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Respiratory Inhibition Induced by Transient Hypertension during Sleep in Unrestrained Cats

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The effects of transient blood pressure elevation, induced by intravenous injection of phenylephrine, were studied in drug-free, unrestrained cats during sleep and waking. Transient hypertension evoked an increase in respiratory cycle duration (T_{tot}), an effect which was most prominent during quiet sleep. Transient hypertension evoked no overall change in inspiratory duration (T_{di}) during any sleep–waking state, although reduction of diaphragmatic EMG amplitude was observed. Thus, the ratio of diaphragmatic activity time to total respiratory cycle duration (T_{di}/T_{tot}) was decreased following blood pressure elevation. Apneic episodes occasionally occurred, and these occurrences were more frequent during sleep states. Apneas induced during quiet sleep were often associated with transient or sustained arousal.

INTRODUCTION

Stimulation of arterial baroreceptors inhibits respiratory activity in anesthetized animal preparations (1, 12, 28, 29). Large increases in blood pressure (BP) can arrest respiration, a finding which led to early investigations of so-called "adrenaline apnea" evoked by intravenous pressor injection

Abbreviations. AW—quiet wakefulness, QS—quiet sleep, REM—rapid eye-movement, MAP—mean arterial pressure, BP—blood pressure, T_{tot}—interval between onsets of diaphragmatic activity, T_{di}—duration of diaphragmatic activity.

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(12). Baroreceptor stimulation affects respiratory cycle timing (9, 16), diaphragmatic activity (4), bronchial tone (12), and the activity of upper airway musculature (20). Transient BP elevation may thus exert a variety of influences on both ventilation and airway resistance.

Baroreflex influences on respiration in intact, unanesthetized animals are relatively unknown. Sleep states greatly affect respiratory patterns (17, 18, 22), airway resistance (19) and arterial baroreflex sensitivity (2, 27). Thus, the interaction between baroreflex activation and respiratory control may vary between different sleep and waking states. Such interactions are of considerable clinical interest, since transient increases in arterial pressure occur during state transitions (8) and during episodes of sleep apnea (25). The object of the present studies was to examine the influence of transient BP elevation on diaphragmatic activity during different sleep–waking states.

METHODS

Eight cats (six female, two male; 2.7 to 3.5 kg) were prepared for chronic recording of sensorimotor EEG, eye movements, EKG, and hippocampal and lateral geniculate activity under pentobarbital anesthesia (Nembutal sodium, Abbott Laboratories; initial dose of 30 mg/kg) as described elsewhere (27). Stainless-steel EMG electrodes were implanted in the crural diaphragm of four cats to monitor inspiratory-related EMG (26). Electrode leads were terminated at a 20-pin miniature connector fixed to the skull with dental acrylic. Arterial and venous catheters were implanted in the femoral vessels and advanced into the descending aorta and inferior vena cava. The vascular lines were passed subcutaneously and exited caudal to the electrode connector. After at least 1 week of recovery, the animals were adapted to sleeping in a shielded, dimly lit chamber, and recordings were initiated. Patency of the catheters was maintained by daily flushing with small volumes of heparinized Ringer's solution.

During recording, the electrode connector atop the cat's head was connected to the polygraph by a cable that allowed the animal to move freely within the recording chamber. Aortic pressure was monitored by connecting the arterial catheter to a transducer (Statham P23Aa) mounted outside the chamber. EMG signals were bandpass filtered to bandpass frequencies from 10 Hz to 1 kHz and passed through an analog signal processor (Service Associates SA 415) which calculated the root-mean-square (RMS) value of the signal amplitude. In all eight animals, respiratory movements of the chest wall were monitored using a piezoelectric strain gauge attached with a band around the lower thorax. All measurements were carried out while the cats spontaneously breathed room air. All physiologic signals and a time code were recorded both on polygraph paper

and on magnetic tape. Animals were observed via a closed circuit video system. Recording sessions lasted 2 to 5 h and took place between 1100 and 1700 h. Polygraph records were scored for sleep–waking states according to standard criteria, i.e., EEG, EOG, hippocampal rhythm, and "ponto-geniculo-occipital wave" activity (22). Three basic sleep–waking states were selected for comparison: quiet wakefulness (AW), quiet or slow-wave sleep (QS), and desynchronized or rapid eye-movement (REM) sleep.

Transient blood pressure (BP) elevation was induced during AW, QS, and REM periods by injecting a small bolus of phenylephrine-HCl through the venous catheter using a syringe pump (Neosynephrine; 10 μg/kg body weight; typical volume 0.3 ml). Resulting changes in BP were measured directly from the calibrated paper record. Mean arterial pressure (MAP) was calculated from measured systolic and diastolic values, according to the formula $MAP = P_{dia} + (P_{sys} - P_{dia})/3$. Measurements of the interval between onsets of diaphragmatic EMG (T_{tot}) and the duration of diaphragmatic activity (T_{di}) were derived from the paper records. Mean T_{tot} and T_{di} were calculated during each trial from four cycles prior to hypertension and from four cycles following onset of BP elevation.

Statistical comparisons of T_{tot} and T_{di} before and after transient BP elevation were based on analyses of variance (ANOVA). Comparisons between sleep–waking states were made using chi-square tests and analysis of variance [BMDP P3F and P2V; (5)]. Multiple linear regression analyses of respiratory variables versus blood pressure were carried out using the BMDP P1R program. The respiratory variables and blood pressure data were *normalized* between animals by calculating z scores according to the formula $z = (x - \mu)/s$, where x is the individual sample value, μ is the mean of all samples for a given set of trials (i.e., runs conducted during AW, QS, and REM sleep), and s is the standard deviation of the mean (14). Using the z-score transformation, a z value of zero indicates the mean value of all samples within a given set of trials. Thus, breath-to-breath variations in respiratory variables could be compared both across sleep–waking states and before and after the hypertensive stimulus.

RESULTS

In intact, unanesthetized cats, phenylephrine injection resulted in transient BP elevation ranging from 15 to 54 mm Hg. In all cases, BP elevation resulted in a significant ($P < 0.01$) slowing of respiration (increased T_{tot}) during each sleep–waking state (Figs. 1, 3; Table 1). This increase in T_{tot} was most pronounced during QS ($P < 0.05$). No significant influence of BP elevation on T_{di} was noted during any sleep–waking state (Figs. 1, 4; Table 2). However, the amplitude of diaphragmatic EMG was decreased, as shown

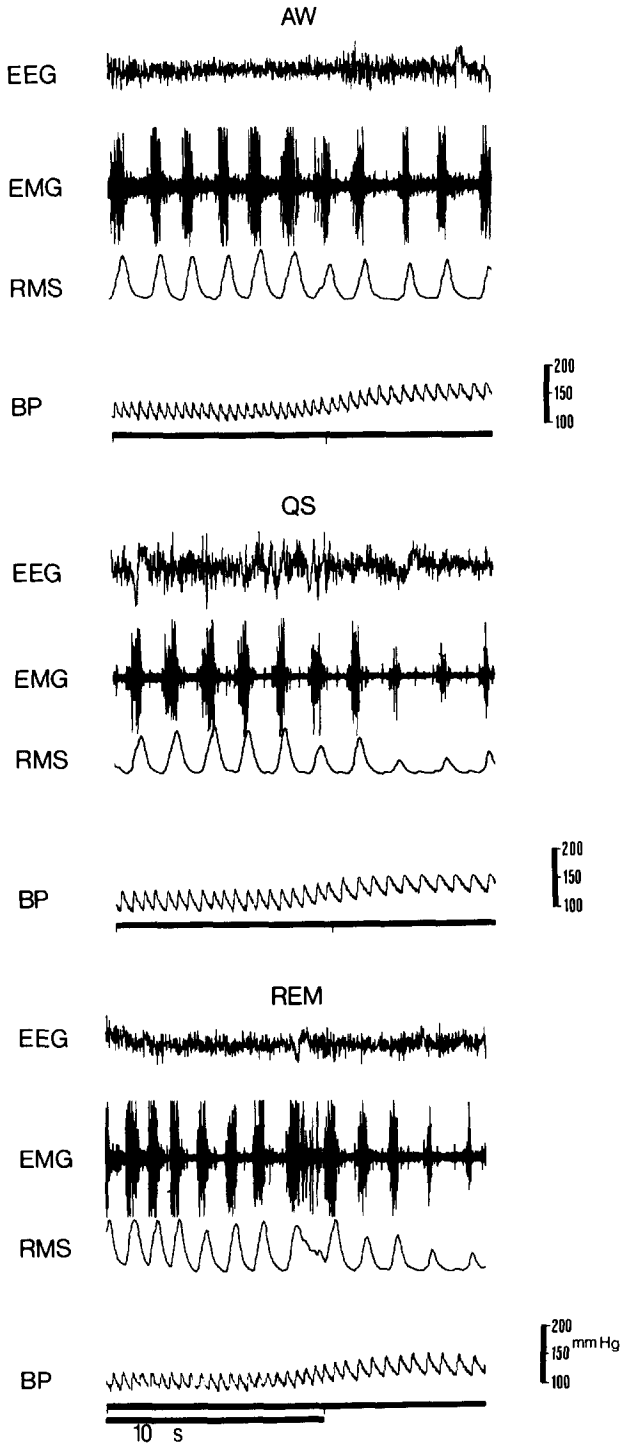
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TABLE 1

Mean Respiratory Interval (T_{tot}) prior to and after Hypertension Induced during Sleep and Waking in Eight Cats

	AW[b]		QS		REM sleep	
Cat	Pre	Post	Pre	Post	Pre	Post
1	1652	1954	1807	2062	1281	1521
	173	68	200	175	246	334
2	878	996	2061	2414	1908	2319
	250	175	300	311	330	401
3	1071	1283	1614	2771	2104	3562
	267	302	663	1286	727	1337
4	1804	2229	2135	2517	1805	2531
	240	169	233	367	329	590
5[a]	1850	2504	1963	2577	1750	2381
	302	545	166	328	140	558
6[a]	1805	2273	2417	4650	1761	3394
	296	351	234	1500	361	1148
7[a]	2230	2910	2695	3410	2009	2492
	644	270	326	320	455	610
8[a]	2346	3185	2783	3500	1726	3361
	570	692	264	310	413	1152

[a] Cats in which diaphragmatic EMG was recorded. Mean values are for four breaths prior to and four breaths after the onset of hypertension, with a minimum of four repetitions for each state in each cat. Values are $\bar{x} \pm$ SD ms.

[b] Abbreviations: AW—awake, QS—quiet sleep, REM—rapid eye-movement.

by a transient decline in the peak amplitude of the RMS EMG output (Fig. 1). The ratio of diaphragmatic activation time to total respiratory cycle length (T_{di}/T_{tot}) was significantly decreased ($P < 0.05$) after BP elevation during each sleep–waking state (Fig. 5; Table 3). This decrease in T_{di}/T_{tot}

FIG. 1. Respiratory effects of transient blood pressure (BP) elevation in an unanesthetized cat during quiet wakefulness (AW), quiet sleep (QS), and active sleep (REM). Phenylephrine was the pressor agent, and the onset of the pressure rise occurred 5 to 7 s after the start of the i.v. injection. Note the transient slowing of respiration for two breaths and the diminished amplitude of diaphragmatic EMG traces and RMS traces following BP elevation. Abbreviations: EEG—electroencephalogram, EMG—diaphragmatic electromyogram, RMS—root-mean-square of diaphragmatic EMG potentials.

TABLE 2

Duration of Diaphragmatic EMG Discharge prior to and after Blood Pressure Elevation during Sleep and Waking States in Four Cats[a]

	AW		QS		REM Sleep	
Cat	Pre	Post	Pre	Post	Pre	Post
5	720	608	775	676	671	600
	110	26	65	47	78	56
6	642	653	845	730	516	456
	79	149	93	79	88	73
7	624	470	714	572	729	542
	42	63	50	32	46	87
8	1094	954	1165	1077	917	881
	149	91	206	151	119	249

[a] Data collection and values as in Table 1.

was comparable in all sleep–waking states (Fig. 5). The influence of elevated BP on T_{tot} and T_{di}/T_{tot} persisted for several respiratory cycles following the onset of hypertension (Figs. 3, 5).

The z-score data for T_{tot} and MAP (Figs. 3, 6) also demonstrated state-related differences in prestimulation measurements. Consistent with previous observations (2, 3, 8, 17, 18) T_{tot} was greater during QS than during AW

TABLE 3

Duty Cycle: T_{di}/T_{tot}[a]

	AW		QS		REM Sleep	
Cat	Pre	Post	Pre	Post	Pre	Post
5	0.39	0.24	0.39	0.26	0.38	0.25
	0.03	0.02	0.03	0.03	0.03	0.02
6	0.36	0.29	0.35	0.16	0.29	0.13
	0.02	0.03	0.03	0.02	0.04	0.03
7	0.28	0.16	0.26	0.17	0.36	0.22
	0.02	0.02	0.03	0.02	0.04	0.03
8	0.47	0.30	0.42	0.31	0.53	0.26
	0.04	0.04	0.03	0.04	0.04	0.03

[a] Data collection and values as in Table 1.

and REM sleep, and MAP was lower during sleep states than during wakefulness.

Hypertensive stimulation occasionally resulted in periods of apnea [apneas defined as $T_{tot} > 2 \times$ mean prestimulation T_{tot} for each trial; (9)] interspersed among more normal cycles (Fig. 2). Apneas occurred more frequently during sleep states (both QS and REM) than during wakefulness (log-linear chi-square test, BMDP 3F: $G = 16.31$, df = 2; $P < 0.001$; Table 4).

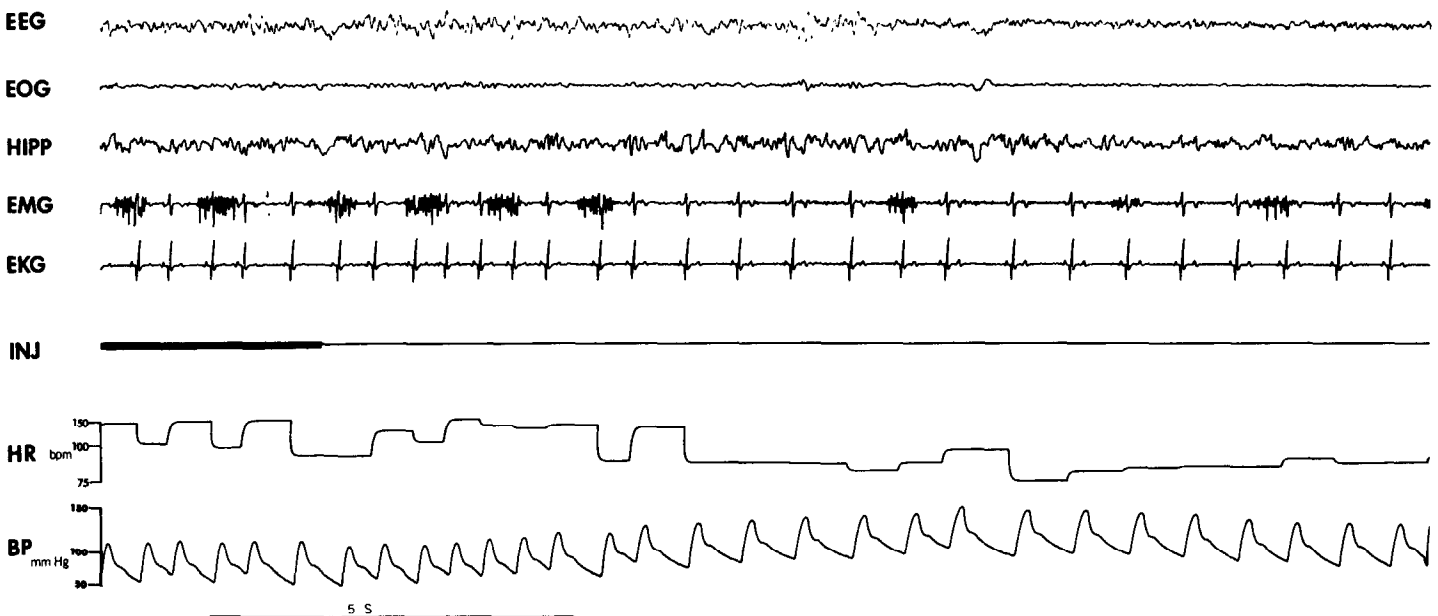
Prestimulation BP was lower during sleep than during waking (BMDP 2V: $F = 14.84$, df = 2; $P < 0.01$). After pressor injection, however, the increase in BP and peak pressures obtained did not differ between sleep and waking states (BMDP 2V; $F = 3.28$, df = 2, $P > 0.06$). In order to assess overall progression of T_{tot}, T_{di}, T_{di}/T_{tot}, and MAP changes on a breath-to-breath basis, z-score conversions were carried out using the overall means for each breath in each state for cats 5 through 8. Breath-by-breath z-score distributions for T_{tot}, T_{di}, T_{di}/T_{tot}, and MAP are shown in Figs. 3–6. Linear regression of respiratory variables with MAP revealed a strong correlation between T_{tot} and MAP ($r = 0.612$, df = 1, $P < 0.001$), a strong negative correlation between T_{di}/T_{tot} and MAP ($r = -0.922$, df = 1, $P < 0.001$), and no correlation between T_{di} and MAP ($r = -0.309$, df = 1, $P > 0.08$).

As noted in a previous study (27), pressor stimulation during quiet sleep occasionally resulted in transient or sustained arousal. Such arousals occurred frequently (but not exclusively) in association with apneic episodes. There was no difference between T_{di}, T_{tot}, and T_{di}/T_{tot} values for normal quiet sleep trials and those which resulted in arousal (Figs. 3–5). No arousals were noted after pressor stimulation during REM sleep.

DISCUSSION

In the intact, drug-free cat, BP increases resulted in a prolongation of the respiratory cycle, a decrease in the ratio of the duration of diaphragmatic activity to total respiratory cycle duration (T_{di}/T_{tot}), and a decrease in the amplitude of diaphragmatic EMG activity. Elevation of BP produced qualitatively similar effects on respiratory activity in each sleep–waking state. However, marked quantitative differences were observed between states. These effects can be divided into two basic components: (i) an effect on respiratory cycle timing, and (ii) an effect on diaphragmatic activation (EMG amplitude).

Acute BP elevation has long been known to evoke changes in respiratory patterns and upper airway muscle activity in acute, anesthetized preparations (12). This effect has been shown to depend on baroreflex mechanisms rather than other reflexes, e.g., alterations in chemoreflex responses due to changes in perfusion of the carotid bodies (1, 12, 29).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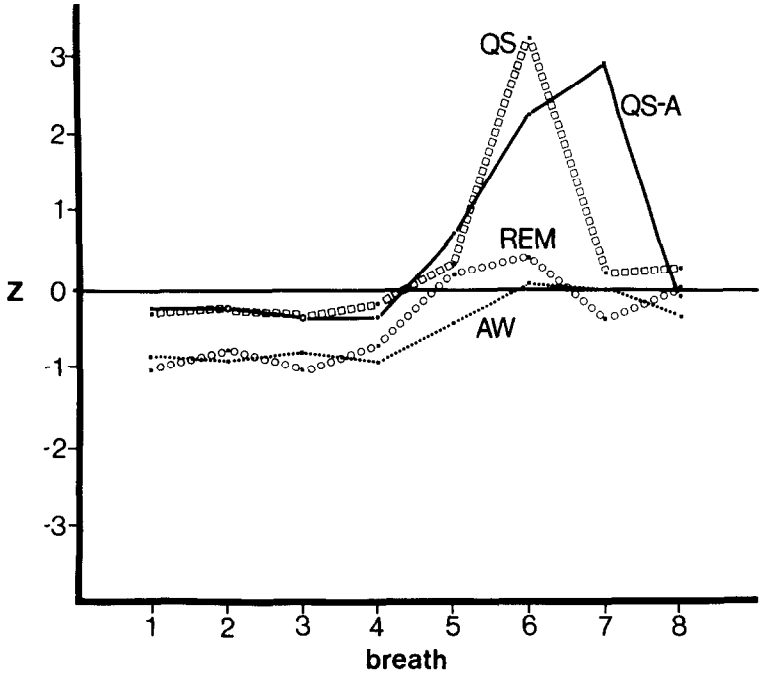

FIG. 3. Overall distribution of T_{tot} values on a breath-by-breath basis. Onset of blood pressure elevation occurred during breath 4. QS-A—arousal from quiet sleep, REM—rapid eye-movement sleep.

Our findings of increased total cycle duration in the drug-free cat are similar to those described by others for anesthetized preparations (9, 12, 16, 20). Therefore, we conclude that a fundamental effect of elevated arterial pressure on the generation of the respiratory rhythm involves a delay in inspiratory onset (i.e., lengthening of T_{tot}). Grunstein *et al.* (9) reported an increase in inspiratory duration following hypertension which, they suggested, produced a modulation of the Hering–Breuer reflex. Although we did not measure inspiratory flow in the present study, we found no lengthening of T_{di} with BP elevation in unanesthetized animals. Similarly, Nishino and Honda (16) and our own observations in anesthetized animals (28) indicate that inspiratory duration is either reduced or unaffected by transient hypertension.

FIG. 2. Apneic period induced by transient hypertension during QS in an unanesthetized cat. These episodes were much more frequent during sleep states. Abbreviations: EOG—eye movement, HIPP—hippocampal EEG, INJ—phenylephrine injection time, HR—heart rate.

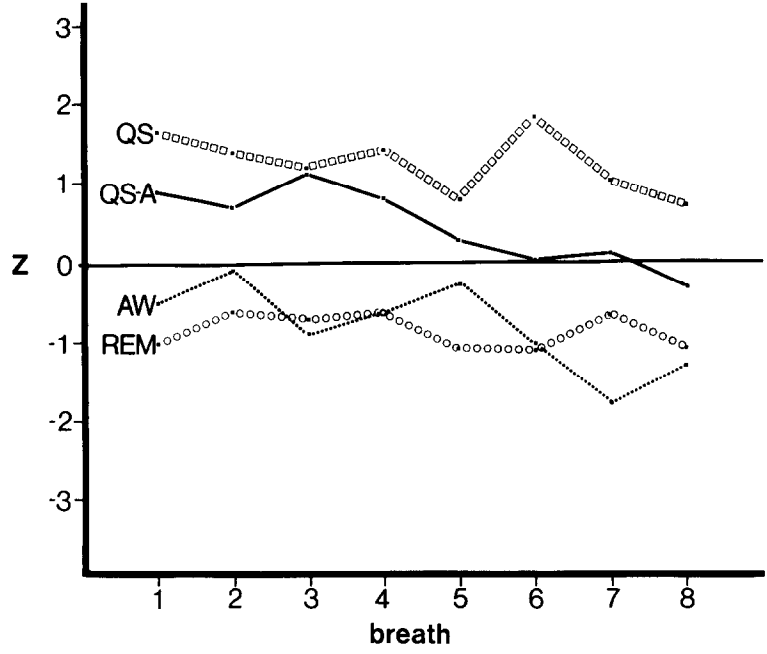

FIG. 4. Overall distribution of T_{di} values on a breath-by-breath basis. Onset of blood pressure elevation occurred during breath 4. Abbreviations and line symbols are the same as in Fig. 3.

Transient hypertension evokes inhibition of activity in upper airway (20) and neck extensor muscles (24). Siegel (24) found that BP plays a major role in regulating atonia mediated by pontomedullary stimulation and suggested that baroreceptor function may be critical to mechanisms producing REM sleep atonia. Thus, the reduction in diaphragmatic EMG may reflect a generalized suppression of motor activity.

Although diaphragmatic EMG suppression and alterations in respiratory cycle timing could be treated as separate phenomena, we suggest that these effects may be closely interrelated. Sears *et al.* (21) propose that enhanced tonic drive to inspiratory and expiratory neurons results in phasic respiratory patterning. If tonic activity is reduced, respiratory rhythm slows until rhythmic activity ceases. Inhibition of tonic activity may thus lead to respiratory slowing. The extent of tonic respiratory activity varies with sleep–waking state (21, 23). Therefore, an interaction between BP effects on respiration and sleep–waking state could be expected.

The effect of BP elevation on T_{tot} and T_{di}/T_{tot} was most pronounced during QS. Although this could result from state-related differences in the magnitude of BP elevation, no such differences were found. Arousal occurs

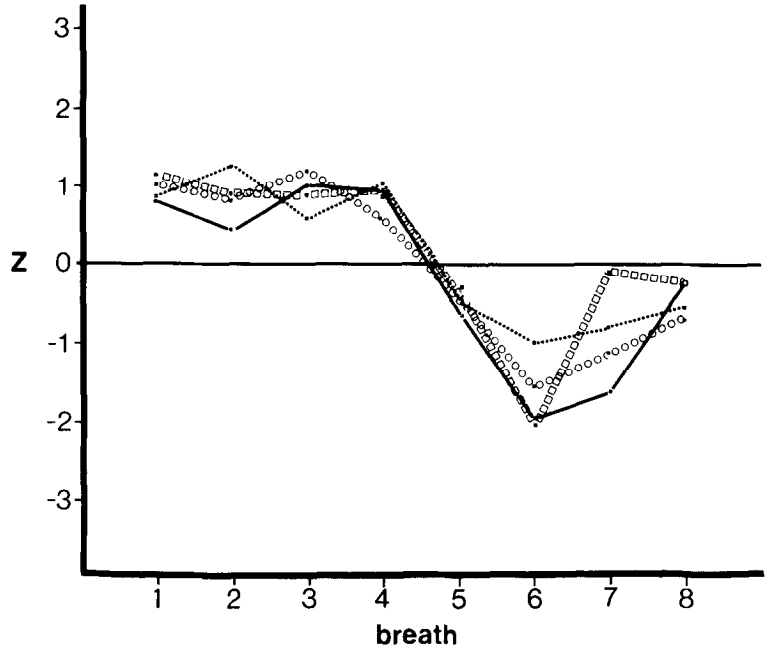

FIG. 5. Overall distribution of duty cycle (T_{di}/T_{tot}) values on a breath-by-breath basis. Onset of BP elevation occurred during breath 4. Abbreviations and line symbols are the same as in Fig. 3.

with BP elevation (3, 6, 27) and is associated with respiratory pattern changes (6, 15). Thus the observed BP effects on respiratory patterns could result from arousal. However, comparison of data from QS periods demonstrated no difference in BP between trials that terminated in arousal and those that did not.

As noted above, no arousals were observed to follow BP elevation during REM sleep. However, it should be emphasized that no attempt was made to determine possible between-state differences in the "thresholds" for arousal. Thus, although arousals associated with hypertension were observed only during QS, it is possible that they could also be induced by hypertensive stimuli during REM sleep, but at a higher threshold. We also did not systematically assess the possibility that hypertension induces arousals more readily at different times during a sleep period (e.g., earlier versus later during QS periods). In this regard, there may also be a difference in arousal thresholds between tonic versus phasic REM periods.

Although BP elevation appears to directly influence pontomedullary mechanisms controlling respiratory cycle timing, a possible role of the forebrain cannot be excluded. REM sleep abolishes the increases in BP and

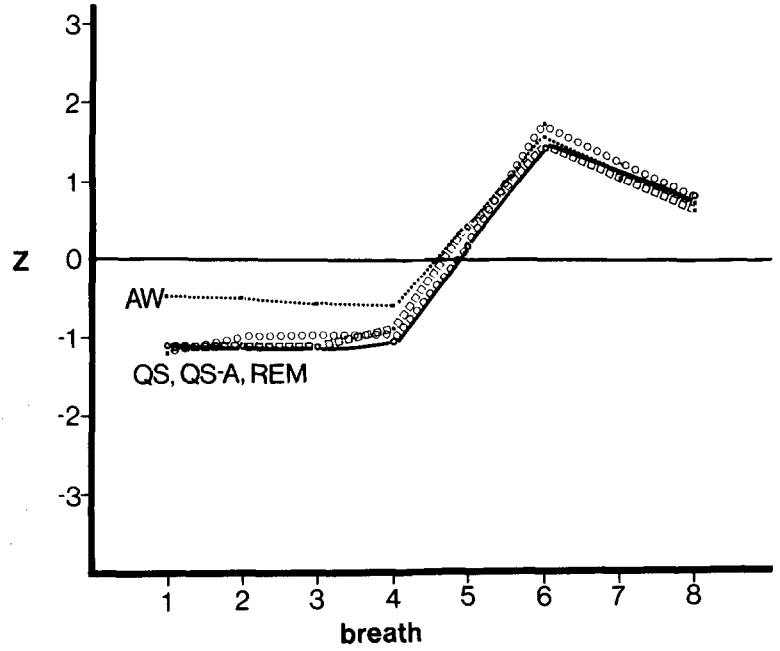

FIG. 6. Overall distribution of mean arterial pressure (MAP) values on a breath-by-breath basis. Onset of blood pressure elevation occurred during breath 4. Abbreviations and line symbols are the same as in Fig. 3.

the changes in respiratory activity evoked by forebrain stimulation (7, 10). Thus, we might expect that BP elevation effects would be greatly reduced in REM sleep; however, the most prominent alterations in respiratory

TABLE 4

Frequency of Occurrence of Apneas after Hypertension Induced during Sleep and Waking States[a]

Cat	AW	QS	REM sleep
1	0/8	0/8	0/4
2	0/6	2/9	0/6
3	0/4	4/9	1/6
4	0/10	2/12	0/4
5	0/4	2/8	0/4
6	0/5	8/12	5/9
7	0/6	6/14	2/8
8	1/6	5/12	3/5

[a] The values are the number of trials producing apneas/total number of trials. This table includes QS trials that terminated in behavioral arousals.

patterns were found in QS, suggesting that the forebrain is less involved in modulating the state response.

The particular respiratory patterns observed to follow rapid elevation of BP have a variety of consequences for cardiorespiratory control. Because vagal activity is "gated" or inhibited by inspiration (11, 13, 27) the lengthening of expiratory time allows a longer period of vagal efferent activity, and thus allows enhanced cardiac slowing. A decrease in negative intrathoracic pressure associated with inspiratory inhibition would result in a decrease in venous return to the heart, contributing to the return of normal BP. A balance between ventilation and perfusion would also be favored by the reduction in cardiac preload and the correction of the increased cardiac afterload.

Transient hypertension may also interact with pathologic mechanisms in sleep apnea syndromes. In addition to phrenic nerve-mediated changes in diaphragmatic activity, BP disturbances can affect upper airway muscle activity through cranial nerve efferent fibers (20). Sleep apnea syndromes are accompanied by a rapid rise in BP (25). In the case of central sleep apnea, BP elevation could exacerbate the inhibition and the pause in respiration. Inhibition of the diaphragm after BP elevation in obstructive apnea, however, could ameliorate the condition by reducing the negative thoracic pressure that contributes to the collapse of the upper airway.

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