

Reversal of apnoea by aspiration reflex in anaesthetized cats

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ABSTRACT: Various flow and timing characteristics of breathing as well as electrocorticographic (ECoG) records were analysed in a model of reversible respiratory failure induced by N₂ inhalation in 27 anaesthetized cats. During the first minute of respiratory arrest, nasopharyngeal stimulation by an elastic nylon fibre elicited a typical sniff- or gasp-like aspiration reflex (in 88.8% of cases), whilst similar tracheo-bronchial irritation evoked a weak cough reaction in one third of cases (34.2%). The aspiration reflex could also be evoked in progressive stages of apnoea characterized by very low and even isoelectric ECoG activity immediately before imminent irreversible respiratory failure, and it could interrupt and replace the periodic gasping sometimes occurring during apnoea. The reflex, comprising powerful inspiration efforts, could be evoked during apnoea many times in succession. Repeated aspiration reflexes alone resulted in recovery from hypoxic apnoea with gradual normalization of ECoG and subsequent restitution of spontaneous breathing even more frequently than it occurred in periodic gasping. Successful resuscitation from hypoxic apnoea by the aspiration reflex in cats suggests that nasopharyngeal stimulation can affect the mechanisms underlying the failure and restitution of breathing at least in some forms of apnoea.

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Spontaneous recovery from apnoea is supposed to be unsuccessful in several pathological processes such as sudden infant death syndrome (SIDS) and central apnoeas of various types [1-5]. Therefore, there have been efforts to study the failure of central control mechanisms of breathing in experimental models. In awake mice, both newborn and adult, hypoxic apnoea induced by inhalation of an anoxic mixture (97% N₂ + 3% CO₂) is usually interrupted by gasping resulting in recovery (autoresuscitation) [6]. In a model of reversible respiratory failure induced by N₂ inhalation [7] the anaesthetized cats mostly die unless artificial ventilation (AV) is applied after 40-60 s of apnoea.

In cats and some other mammals there is a sniff- or gasp-like aspiration reflex comprising powerful inspiratory efforts [8-12]. The reflex elicited by mechanical stimulation of the nasopharynx can interrupt various types of apnoea [13] and it persists even in very deep stages of general anaesthesia, when cough and other respiratory reflexes have already disappeared [14].

Cough without previous arousal could not be elicited by airways' stimulation during rapid eye movement (REM) and slow wave sleep in dogs [15] and it failed to increase in parallel with the ventilation during longer-lasting hypoxia in awake cats [16], indicating functional differences between the central mechanisms of eupnoeic respiration and cough [10, 15]. In spite of its great

significance, the occurrence of the aspiration and cough reflexes has not been studied systematically during hypoxic apnoea.

The aim of our study was to investigate in anaesthetized cats 1) whether the aspiration and cough reflexes can be elicited by airways' stimulation during the respiratory arrest induced by N₂ inhalation; 2) whether repeated induction of the aspiration reflex alone (without AV), may reverse the hypoxic apnoea and protect the anaesthetized cats from death, as occurs in awake mice with gasping developing in hypoxic respiratory arrest accompanied by hypercapnia; and 3) how the provocation of the aspiration reflex resulting in recovery from apnoea will be reflected in electrocorticogram (ECoG) and other polygraphic recordings.

Methods

The experiments were performed in 27 adult cats (1.9-3.6 kg) anaesthetized with sodium pentobarbitone (Pentobarbital Spofa 40 mg·kg⁻¹, given intraperitoneally). Supplementary doses of anaesthetic were given when required. A tracheal catheter was introduced to allow spontaneous breathing of either room air or pure N₂ from a cylinder, or to apply AV when needed. A wide ventrolateral pharyngostomy was performed to

allow mechanical stimulation of the nasopharynx, using a 0.3 mm diameter elastic nylon fibre, and to elicit the aspiration reflex [8]. The tracheobronchial mucosa was stimulated with a similar nylon fibre fixed in a small hole made in the tracheal catheter in order to induce cough.

Three series of experiments were performed to study the changes in various respiratory and neurophysiological parameters. In the first series, the tracheal catheter was connected to a Fleisch pneumotachograph (LVK-11, Medicor) for the measurement of airflow (\dot{V}) in 12 cats. The end-tidal pressure of O_2 and/or CO_2 in the respired air (PET_{O_2} and PET_{CO_2}) was occasionally measured by an oxymeter and capnograph (Datex). The electrocardiogram (ECG) and systemic blood pressure (BP) in the femoral artery were also monitored simultaneously in some experiments. The variables were recorded on a 6-channel polygraph (Chirastar 60). The air-flow signal was also simultaneously recorded on a magnetic tape (EAM 350 Tesla). After digitalization at a frequency of 100 Hz the flow signal was evaluated by microcomputers using a program in Basic and statistical analysis (Statgraphics). A total of 25 variables comprising various flow, volume and timing parameters as well as some quotients calculated from them, were obtained for each inspiro-expiratory cycle [7].

After control recordings, the cats were made to inhale pure N_2 from a bottle (2 l) filled from a cylinder at a constant flow ($3 \text{ l}\cdot\text{min}^{-1}$) until respiration stopped. After 10 s of apnoea the bottle was disconnected and access to room air was arranged. During respiratory arrest the elicibility of the aspiration and cough reflexes was tested using 1–3 mechanical probing of the nasopharyngeal or tracheobronchial region, performed under visual control in random order. These irritations lasted 1–5 s and were repeated at intervals of approximately 10–15 s and the immediate reactions were evaluated. When several nasopharyngeal stimulations failed to evoke the aspiration reflex during apnoea and clear-cut mydriasis indicating that an imminent brainstem paralysis had developed, a mandatory type of AV with air was induced (Bird, Mark 7). After three spontaneous breaths, the cat was weaned from the respirator and spontaneous breathing lasting 20 min was allowed for recovery.

In a second series of seven cats, additional experiments were performed to measure airflow using the same experimental techniques described above. The aim of investigations was to test whether the aspiration reflex alone could increase cough reactivity and whether it could change or interrupt the periodic gasping occurring sometimes during apnoea. The incidence of

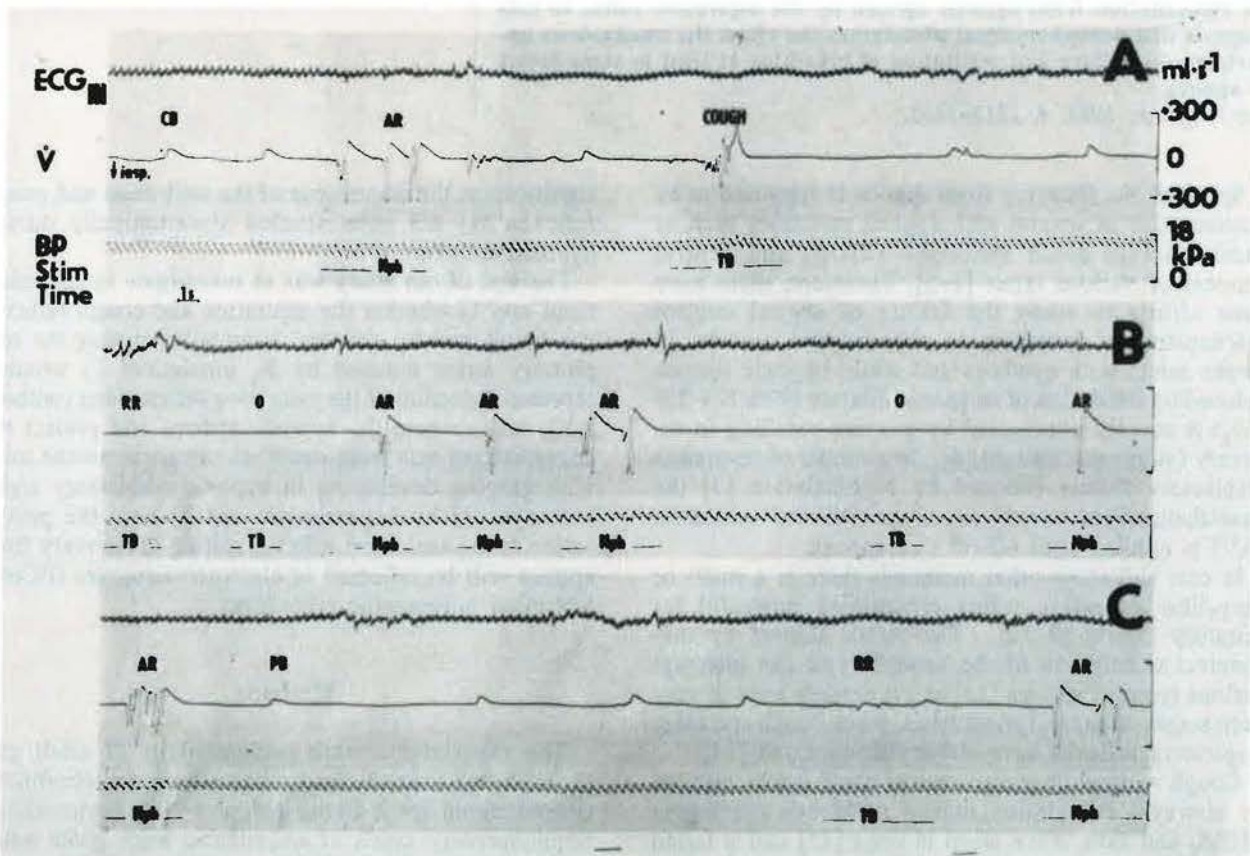


Fig. 1. — Recording of electrocardiogram (ECG III), airflow (\dot{V}) and blood pressure (BP) in control conditions (panel A), and after 2 min (panel B), and 3.7 min (panel C) from the beginning of hypoxic apnoea. Nasopharyngeal stimulation (NPh) elicits constantly throughout apnoea a typical aspiration reflex (AR) sometimes followed by a provoked breath (PB in panel C), whilst tracheobronchial irritation (TB) evokes only a small reflex respiratory change (RR), or no reaction (0) when compared to the control breath (CB) and control reactions (AR and Cough) in panel A.

spontaneous gasping, leading in our conditions to recovery, was also investigated.

In the third series of experiments in eight anaesthetized cats, the electromyogram (EMG) of an inspiratory intercostal muscle (Th 5–6) and four leads of standard unipolar ECoG were recorded in addition to the pneumotachogram, capnogram and ECG on an 8-channel polygraph (Bioscript, RFT). This series was intended to investigate mainly the changes in ECoG and the occurrence of gasping during more advanced stages of hypoxic apnoea. Therefore, no tracheobronchial stimulation was tested and resuscitation using nasopharyngeal stimulation or AV was induced only after an expressive decrease in the frequency of ECoG (delta waves) accompanied by clear-cut mydriasis, indicating incipient brainstem paralysis, i.e. usually after 50–60 s of apnoea. A low-resistance inspiro-expiratory valve, promoting the immediate switch to fresh air or to AV with O₂, facilitated a prompt and effective resuscitation in all cases.

Statistical analysis was performed using Student's t-test and the Chi-squared test. The results of experiments are given as mean±SEM.

Results

Airways' reflexes during hypoxic apnoea

Figure 1 illustrates an experiment in which reactions to airways' stimulation were recorded in control conditions (panel A) and during N₂-induced apnoea (panels B and C). During respiratory arrest the aspiration reflex was well preserved, whilst the reactivity to tracheobronchial stimulation in this case was very poor (a reflex change in respiration or no reaction at all). The elicibility of the airways' reflexes was tested altogether in 46 apnoeic attacks in 12 cats (first series of experiments). Tab. 1 compares the number of typical positive and negative reactions to nasopharyngeal and tracheobronchial stimulations, respectively, performed in four successive attacks in the same cats. The results indicate that the ability to respond does not differ significantly (Chi-squared, $p>0.05$) in the early and subsequent attacks for both the tracheobronchial and nasopharyngeal stimulations. The results from all 46 apnoeic attacks taken together indicate that the number of fully elicited aspiration reflexes (90%) from 220 nasopharyngeal stimulations is significantly higher ($p<0.001$) than the number of weak cough reactions (46%) evoked by 126 tracheobronchial irritations.

Figure 2 illustrates the time course of changes in ability to respond given as the percentage of positive reactions (fractional value) and grouped separately for the consecutive 30 s intervals during the respiratory arrest. The results indicate that while nasopharyngeal stimulations usually evoked the aspiration reflex in the first 30 s of apnoea, (in 89.8% of tests), tracheobronchial irritations induced the cough reaction in only 28.1% of attempts. Also during the whole first minute of respiratory arrest, the aspiration reflex was present fairly

constantly (in 88.8% of stimulations). The percentage of positive reactions increased exponentially ($R=0.986$) to reach the calculated 100% reactivity in 150 s. On the contrary, cough reaction during the first minute was induced in only 34.2% of tests ($p<0.001$) and its percentage values during the prolongation of apnoea increased linearly ($R=0.998$) with the calculated 100% reactivity in 223.5 s.

Table 1. – The numbers of positive (+) and negative (-) reactions to 220 nasopharyngeal (NPh) and 126 tracheobronchial (TB) stimulations in four successive apnoeic attacks (I–IV), respectively

Apnoea Stimulation		I	II	III	IV	Sum
NPh	+	64	43	45	46	198
	-	3	7	7	5	22
	Sum	67	50	52	51	220
TB	+	17	11	18	12	58
	-	20	17	18	13	68
	Sum	37	28	36	25	126

The results from 46 attacks in 12 cats indicate no significant differences in the ability to respond in early and subsequent attacks (NPh: $\chi^2 = 3.8467$; $p>0.05$ and TB: $\chi^2 = 0.7802$, $p>0.05$).

The results from the first series of experiments indicate that tracheobronchial stimulation resulted in weak but positive cough reactions less frequently when tested before the aspiration reflex (6 out of 38 trials) compared to 13 positive reactions to 35 stimulations applied after the reflex (Chi squared = 4.315, $p<0.05$). However, the latency of the response to tracheobronchial stimulation applied after the aspiration reflex was increased (47.7 ± 2.9 s) compared to the latency for probing before the reflex (25.8 ± 5.8 s, $p<0.01$). Figure 3 clearly demonstrates that a series of aspiration reflexes elicited by frequent nasopharyngeal irritations lasting 17 s can change the negative response to tracheobronchial stimulation to a weak cough reaction.

A computerized analysis of various parameters obtained from pneumotachographic records of 12 cough reactions and 42 typical aspiration reflexes evoked during hypoxic apnoea enabled their comparison with the controls (fig. 4). In the cough induced during apnoea there is a very marked decrease in respiratory parameters such as peak expiratory flow (PEF), expiratory volume/expiratory duration ratio (V_{TE}/T_E), tidal volume (V_T) and minute ventilation (V_E) and prolongation of T_E and time to PEF (TPEF) without significant changes in their inspiratory counterparts. These results clearly demonstrate an apparent inhibition of the expiratory effort in the cough reaction elicited during respiratory arrest.

Compared to the controls, the aspiration reflex induced during apnoea is characterized by both a smaller decrease in peak inspiratory flow (PIF), and the ratio of inspiratory tidal volume to inspiratory duration V_{Ti}/T_i and an prolongation in T_E . But the insignificant

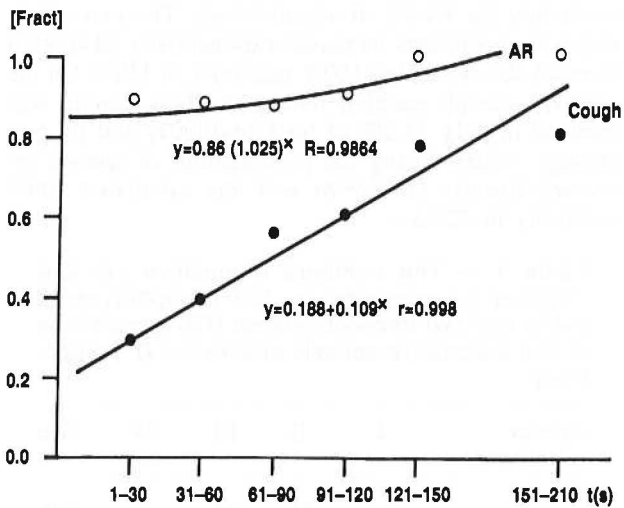


Fig. 2. - Elicitability of the aspiration (AR) and cough reflexes (Cough) indicated as fractional values of positive reactions to 220 nasopharyngeal and 126 tracheobronchial stimulations averaged for consecutive 30 s intervals in the course of hypoxic apnoea in 12 cats.

changes in both T_I and the time to PIF (TPIF) and in frequency (F) as well as in tidal volume (V_T) and \dot{V}_E suggest preserved ability for fast and yet strong active inspiratory efforts.

The aspiration reflex, gasping and recovery from apnoea

Repeated provocation of the aspiration reflex alone (without AV) managed to resuscitate cats from 36 out of 46 apnoeic attacks (78.3%) in the first series of experiments. In about 8% of cases when the usual nasopharyngeal stimulation with 0.3 mm diameter elastic nylon fibre failed to elicit the aspiration reflex repeatedly, stronger irritation by a catheter usually provoked at first a solitary reflex gasp and later resulted in the aspiration reflex with subsequent resuscitation (fig. 3).

In 10 out of 46 apnoeic attacks (21.7%) induced by hypoxia the aspiration reflex could not be provoked by repeated nasopharyngeal stimulations, and extensive mydriasis indicating development of an imminent brainstem paralysis. In four of these cases, AV induced

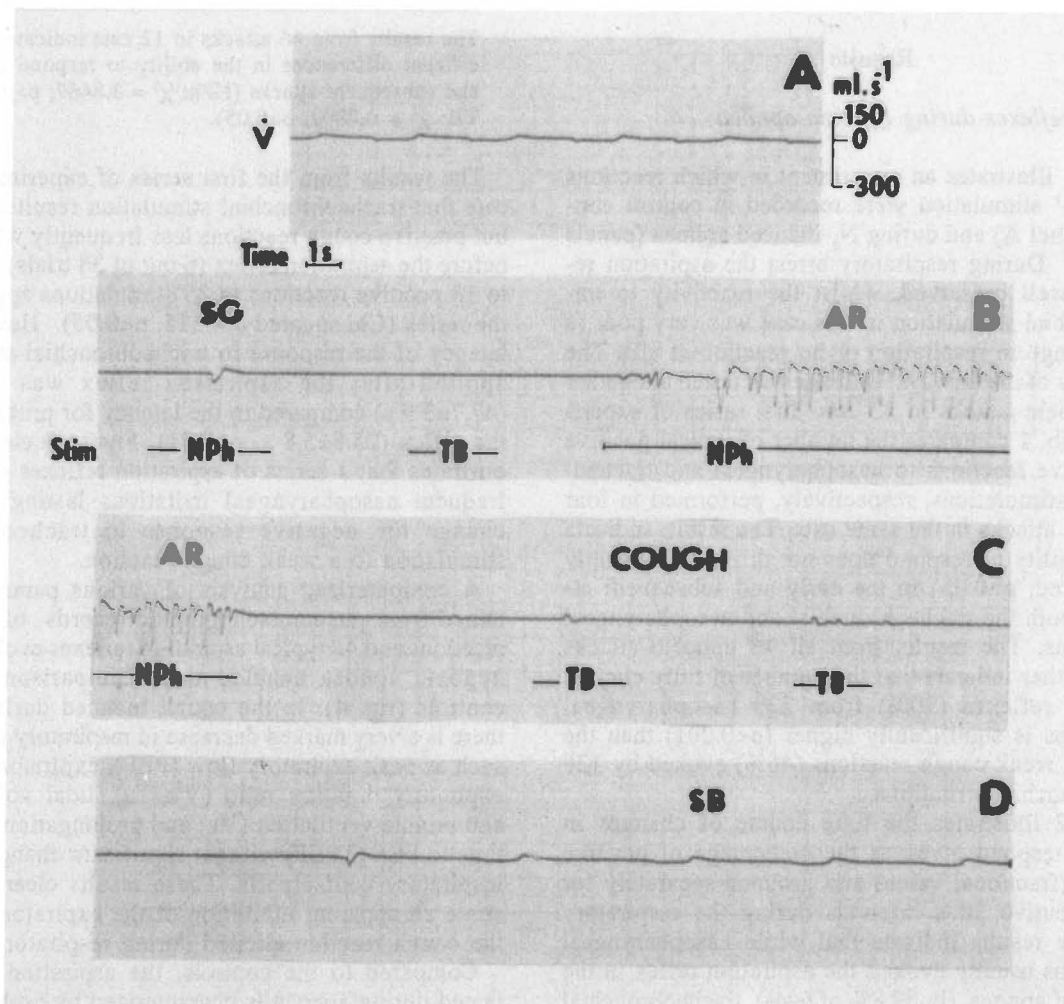


Fig. 3. - Changes in airflow (\dot{V}) during various respiratory reactions to nasopharyngeal (NPh) and tracheobronchial (TB) stimulations during hypoxic apnoea (panels B, C and D are continuous records) compared to control breathing (panel A). SG: solitary gasp; AR: series of aspiration reflexes; Cough: small cough reactions to TB stimulation after the AR but no reaction before the AR; SB: spontaneous breathing.

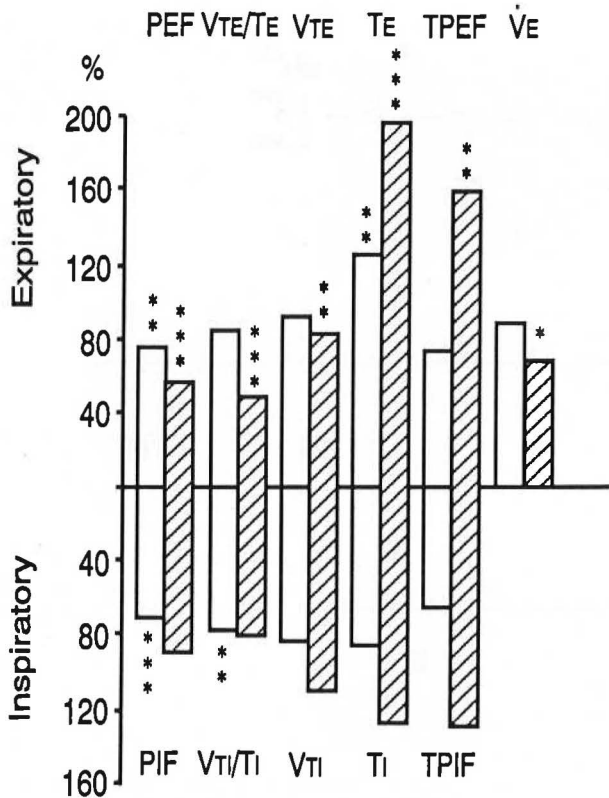


Fig. 4. - Inspiratory and expiratory values of selected flow, volume and timing parameters in 42 aspiration (open columns) and 12 cough reflexes (hatched columns) elicited during hypoxic apnoea compared to controls taken as 100%. PIF: peak inspiratory flow; PEF: peak expiratory flow; V_{Ti} and V_{Te} : tidal volume measured during inspiration and expiration respectively, T_i and T_e : time of inspiration and expiration respectively, V_{Ti}/T_i and V_{Te}/T_e : mean inspiratory and expiratory flow respectively, TPIF: and TPEF: time to reach PIF and PEF respectively, VE: minute ventilation. Significant differences compared to controls: *: $p < 0.05$; **: $p < 0.01$; ***: $p < 0.001$.

after 60.0 ± 5.8 s of apnoea resulted in recovery. There was no recovery in the remaining six attacks, where after longer-lasting unsuccessful provocations of the aspiration reflex, AV was induced significantly later (90.0 ± 10.3 s from the beginning of apnoea ($p < 0.05$)).

In our three series of experiments there was a variable occurrence of gasping depending on the conditions. No periodic gasping occurred in 36 cases of the first series successfully resuscitated from earlier stages of hypoxic apnoea by repeated provocation of the aspiration reflex, because this prevented the development of gasping. Except for a few solitary gasps no periodic gasping appeared even in six fatal cases of the first series where the start of AV was postponed to 90 s of apnoea.

Additional experiments performed in seven cats of the second series gave some further information on both gasping and its relationship to the aspiration reflex. In apnoeic attacks, where the nasopharyngeal stimulations and few solitary gasps were too weak to resuscitate the cats, AV was induced at the onset of extreme mydriasis

(92.8 ± 7.3 s from the beginning of apnoea). This usually resulted in recovery, and periodic gasping occurred in 53.8% of cases. Figure 5 demonstrates a case where weaning from the respirator after these gasps superimposed on AV allowed periodic gasping to develop fully. These gasps occurring in a regular rhythm could be transiently interrupted and replaced, especially to the end of paroxysm, by a series of aspiration reflexes or reflex gasps elicited by frequently repeated nasopharyngeal stimulations. After reappearance the gasping had a lower frequency and it continued until spontaneous breathing started. At the stage of periodic gasping there was no cough reaction to tracheobronchial stimulation.

In order to assess further the rate of successful auto-resuscitation by spontaneous gasping in our model using the same animals, an additional N_2 inhalation was performed in six cats of the second series at the end of the experiment. The cats were left without any treatment for 10–15 min before and after the inhalation. Spontaneous recovery by gasping was observed in only one case (16.7%). Similar results were also obtained in the third series of 40 experiments in eight cats devoted to the analysis of the ECoG changes and occurrence of gasping. Here, the progressive stages of apnoea with very marked mydriasis and delta waves were allowed to develop before any resuscitation using the aspiration reflex and/or AV was induced. Table 2 summarizing the results of 40 experiments in the third series indicates that there was always a complete recovery from such progressive stages of hypoxic apnoea. This was realized by spontaneous periodic gasping in 20% of cases, by the aspiration reflex in 27.5% and by the aspiration reflex supplementing ineffective solitary gasps in 7.5%. On another 45% of occasions, the flattening of ECoG activity and marked mydriasis required the induction of AV to save the animals.

ECoG manifestation of the aspiration reflex

Figure 6 A illustrates the development of a reversible apnoeic attack induced by hypoxia where the ECoG, pneumotachogram, capnogram and intercostal EMG were simultaneously recorded. After 55 s from the start of N_2 inhalation, EMG and airflow disappeared and $PETCO_2$ decreased to zero demonstrating the onset of apnoea. At this moment, there was still a marked ECoG activity which later gradually decreased during apnoea. Figure 6 B shows continuation of the same apnoeic attack where after 55 s of respiratory arrest the ECoG even became isoelectrical, and in spite of this nasopharyngeal stimulation evoked a typical aspiration reflex. This was manifested in short bursts of powerful inspiratory EMG activity as well as in typical inspiro-expiratory airflow changes resembling a gasp reaction. The reflex was often followed by both large reticular discharges and a subsequent increase in ECoG activity resulting in the restitution of spontaneous breathing later on. Similar changes in ECoG including reticular discharges also occurred after periodic gasps (table 2).

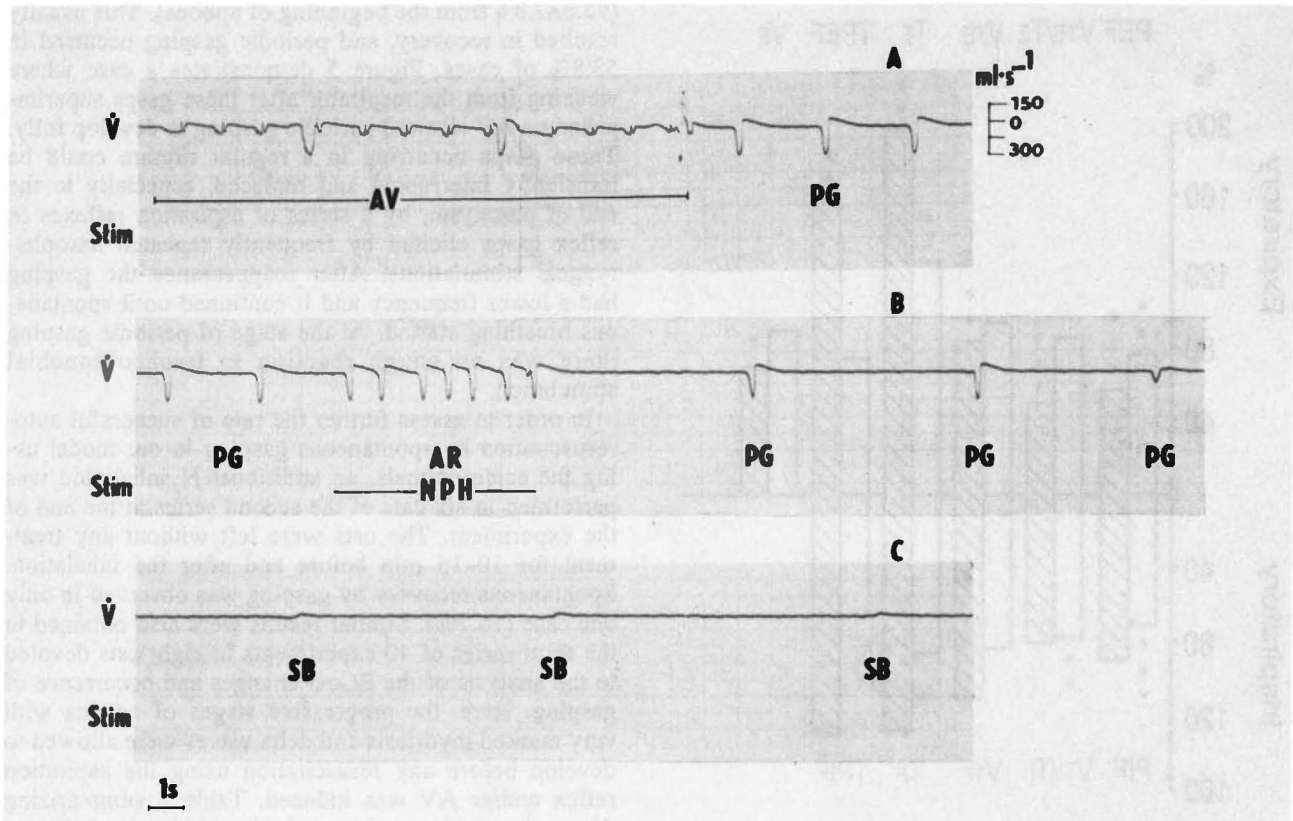


Fig. 5. — Changes in airflow (\dot{V}) during artificial ventilation (AV), consecutive periodic gasping (PG), aspiration reflex (AR) elicited by nasopharyngeal stimulation (NPh) and spontaneous breaths (SB) in an anaesthetized cat resuscitated from progressive hypoxic apnoea. Panels A, B and C are continuous records. Note the transient interruption and replacement of the periodic gasping by the aspiration reflex.

Table 2. — Recovery from progressive hypoxic apnoea to the first spontaneous breath (SB) by aspiration reflex (AR), gasping (G) and artificial ventilation (AV) in various combinations

	Presence (+) or absence (-) of AR and G in combinations		Cases		ECoG changes induced by		Type of recovery	Duration of apnoea to	
			n	%	G	AR		AV start s	first SB s
1	G -	AR +	11	27.5	0	+	AR → SB		106±13.5
2	G -	AR +	2	5	0	±	AR → AV → SB	87.5±3.5	134±18
3	G -	AR -	10	25	0	0	AV → SB	72.1±10.5	116.1±24.2
4	G +		8	20	+		PG → SB		103.5±13.5
5	G +		5	12.5	0		G → AV → SB	71.2±6.7	115±12.5
6	G +	AR +	3	7.5	0	+	G+AR → SB		114±6.7
7	G +	AR +	1	2.5	0	0	G+AR → AV → SB	66	93

Results from 40 experiments in 8 anaesthetized cats. PG: periodic gasping appearing after 47.1±10.9 s of apnoea. G+: presence of solitary, small or ineffective gasps (without autoresuscitation or changes in ECoG); ECoG: electrocorticogram.

Evaluation of ECoG records (segments of 25 s duration) before and after 11 successful nasopharyngeal stimulations in the course of severe apnoeic attacks in eight cats revealed arousal and some other effects of the aspiration reflex. The reflex elicited 53.2±1.6 s from the beginning of apnoea resulted first in an insignificant increase of the mean ECoG frequency

from 1.39±0.8 Hz to 1.53±0.6 Hz ($p>0.05$). Approximately 15 s later there was a marked rise in frequency to 3.6±0.3 Hz ($p<0.05$). Repeated inductions of the aspiration reflex during the next 50 s resulted in ECoG normalization followed by the restitution of spontaneous breathing at the level of ECoG activity about 4.8 Hz.

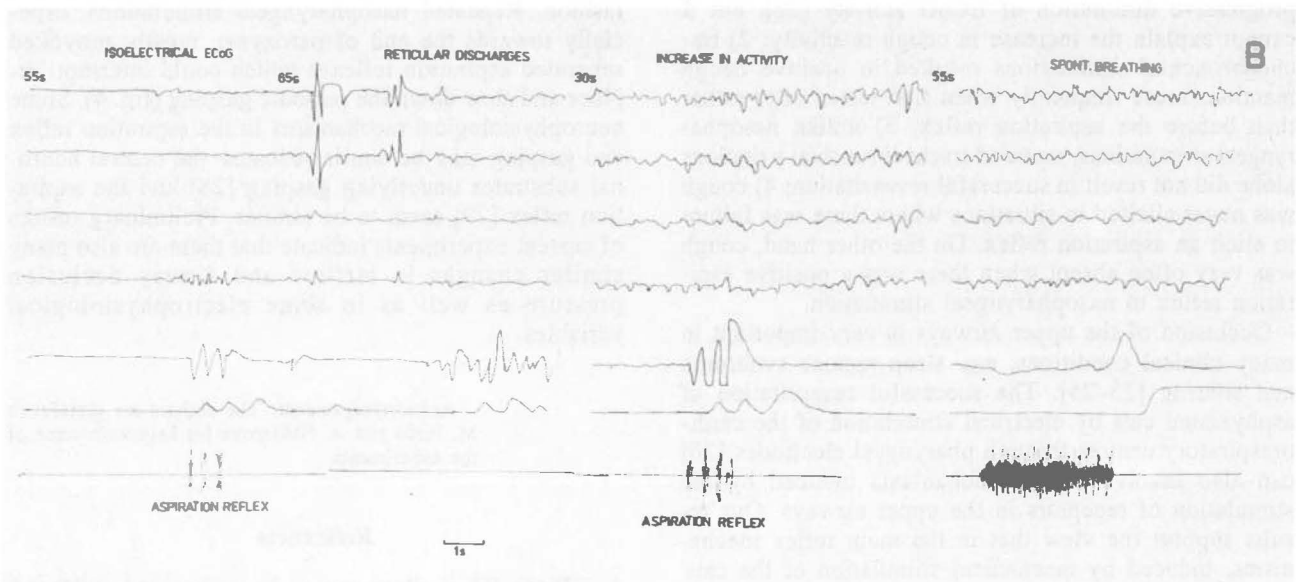
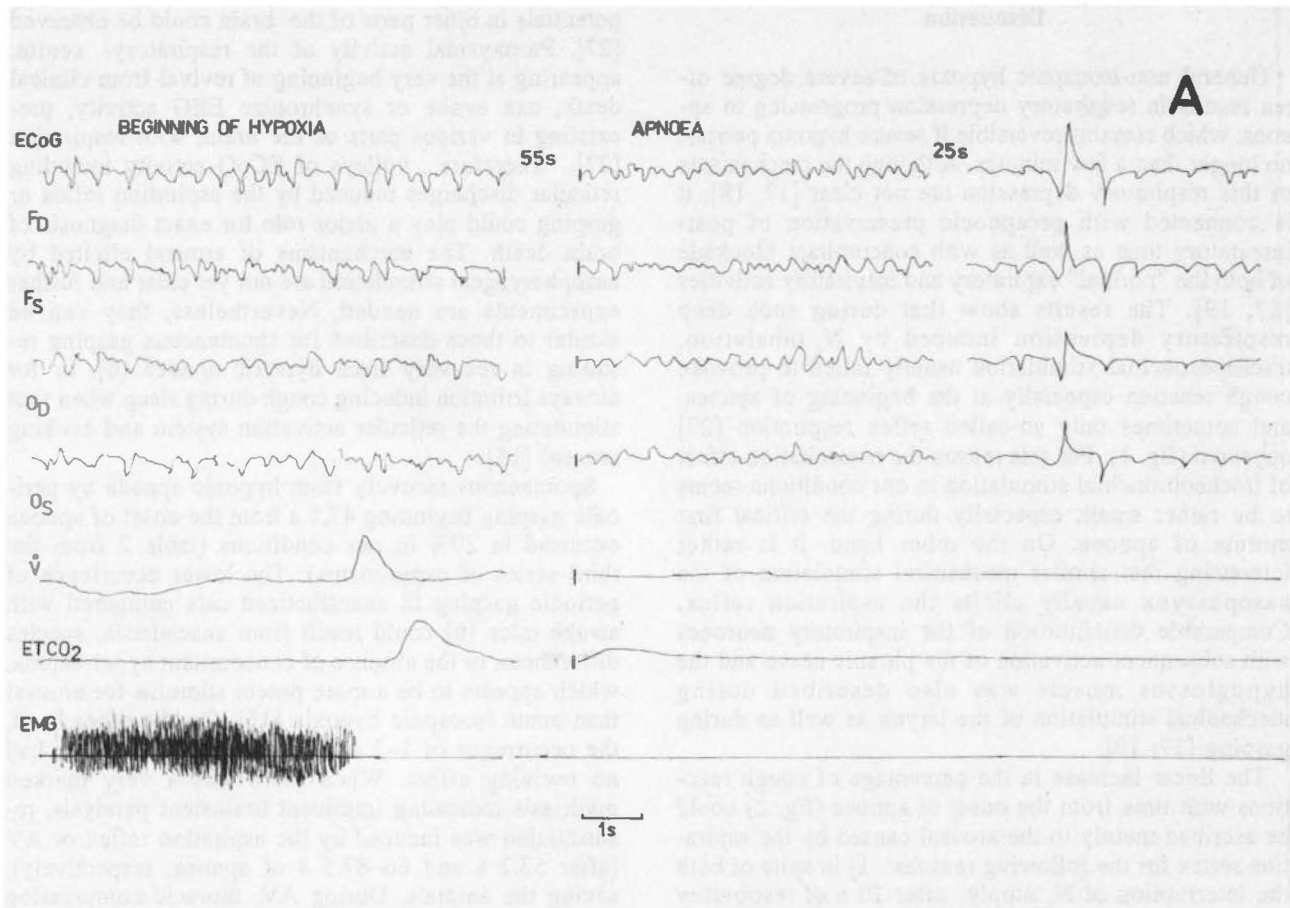


Fig. 6. - Changes in unipolar electrocorticograms (ECoG), during reversible hypoxic apnoea. FD: right frontal; FS: left frontal; OD: right occipital; OS: left occipital; \dot{V} : airflow; ETco₂: end-tidal CO₂; EMG: intercostal electromyogram. The time of particular phases are in seconds, indicated above the first channel. Panel A: the beginning of hypoxic ventilation (one inspiro-expiratory cycle on the left side), onset of apnoea 55 s after the start of hypoxia (middle part) and continuation of apnoea with large reticular discharges 25 s after the beginning of apnoea (right side). Panel B: reversal by the aspiration reflex of respiratory arrest with isoelectric ECoG resulting in reticular discharges (after 65 s of apnoea) and increase in ECoG activity (30 s after repeated aspiration reflexes), and spontaneous breathing (following further aspiration reflexes 55 s later). The ECoG, \dot{V} , ETco₂ and EMG traces are original records overtraced to increase contrast.

Discussion

General non-isocapnic hypoxia of severe degree often results in respiratory depression progressing to apnoea, which remains reversible if severe hypoxia persists no longer than a few minutes. Although the mechanisms of this respiratory depression are not clear [17, 18], it is connected with preapnoeic preservation of post-inspiratory tone as well as with concomitant blockade of both the "normal" expiratory and inspiratory activities [17, 19]. The results show that during such deep respiratory depression induced by N₂ inhalation, tracheobronchial stimulation usually failed to provoke cough reaction especially at the beginning of apnoea, and sometimes only so-called reflex respiration [20] appeared (fig. 1). For this reason the resuscitation effect of tracheobronchial stimulation in our conditions seems to be rather small, especially during the critical first minute of apnoea. On the other hand, it is rather interesting that similar mechanical stimulation of the nasopharynx usually elicits the aspiration reflex. Comparable disinhibition of the inspiratory neurones with subsequent activation of the phrenic nerve and the hypoglossus muscle was also described during mechanical stimulation of the larynx as well as during gasping [17, 19].

The linear increase in the percentage of cough reactions with time from the onset of apnoea (fig. 2) could be ascribed mainly to the arousal caused by the aspiration reflex for the following reasons: 1) in spite of both the interruption of N₂ supply after 10 s of respiratory arrest and the access of O₂ from air by diffusion, the arterial oxygen tension (Pao₂) slowly decreased further during the proceeding apnoea [7, 21]. This resulted in progressive diminution of ECoG activity [22], but it cannot explain the increase in cough reactivity; 2) tracheobronchial stimulations resulted in positive cough reactions more frequently when first tested after rather than before the aspiration reflex; 3) unlike nasopharyngeal stimulations, repeated tracheobronchial irritations alone did not result in successful resuscitation; 4) cough was never elicited in situations where there was failure to elicit an aspiration reflex. On the other hand, cough was very often absent when there was a positive aspiration reflex to nasopharyngeal stimulation.

Occlusion of the upper airways is very important in many clinical conditions, e.g. sleep apnoea syndrome and snoring [23–25]. The successful resuscitation of asphyxiated cats by electrical stimulation of the cardiorespiratory centres through pharyngeal electrodes [26] can also involve reflex mechanisms induced by the stimulation of receptors in the upper airways. Our results support the view that in the main reflex mechanisms, induced by mechanical stimulation of the cats' nasopharynx and provoking both the aspiration reflex and signs of arousal (increase in ECoG frequency and reticular discharges), may have some implications for apnoea of various types.

Action potential volleys can be recorded in the medullary respiratory centres during the advanced stages of clinical death, when no sign of ventilation or action

potentials in other parts of the brain could be observed [27]. Paroxysmal activity of the respiratory centre, appearing at the very beginning of revival from clinical death, can evoke or synchronize EEG activity, pre-existing in various parts of the brain, with respiration [27]. Therefore, volleys of ECoG activity including reticular discharges induced by the aspiration reflex or gasping could play a major role for exact diagnosis of brain death. The mechanisms of arousal elicited by nasopharyngeal stimulation are not yet clear and further experiments are needed. Nevertheless, they can be similar to those described for spontaneous gasping resulting in recovery from hypoxic apnoea [6], or for airways irritation inducing cough during sleep when first stimulating the reticular activation system and evoking arousal [15].

Spontaneous recovery from hypoxic apnoea by periodic gasping beginning 47.1 s from the onset of apnoea occurred in 20% in our conditions (table 2 from the third series of experiments). The lower occurrence of periodic gasping in anaesthetized cats compared with awake mice [6] could result from anaesthesia, species differences, or the absence of concomitant hypercapnia, which appears to be a more potent stimulus for arousal than acute isocapnic hypoxia [15]. On the other hand, the occurrence of 1–3 small and ineffective gasps had no reviving effect. When there was a very marked mydriasis indicating imminent brainstem paralysis, resuscitation was induced by the aspiration reflex or AV (after 53.2 s and 66–87.5 s of apnoea, respectively), saving the animals. During AV, thoracic compression synchronized with the inflation phase of the pump or supplementary nasopharyngeal stimulation seemed to facilitate the onset of paroxysmal gasping in a reflex fashion. Repeated nasopharyngeal stimulations, especially towards the end of paroxysm, mostly provoked separated aspiration reflexes which could interrupt, replace and slow down the periodic gasping (fig. 4). Some neurophysiological mechanisms in the aspiration reflex and gasping may be similar because the central neuronal substrates underlying gasping [28] and the aspiration reflex [29] seem to be similar. Preliminary results of current experiments indicate that there are also many similar changes in airflow and airway occlusion pressure as well as in some electrophysiological variables.

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Réversibilité de l'apnée hypoxique grâce au réflexe d'aspiration chez les chats anesthésiés. Z. Tomori, R. Beňačka, V. Donič, R. Tkáčová.

RÉSUMÉ: Nous avons analysé, chez 27 chats anesthésiés, dans une modèle d'insuffisance respiratoire réversible induite par l'inhalation de N₂, différentes caractéristiques de débits et de temps respiratoires, ainsi que les enregistrements électrocorticographiques. Pendant la première minute d'arrêt respiratoire, la stimulation naso-pharyngée au moyen d'une fibre en nylon élastique a induit un réflexe d'aspiration typique ressemblant au reniflement ou au gasping (dans 88.8% des cas), alors qu'une irritation trachéo-bronchique similaire évoquait une réaction tussigène faible dans une tiers des cas (34.2%). Le réflexe d'aspiration a également peut être provoqué dans les stades progressifs d'apnée caractérisés par une activité électrocorticographique très basse et même iso-électrique, immédiatement avant la décompensation respiratoire imminente et irréversible. Il pouvait également interrompre et remplacer le gasping paradoxal survenant parfois pendant l'apnée. Ce réflexe, qui comporte de puissants efforts inspiratoires, a pu être provoqué pendant l'apnée à de nombreuses reprises successives. Les réflexes d'aspiration répétés entraînent une récupération de l'apnée hypoxique, avec normalisation graduelle de l'électrocorticogramme et réapparition subséquente d'une respiration spontanée, même plus fréquemment que ce n'est le cas au cours de gasping paradoxal. Une réanimation réussie d'une apnée hypoxique grâce au réflexe d'aspiration chez le chat, suggère qu'une stimulation naso-pharyngée pourrait influencer les mécanismes sous-jacents à la décompensation respiratoire et à la récupération de la respiration, tout au moins dans certaines formes d'apnée.

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