Morgan transfer test machine connected in series to a tussometer to measure cough expired volume.

breath display of inspired and expired lung volume. The subject held the mask firmly over the nose and mouth, carefully preventing any air leaks. The respiratory excursions were displayed continuously on the computer screen of the Morgan transfer test machine. The subject was asked to inhale to maximum capacity (TLC) and then to cough as forcefully as possible, expelling as much air as possible. Further cough manoeuvres were performed in a similar way but in such a manner that every subsequent attempt was at an inspiratory lung volume of 500–1000 ml less than the preceding attempt; the final attempt was near the functional residual capacity (FRC). The varying inspiratory volumes were obtained with the subjects inspiring slowly until the required volume was reached, as indicated on the oscilloscope screen.

The pneumotachograph system used in this study consists of a fine mesh through which the subject coughed. The pressure differential across this was used to derive air flow with a Gaeltec flowmeter. Using an analogue-to-digital converter and a BBC master computer, this is displayed in graphical form (fig. 1) as a flow–time wave. The output of this system was calibrated against that of a standard flowmeter (series tube size 47 k, Rotameter Manufacturing Co. Ltd, Croydon, England) for air flows up to 1100 litre min^{-1}. A strong positive relationship was seen between pneumotachograph output and flowmeter readings ($r^2 = 98.5\%$, $P = 0.0001$).

For each cough manoeuvre, CPFR, PVT (from the flow–time wave) and CEV (measured by Morgan transfer test machine) were recorded. Relationships between CPFR and PVT, CEV and CPFR, and CEV and PVT were determined by calculating the coefficients of correlation and subsequent analysis for significance using the SPSS statistical package.

RESULTS

The subjects were aged 28–36 yr, height 1.67–1.82 m (mean 1.75 m) and weight 66–88 kg (mean 74.8 kg). Two of our volunteers had a history of exercise-induced asthma for which they took bronchodilator inhalers when indicated; they had no evidence of bronchospasm before, during or after the investigation. Otherwise, none of the subjects had a history of cardiovascular, respiratory, neurological or any other systemic disease. The subjects had no difficulty in performing the cough manoeuvres. In all, 54 cough attempts were made with six subjects performing at five different volumes and four subjects
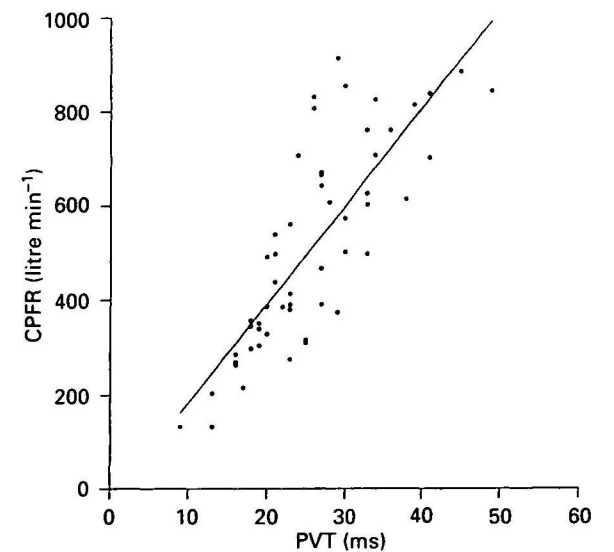

FIG. 3. Relationship between cough peak flow rate (CPFR) and peak velocity time (PVT) during 54 cough attempts made by 10 volunteers at varying lung volumes ($r = 0.81$).
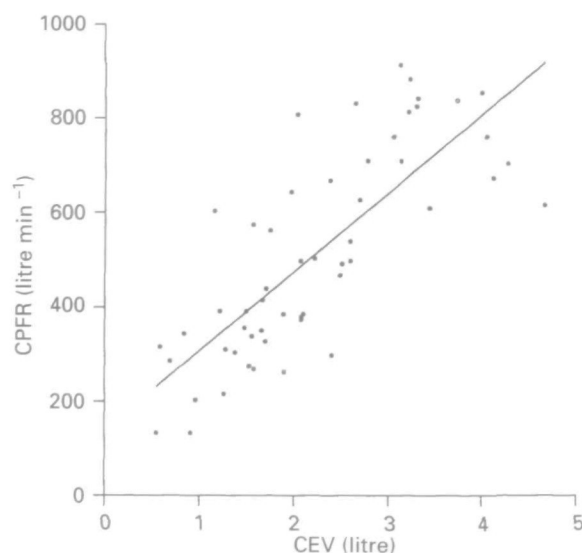
FIG. 4. Relationship between cough peak flow rate (CPFR) and cough expired volume (CEV) during 54 cough attempts made by 10 volunteers ($r = 0.78$).
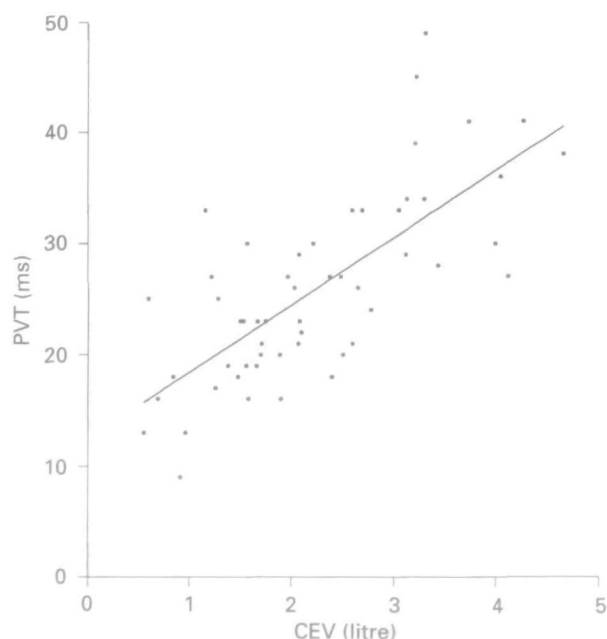

FIG. 5. Relationship between cough expired volume (CEV) and peak velocity time (PVT) during 54 cough attempts made by 10 volunteers ($r = 0.71$).

performing at six different volumes. Data from all the 54 cough manoeuvres were pooled. The relationships between CPFR, PVT and CEV are illustrated in figures 3–5. Highly significant ($P < 0.001$) correlations were found between CPFR and PVT ($r = 0.81$), CEV and CPFR ($r = 0.78$) and CEV and PVT ($r = 0.71$).

DISCUSSION

Our results indicate that PVT varied directly with CPFR, which in turn was determined by the exhaled volume during a voluntary cough manoeuvre. Knudson, Mead and Knudson [9] have shown that, during the expiratory phase of a cough, two distinct components contribute to the initial expiratory flow:

the airway and pulmonary components. They have different sites of origin and different time courses and mechanics. Because alveolar pressure exceeds that at the airway opening, air begins to flow from the pulmonary parenchyma (pulmonary component). At the same time, because pleural pressure exceeds that within the central airways (which are now open to the atmosphere), the airways are subjected to the dynamic compression and collapse abruptly (airway component). This may be seen as a transient flow "spike" at the beginning of the expiratory flow curve (fig. 1), referred to as supramaximal expiratory flow, superimposed on more sustained flow from parenchyma. Thus CPFR measured in our study may have two different components, although the contribution of each of the pulmonary and airway components to the total value is not clear.

During a forced expiratory manoeuvre (not a cough), the maximum expiratory flow–volume curve (MEFV) describes the relationship between expired volume and maximum flow (Vmax) which cannot be exceeded at that volume [11–13]. Vmax during a forced expiration is the same as the pulmonary component of the cough peak–flow wave. The MEFV curve indicates that the upper limit to expiratory flow during a forced expiration is normally set not by muscle strength or effort, but by a flow-limiting mechanism operating in the lung and intrathroacic airways [14, 15], and that maximal expiratory flow is proportional to the expired volume [16]. Our study shows that there is a direct and strong relationship between CPFR and CEV during a voluntary cough. As we are unable to differentiate between the pulmonary and airway components of CPFR in our study, we are unable to determine if the pulmonary or airway components of cough peak–flow wave, or both, bear a consistent relationship with CEV. The volume of airway undergoing dynamic compression and subsequent volume decrease during a cough manoeuvre depends on the location of the equal pressure point (EPP) [9], which in turn depends on lung volume [17]. The volume decrease is not great, but because of a very short time constant, instantaneous flows may be large. In theory, for example, a volume change of 50 ml in 10 ms results in a mean flow during that period of 300 litre min^{-1}. However, experiments by Knudson, Mead and Knudson [9] have demonstrated that the volume displaced from the airways may vary from 50–150 ml, but has no predictable relationship with the change in lung volume.

The relationship between PVT and CPFR has not been described previously. Knudson, Mead and Knudson [9] demonstrated that the time required to reach peak flow upon initiating a cough is 30–35 ms in most instances. Maximum volume accelerations were 300 litre s^{-2}. It has been suggested that cough is initiated by rapid abduction of the arytenoid cartilages, an active phenomenon involving muscle contraction [18]. Using high speed photographic techniques, von Leden and co-workers [18, 19] were able to demonstrate that a laryngeal opening time of about 25–30 ms is of sufficient magnitude to account for the time observed to reach peak flow in the

cough. The early concavity of the flow–time curve is suggestive of diminishing flow resistance offered by widening of the glottis during this initial phase. Based on these observations, previous workers [9] suggested that the time to reach peak flow on a flow–time curve during a cough is dependent on laryngeal muscle activity. This suggestion was supported by later findings in patients with vocal cord palsy [7]. The PVT in these patients was significantly greater (45–214 ms) compared with normal controls (24–51 ms); CPFR was similar in both groups. These studies imply that glottic function is the main determinant of PVT. Our results show that CEV and CPFR are other variables which need to be controlled if such an interpretation is to be made.

Another possible variable, which we have not measured in our study, is effort or muscle strength. In theory, a change in driving pressure (pleural pressure) subsequent to a change in effort can alter the slope of the initial rise of the flow–time curve, thereby changing the CPFR–PVT relationship. Arora and Gal [20] recorded smaller pleural pressures when their subjects coughed at FRC compared with coughing at TLC. In addition, flow rates during coughs initiated at FRC did not appear to be altered by curarization and exhibited distinct peak flow transients despite a marked reduction in pleural pressure. Knudson, Mead and Knudson [9] have shown that volume acceleration at the beginning of a cough is independent of volume (and, therefore, of pleural pressure [20]) at which the manoeuvre is started. It has also been shown that CPFR is achieved much earlier than peak pressure during coughing [15] and a maximum voluntary ventilation manoeuvre [9]. Based on these studies, it appears that effort is not a major determinant of the initial events in a flow–time wave after a cough, especially if the manoeuvre is started at smaller lung volumes. However, this remains to be confirmed.

The question that needs to be examined is how tussometry can be used and its results interpreted to study the effect of residual anaesthesia, sedation or instrumentation of the upper airway. One limitation is that the technique requires a reasonably cooperative patient who is able to generate a voluntary cough and thus this technique cannot be used during the earlier phases of recovery. When a voluntary cough can be performed, changes in PVT reflect changes in glottic function, provided that CPFR remains unchanged. Interpretation may not be as simple if both PVT and CPFR change. If PVT increases with a decrease in CPFR, this would favour impaired glottic function. However, if both PVT and CPFR change in the same direction, it may be difficult to make any interpretation regarding glottic function. Measurement of maximum volume acceleration, CPFR:PVT ratio or the maximum slope at the beginning of the flow–time curve, may provide

information on glottic function which would be independent of the other variables.

We conclude that PVT changes in the same direction as CPFR and that both PVT and CPFR are determined by the exhaled lung volume during a voluntary cough manoeuvre. These relationships should be taken into consideration when interpreting the results obtained by tussometry to assess laryngeal function.

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