TITLE: CHEST WALL MECHANICS DURING PROPOFOL ANESTHESIA

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Inhalation anesthesia has been reported to preferentially depress rib cage muscle activity[1], while opposite effects have been observed with intravenous hypnotics[2,3]. The aim of the study was to evaluate the effects of propofol on chest wall mechanics.

Eight ASA PSI unpremedicated patients were studied preoperatively after informed consent and approval of the ethics committee. Anesthesia was induced and maintained with propofol as the sole agent[4]. Tracheal intubation was facilitated with lidocaïne 5% spray. End-tidal CO_2 pressure ($PetCO_2$) was measured with a Datex Capnomac capnometer. Tidal changes in rib cage (RC) and abdomen (AB) circumferences were measured in both awake and anesthetized states using differential linear transducers. A calibration procedure was performed in both states by matching the RC and AB signals to the volume changes measured with a pneumotachograph. All tidal volumes (VT) estimated by the indirect method were within ± 5% of simultaneous spirometric VTs. Respiratory swings in gastric pressure (Pga) were measured using a balloon catheter linked to a Validyne transducer. Then the following variables were obtained in each patient as the mean of all respiratory cycles during a 5-minute period in both states: VT, inspiratory time (TI), mean inspiratory flow (VT/TI), AB contribution to VT (VAB/VT), Pga/TI and abdominal compliance (VAB/Pga).

Results are expressed as mean ± SD and compared by the paired Student's t test (*: p<0.05).

		AWAKE	PROPOFOL
VT	(ml):	279 ± 78	168 ± 52 *
TI	(s):	1.61 ± 0.62	1.12 ± 1.12*
VT/TI	(ml/s):	182 ± 33	148 ± 21*
VAB/VT	(%):	60.5 ± 13.7	37.4 ± 18.1*
Pga/TI	(cmH_2O/s):	1.49 ± 0.42	1.49 ± 0.74*
VAB/Pga	(ml/cmH_2O):	69 ± 9	34 ± 12 *
$PetCO_2$	(mmHg):	38 ± 2	52 ± 4

Propofol anesthesia is associated with a fall in VT which results from the decreases of VT/TI and TI. A reduced abdominal contribution to VT accounts for the decreased VT/TI. Such impairment of inspiratory abdominal expansion has previously been reported with other iv agents and related to intercostal muscle recruitment[3] or diaphragm depression[2]. This study shows that a decrease in abdominal compliance is involved during propofol anesthesia and suggests a recruitment of abdominal muscles.

References
1.Tusiewicz K et al. Anesthesiology 1977;47: 327-337
2.Morel DR et al. J Appl Physiol 1984; 57: 1104-1110
3.Mankikian B et al. Anesthesiology 1986;65:492-99
4.Roberts FL et al. Anaesthesia 1988; 43(S): 14-17

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TITLE: DIFFERENTIAL EFFECTS OF MIDAZOLAM ON INSPIRATORY MUSCLES

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Midazolam (M) at sedative doses alters chest wall mechanics and reduces tidal volume (VT). It has been claimed, by studying thoracoabdominal motions, that the reduced VT was mediated by a decrease in abdominal contribution while ribcage motion was spared.[1] However, respiratory muscle functions were not directly assessed. Furthermore, these data contrast with the increased work of breathing reported following M in relationship with an increase in upper airway resistance[2], suggesting an increase rather than a decrease respiratory muscle activity. The aim of the present study was to determine the effects of M on intercostal and diaphragm muscle function.

Seven healthy volunteers (30±3 yrs) were studied. The protocol was approved by the local Clinical Investigation Comittee.The subjects laid supine, breathing through a face mask attached to a pneumotachograph. Esophageal and gastric pressures were measured with two balloon catheter systems connected to pressure transducers. Changes in esophageal (ΔPpl) and in gastric (ΔPga) pressures were recorded during tidal breathing. ΔPpl was used as an index of the activity of inspiratory muscles (intercostal and diaphragm). The ratio ΔPga/ΔPpl was calculated and taken as an index of the relative contribution of the diaphragm to the breathing process. Electromyographic activity of the third intercostal parasternal muscle was recorded using needle electrode. Raw signals was integrated, and electrical activity of intercostal muscle was assessed by measuring peak height electromyogram (EMGi).The following parameters: respiratory rate (RR), tidal volume (VT), ΔPpl, ΔPga, ΔPga/ΔPpl, and peak height EMGi, were recorded before IV Midazolam injection (0,1 mg/kg) (T0) . Measures were repeated 5 (T5) and 10 (T10) min later.Then IV Flumazenil (1mg) was administered and a new set of measures was performed (TF). Anova was used for statistical analysis.

Changes in RR,VT, ΔPpl, ΔPga/ΔPpl, and peak height EMGi are summarized in table. Following M, ΔPga/ΔPpl was reduced, while a dramatic increase in ΔPpl and in peak height EMGi was observed . All parameters return to T0 values after Flumazenil.

TABLE : mean±SEM. * p< 0,05, ** p< 0,01 vs T0.
° p < 0,05, °° p<0,01 vs TF.

	T0	T5	T10	TF
RR (c/min)	12,6±1,5	*° 15,4±0,9	*° 15,1±0,5	12,9±0,9
VT (ml)	734±67	** °° 464±46	** °° 450±66	681±37
ΔPpl (cm H2O)	5,5±0,5	*° 14,2±3,1	** °° 17,4±4,5	5,7±0,5
ΔPga/ΔPpl (%)	64±8	** °° 16,2±3,9	** °° 15,7±4,4	61,2±8,4
EMGi (arbitrary units)	6,4±0,5	14,7±6	*° 17,8±5,1	6,2±0,8

Our data demonstrate that M has a differential effect on respiratory muscle groups. Indeed it produces a marked depression in diaphragmatic strength generation, whereas it dramatically increases intercostal muscle activity. These findings suggest that in patients with impaired rib cage expansion, M at sedative dose could lead to a severe ventilatory depression .

References:
1. J. Appl. Physiol. 57 : 1104-1110,1984
2. Anesthesiology . 71 : A1100, 1989