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EFFECT OF RESPIRATORY APPARATUS ON TIMING AND DEPTH OF BREATHING IN MAN

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Abstract. The effects on breathing of (1) nose-clips, with and without mouthpieces, (2) tube breathing with incremental dead spaces up to 600 ml, and (3) a Venturi mask with a high air inflow reducing the effective dead space to zero were measured with respiratory inductance plethysmography. The effective dead spaces of the lengths of tubing were 25% smaller than their water-displacement volumes. The principal response to the increase in dead space was an increase in VT without any change in frequency or timing. The increase in $\dot{V}E$ was 16% greater than the ventilation of the effective dead space. The facemask with a high flow of air did not affect the pattern of breathing. External devices alone (nose-clips, mouthpieces) by themselves brought about a significant increase in VT ($+10\%$, $P < 0.05$) and a small drop in frequency, which in the case of a nose-clip alone was large enough to be significant (-15%, $P < 0.05$). The increase in DS with the change from nasal to oral breathing may have contributed to the accompanying increase in VT; the fall in frequency with the nose-clip alone might have been caused by stimulation of receptors in the trigeminal area.

Breathing pattern	Human	Respiratory frequency
Dead space	Plethysmography	Ventilation

The pattern of breathing may be affected by a variety of external stimuli including the method with which it is observed. Several devices have been developed for measuring minute ventilation ($\dot{V}E$) and tidal volume (VT) which leave the mouth and nose undisturbed. Using chest and abdominal magnetometers, Gilbert *et al.* (1972) found that with a mouthpiece and nose-clip only (no additional external dead space) respiratory frequency fell and tidal volume rose. On the other hand, in later studies, with inductance plethysmography (Sackner *et al.*, 1980) or a head canopy (Askanazi *et al.*, 1980) respiratory frequency was essentially unchanged ($\pm 5\%$). Tidal volume and minute ventilation rose by about 20%.

The reason for these changes is not clear. Unimpeded ventilation at rest occurs mainly through the nose in normal subjects. Cadaver studies of the water displacement

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volume of the extra-thoracic airways excluding the naso-pharynx and nose suggest that the anatomical dead space of the mouth and larynx is 50 ± 25 ml (Nunn *et al.*, 1959). With obligate mouth breathing, therefore, there will be at most (assuming nasal dead space to be small) an increase in dead space from the oral cavity of the order of 40 ml. Nevertheless, VT increased by > 100 ml with the switch from nasal to oral breathing (Sackner *et al.*, 1980; Askanazi *et al.*, 1980). An additional explanation is that the unpleasant sensation associated with nose-clips and mouthpieces, via the trigeminal nerve, is a ventilatory stimulant. Hirsch and Bishop (1982) matched the dead spaces (allowing 50 ml for the oral cavity) for subjects breathing with a mouthpiece, nose-clip and facemask. With mouthpiece and nose-clip, subjects decreased respiratory frequency and increased VT (without change of $\dot{V}E$) relative to mask breathing. When the nose-clip was worn under the mask, there was only a small fall in frequency and $\dot{V}E$, so that nasal stimulation accounted for only a minor part of the mouthpiece–nose-clip effect.

In an attempt to relate the changes induced by the switch from nasal to oral breathing to the effects of an external dead space, we used respiratory inductance plethysmography (RIP) to measure resting ventilation in normal subjects unimpeded and with masks, cotton-wool nasal plugs, nose-clips and mouthpieces with and without instrumental dead spaces of up to 600 ml. The effect of facial stimulation alone was studied with a facemask of the Venturi type, which, when provided with a high flow of air, had an effective dead space of zero.

Methods

SUBJECTS AND PROTOCOL

Series I. Ten volunteers (six men, four women, age range 23–46, mean FEV_1 102% pred. SD 8, mean VC 103% pred. SD 7) from the staff of the department were studied. None had any history of cardiopulmonary disease, although two were smokers. These subjects, who were unaware of the specific purpose of the experiment, were studied twice, on separate days, at the same time of day, and 2–4 h after the last meal or caffeinated drink. Ventilation and its subdivisions were monitored at rest, in the erect position in a purpose built chair (subjects 1–6), or semi-recumbent on a bed (subjects 7–10) using a respiratory inductance plethysmograph (Respitrace, Ambulatory Monitoring Inc., Ardsley, New York) in the following situations: (a) unimpeded with mouth and nose free, (b) with a Venturi mask (Vickers Medical Mask III), (c) with a nose-clip and mouthpiece (external dead space (DS) = 10 ml), and with nose-clip, mouthpiece and widebore tubing (internal diameter 2.9 cm) to create DS of (d) 80 ml, (e) 350 ml, and (f) 600 ml.

During (b) the flow of air (40 L/min) was greater than the mean inspiratory flow of every subject and effectively reduced DS to zero. During (c) subjects 7–10 wore the nose-clip but no mouthpiece. The resistance of the wide-bore tubing was < 0.2 cm

$H_2O \cdot L^{-1} \cdot sec^{-1}$ at a flow of 80 L/min. The actual volume of each length of tubing was measured by water displacement. The effective dead spaces of the 80 ml, 350 ml, and 600 ml tubes, measured at flow rates of 30 L/min using a single breath technique (Fowler, 1948), were 50 ml, 290 ml, and 440 ml respectively. In subjects 1–5, 'arterial' P_{CO_2} (Pa_{CO_2}) was estimated transcutaneously with an electrode attached to the forearm (Radiometer, Copenhagen). Previous experience from this laboratory showed that this electrode estimated Pa_{CO_2} with 95% confidence limits of ± 6.7 mmHg and a correlation coefficient of 0.95, and that it followed step changes more accurately (Stradling *et al.*, 1983).

Series II. Eight subjects (including two from the previous series) with similar age range and lung function were studied on one occasion only, sitting in the chair, semi-fasting. Ventilation and its sub-divisions were measured: unimpeded (control (C)), with cotton-wool nasal plugs (CW), with a nose-clip (NC), and with a nose-clip and mouthpiece (MP).

During both series radio programmes were played to distract the subjects from their breathing. Each test condition (except (f) of series I, which came last) was presented in a randomised order and lasted 6 min followed by a gap of 5 min to allow a return to normal breathing.

Ventilatory monitoring. RIP was calibrated using a single-posture technique previously described (Mannix *et al.*, 1984) and was validated against simultaneous spirometry by comparing the tidal volumes of twenty breaths measured by RIP (VT_{RIP}) with those from the spirometer (VT_{SP}) measured simultaneously but independently before and after each test condition. The ratio of these tidal volume measurements (VT_{RIP}/VT_{SP}) provided confirmation that the calibration was adequate and a factor by which volume measurements during the test runs could be corrected. The overall mean VT_{RIP}/VT_{SP} ratio was 1.03 (SD = 0.09).

Data collection and analysis. Signals from RIP were collected and analysed by an Apple II computer to provide on-line breath by breath values for VT, $\dot{V}E$, frequency (f), inspiratory duty cycle (TI/TTOT), inspiratory duration (TI), expiratory duration (TE), mean inspiratory flow (VT/TI) and the abdominal contribution (as %) to VT (AB). These data were displayed on a monitor and a chart recorder. Data from the last 2 min of each test condition were collected and averaged for later analysis. The significance of differences between the group means of each test run was assessed by analysis of variance using the Newman–Keuls test for multiple comparisons whenever significant differences were observed.

Results

Subjects 7–10 of Series 1 were studied semi-recumbent on a bed and during test condition (c) wore only the nose-clip. Analysis of their data during this period revealed a decrease in frequency that did not occur in subjects 1–6. As this may have been due to the small change in posture and not just the nose-clip, the data of the two groups were analysed separately. Series II was designed to look more closely at the nose-clip effect.

Breathing unimpeded or with a high flow mask. The mean values of ventilatory variables at rest are displayed in table 1 alongside those obtained when breathing through the zero dead-space Venturi mask. There was no significant difference between them for either group. All subjects spontaneously breathed through the nose during both tests so that effective dead spaces were similar.

The effect of adding DS. Figure 1 shows the mean values for $\dot{V}E$ and its components VT/TI and TI/TTOT in subjects 1–6 whilst breathing through different instrumental dead spaces. VT, TI and TE are plotted in fig. 2. These figures show how the response to increasing dead space is an increase in VT/TI with little or no change in timing. The nose-clip and mouthpiece alone caused a significant increase in VT (11% SD 11, $P < 0.05$) above that while breathing air. With the exception of the alteration in timing in period (c) similar changes were seen in subjects 7–10.

External dead space ventilation (DS) was calculated ($\dot{D}S = DS \times f$) and compared to the increase in $\dot{V}E$ ($\Delta\dot{V}E$) brought about by the addition of DS (taking control $\dot{V}E$ as that obtained with nose-clip and mouthpiece alone, fig. 3). The net increase in ventilation due to increasing DS is the difference between these two values (*i.e.* $\Delta\dot{V}E - \dot{D}S$). Using

TABLE 1

Effects on resting breathing of a Venturi face mask (Vickers Ventimask) flushed with air at 40 L/min (Mask) compared with no mask (Control). $\dot{V}E$ = minute ventilation; VT = tidal volume; f = respiratory frequency; TI/TTOT = inspiratory time as a fraction of total cycle time; VT/TI = mean inspiratory flow; TI and TE = inspiratory and expiratory times. ABDO = fraction of movement from abdomen contributing to total volume. Average values over last 2 min of 6 min observation period. Mean ± SEM.

	Control (n = 6)	Mask (n = 6)	Control (n = 4)	Mask (n = 4)
$\dot{V}E$ (L/min)	5.9 ± 0.4	6.0 ± 0.5	5.4 ± 0.5	5.6 ± 0.5
VT (L)	0.40 ± 0.05	0.41 ± 0.06	0.35 ± 0.02	0.36 ± 0.03
f (min^{-1})	15.3 ± 0.5	15.5 ± 0.5	15.5 ± 1.5	15.7 ± 1.5
VT/TI (L/min)	15.5 ± 1.0	15.9 ± 1.3	15.6 ± 1.5	15.7 ± 1.2
TI/TTOT	0.38 ± 0.02	0.38 ± 0.02	0.36 ± 0.04	0.36 ± 0.03
TI (sec)	1.53 ± 0.09	1.51 ± 0.1	1.38 ± 0.06	1.41 ± 0.04
TE (sec)	2.51 ± 0.2	2.48 ± 0.2	2.63 ± 0.4	2.55 ± 0.3
ABDO	0.38 ± 0.05	0.35 ± 0.04	0.42 ± 0.08	0.44 ± 0.07

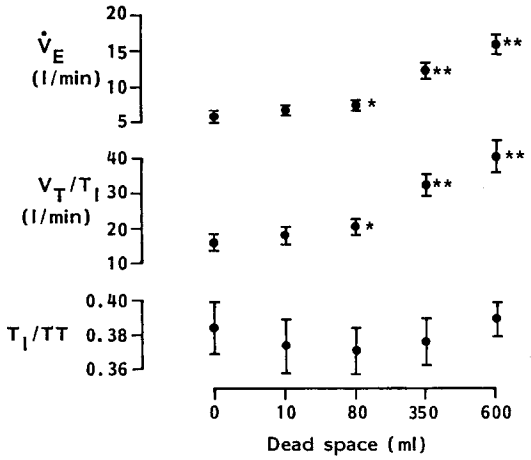

Fig. 1. Effect of addition of external dead space on minute ventilation ($\dot{V}E$) and mean inspiratory flow (VT/TI) and the inspiratory duty cycle (TI/TTOT) in 6 normal subjects in Series I with instrumental dead spaces (DS) of 0, 10, 80, 350, 600 (*i.e.* during periods (a), and (c)–(f); see text for details). Values averaged over last 2 min of each 6 min period: mean ± SEM. Significant differences from DS 0 ml: * $P < 0.05$; ** $P < 0.01$.

values of effective dead space volume obtained by the Fowler single breath technique (Fowler, 1948), the external dead spaces of 80, 350 and 600 ml caused net increases in ventilation of 3%, 19%, and 26% of $\dot{V}D$S. Transcutaneous CO_2 in the 5 subjects who were measured rose significantly only with dead space volumes of 350 and 600 ml ($P < 0.001$; fig. 3).

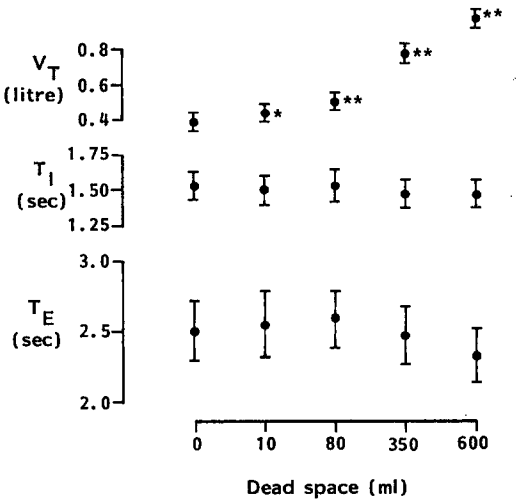

Fig. 2. Effect of addition of external dead space on tidal volume (VT) and inspiratory (TI) and expiratory (TE) time in Series I. Subjects, symbols and periods as in fig. 1.

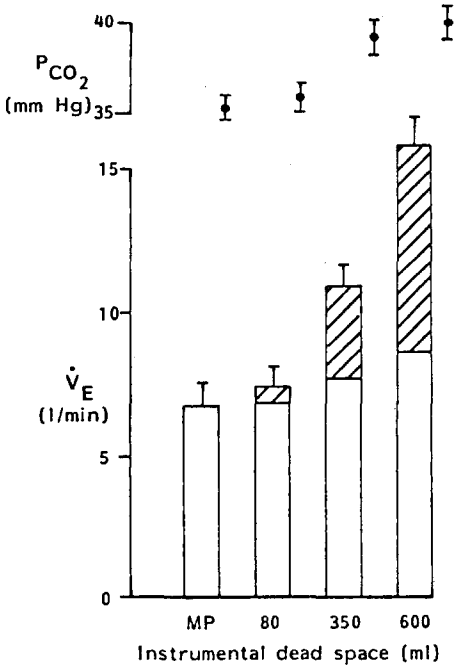

Fig. 3. Transcutaneous P_{CO_2} (Ptc_{CO_2}) and minute ventilation ($\dot{V}E$) as a function of instrumental dead space. MP = mouthpiece. The shaded area represents the $\dot{V}E$ required to overcome the *effective* instrumental dead space (Fowler technique). Note *net* ventilation ($\dot{V}E$, unshaded) also increases. For Ptc_{CO_2} n = 5, and for $\dot{V}E$ n = 6: mean ± SEM.

The effect of change from nose to mouth breathing (Series II). The change from nasal to oral breathing in series II (table 2) was accompanied by a similar increase in VT in each of the three conditions (14–16%, $P < 0.05$). Changes in minute ventilation were, however, very small (range −2% to +2%, P = NS). With the nose-clip only (NC) there was a significant reduction in frequency (−15% SD ± 15, $P < 0.05$) with increases in

TABLE 2

Effects on resting breathing of devices occluding the nose (cotton wool and nose-clip) and with and without a mouthpiece. 8 subjects. Symbols and values as for table 1. n = 8. (* different from control, $P < 0.05$.)

	Control	Cotton wool	Nose-clip	Nose-clip + Mouthpiece
$\dot{V}E$ (L/min)	5.9 ± 0.6	6.1 ± 0.4	5.8 ± 0.6	6.03 ± 0.4
VT (L)	0.42 ± 0.02	0.48 ± 0.04*	0.48 ± 0.03*	0.48 ± 0.03*
f (min^{-1})	14.1 ± 1.2	13.0 ± 1.0	12.3 ± 1.2*	12.8 ± 0.9
VT/TI (L/min)	16.0 ± 1.3	17.9 ± 1.7	16.1 ± 1.7	17.3 ± 1.2
TI/TTOT	0.373 ± 0.02	0.350 ± 0.02	0.37 ± 0.02	0.35 ± 0.01
TI (sec)	1.61 ± 0.1	1.64 ± 0.1	1.92 ± 0.2*	1.72 ± 0.1
TE (sec)	2.76 ± 0.2	3.11 ± 0.3	3.25 ± 0.3	3.15 ± 0.2
ABDO	0.34 ± 0.04	0.35 ± 0.04	0.34 ± 0.03	0.34 ± 0.04

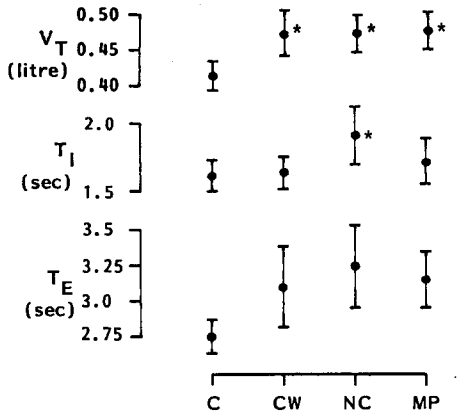

Fig. 4. Effect of nasal plugging and mouthpieces on tidal volume (VT) and inspiratory (TI) and expiratory (TE) time in Series II. n = 8. Mean ± SEM. Significant differences from C: * $P < 0.05$.

TI (17% SD ± 26, $P < 0.05$) and TE (20% SD ± 22, P = NS) (fig. 4). With mouthpiece and nose-clip together and with nasal cotton wool plugs alone there were smaller reductions in frequency when compared to control (−7% SD ± 13, P = NS; −10% SD ± 12, P = NS, fig. 4). The increases in VT compared to unimpeded breathing were secondary to changes in both TI and VT/TI.

Discussion

Breathing with a facemask. The effect of a facemask delivering a high flow of air has not been reported before. This device has been used to deliver different gas mixtures when ventilation was being monitored non-invasively (Mannix *et al.*, 1984). Freedman *et al.* (1983) have shown that similar equipment made no difference to resting breathing in acutely ill hypoxic asthmatics. The data (table 1) confirm that in normals also this type of stimulation has no effect on resting ventilation, nor was there any change in transcutaneous P_{CO_2}.

The effect of added instrumental dead space. The increases in VT and $\dot{V}E$ following increases of DS were not accompanied by significant changes in the timing of respiration. Similar effects of increases in DS of 150 ml or more were first noted by Stannard and Russ (1948). In five subjects, transcutaneous P_{CO_2} increased, in agreement with others, who have shown that increasing DS is accompanied by a small but proportionate increase in alveolar P_{CO_2} (Stannard and Russ, 1948; Schwartz *et al.*, 1957; Ward and Whipp, 1980) and somewhat larger falls in alveolar P_{O_2} (Goode *et al.*, 1969). The latter group have shown that the combined stimuli of hypercapnia and hypoxia are sufficient to cause this increase in ventilation, at least whilst breathing air through a dead space

of about a litre. In fact, the change in the pattern of breathing when stimulated by an external dead space (figs. 1 and 2) is the same as the response to hypoxia and hypercapnia (Mannix *et al.*, 1984).

It is of interest that, in this study, the increase in ventilation was greater than that required to ventilate the effective dead space (fig. 3). Thus the increase of 'arterial' (transcutaneous) P_{CO_2} occurred despite adequate ventilation of this dead space. Clearly, this conclusion is dependent on the value taken for effective dead space. The water-displacement values, used by Ward and Whipp (1980), for example, overestimate DS so that their conclusion that the increase in $\dot{V}E$ matches the increases in DS is not valid. Jones *et al.* (1971) achieved adequate mixing of air in the added dead space by means of a turbine and found that the increase in $\dot{V}E$ was less than that expected from the size of the DS. Nevertheless, the resistance of their circuit was high (1–1.4 cm $H_2O \cdot L^{-1} \cdot sec^{-1}$). Sackner *et al.* (1980) measured instrumental dead spaces with the Fowler technique, and, although the design of their study was different, the results were similar *i.e.* ventilation was greater than that required to clear the DS. Thus the increase in P_{CO_2} (fig. 3) must reflect either worsening $\dot{V}_A/\dot{Q}$ relationships or increased CO_2 production caused by the extra work of breathing. With the 600 ml DS, $\dot{V}_{CO_2}$ increased in one subject by 17%, hardly sufficient to raise Pa_{CO_2} by 4 mmHg (fig. 3) in the face of a net increase of $\dot{V}E$ of 1.9 L/min.

The effect of the change from nasal to oral breathing. The use of cotton wool plugs was the least invasive way of switching to oral breathing without actually asking the subject to maintain this voluntarily. The plugs caused no obvious discomfort and permitted actions such as swallowing which are not so easily performed with nose-clips and mouthpieces.

The switch from nasal to oral breathing was accompanied by a consistent 60 ml increase in VT irrespective of how the nose was occluded or whether a mouthpiece was inserted (table 2). Sixty ml corresponds closely with the estimates of the volume of the buccal cavity (Nunn *et al.*, 1959). Nevertheless, $\dot{V}E$ did not increase by the expected amount (0.84 L/min) because respiratory frequency fell. With the nose-clip alone there were significant changes in timing. Weissman *et al.* (1984), using a head canopy, also found changes in timing with application of a nose-clip alone. On the other hand Hirsch and Bishop (1982) did not find a significant fall in frequency when a nose-clip was applied under a facemask compared to breathing with the mask alone.

Mouthpieces impose uncomfortable constraints which might be expected to affect the pattern of breathing. Druz and Sharp (1981) have shown that these devices can cause an increase in electromyographic activity in the diaphragm, the scalene and sternocleidomastoid muscles. Interestingly, they also observed a drop in frequency accompanying the application of a mouthpiece (and, presumably, a nose-clip). It is well established that inhibition of respiration in primates, sub-primates and diving mammals can be elicited by stimulation of trigeminal receptors in this area (Daly, 1984). The nose also acts to warm, humidify and filter inspired air. These functions may be lost during oral breathing and result in irritation of the oropharynx and airways. The effects of this on breathing

are unknown, but chemical stimuli applied to the larynx may elicit inhibitory responses similar to those described above (Szereda-Przestaszewska and Widdicombe, 1973).

A further factor, which may induce a change in breathing pattern following the switch from nasal to oral breathing, is an alteration in airways resistance. Total airflow resistance during nasal breathing is reduced by 70–80% by breathing through mouthpieces, which necessarily widen the oral aperture (Cole *et al.*, 1982). However, without a mouthpiece, the oral cavity may remain sufficiently narrow for its airflow resistance to be as great as that of the nose until tidal flows increase considerably (Niinimaa *et al.*, 1981). Airflow resistance with a mouthpiece and a nose-clip may therefore be much less than with a nose-clip alone or with nasal breathing. Application of light external resistive loads (3.0 cm $H_2O \cdot L^{-1} \cdot sec^{-1}$) cause small but significant increases in V_T and T_I with decreases in frequency but no change in minute ventilation (Daubenspeck, 1981). If obstructing the nose caused an important decrease in airflow resistance, changes opposite to those described, *i.e.* a fall in T_I, would have been expected. If changes in resistance do occur in the transition from nasal to oral breathing, these do not affect breathing at rest in any major way.

In conclusion, alterations in the pattern of breathing are associated with the equipment commonly used to measure it. Increases in dead space resulting from the change from nasal to oral breathing may contribute to increases in tidal volume, but any increase in minute ventilation is limited by a small drop in frequency, which may be due to the discomfort of the equipment itself. A nose-clip appears to be particularly effective in slowing breathing possibly due to stimulation of trigeminal receptors.

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