

## THE EFFECT OF VOLUNTARY BREATHING ON REACTION TIME

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**Abstract**—The effect of voluntary changes in inspiratory duration on reaction time was studied in 17 normal subjects. All received a visual feedback on inspiratory duration and successively adjusted this variable to two target values which were respectively higher and slightly lower than resting level. The order of the two tasks was counterbalanced across subjects. Reaction times to auditory stimuli were measured during a key-pressing task immediately after voluntary breathing. Voluntary control of inspiration induced concomitant changes in breathing period and minute ventilation. Per cent changes in these breathing variables and in reaction times under the two conditions were analysed. Correlations were significant for period not for minute ventilation. This effect was probably due to neuromuscular rather than humoral factors.

### INTRODUCTION

BECAUSE they provide a non-pharmacological treatment of many psychophysiological disorders such as hypertension or insomnia [1–4], cognitive-behavioural therapies have raised great hopes in both therapists and patients. These hopes seem to be partially fulfilled in current clinical practice, although considerable individual differences in clinical results are consistently observed. Despite a host of controversial issues, there is a nearly universal consensus concerning both these therapies and traditional practices such as yoga that one of the most critical forms of behavioural access to psychophysiological processes is voluntary breathing. As a general rule, patients are trained to adopt a slow and deep pattern of breathing, either just prior to the acute episodes of their disorder (panic attack, agoraphobia, asthma) or in a permanent fashion. Clinical studies [5, 6] assessing the efficacy of slow breathing have shown a decrease in the frequency of symptoms in subjects who practised this pattern of breathing. However, the interpretation of these findings has often been hindered by a lack of investigations into the physiological effects of slow breathing and by the combined use of breathing therapy with standard medications.

With a few notable exceptions in experimental research, the scientific basis for these practices has received little attention, and few data actually show that slow breathing has any influence on internal states. Typically, studies consisted of instructing normal subjects placed in an artificial stressful situation (e.g. threat of an electric shock) to breathe at a low frequency. Standard physiological indices of arousal (skin conductance, heart rate, finger pulse volume, blood pressure) were measured. Some of these physiological variables displayed significant changes after slow breathing, but as a rule, no consistent variations were observed. In one study [7] slow breathing reduced skin resistance and pulse volume, but not heart rate. In another study [8], consistent changes were seen for skin resistance but not for blood

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pressure or heart rate. In both studies, the authors concluded that breathing was effective for reducing arousal, a contention which may be considered doubtful or valid, according to how arousal is conceptualized and defined (see Refs [9–11]). But whatever the case, these results do not support the concept that slow breathing has a uniform physiological effect. They rather suggest that breathing may differentially affect cognitive, physiological and behavioural variables. Accordingly, each of these possible influences of breathing requires investigation.

In the present study, we focused on behavioural effects and investigated whether voluntary changes in breathing pattern may influence reaction time (*RT*). This variable has often been considered as a behavioural correlate of arousal [10–12], heightened levels of arousal being associated with shorter *RT*. If slow breathing reduces arousal, an increase in *RT* would ensue. However, this prediction may be hampered by two complicating factors which were carefully attended for in our experiment.

The first factor is that breathing patterns vary widely across individuals. This has been consistently demonstrated in the physiological literature [12]. Breathing pattern is usually characterized by the frequency of breathing cycles ( $f$ ), their duration ( $T_{TOT}$ ), the volume of inspired air (tidal volume,  $V_T$ ) and minute ventilation  $V$  (product of  $f$  by  $V_T$ ). Because of the large inter-individual differences in these variables, the absolute level of a given variable is often less informative than the difference between this level and an individual reference level [13, 14]. In the present context, the extent to which frequency must be changed to bring about behavioural effects is presumably highly specific for each individual, a fact which may explain the conflicting results of previous studies. Accordingly, per cent changes in breathing parameters can be expected to predict *RT* more accurately than absolute values.

The second factor is that subjects asked to change one breathing variable tend to change their whole breathing pattern [13, 14]. In this study, we attempted to determine the specific influence of various breathing variables (tidal volume, period, minute ventilation) by looking for correlations between these variables and *RTs*. Assuming that the influence of breathing on behaviour is predominantly mediated by the periodic activity of the neural structures involved in spontaneous or voluntary breathing, time parameters (frequency, periods) would be expected to play a leading role in this process and, accordingly, to display the strongest correlations with *RTs*. Conversely, if the effect of breathing is predominantly mediated by alveolar ventilation and arterial gases, the closest correlations would occur between minute ventilation and *RTs*.

## METHODS

### *Subjects*

Twenty-one healthy subjects volunteered for this experiment. All were undergraduate students, mainly of medicine. They were not informed of the purpose of the experiment. None was familiar with breathing experiments. Four subjects were unable to complete a normal session: one was interrupted by a technical failure, one requested complementary information during testing, and two did not follow instructions concerning the reaction time task. The remaining 17 subjects (9 males and 8 females, mean age  $21.8 \pm 4.4$  yr) each completed one experimental session.

### *Apparatus*

The experimental setup was composed of a heated Fleisch pneumotachograph (No. 2) with its pressure transducer (Schlumberger CH510510, No. 13, conditioner CA1065), an analog processing device built in the laboratory which detect transitions between inspiration and expiration and integrated the flow signal for the calculation of tidal volume ( $V_T$ ) and minute ventilation ( $V$ ), an analog-to-digital converter (Selia

PA300), a microcomputer (Olivetti M24), and a memory oscilloscope (Tektronix 5103N). An airtight facial mask attached to the pneumotachograph was then suspended at the appropriate height and fastened to the subject's head (no leakage was observed). The two inputs to the computer were  $V_T$  and a signal from a relay that shifted whenever the flow crossed zero. The computer used this binary signal to compute inspiratory and expiratory durations ( $T_I$  and  $T_E$ , respectively), and the total duration of each cycle ( $T_{TOT} = T_I + T_E$ ). The sampling frequency for this signal was about 200 Hz. Computer measures of time were provided by the analog-to-digital converter and were electronically validated with the memory oscilloscope. Volume was calibrated before each session in Ambient Temperature, Pressure, and Saturation (ATPS) conditions using a sinusoidal pump built in the laboratory (frequency: 12 l/min). All volume measurements were then converted into BTPS units (Body Temperature, Pressure and Saturation). The 50 msec auditory stimuli were generated at an easily audible volume by the computer. The intertrial period varied randomly from 2 to 22 sec. The reaction time task consisted of depressing one particular key of the computer keyboard placed near the right arm of the subject's chair. Visual feedback used in the controlled breathing task was displayed on the computer monitor. A horizontal segment moved up to a position corresponding to the duration of the preceding  $T_I$ s (terminal, continuous, and proportional feedback). Two horizontal lines (minimum and maximum values) represented the target. The distance between the two lines corresponded to an interval of 0.2 sec. The screen resolution allowed 200 vertical steps corresponding to four seconds. Computer programs were written in Basic and compiled by Quick Basic Compiler (Microsoft).

### Procedure

Subjects were tested individually. Each subject was seated in an armchair opposite the computer display with his/her right hand resting comfortably on a support. The subject was shown how to operate the key and instructed to press it as quickly as possible whenever a sound was delivered by the computer. Twenty practice trials were then run. Next, the subject was given standardized instructions. First, s/he was asked to read a text chosen by the experimenter for its neutral emotional impact. While the subject was reading, the experimenter monitored breathing signals on the oscilloscope and numerical values of  $T_I$ ,  $T_E$  and  $V_T$  on the computer display. After allowing a 10-min period for the subject to adapt the apparatus, 20 values were collected for the resting  $T_I$ . Each subject was informed of the significance of the respiratory visual feedback, and instructed to keep the moving segment inside the target delimited by the two horizontal lines. To avoid respiratory discomfort due to hyperventilation, the subjects were warned against any excessive increase in  $V_T$ . The session was then divided into two phases comprising a period of voluntary breathing followed by 20 *RT* trials. The two periods of voluntary breathing involved two different target  $T_I$  values which were calculated individually by multiplying the resting  $T_I$  by 0.8 or 2. These two targets are designated as  $T_I^-$  and  $T_I^+$ , respectively.  $T_I^-$  was therefore slightly shorter than the resting  $T_I$ .  $T_I^+$  was notably higher than the resting  $T_I$ . The order of the two targets was counter-balanced across the subjects. During the first 15 cycles of each breathing training period, a continuous visual feedback, represented by the moving segment, informed the subject of the duration of each inspiration. Then, feedback was provided every 15 sec until the end of the task (which lasted about 5 min). This intermittent feedback represented the mean value of the preceding  $T_I$ s since the last presentation of 15 sec earlier. The end of each breathing task was indicated on the monitor and 20 auditory stimuli were then delivered. Because the stimuli were delivered after the breathing task was over, the *RT*s to these stimuli reflected internal state modifications induced by breathing, not the degree of difficulty met in controlling  $T_I$ . Total duration of the session was about 50 min.

### Data reduction and analysis

Performance during the breathing tasks was scored by the conventional Absolute Error (AE) index:  $AE^+ = \Sigma |T_{i,k} - T_I^+| / N$ , and  $AE^- = \Sigma |T_{i,k} - T_I^-| / N$ , where  $T_{i,k}$  are the successive  $T_I$ s and  $N$  the number of cycles.  $AE^+$  and  $AE^-$  value were then expressed as a per cent of the corresponding individual target ( $T_I^+$ ,  $T_I^-$  respectively). The regularity of breathing was indexed by the coefficient of variation of the duration of the breathing cycles (standard deviation/mean value, noted *CV*) during a given training period. For each subject, *RT*s were averaged for each of the two series of 20 values which followed each of the two breathing tasks ( $T_I^+$  and  $T_I^-$ ). These two mean values were noted  $RT^+$  and  $RT^-$ . For each subject, the differences in *RT* measured after the  $T_I^+$  and  $T_I^-$  tasks were expressed as the per cent variation:  $\Delta RT = 100 \times \{RT^+ - RT^-\} / RT^-$ . The same formula and notation was applied to  $T_{TOT}$ ,  $T_I / T_{TOT}$ ,  $V_T$ ,  $V$ , and *CV*, yielding  $\Delta T_{TOT}$ ,  $\Delta VT$ ,  $\Delta V$ , and  $\Delta CV$  respectively. Statview and VAREDI (University René-Descartes) software provided descriptive statistics, product-moment correlations, and analyses of variance (ANOVAs).

## RESULTS

In all the subjects but one (CS, who met difficulties with the  $T_1^-$  task) AE values were under 25% during the breathing tasks (Table I); this denotes a good level of accuracy, as compared with previous studies [12, 13]. Mean AE were similar for the two breathing tasks.

TABLE I.—ABSOLUTE ERROR FOR  $T_1$  CONTROL AND REACTION TIMES (msec)

Subject	AE <sup>-</sup>	AE <sup>+</sup>	RT <sup>-</sup>	RT <sup>+</sup>
RI	24	19	338	306
CA	8	13	280	260
DE	10	12	306	334
BA	10	11	290	301
AI	17	18	236	236
GU	14	19	323	306
BO	7	13	314	288
GR	10	17	325	339
BE	15	15	266	275
EV	22	14	311	308
LA	7	21	309	308
DL	9	11	301	325
NI	9	13	266	278
EN	24	15	288	308
FR	17	12	308	313
DU	10	11	279	286
CS	37	23	307	287
<i>M</i>	14.70	15.12	296.8	297.5
<i>SD</i>	8.11	3.74	25.58	26.15

The two mean *RT* values (determined after each breathing task) were nearly equal, showing that there was no general tendency towards a decrease or increase in *RT* after an increase in  $T_1$ . This was confirmed by standard ANOVA. No significant effects of the target  $T_1$  ( $T_1^+$  versus  $T_1^-$ ) or of the order of the two tasks ( $T_1^+$  before  $T_1^-$ , or the reverse) on *RT* were observed. No trend was observed within each run of *RT* trials. This also suggests that learning, habituation, and fatigue were not influential factors, although an alternative hypothesis is that the effects of these factors cancelled each other out.

The subjects spontaneously modified  $T_{TOT}$ ,  $T_1/T_{TOT}$ ,  $V_T$ , and  $V$ , as a consequence of their voluntary change in  $T_1$  (Table II). As a rule, the subjects had lower  $V_T$  values and higher minute ventilation values during  $T_1^-$  than during  $T_1^+$ , which means that the decrease in frequency associated with long  $T_1$ s was not compensated for by a corresponding increase in  $V_T$ . The  $T_1/T_{TOT}$  ratio increased in all but two subjects (EV, who markedly hyperventilated and CS). These two subjects performed poorly on the  $T_1^-$  tasks (22 and 37%, respectively) and their particular patterns of performance yielded extremely high  $\Delta T_{TOT}$  values.

Figure 1 shows the relationships between  $\Delta T_{TOT}$ ,  $\Delta T_1/T_{TOT}$ ,  $\Delta V_T$ ,  $\Delta V$ , and  $\Delta RT$ . Below a threshold  $T_{TOT}$  increase (which was exceeded by EV and CS),  $\Delta RT$  and  $\Delta T_{TOT}$  were significantly correlated ( $r = 0.554$ ,  $p = 0.025$ ) as were  $\Delta RT$  and  $\Delta T_1/T_{TOT}$  ( $r = 0.583$ ,  $p = 0.019$ ). This latter result was due to the close or perfect

TABLE II.—VENTILATORY DATA\*

Subject	$T_1^-$	$T_1^+$	$T_{TOT}^-$	$T_{TOT}^+$	$CV^-$	$CV^+$	$T_1/T_{TOT}^-$	$T_1/T_{TOT}^+$	$V_T^-$	$V_T^+$	$V^-$	$V^+$
RI	1005	2513	3390	5432	17	20	0.296	0.463	627	901	11.10	9.95
CA	1372	3432	3197	6174	8	14	0.429	0.556	400	545	7.51	5.27
DE	886	2215	1756	4324	16	15	0.505	0.512	391	761	13.36	10.56
BA	1361	3403	2979	6682	13	9	0.457	0.509	681	1165	13.72	10.41
AI	1000	2502	2313	5120	20	19	0.432	0.489	447	860	11.60	10.08
GU	916	2292	2212	4437	17	22	0.414	0.517	450	961	12.21	12.99
BO	953	2384	2113	4325	11	11	0.451	0.551	539	833	15.31	11.57
GR	816	2042	1826	4384	13	16	0.447	0.466	317	609	10.42	8.33
BE	1512	3700	4014	7846	18	23	0.377	0.472	708	1212	10.58	9.27
EV	1016	2542	1971	6145	27	20	0.515	0.414	599	697	18.23	6.81
LA	1359	3398	2975	5345	10	14	0.457	0.636	513	743	10.35	8.34
DL	1423	3557	3387	7264	10	14	0.420	0.490	638	1285	11.30	10.61
NI	1260	3150	2698	5110	13	13	0.467	0.616	595	901	13.23	10.58
EN	1503	3700	3442	6690	26	17	0.437	0.553	959	1071	16.71	9.60
FR	893	2233	1943	4336	29	14	0.460	0.515	322	740	9.94	10.24
DU	1269	3174	2555	6050	23	13	0.497	0.525	637	898	14.94	8.88
CS	1513	3700	2382	7520	36	22	0.635	0.492	487	1166	12.24	9.30
<i>M</i>	1179	2937	2656	5717	18.05	16.23	0.453	0.516	547.6	902.8	12.51	9.58
<i>SD</i>	251.6	610.9	671	1191	7.81	4.12	0.069	0.055	161.1	216.1	2.69	1.77

\*Time values are given in msec,  $V_T$  are given in ml, and  $V$  in l/min.  $CV$  and  $T_1/T_{TOT}$  are dimensionless.

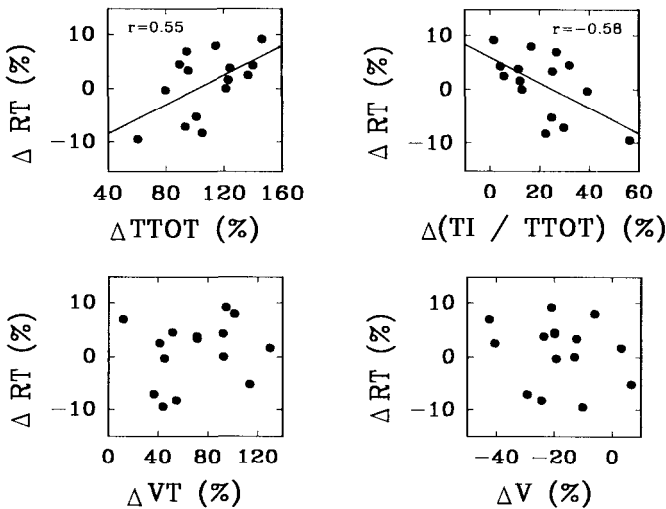


FIG. 1. Regression of individual changes in RT on changes in breathing variables. For clarity, two subjects are not represented: *EV* ( $\Delta T_{TOT} = 212$ ;  $\Delta T_1/T_{TOT} = -20$ ;  $\Delta V_T = 16$ ;  $\Delta V = -63$ ;  $\Delta RT = -1$ ) and *CS* ( $\Delta T_{TOT} = 216$ ;  $\Delta T_1/T_{TOT} = -22$ ;  $\Delta V_T = 139$ ;  $\Delta V = -24$ ;  $\Delta RT = -6$ ).

correlation between  $\Delta T_{TOT}$  and  $\Delta T_1/T_{TOT}$  ( $r = 0.99$ ), which is a mathematical consequence of the fact that individual targets, and therefore actual  $T_1$ s, were in the same ratio for all the subjects. For the same reason, the variance for  $\Delta T_1$  was very low and no correlations were found between  $\Delta RT$  and  $\Delta T_1$ . Contrary to  $\Delta T_{TOT}$  and  $\Delta T_1/T_{TOT}$ , neither  $\Delta V_T$  nor  $\Delta V$  was significantly correlated with  $\Delta RT$ . The fact that

significant correlations were observed for  $\Delta T_{\text{TOT}}$  (and  $\Delta T_1 / T_{\text{TOT}}$ ), but not  $T_{\text{TOT}}$  or  $T_1 / T_{\text{TOT}}$  confirms that the absolute levels of these variables do not predict  $RT$  as accurately as per cent changes.

Correlations between  $\Delta RT$  and  $\Delta CV$  and between  $\Delta RT$  and  $\Delta AE$  were not significant but direct observation of the data suggested that the subjects who exhibited longer  $RT$ s (i.e.  $\Delta RT \geq 0$ ) after practising the  $T_1^+$  task were in general those subjects who tended to adopt an irregular pattern of breathing, i.e. who had a negative  $\Delta CV$ . A nonparametric test (Exact Fisher test on the signs of  $\Delta RT$  and  $\Delta CV$ ) confirmed this observation ( $p < 0.042$ ).

#### DISCUSSION

The pattern of correlations suggests that increasing  $T_{\text{TOT}}$ , i.e. reducing breathing frequency, tends to decrease reaction time, but that other factors brought about by the change in frequency may not only influence performance, but even reverse the effect on performance of slow breathing. One of these parameters is breathing regularity, which may decrease whenever a subject is asked to adopt an unusual pattern of breathing, especially if this subject is untrained. As a rule, the subjects who did not increase  $RT$  after slow breathing (7 out of 17) also adopted an irregular pattern of breathing. Thus slow breathing may influence  $RT$ , provided that the subject is able to adopt a regular pattern easily. Longer training may possibly enable subjects to reduce breathing frequency without increasing variability, which would enhance the effect of slow breathing. When the new pattern of breathing departed exaggeratedly from the spontaneous one, as occurred in two of our subjects, no effect of breathing on  $RT$  occurred. Conversely, subjects who used a regular and slow pattern exhibit this effect after a surprisingly short period of voluntary breathing (less than five minutes for each exercise). This result sheds light on both the power and the limits of voluntary breathing effects on behaviour.

A number of neural or humoral causes may explain this effect of breathing. Since arterial  $\text{PCO}_2$  is involved in the production of slow EEG waves, and strongly affects the EEG spectrum [6], the influence of breathing may stem from changes in alveolar ventilation (see Ref. [15]). Alternatively, slow and regular breathing could bring about a pacing effect similar to those observed in many slow and periodic movements, whether active or passive, such as rocking. A conditioning hypothesis would be based on the fact that, as a general rule, low responsiveness is associated with slow and regular breathing. This pattern of breathing, even if it does not affect responsiveness in an unconditioned fashion, may act as a conditioned stimulus. Finally, psychophysiological effects of slow breathing may be due to its influence on other autonomous functions [16–19]. Our results are uninformative here, because of the lack of measurement of  $\text{PCO}_2$ , or EEG. Specifically designed experiments should be done to clarify the phenomenon observed here. However, contrary to  $T_{\text{TOT}}$  and  $T_1 / T_{\text{TOT}}$ , changes in  $V_T$  and  $V$  were not significantly correlated with  $RT$  changes. This suggests that  $\text{PCO}_2$  may be less crucial than breathing motor activity itself, at least in the initial stages of the process and provides some support to the neuromuscular pacing hypothesis, which assumes that changes in the level of responsiveness provoked by breathing would be assimilable to the pacing effect of many slow and periodic movements. Whether any motor activity would induce similar changes in  $RT$  or whether

these changes are specific to breathing cannot be deduced from our results. This issue would require further experiments.

It is worthwhile recalling that well before respiratory physiologists made extensive use of  $T_I/T_{TOT}$ , as an index of respiratory center activities,  $T_I/T_{TOT}$  or  $T_I/T_E$  were cited in psychological texts dating back to the beginning of this century as an index of attention [20], and were then progressively abandoned because of difficulties of interpretation. The negative correlation between  $\Delta T_I/T_{TOT}$  and  $\Delta RT$  observed here may perhaps contribute to reviving interest in this index, although its psychophysiological significance remains uncertain. From a neurophysiological point of view, analysing  $T_I/T_{TOT}$  in terms of respiratory centers would be irrelevant here, because during voluntary control, the oscillatory activity of the respiratory neurones is inhibited at the output stage of the bulbopontine centers. Moreover  $T_{TOT}$  and  $T_I/T_{TOT}$  are closely related in this experiment where  $T_I$  was imposed, and it is very difficult to determine whether their effects on behaviour are really underpinned by different processes.

In conclusion, this study lends some experimental support to the widespread belief that slow breathing may influence psychophysiological processes, and it points to two preconditions for this effect to occur: breathing needs to stay regular and should not depart too greatly from its resting pattern, at least in untrained subjects. The correlations observed here suggest that these changes are not predominantly mediated by changes in arterial gases, but rather are affected by neural influences associated with breathing activity. Finally, it must be stressed that breathing is a complex movement, and that any change in one of its kinematic components affects the whole pattern. An effect predicted from the voluntary control of a set of determined variables (usually the frequency, the tidal volume and thoraco-abdominal distribution) might be thwarted by the influence of the remaining variables.

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