Thoracic Gas Volume and Chest-Abdomen Dimensions during Anesthesia and Muscle Paralysis

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The aim was to study thoracic gas volume (TGV) in anesthetized and paralyzed subjects (n = 16) and to analyze any change in TGV in terms of thoraco-abdominal dimensions. TGV was measured by means of a body plethysmograph, using a super syringe for lung inflation during muscle paralysis. The thoraco-abdominal shape at the mamillary and umbilical levels was studied by means of magnetometry for the assessment of antero-posterior diameters (A-P), a strain gauge technique for measuring circumferences and respiratory inductive plethysmography for the recording of cross-sectional areas. TGV was reduced by an average of 28 per cent, from 2.38 l awake to 1.72 l during pentothal anesthesia and spontaneous breathing. No change in A-P diameters or cross-sectional areas were noticed. Muscle paralysis and mechanical ventilation caused no further change in TGV, nor were there any changes in A-P diameters, circumferences or cross-sectional areas of the chest or abdomen. The rib cage contribution to the tidal breath was significantly increased, from 41 per cent to 62 per cent of the tidal volume. The result of a lowered TGV during anesthesia concurrently with no measurable change in chest-abdomen dimensions remains a paradox not yet fully understood. (Key words: Anesthetics, intravenous: thiopental. Lung: thoracic gas volume. Measurement techniques: plethysmography. Ventilation: anesthetics, effect of; diaphragm; gas trapping; intermittent positive pressure breathing.)

THE INFLUENCE OF ANESTHESIA on the resting lung volume has been the subject of several studies. Most of these have shown a decrease in the resting level[1-7] but an unaltered lung volume has also been reported.[8] The technique used almost exclusively has been gas dilution (mainly multiple breath nitrogen washout). However, this will not detect any gas trapped behind occluded airways. Indeed, Don et al.[9] measured trapped gas in anesthetized subjects and found it to average 150 ml. Varying degrees of gas trapping may thus help to explain divergent findings on resting lung volume during anesthesia. Its determination by body plethysmography would be a superior technique, since it measures all gas in the chest, whether trapped or not (i.e., thoracic gas volume, TGV). In a study on anesthetized subjects using a modified tank

respirator as plethysmograph and Boyle's law according to Dubois et al.,[10] a reduced TGV was demonstrated during anesthesia and spontaneous breathing. No further change in the respiratory level was noticed when the patient was paralyzed. This was considered to indicate an unaltered TGV with paralysis, although no direct measure of TGV had been obtained. Additional studies under this condition remain to be done. Moreover, two recent reports on the chest and abdomen dimensions seem to question present knowledge on resting lung volume in anesthetized subjects. Thus, Vellody et al.,[11] using magnetometry, and Jones et al.,[12] using strain gauge technique, found an unaltered thoraco-abdominal configuration with anesthesia, which may conflict with a reduced lung volume.

Thus, we considered it important to reevaluate the analysis of resting lung volume in anesthetized subjects simultaneously with measurements of the rib-cage and abdomen dimensions. This was accomplished by using a specially designed body plethysmograph for the determination of TGV, together with mercury bands and magnetometers for the assessment of the thoraco-abdominal shape. In supplementary measurements, cross-sectional areas of the chest and abdomen were also measured by respiratory inductive plethysmography. Measurements were done during spontaneous breathing awake and anesthetized, as well as during muscle paralysis, and TGV was measured directly even during the paralyzed state.

Material and Methods

Sixteen subjects (seven males and nine females) were studied. All were surgical patients considered free from cardiopulmonary disease as judged by clinical examination, chest x-ray, and electrocardiogram. Age varied from 21 to 61 years, height from 152 to 181 cm, and weight from 60 to 103 kg. Only one patient was a smoker. The measurements were performed prior to and during anesthesia before surgery had commenced. Informed consent was obtained in each case. The ethical committee at the University Hospital, Linköping, had approved the study. There were no complications.

The patients were premedicated with 10 mg diazepam by mouth. Just before the induction of anesthesia, 0.5 mg atropine was given iv. The patients were anesthetized with thiopentone (3–5 mg/kg). A local anesthetic (lidocaine, 2 per cent) was sprayed into the larynx and the

trachea. Orotracheal intubation was performed with the aid of succinylcholine (1 mg/kg). During the study which required 20–30 min, thiopentone (1 mg/ml saline) was slowly infused (200–600 mg), to prevent return of the lid reflexes. The patients were ventilated with a mixture of air and oxygen at an F_{IO_2} of 0.3–0.35. Additional doses of succinylcholine (1 mg/kg) were given when measurements were to be obtained during muscle paralysis. In part of the study (respiratory inductive plethysmography), the intravenous anesthesia was succeeded by inhalation of 1.5 per cent halothane for a period of 10 min.

TGV

A specially designed body plethysmograph was used for the study. The box was made of Lucite® and had the shape of a half cylinder. Its volume was 485 l. The subject lay supine with the whole body, including the head, within the box. The box had blockable pipes and lines for the anesthetic tubings and the catheters for pressure recordings. Volume variations were detected by a pneumotachograph screen (Fleisch No. 5, pressure drop 1 mm H_2O for flow rate of 1.5 l/s). The pressure drop was sensed by a differential pressure transducer (EMT 32, Siemens-Elema). The atmospheric port of the transducer was connected to a compensation chamber with the same acoustical time constant as the body plethysmograph. The frequency response was flat up to 7 Hz which was considered enough for the present measurements. Mouth pressure was recorded with a pressure transducer (EMT 34, Siemens-Elema) and a remotely controlled shutter was connected to the mouth-piece. Pressure and volume were displayed on an X-Y-storage oscilloscope (Tektronix® 564) and photographed by a Polaroid camera. Measurements were first done during spontaneous breathing awake. After the box had been shut, time was allowed for thermal equilibration which took about 3–4 min. Panting maneuvers were performed with the mouthpiece occluded by the shutter, according to DuBois et al.[10] The patient was anesthetized and intubated, and when he had resumed breathing spontaneously the box was closed again and the measurements were repeated after 3–4 min of thermal equilibration. In six patients the respiratory efforts against the closed shutter were large enough to allow for an analysis of TGV (an example is shown in fig. 1).

Care was taken to secure a "true" end-expiratory resting level by following both the spirographic signal from the pneumotachograph and the magnetometer and strain gauge signals (see below). When the subject was breathing quietly and with a steady end-expiratory level, measurements were performed. The shutter was occluded at the end of a normal expiration. However, in some measurements the occlusion occurred slightly above the true

resting level. This could be corrected for by reading on the spirographic recording the point where the shutter had been occluded.

Measurements of TGV during anesthesia and muscle paralysis were also performed by body plethysmography, using the box described above. The technique used has been described in another paper[13] and will only be summarized here. With the patient lying in the box the respiratory tubings were opened to atmosphere to ascertain a true resting expiratory level. The lungs were then inflated with 1–2 l of air by means of a syringe. The rear space of the syringe was connected to the box. Accordingly, when the patient was inflated by means of the syringe, an equal amount of gas was sucked from the box. Compression of gas in the lungs (and in tubings) during the inflation resulted in a gas flow into the box, sensed by the pneumotachograph screen. This inflow of gas depends on the lung volume and the alveolar pressure. If the inflated gas differs from body temperature and is not saturated with water, its heating and humidification cause an increased expansion of the chest and so affect the net flow of gas into or out from the box. The following equation describes the calculation of TGV by body plethysmography and manual inflation:

$$TGV = \frac{\Delta V \times (1 - P) - (\Delta V^1 - V_{net})}{P - 1} \quad (1)$$

where

V = inflation volume;

$$P = \frac{(P_B - P_{H_2O_{BT}})}{(P_B - P_{H_2O_{BT}} + \Delta P)} \; ; \quad P_B = \text{barometric pressure,}$$

$P_{H_2O_{BT}}$ = water vapor pressure at body temperature,

ΔP = change in alveolar pressure;

ΔV^1 = the increment of ΔV caused by conversion from ATPS to BTPS, i.e.,

$$\Delta V^1 = \Delta V \frac{(P_B - P_{H_2O_{BT}})}{(P_B - P_{H_2O_{AT}})} \cdot \frac{(273 + AT)}{(273 + BT)} - 1$$

$P_{H_2O_{AT}}$ = water vapor pressure at ambient temperature,

AT = ambient temperature,

BT = body temperature;

V_{net} = change in box volume, i.e., the inflow or outflow of gas to or from the plethysmograph with the inflation of the subject;

V_{app} = volume of tubings connecting the syringe and subject.

FIG. 1. Typical pressure-volume recordings during panting against an occluded shutter awake and anesthetized (left and middle panels) and during mechanical inflation-deflation with the subject anesthetized and paralyzed (right panel). Note the steeper slope, indicating a lower TGV, during anesthesia than awake (left and mid panels). Numbers in right panel denote: 1) start of inflation, 2) end-inspiration, 3) point obtained after correction for gas expansion due to increase in temperature and humidity. Note that the slope of a line drawn through points 1 and 3 is rather similar to the slope shown in the mid panel. In practice the volume difference between points 1 and 2 (V_{net}) was used for the calculation of TGV according to the equation 1.
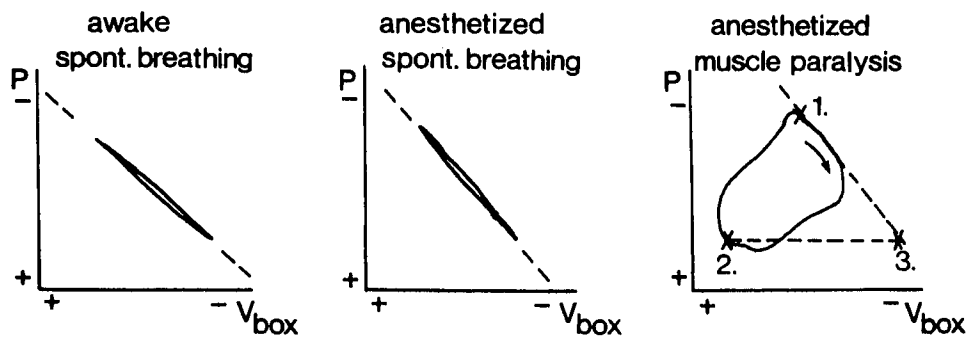
Δ V was measured from the movement of the plunger of the syringe. ΔV^1 was calculated from Δ V, temperature and humidity of ambient air, and body temperature. A thermometer, hygrometer, and barometer were located in the room where the study was conducted. Figure 1 shows a typical X-Y-recording of the simultaneous box volume and tracheal pressure changes during the manual inflation. A curve is obtained and the shape of it depends on the pressure change and the net effect of the reduction in "box volume" caused by gas compression within the chest and the increase in "box volume" as a consequence of the heating and humidification of the inflated gas.

The validation of the plethysmographic technique for determining TGV during muscle paralysis had been performed earlier and a coefficient of variation of less than 5 per cent was obtained in measurements on anesthetized subjects with healthy lungs.[13]

CHEST-ABDOMEN DIMENSIONS

The circumferences of the chest and the abdomen were measured at the mamillary and umbilical levels by a tape measure. The tidal variation in these circumferences was measured by strain gauge technique (mercury bands: Allhabo, Stockholm, amplifiers: 270, Parks Electronics Laboratory, Beaverton, Oregon). Free sliding of the mercury bands on the body surface was ensured by smearing vaseline on the body and by placing the subject on pillows with gaps for the mercury bands. The bands were connected to home-built bridges similar to slide rules, mounted on the front of the chest and the abdomen. The bridge enabled adjustment of the strain of the band and permitted the calibration of the output signal.

Strain gauges of different lengths were available to fit each individual. The strain was so adjusted that the circumference could be reduced by 6–7 cm with no slackening of the band. Model tests on linearity showed an accurate recording within 6 per cent of true change and

a resolution of better than 0.1 cm. Moreover, if the band was distended nonuniformly (part of it being strained and another part being relaxed) an error was introduced which at most added 0.2 cm to the true value. Thermal and other possible causes of drift appeared negligible, the coefficient of variation of recording a constant tidal volume (500 ml) over a 5-min period in paralyzed humans being 2.4 and 3.1 per cent, chest and abdomen, respectively.

Antero-posterior diameters of the rib cage and abdomen were also measured at the mamillary and umbilical levels. The strengths of magnetic fields generated by electromagnets fixed to the body surface were detected by coils at the opposite surface.[14] Calibration of the magnetometer signals was performed after each experiment by moving one coil step-by-step to and from the other coil along a measure. The resolution in model experiments was better than 0.1 cm (the limit was rather set by the calibration device). Rotation of the magnetometer caused an overestimation of the distance, but by less than 0.5 per cent when the angle was within 8°. All signals were registered on a 6-channel ink-jet recorder (Mingograf® 81, Siemens-Elema). Clinical tests on reproducibility of a 500-ml tidal volume recording over a 5-min period showed a coefficient of variation of 2.9 and 3.6 per cent, chest and abdomen, respectively.

In five subjects the separate contributions of the rib cage and the abdomen to the tidal volume were calculated. This required volumetric calibration of the magnetometer signals, performed as isovolume maneuvers[14] both when awake and when anesthetized. When awake, the subject was instructed to contract the abdominal muscles against a closed glottis and during artificial ventilation, the examiner gently pressed his hand against the subject's abdomen at different lung volumes. In this way equal volume changes of the chest and the abdomen, but with opposite signs, were obtained. The magnetometer signals were displayed on an X-Y-storage oscilloscope (564B with amplifiers 2A 63, Tektronix Inc.) with rib

cage diameter on one axis and abdominal diameter on the other. With each isovolume maneuver a flat loop was registered. The slope of the loop and the vertical and horizontal distances between loops obtained at different lung volumes were measured. The difference in lung volume was obtained by measuring the expired volume between two isovolume maneuvers by a spirometer (Vitalograph Ltd.). Knowing the volume changes, the deflections on the oscilloscope face could be calibrated in terms of rib cage and abdomen displacements. Measures were taken to avoid distorted signals from the magnetometers by pressing over a large area of the abdomen. The five patients who performed isovolume maneuvers were all nonobese.

Assuming similar relaxation curves of the chest-abdomen awake and anesthetized, the volumetric calibration can be accomplished also without isovolume maneuvers, thus eliminating any error introduced by manually pressing on the abdomen. This is accomplished by solving two second-degree equations on tidal volume and rib cage and abdomen A-P diameter variation, after equalization of the output signals from the two pairs of magnetometers (M. Goldman, personal communication). Thus, as an example, if in the awake subject the rib cage, (rc) signal corresponds to 1 unit and the abdomen (ab) signal to 4 units and the tidal volume is measured to 500 ml, then 1 rc + 4 ab = 500 ml. Similarily, if in the anesthetized subject the rc and ab deflections correspond to 3 and 2 units, respectively, and tidal volume is 750 ml, then: 3 rc + 2 ab = 750 ml. Solving for rc gives 200 ml and ab equals 75 ml. The two calibration procedures gave rather similar results, supporting the results here presented.

CROSS-SECTIONAL AREAS OF CHEST AND ABDOMEN

Cross-sectional areas of the chest and abdomen were measured in supplementary studies by means of respiratory inductive plethysmography.§ Two transducers, zig-zag coils of wire attached to an elastic bandage, were mounted around the body at the mamillary and umbilical levels, respectively. The coils were connected to an oscillator. The frequencies of the oscillations were varied by changes in the inductance of the coils due to changes in their cross-sectional areas during ventilation. The signals were converted to DC voltage. Volumetric and partitioning into rib cage and abdomen movements were performed by measuring the voltage deflections from the rib cage and abdomen coils when breathing at two different rib cage contributions. The latter was ascertained

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§ Watson H: The technology of respiratory inductive plethysmography. Third International Symposium on Ambulatory Monitoring, 1979.

by breathing in the supine and upright positions at known tidal volumes. Two second degree equations could then be solved, in analogy with the partitioning during magnetometry (see above). The respiratory inductance equipment was tested in humans during mechanical ventilation with different tidal volumes. The reproducibility of recording a constant tidal volume of 500 ml was better than 4 per cent both for the chest and the abdomen. The deflections were somewhat nonlinear so that a calibration with a 500-ml tidal volume caused an underestimation of a tidal breath of 250 ml by 6 per cent. Results in the present study have been corrected for this error. The oscillator was sensitive to temperature changes. Care was therefore taken to maintain its temperature constant. A built-in thermistor enabled the continuous recording of temperature.

STATISTICS

The statistical analysis consisted of the calculation of means and standard errors. The degree of significance of results was calculated with Student's t test.

Results

SPONTANEOUS BREATHING AWAKE *VS.* MECHANICAL VENTILATION DURING ANESTHESIA AND MUSCLE PARALYSIS

TGV and chest-abdomen dimensions in the supine, awake, spontaneously breathing subjects are shown in table 1. TGV was on average 0.6 l less than the predicted normal value for sitting or standing subjects.[15] This lower value is in good agreement with the well-known reduction in resting lung level with change from upright to supine posture.[16] The antero-posterior and circumferential tidal excursions of the rib cage at the mamillary level were approximately half those of the abdomen at the umbilical level. The five patients, in whom isovolume maneuvers were performed, showed a rib cage contribution to the tidal breathing of, on average, 41 per cent (range: 26–55 per cent; see table 2).

After 10 min of mechanical ventilation under anesthesia and muscle paralysis new recordings were performed. These showed that TGV was reduced by an average of 0.7 l (28 per cent decrease of initial value) (table 2, fig. 2). Despite the lowered TGV, the antero-posterior diameter and the circumferences of the rib cage and of the abdomen were not significantly altered (table 1). The rib cage antero-posterior and circumferential tidal excursions were proportionately increased during mechanical ventilation, and the five subjects in whom isovolume maneuvers were performed displayed an increase in the rib cage contribution to the tidal breath, the average amounting to 62 per cent (range: 44–78 per cent; table 2).

THORACIC GAS VOLUMES DURING ANESTHESIA

TABLE 1. TGV, Chest and Abdomen Dimensions and Their Tidal Variations in the Awake Subject, Breathing Spontaneously and during Barbiturate Anesthesia and Muscle Paralysis, n = 10

		Rib Cage				Abdomen			
		Dimensions at TGV		Tidal Excursions		Dimensions at TGV		Tidal Excursions	
	TGV (l)	A-P diameter (cm)	Circumference (cm)	A-P diameter (cm)	Circumference (cm)	A-P diameter (cm)	Circumference (cm)	A-P diameter (cm)	Circumference (cm)
Awake, spontaneously breathing	2.44 ± 0.16	22.8 ± 0.08	94.8 ± 5.4	0.21 ± 0.04	0.72 ± 0.21	23.5 ± 1.9	93.7 ± 7.1	0.52 ± 0.09	1.34 ± 0.32
Anesthetized, muscle paralysis	1.78 ± 0.13	22.8 ± 0.7	94.2 ± 4.9	0.50 ± 0.07	0.99 ± 0.23	23.3 ± 1.7	93.4 ± 6.6	0.60 ± 0.12	1.18 ± 0.31
Significance awake-anesthetized, P	< 0.001	N.S.	N.S.	<0.001	<0.05	N.S.	N.S.	N.S.	N.S.

The end-expiratory level increased in several subjects during mechanical ventilation as compared to the resting level during apnea. The increase occurred in both the chest and the abdomen dimensions. The increase depended on the respiratory frequency, the size of the breath and the expiratory resistance of the ventilatory equipment used. In general, the increase amounted to less than 0.1 l, and at most 0.2 l. It has not been included in the calculation of TGV.

In nine patients, TGV and chest-abdomen antero-posterior diameters were measured once more about 30 min after induction of anesthesia. There was no significant change between 10 and 30 min of anesthesia in either TGV (1.72 ± 0.22 and 1.66 ± 0.31 l, respectively) or the antero-posterior diameters (rib cage: 23.0 ± 2.1 and 23.0 ± 1.9 cm, respectively; abdomen: 23.8 ± 2.2 and 23.5 ± 2.1 cm, respectively).

TABLE 2. Rib Cage Contribution to Tidal Breathing As Measured in Different Studies on Supine Subjects during Spontaneous Breathing Awake, Spontaneous Breathing under Anesthesia and during Manual Inflation and Anesthesia

Authors and Type of Anesthesia	Rib cage contribution, Per Cent		
	Awake	Anesthetized	Manual Inflation
Grimby et al., 1975[24] (thiopentone drip + fentanyl)	40		73
Tusiewicz et al., 1977[25] (halothane 0.8–0.9 per cent)	43	19	
Vellody et al., 1977[11] (thiopental + ?)	25		48
Jones et al., 1979[12] (halothane 1 per cent)	15	~8	27
Present study (Thiopentone drip)	41		62

SPONTANEOUS BREATHING AWAKE VS. SPONTANEOUS BREATHING DURING ANESTHESIA

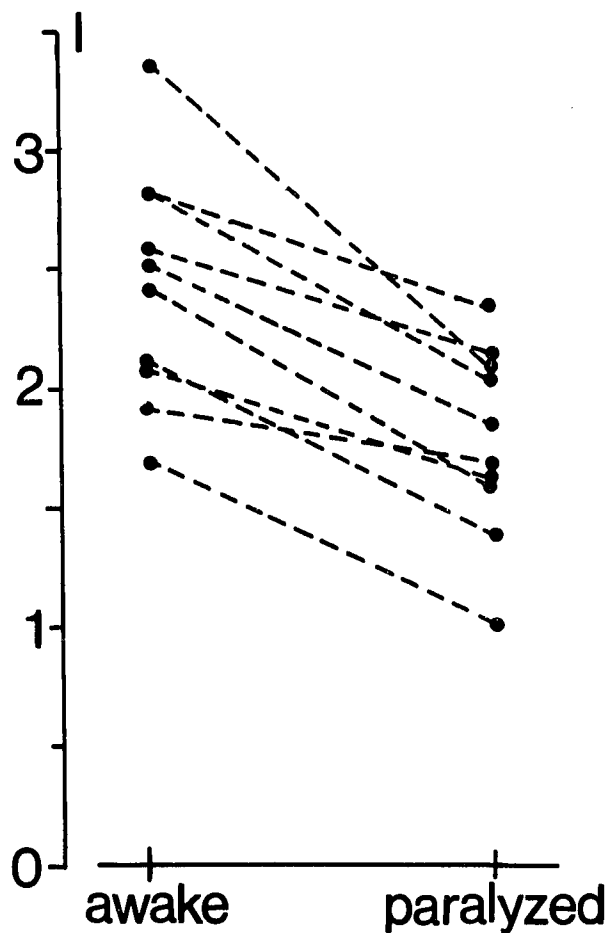TGV and antero-posterior diameters of the rib cage and abdomen were also measured in six subjects both

FIG. 2. TGV in awake and then anesthetized and paralyzed subjects.

when awake and when anesthetized, breathing sponta-
neously. The measurements were performed as soon as
breathing had reappeared after induction of anesthesia
and intubation, plus another 3 min for thermal equili-
bration in the closed box. The results are shown in table
3 together with data during anesthesia and muscle pa-
ralysis. TGV was reduced by 0.6–0.7 l with anesthesia
and spontaneous breathing and no further change was
recorded when the patient was paralyzed. There was no
significant change in the antero-posterior diameters of
the rib cage or abdomen when the patient was anesthe-
tized, nor when he was paralyzed. There was a slight
increase in the tidal excursions of the rib cage when
breathing reappeared during thiopental anesthesia after
intubation, while abdominal excursions were the same
as under awake conditions. However, since no meaning-
ful isovolume maneuver could be obtained from the anes-
thetized patients, a true partitioning of the tidal breath
into the rib cage and abdomen displacement could not
be performed. With muscle paralysis and mechanical
inflation the rib cage antero-posterior tidal excursion was
more than doubled, concurrently with an unaltered ab-
dominal tidal excursion.

SUPPLEMENTARY RECORDINGS OF CROSS-SECTIONAL AREAS AT MAMILLARY AND UMBILICAL LEVELS

The cross-sectional areas of chest and abdomen re-
mained essentially unaltered during intravenous anes-
thesia, both when breathing spontaneously and during
mechanical ventilation. Thus, computed rib cage volume
was altered by an average of −60 ± 30 ml (SE) during
spontaneous breathing and by −30 ± 25 ml during me-
chanical ventilation compared to initial values awake
($P > 0.05$ in both cases). Corresponding figures for ab-

dominal volume was +20 ± 35 ml and +15 ± 30 ml,
respectively ($P > 0.05$ under both conditions). Nor was
there any measurable change in either volume when
halothane was added to the inspirate.

Discussion

A modified body plethysmographic technique has been
used in the present study, allowing lung-volume mea-
surements in anesthetized subjects breathing sponta-
neously as well as being ventilated mechanically. Si-
multaneous measurements of thoraco-abdominal dimen-
sions by magnetometry and strain gauge technique have
also been performed, thus permitting a comparative anal-
ysis of lung volume and chest-abdomen shape. The major
findings were a reduced TGV during anesthesia con-
currently with unaltered antero-posterior diameters and
circumferences of the rib cage and abdomen, whether
ventilation was spontaneous or mechanical. Additional
measurements of cross-sectional areas of the chest and
the abdomen also did not reveal any change with anes-
thesia, whether intravenous or inhalational. The reduc-
tion in TGV appeared as soon as measurements could
be performed, i.e., approximately 10 min after induction,
while no further change was observed 20 min later. Thus,
the change in TGV seems to be a rather rapid phenom-
enon, not a slow shift in the resting level. The seemingly
paradox, reduced TGV but no measurable change in
chest-abdomen shape, is discussed in the following sec-
tions.

TGV

Failure to measure gas behind occluded airways may
cause an underestimation of the gas volume in the lung.[9]
Anesthesia may promote airway closure within or above

TABLE 3. TGV, Antero-posterior Diameters (A-P) and Tidal Excursions during Spontaneous Breathing Awake and Anesthetized, and after Muscle Paralysis (n = 6; means ± SEM)

	TGV (l)	A-P Diameters		A-P Tidal Excursions	
		Rib Cage (cm)	Abdomen (cm)	Rib Cage (cm)	Abdomen (cm)
Awake, spontaneous breathing	2.38 ± 0.31	23.1 ± 1.7	26.7 ± 2.9	0.22 ± 0.03	0.62 ± 0.10
Anesthetized, spontaneous breathing	1.72 ± 0.23	23.4 ± 1.8	26.1 ± 2.5	0.33 ± 0.06	0.65 ± 0.28
Anesthetized, paralyzed	1.75 ± 0.23	23.0 ± 1.4	25.9 ± 2.6	0.50 ± 0.06	0.61 ± 0.14
Significance					
Awake-anesthetized spontaneous breathing	<0.01	N.S.	N.S.	<0.05	N.S.
Awake-anesthetized paralyzed	<0.01	N.S.	N.S.	<0.01	N.S.
Anesthetized spontaneous breathing-paralyzed	N.S.	N.S.	N.S.	N.S.	N.S.

the tidal volume.[17-19] and so trap gas. However, the use of Boyle's law in body plethysmography means that trapped gas is included in the measurement. This should hold true also during the mechanical inflation of the lung during muscle paralysis for assessing TGV, provided the lung behaves as an isotropic tissue.[13,20]

Abdominal gas may influence the determination of lung volume by body plethysmography, but negligibly so during spontaneous breathing at lung volumes near the resting level.[21] In the paralyzed, mechanically ventilated subject, abdominal gas presumably contributes less than 100 ml to the measured TGV. Pulmonary gas exchange may also affect the measurement of TGV, although the effect has been shown to be small for most situations. However, to reduce any effect to a minimum, anesthesia was induced and maintained by intravenously administered agents. It seems therefore unlikely that any significant error has been introduced in the measurement of TGV.

CHEST-ABDOMEN SHAPE

Rather high resolution and reproducibility characterized both the strain gauge and the magnetometer in model tests. However, human tests may introduce non-uniform tension of the band and rotation of the magnetometer which may obscure the measurement. A non-uniform reduction of the strain of the mercury band would result in a higher output than if the band were relaxed uniformly, even if band lengths were equal under both conditions. The overestimation would be at most 0.2 cm. However, no statistical significance was achieved between awake and anesthetized conditions, even after subtraction by 0.2 cm from all circumferences measured under anesthesia. Rotation of the magnetometer must reach 8° to cause an overestimation of the diameter by 0.1 cm (A-P diameter: 23 cm). Allowance for such a rotation from awake to anesthetized states did not create any significant difference in the diameters between the two conditions.

The change in circumference and diameter that would cause a reduction in TGV by 660 ml (effect of anesthesia in the present study) can be calculated, under certain assumptions. Using the presently measured abdomen circumference (see table 1) and considering the abdomen as a cylinder with a height of 20 cm, the circumference must be diminished by 2.2 cm to cause a reduction in TGV by 660 ml (compared to the measured nonsignificant change of 0.3 cm). It can be noticed that if the reduction in TGV is caused by equal reductions in chest and abdomen volume, and if rib cage is assumed to have a cylindrical height of 30 cm, then its circumference need not be reduced by more than 0.8 cm which is a little more than the *insignificant* change measured in the present study. One would then anticipate a reduction of the

abdomen circumference of at least 1.1 cm (assuming an abdomen height of 20 cm) which is far more than the present result. Similarly, the A-P diameters need to diminish by 0.52 and 0.46 cm, chest and abdomen, respectively to, in equal proportions, cause the measured reduction in TGV. This appears to be more than what should pass unnoticed by artefactual recordings.

Assuming an elliptic instead of a circular circumference, it can be shown that a volume change will cause an even greater variation in the circumference. Similarly, the elliptic base causes greater A-P diameter variation for a given change in volume than does the circular base.

A change in the cross-sectional area from circular to elliptic would cause a volume reduction with no necessary change in circumference or A-P diameter. However, this possibility appears to have been excluded by the findings of unaltered areas at the mamillary and umbilical levels by inductive plethysmography. The possibility remains that the shape of the chest or abdomen someplace other than at the recording levels has been altered. However, Konno and Mead[14] showed that in awake subjects chest and abdomen behave as systems with only one degree of freedom. This means that only one measuring point is required to adequately describe a dimensional change in that system. That anesthesia and muscle paralysis would introduce further freedoms is rather unlikely, uncoordinated muscle activity is what is required to do so.

REDUCED TGV-UNALTERED CHEST-ABDOMEN SHAPE

Our analysis has been based on the assumption that one measuring point suffices to record dimensional changes of the chest or abdomen.[14] With this *limitation* kept in mind it can be said that our analysis points to chest-abdomen changes which should be large enough to be detected, were they the only cause of the reduced TGV. It should be stressed here that a cranial displacement of the diaphragm with anesthesia, as shown by Froese and Bryan,[22] may cause a reduced TGV with no change in rib cage dimensions. However, a concomitant reduction in abdominal dimensions would be expected, were the shift in diaphragm the only cause of a diminished TGV. However, if fluid accumulated in the chest or the abdomen (*e.g.*, central blood pooling) a reduction in TGV might occur with no simultaneous change in thoraco-abdominal shape. A central fluid accumulation in conjunction with reduced thoraco-abdominal dimensions need not conflict with our analysis since the reduction in chest-abdomen diameters and circumferences may then be too small to be detectable. However, fluid shift as an explanation of the reduced TGV during anesthesia is pure conjecture though it does appear to be an interesting subject for future research.

TIDAL BREATH

The presently measured increase in rib cage displacement with anesthesia and mechanical ventilation is in agreement with previous reports which are summarized in table 2. However, the fractional contribution of the rib cage varied between the studies from 25 to 43 per cent in awake, supine subjects as measured by magnetometry,[11,23,24] while a contribution of only 15 per cent was measured by a strain gauge technique.[12] The rib cage contribution has been shown to be reduced during spontaneous breathing under anesthesia.[12,24] The results in the present study were rather an increase in the rib cage movement as measured by magnetometry but the findings have not been transferred to volume contribution since no isovolume maneuver could be performed. However, the possibility remains that the different anesthetic procedure in the present study (thiopentone drip) compared to the previous studies (halothane anesthesia) contributed to the different results.

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