Effect of age on sleep onset-related changes in respiratory pump and upper airway muscle function

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Worsnop, Christopher, Amanda Kay, Young Kim, John Trinder, and Robert Pierce. Effect of age on sleep onset-related changes in respiratory pump and upper airway muscle function. J Appl Physiol 88: 1831–1839, 2000.—In normal young men, there is an abrupt fall in ventilation (Ve), a rise in upper airway resistance (UAR), and falls in the activities of the diaphragm (Di), intercostals (IC), genioglossus (GG), and tensor palatini (TP) at sleep onset. On waking, there is an abrupt increase in Ve and fall in UAR and an increase in the activities of Di, IC, GG, and TP. The aim of this study was to determine whether these changes are age dependent. Nine men aged 20 to 25 yr were compared with nine men aged 42 to 67 yr. Airflow, UAR, Di, and IC surface electromyograms (EMGs) and the intramuscular EMGs of GG and TP were recorded. It was found that the falls in IC, GG, and TP at the transition from α to θ electroencephalogram (EEG) activity were significantly greater in the older than in the younger men (P < 0.05) and that the fall in Di was also greater, although this was only marginally significant (P = 0.15). The rise in GG at θ-to-α transitions was also greater in the older than in the younger men, and there was a trend for TP to be higher.

IN NORMAL YOUNG MEN, there is an abrupt fall in ventilation (Ve; Refs. 8, 32) and rise in upper airway resistance (UAR; Refs. 14, 15) at the transition from α to θ electroencephalogram (EEG) activity, that is, a transition from wakefulness to sleep. As sleep progresses, Ve remains depressed at a stable level, but UAR continues to rise until a stable level is reached in slow-wave sleep (16). When there is an awakening during sleep onset, characterized by a change in EEG activity from θ back to α, there is an abrupt increase in Ve and fall in UAR (14, 15).

The changes in Ve and UAR during sleep onset are accompanied by changes in the electromyogram (EMG) activities of respiratory muscles. The diaphragm (Di) and intercostal (IC) activities fall abruptly at α-to-θ transitions and increase abruptly at θ-to-α transitions. The activities of the upper airway muscles, genioglos-

The prevalence of OSA is greater in middle-aged and older men compared with young men (1, 3–7, 12, 25, 27,
This is in part related to an increase in body fat in older men (2, 5–7, 26, 36). Older subjects have also been shown to have increased pharyngeal resistance during wakefulness that was not due to differences in weight (35) and to have greater fluctuations in UAR than younger subjects during both wakefulness and non-rapid eye movement sleep (13). The ventilatory responses and the responses in the pressure generated in the first 0.1 s after airway closure during inspiration (P0.1; used as an indicator of central respiratory drive) to hypoxia and hypercapnia are reduced in the elderly. Given that these differences between older and younger subjects cannot be explained by differences in lung mechanics or Di strength (18, 24), they may occur because of reduced neural input to respiratory muscles. Naifeh et al. (21) found the ventilatory response to CO2 to be the same in older subjects compared with younger subjects, although their younger subjects were older than those in the other studies.

This study was undertaken to directly assess the effects of age on the changes in V˙E, UAR, and the EMG activities of Di, IC, GG, and TP. Obesity can have a confounding effect on upper airway muscle activity; it has been shown that obese subjects without OSA have greater GG EMG activity during non-rapid eye movement sleep than during wakefulness, whereas subjects who are not obese have the same GG activity in stable sleep and wakefulness (28). To avoid this confounding effect, we studied only subjects who had a normal body mass index (BMI). Initially, they were asked to remain awake for ~10 min before falling asleep so that some baseline α EEG activity could be collected. To obtain multiple sleep onsets, they were woken once stable stage 2 sleep had been observed and were then allowed to fall asleep again. This procedure was repeated until ~4 h of data had been collected.

Sleep, EMG, and respiratory measurements were recorded with a 16-channel Grass polygraph (model 7D). Occipital EEG, all EMGs, airflow, and pressure measurements were also recorded on an IBM-compatible 486 PC. Central (C3/A2) and occipital (O2/A2) EEG and EOG were recorded. For each subject, the occipital EEG activity during each breath was assessed as being predominantly α or θ, as previously described (14). Briefly, for each subject, 10 min of α and 10 min of θ were visually identified. This included 100–150 breaths during α and 100–150 breaths during θ. For each breath in these two periods, the frequencies of all negative peak-to-peak intervals in the EEG in the 0.3- to 50-Hz range were determined, and these intervals were divided into those >8 Hz, that is, 0.125 s, and those <8 Hz. For each breath, a ratio of the number of EEG intervals >8 Hz to the total number of intervals was calculated. The distributions of EEG ratios for the breaths in the selected 10 min of α and for the breaths in the 10 min of θ were then plotted. The point of intersection between these two distributions was identified, and the ratio corresponding to this point of intersection became the criterion ratio for that subject. Thus any breath that had a ratio below the criterion ratio was classified as occurring during α EEG activity, and any breath with a ratio above the criterion ratio was classified as occurring during θ EEG activity. This criterion ratio was then used to classify all breaths for that subject as occurring either during α or θ EEG activity. This process was repeated for each subject. It is illustrated in Fig. 1.

In addition, the sleep period was classified into three phases. Phase 1 was defined as wakefulness before the appearance of θ, that being defined as at least three out of five consecutive breaths having predominantly θ activity. Phase 2 was defined as the period from the first appearance of θ to the first sleep spindle or K complex. Phase 3 was defined as the period from the first sleep spindle or K complex to the attainment of stable stage 2 sleep. The development of sleep was described in terms of these phases rather than as stages of sleep because the changes between phases can be identified more precisely in terms of differences between the phases, which are arbitrarily defined as stages of sleep. In this study, the occipital EEG activity was visually identified. This included 100–150 breaths during each of the three phases, and these intervals were divided into those >8 Hz, that is, 0.125 s, and those <8 Hz. For each breath, a ratio of the number of EEG intervals >8 Hz to the total number of intervals was calculated. The distributions of EEG ratios for the breaths in the selected 10 min of α and for the breaths in the 10 min of θ were then plotted. The point of intersection between these two distributions was identified, and the ratio corresponding to this point of intersection became the criterion ratio for that subject. Thus any breath that had a ratio below the criterion ratio was classified as occurring during α EEG activity, and any breath with a ratio above the criterion ratio was classified as occurring during θ EEG activity. This criterion ratio was then used to classify all breaths for that subject as occurring either during α or θ EEG activity. This process was repeated for each subject. It is illustrated in Fig. 1.

EMG recordings. Diaphragmatic EMG was recorded with gold-plated, cup-shaped surface electrodes placed over the subcostal margin anteriorly, and external intercostal EMG was recorded with surface electrodes placed over the sixth intercostal space laterally. Fine wire intramuscular electrodes were used to record GG and TP EMGs. The wire was stainless steel, 3/1,000 in. thick, with a 1/1,000-in. Teflon coating. The Teflon was stripped from the end of the wire for 1.5 mm, and a 1-mm hook was fashioned in the end of the wire. The wires were inserted into the muscles perorally with hypodermic needles. The sites of insertion were anesthetized with a small amount of 2% lidocaine gel. While the electrodes were being inserted, the visual and auditory EMG signals were monitored to ensure that the electrodes were placed in muscle. To confirm that the electrodes were in the correct muscle, a series of maneuvers that have previously been shown to elicit responses from GG and TP was performed (19,
Fig. 1. An illustration of how criterion ratio is determined. A: a single breath from a period of θ activity. *Peak-to-peak intervals >8 Hz (0.125 s) | Peak-to-peak intervals <8 Hz (0.125 s). The ratio of intervals >8 Hz/total intervals for this breath of θ activity is 11/16, which is 0.69. A ratio such as this is calculated for every breath in a 10-min period of typical θ activity and then plotted in a frequency distribution. This process is repeated for a 10-min period of typical α activity. B: diagram of a theoretical plot of frequency distributions of ratios for each breath in the 10-min period of typical θ activity and for each breath in the 10-min period of typical α activity. Arrow marks ratio that separates the two distributions. This is the criterion ratio. (This is an illustration only and not based on real data.) EEG, electroencephalogram.

30). Jaw opening, jaw protrusion, blowing, sucking, swallowing, nasal breathing, and increased tidal volume produced increases in the EMG activity of TP. Tongue protrusion, the Muller maneuver, swallowing, and increased tidal volume produced increased EMG activity in GG. Sections of the recording containing movement and other artifacts were removed before analysis. Furthermore, 100- to 160-ms sections of the Di and IC surface EMGs containing QRS complexes were removed and replaced by the data points in the 50- to 80-ms periods before and after the QRS complex using computer software. The raw EMG signals for all muscles were then integrated by using a 100-ms moving time average (MTA). For each muscle, several values were calculated on a breath-by-breath basis: 1) For each breath, the preceding expiration was divided into 10 equal time periods, and the mean EMG amplitude from the period with the lowest mean amplitude was used as the tonic activity for that breath. 2) Phasic activity was defined as the area under the inspiratory MTA curve above the tonic activity level identified in the previous expiratory phase. 3) Total inspiratory activity was calculated as the total area under the inspiratory MTA curve. It should be noted that because tonic activity was defined as the lowest level of activity during expiration, statistically all muscles were identified as having phasic activity.

Measurement of ventilation. An oronasal mask with an air-filled cushion was strapped to the head tightly enough to eliminate any leaks. A heated pneumotachograph (Morgan) was attached to the mask. The dead space of the mask and pneumotachograph was 120 ml. The pneumotachograph was connected to a differential pressure transducer (Validyne model DP45–14) and to a carrier demodulator (Validyne CD75) that converted the output to a voltage signal. Airflow was calibrated with a flowmeter (Shorate 1355). The airflow signal was analyzed off-line to calculate extrapolated minute ventilation for each breath.

Measurement of UAR. Simultaneous recordings of mask pressure, epiglottic pressure, and airflow were used to calculate UAR. Mask pressure was recorded via a pressure transducer (Validyne DP45–28) and carrier demodulator (Validyne CD15). The other side of the pressure transducer was connected to an equal length of tubing left open to the atmosphere. Epiglottic pressure was measured with a transducer-tipped catheter (Millar model MPC-500) inserted through the nose and advanced until the tip was 1 cm below the base of the tongue visualized through the mouth without the tongue protruded. The nostril was premixed with 0.05% oxymetazoline hydrochloride spray and 2% lidocaine gel. Computer software was used to calculate the pressure gradient across the upper airway from epiglottis to mask and to zero this pressure differential at the end of inspiration and the end of expiration, the points of zero flow. The phasing and time constants of the epiglottic pressure catheter and mask pressure measurements were adjusted to coincide. Although a number of resistance measurements were generated by the software, the UAR reported was the resistance at peak airflow.

Data analysis. Once each breath had been classified as occurring during α or θ EEG activity, computer software was used to identify sets of consecutive α or θ breaths occurring at either side of α-to-θ transitions and of θ-to-α transitions. Thus for each transition four to ten breaths were identified, two to five consecutive α breaths and two to five consecutive θ breaths. Each of these breaths then had an identifiable position within a transition from −5 to +5. For each subject, the parameters of interest were averaged for each breath position. These parameters were V˙E, UAR at peak flow, and the EMG activities of Di, IC, GG, and TP expressed as arbitrary units. The changes in these parameters between wake and sleep were determined by the changes in EEG activity between α and θ without reference to changes in the respiratory parameters. Each subject had to have data from at least five breaths at a particular breath position for those data to be included, so that an aberrant breath from one subject would not unduly bias the group data. Changes beyond five posttransition breaths were also studied, but data from phases 2 and 3 needed to be combined, and for this latter analysis there was no minimum requirement for the number of data points at a particular breath position.

Because raw score EMG units are arbitrary and depend on degrees of amplification, group data can be excessively influenced by one subject. Therefore, we expressed the EMG activity for each posttransition breath as a percentage of the pretransition baseline level. This baseline was defined as the average of the breaths −5 to −2 for each of the four types of transition, i.e., α-to-θ in phase 2, α-to-θ in phase 3, θ-to-α in phase 2, and θ-to-α in phase 3. Four pretransition breaths were chosen as the baseline to overcome the inherent variability seen in any physiological parameter. Using the −5 to −2 breaths as a comparison for the posttransition breaths better reflects any changes at transitions than comparing each of the posttransition breaths with a single pretransition breath. Breath position −1 was not used in the determination of the baseline because a breath in the −1 or −2 position may have the change in EEG activity occurring during it and so may not be purely α or θ activity. It should be noted that the lack of precision in the classification of breaths at transitions results in some smoothing of the data over the transition and can
create the impression that changes in the parameters at transitions have commenced before the $\alpha$-to-$\theta$ transition.

Statistics. To determine whether there were significant changes at $\alpha$-to-$\theta$ and $\theta$-to-$\alpha$ transitions, single sample $t$-tests were performed comparing the mean data from all of the subjects at each of the first five posttransition breath positions with a reference value of 100 for $\dot{V}E$, UAR, and the EMG activities of the four muscles for phase 2 and for phase 3. A $2 \times 2 \times 5$ ANOVA with repeated measures on phase and breath position was used to assess the effect of age, phase, and breath position on $\dot{V}E$, UAR, and the EMG activities of the four muscles. The breath position data for each parameter were the values at each of the first five posttransition $\theta$ breaths at $\alpha$-to-$\theta$ transitions; there was a separate $2 \times 2 \times 5$ ANOVA for the five posttransition $\alpha$ breaths at $\theta$-to-$\alpha$ transitions. To assess the effect of age and breath position in the 20 posttransition $\theta$ breaths at $\alpha$-to-$\theta$ transitions, a $2 \times 20$ ANOVA was performed.

RESULTS

An example of the raw data from an individual older subject is shown in Fig. 2. It illustrates the dramatic fall in activities of the four muscles at an $\alpha$-to-$\theta$ transition and the precision with which the changes are associated with the state changes.

The group data for all subjects are shown in Tables 1 and 2. The group data for the younger and older subjects are illustrated in Figs. 3 and 4. At $\alpha$-to-$\theta$ transitions, $\dot{V}E$ in each of the five $\theta$ breaths immediately after the transition was significantly lower than the

Table 1. Group data at each of the five $\theta$ breaths after a transition from $\alpha$ to $\theta$

<table>
<thead>
<tr>
<th></th>
<th>Phase 2</th>
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<th>Phase 3</th>
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<tbody>
<tr>
<td></td>
<td>+1</td>
<td>+2</td>
<td>+3</td>
<td>+4</td>
</tr>
<tr>
<td>$\dot{V}E$</td>
<td>83 ± 10</td>
<td>80 ± 12</td>
<td>80 ± 15</td>
<td>84 ± 15</td>
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<tr>
<td>UAR</td>
<td>112 ± 14</td>
<td>140 ± 47</td>
<td>152 ± 62</td>
<td>185 ± 113</td>
</tr>
<tr>
<td>$\dot{D}i$</td>
<td>82 ± 15</td>
<td>81 ± 20</td>
<td>83 ± 19</td>
<td>86 ± 18</td>
</tr>
<tr>
<td>$\dot{I}C$</td>
<td>84 ± 16</td>
<td>87 ± 17</td>
<td>90 ± 22</td>
<td>99 ± 34</td>
</tr>
<tr>
<td>$\dot{G}G$</td>
<td>76 ± 17</td>
<td>77 ± 20</td>
<td>81 ± 26</td>
<td>96 ± 33</td>
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<tr>
<td>TP</td>
<td>73 ± 17</td>
<td>73 ± 26</td>
<td>61 ± 25</td>
<td>56 ± 18</td>
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<td></td>
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<td></td>
<td>+1</td>
<td>+2</td>
<td>+3</td>
<td>+4</td>
</tr>
<tr>
<td>$\dot{V}E$</td>
<td>78 ± 12</td>
<td>69 ± 16</td>
<td>66 ± 16</td>
<td>65 ± 16</td>
</tr>
<tr>
<td>UAR</td>
<td>115 ± 30</td>
<td>166 ± 74</td>
<td>234 ± 167</td>
<td>295 ± 343</td>
</tr>
<tr>
<td>$\dot{D}i$</td>
<td>78 ± 22</td>
<td>71 ± 21</td>
<td>72 ± 20</td>
<td>74 ± 22</td>
</tr>
<tr>
<td>$\dot{I}C$</td>
<td>80 ± 22</td>
<td>80 ± 30</td>
<td>84 ± 23</td>
<td>88 ± 34</td>
</tr>
<tr>
<td>$\dot{G}G$</td>
<td>74 ± 19</td>
<td>72 ± 24</td>
<td>82 ± 26</td>
<td>86 ± 25</td>
</tr>
<tr>
<td>TP</td>
<td>76 ± 41</td>
<td>64 ± 35</td>
<td>60 ± 37</td>
<td>68 ± 43</td>
</tr>
</tbody>
</table>

Values are means ± SD expressed as a percentage of the fifth to second breaths of $\alpha$ electroencephalogram activity immediately before a transition; $\dot{V}E$, ventilation; UAR, upper airway resistance; $\dot{D}i$, diaphragm electromyogram (EMG) activity; $\dot{I}C$, intercostal EMG activity; $\dot{G}G$, genioglossal EMG activity; TP, tensor palatini EMG activity.
average of the five preceding \( \alpha \) breaths in both phases 2 and 3, whereas UAR was significantly higher in each \( \theta \) breath. The EMG activity of Di was significantly lower across each of the five \( \theta \) breaths than the preceding five \( \alpha \) breaths in phases 2 and 3. IC activity was significantly lower in each of the first two \( \theta \) breaths in phase 2 and significantly lower in four of the five \( \theta \) breaths in phase 3. GG activity was significantly lower in each of the first three \( \theta \) breaths in phase 2 and significantly lower in all the five \( \theta \) breaths in phase 3. TP activity was significantly lower in all the five \( \theta \) breaths in both phases 2 and 3.

At \( \theta \)-to-\( \alpha \) transitions for all subjects, \( \dot{V}e \) was significantly higher and UAR was significantly lower in each of the five \( \alpha \) breaths than the preceding five \( \theta \) breaths in both phases 2 and 3. There was a trend for Di activity to be higher in each of the first four \( \alpha \) breaths in phase 2, and Di activity was significantly higher in the first three \( \alpha \) breaths in phase 3. IC activity did not differ significantly between the \( \alpha \) and \( \theta \) breaths. GG activity was significantly higher in the first posttransition breath in phase 2, and in the first two posttransition breaths in phase 3. TP activity was significantly higher in each of the posttransition \( \alpha \) breaths in phase 3.

With respect to the age effects, there were significant group effects showing greater changes at \( \alpha \)-to-\( \theta \) transitions in the older subjects for UAR, IC, GG, and TP, and a trend for \( \dot{V}e \) (\( P = 0.09 \)) and Di (\( P = 0.15 \)), and significant phase effects showing that phase 3 changed more than phase 2 for \( \dot{V}e \), UAR, Di, and IC but not GG and TP. There was no difference in IC EMG activity between \( \alpha \) and \( \theta \) in the younger subjects, yet its activity was lower in \( \theta \) than in \( \alpha \) in the older subjects.

The 2 \( \times \) 20 ANOVA on the 20 posttransition breath data at \( \alpha \)-to-\( \theta \) transitions showed that there was a significant age group effect showing lower activity in the posttransition \( \theta \) breaths in the older subjects for IC and UAR, but not for \( \dot{V}e \), Di, GG, or TP.

With respect to the \( \theta \)-to-\( \alpha \) transition data, there was a significant age group effect for UAR and GG and a trend for \( \dot{V}e \) (\( P = 0.08 \)) and TP (\( P = 0.08 \)), but not for Di or IC, and a significant phase effect for \( \dot{V}e \), UAR, and Di and trends for GG (\( P = 0.09 \)) and TP (\( P = 0.10 \)), but not for IC.

**DISCUSSION**

This study has shown that during sleep onset the decreases in IC, GG, and TP activities and the increases in UAR are greater in older than younger normal men, and there were trends for \( \dot{V}e \) and Di activity to be lower. On waking from sleep, the increase in GG and fall in UAR was greater in older than younger men, and there were trends for \( \dot{V}e \) and TP activity to be higher. The falls in \( \dot{V}e \) and EMG activity and rise in UAR at \( \alpha \)-to-\( \theta \) transitions and rises and falls at \( \theta \)-to-\( \alpha \) transitions were greater in phase 3 than in phase 2, although not all of these differences were statistically significant. These phase differences were not different between the older and younger groups.

The demonstrated greater fall in the activities of respiratory muscles at \( \alpha \)-to-\( \theta \) transitions in older men compared with younger men could occur by two mechanisms. One possibility is that older men have greater muscle activity during wakefulness yet fall to the same level as the younger men during sleep. Alternatively, the muscle activities may be similar during wakefulness, but the older men have a lower level during sleep. As the EMG is recorded in arbitrary units, it is possible to directly compare only the relative changes in EMG activities between the two groups and not the absolute activities of the muscles in either sleep or wakefulness. Thus our data cannot distinguish between the two explanations. Also, it is not possible to relate EMG activity directly to UAR because UAR is dependent not only on neural activity and upper airway muscle activity but also on the mechanical output of individual muscles, the interactions between the upper airway muscles, the anatomy of the upper airway, and the driving force during inspiration. Thus examining the UAR of the two groups does not help in determining why there is a difference in the changes at sleep onset between older and younger men.

Nevertheless, irrespective of the relative activities in wakefulness, it is possible that the greater fall in upper airway muscle activity during sleep onset in older men may contribute to the greater prevalence of OSA in older men. The older men in our study had normal BMIs and did not have sleep apnea, but if they were predisposed to having sleep apnea because of truncal obesity or some other cause of a narrow or more compliant upper airway, the greater fall in upper airway muscle activity might have been critical and led to sleep apnea, whereas in a younger man with the same degree of upper airway functional narrowness, but a lesser fall in upper airway muscle activity during sleep onset, sleep apnea might not have resulted. Our data also support the hypothesis that the higher prevalence of periodic breathing in the elderly may be
Fig. 3. Expiratory V̇, upper airway resistance (UAR), and total inspiratory EMG activities for the Di, IC, GG, and TP at α-to-θ transitions of 9 older men compared with 9 younger men. EMG data are expressed as a percentage of average activity in −5 to −2 breaths for each transition. Only data from 5 breaths just before and from 5 breaths just after each transition are included. Each subject had to have at least 5 data points at a particular breath position for his data to be included at that position. Vertical lines mark EEG transitions from α to θ.
explained by respiratory instability associated with changes in state (23). As older men have greater changes in the activity of their respiratory muscles associated with state changes, fluctuations in state during the sleep onset period would produce greater fluctuations in $V_E$, predisposing to periodic breathing.

The difference between the fall in Di, GG, and TP activities in older and younger men would appear to be
a transient phenomenon because it was confined to the first five breaths of $\theta$, but no difference was found when the first 20 $\theta$ breaths were assessed. This finding is consistent with other studies (28) showing no difference in GG activity in stable sleep between young and older thin men given that their study did not specifically examine the first few breaths after transition. The lack of a difference in V\text{\textsubscript{E}} is also consistent with the finding (27) that the difference in V\text{\textsubscript{E}} between quiet wakefulness and established stable sleep was, the same in young men as older men, although V\text{\textsubscript{E}} was more variable in both sleep and wakefulness in the older men. It would thus appear that the direct sleep influence on both respiratory pump and upper airway muscles leading to an immediate fall in their activities is greater in older than younger men. These differences then disappear in a time frame consistent with the effects of reflexes to chemical and mechanical factors beginning to influence the respiratory muscles so that difference in activity of the muscles between the age groups is no longer apparent.

We deliberately chose to compare a group of middle-aged men with a young group rather than study an elderly group because other studies have indicated that sleep-related influences on respiratory variables change as they become elderly (17, 25). There was no relationship between age and sleep-disordered breathing in males over the age of 60 yr in two studies (30). It would thus appear that the direct sleep influence on both respiratory pump and upper airway muscles during sleep onset is greater in older than younger men. These differences may only last for 2 or 3 breaths, and the periods of $\alpha$-to-$\theta$ transitions some of the $-1$ $\alpha$ breaths will contain some $\theta$ activity and some of the $+1$ $\theta$ breaths will contain some $\alpha$ activity. This is the reason that the baseline chosen was $-5$ to $-2$ breaths rather than $-5$ to $-1$ breaths.

The postransition data were compared with the data from several pretransition breaths because there was some breath-to-breath variability during stable $\alpha$ activity and stable $\theta$ activity. This can be regarded as physiological noise. If the postransition breath data were compared with various individual pretransition breath data, the effect of a change in state on the relevant parameter could be over- or underestimated. To avoid this problem, it was decided to determine whether there was a significant change in the postransition breaths relative to the pretransition baseline defined above.

In summary, at transitions from $\alpha$ EEG activity to $\theta$ activity, there are significant falls in V\text{\textsubscript{E}} and the EMG activities for Di, IC, GG, and TP and a rise in UAR. The changes in V\text{\textsubscript{E}} and UAR and the activities of all the muscles were greater in normal older men than in normal younger men. At transitions from $\theta$-to-$\alpha$ activity, there was an increase in V\text{\textsubscript{E}}, a fall in UAR, and increases in the activities of Di, GG, and TP in phase 3. The changes in V\text{\textsubscript{E}}, UAR, GG, and TP were greater in the older than in the younger men. These differences may help explain the greater prevalence of sleep-disordered breathing with increasing age.

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