Large tidal volume ventilation improves pulmonary gas exchange during lower abdominal surgery in Trendelenburg's position

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Impaired pulmonary gas exchange is a common complication of general anaesthesia. Periodic hyperinflation of the lungs and large tidal volume ventilation were the first preventive measures to be widely embraced, but their effectiveness in clinical practice has never been clearly established by controlled clinical studies. To assess their effects in high-risk patients we studied 24 adults having lower abdominal gynaecological surgery in the Trendelenburg (head down) position. Pulmonary oxygen exchange was determined during four steady-states: awake control (AC), after 30 min of conventional tidal volume (CVT, 7.5 ml · kg^{-1}) or high tidal volume (HVT, 12.7 ml · kg^{-1}) ventilation, introduced in random order, and five minutes after manual hyperinflations (HI) of the lungs. The patients' lungs were ventilated with air/O_2 by an Ohmeda volume-controlled ventilator via a circle system. The F_IO_2 was controlled at 0.5, and F_ETCO_2 was controlled by adding dead space during HVT. Arterial blood gas analysis was used to calculate the oxygen tension-based indices of gas exchange.

There was significant deterioration of (A-a)DO_2 at 30 min in Group A, whose lungs were first ventilated with CVT (81.6 ± 7.2 to 166.8 ± 13.7 mmHg, $P < 0.001$); but not in Group B, whose lungs were first ventilated with HVT (77.0 ± 9.9 to 104.4 ± 16.8 mmHg). When Group A and B data were pooled there was no difference between randomized CVT and HVT, but improvement occurred after HI. In this model of compromised O_2 exchange large inflation volumes (HVT and HI) were of considerable clinical benefit, HVT prevented and HI reversed the gas exchange disorder.

L'anesthésie générale perturbe souvent les échanges gazeux pulmonaires. L'hyperinflation intermittente des poumons et la ventilation pulmonaire à grand volume courant ont très tôt servi à pallier à ce problème même si leur efficacité n'a jamais été démontrée en clinique. Nous avons mesuré l'impact de ces deux manoeuvres chez 24 patientes en Trendelenburg lors d'interventions chirurgicales abdominales basses (gynécologiques). On mesurait le transfert pulmonaire de l'oxygène à quatre occasions : avant l'induction de l'anesthésie (EV), après 30 min de ventilation avec volume courant conventionnel (VcC, 7,5 ml · kg^{-1}) ou avec grand volume courant (VcG, 12,7 ml · kg^{-1}), l'ordre étant déterminé au hasard, et cinq minutes après hyperinflation pulmonaire (HP). C'est un ventilateur volumétrique d'Ohmeda branché sur un système anesthésique en cercle qui assurait la ventilation pulmonaire avec un mélange d'air et d'oxygène, une F_IO_2 de 0,5 et une F_ETCO_2 ajustée grâce à l'ajout d'espace mort lors de la VcG. On mesurait les gaz artériels pour calculer le gradient alvéolo-artériel pour l'oxygène ((A-a) DO_2).

On notait une détérioration significative de ce dernier à 30 min chez le groupe A, ventilation en VcC d'abord (81,6 ± 7,2 à 166,8 ± 13,7 mmHg, $P < 0,001$) mais non chez le groupe B, ventilation en VcG d'abord (77,0 ± 9,9 à 104,4 ± 16,8 mmHg). Toutefois, une fois regroupées les données des groupes A et B, on n'identifiait pas de différence entre le VcC et le VcG, mais on notait une amélioration après HP. Avec ce modèle clinique où l'oxygénation était compromise, on a démontré que de grandes insufflations pulmonaires (VcG et HP) offraient un

Key words

LUNG: gas exchange, shunting;
VENTILATION: artificial, tidal volume, oxygen tension (gradients), shunting;
OXYGEN: blood levels, gradients, tension.

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avantage considérable ; l'usage de grands volumes courants prévenait et l'hyperinflation pulmonaire corrigeait les anomalies des échanges gazeux.

Impaired pulmonary gas exchange frequently complicates general anaesthesia,[1] particularly during abdominal or thoracic surgery and in lateral or head down positions. The underlying mechanisms include reduced functional residual capacity (FRC),[2,3] airway closure[4] and dependent atelectasis;[5] the common denominator is increased physiological shunt fraction ($\dot{Q}s/\dot{Q}T$). There is no consensus on how these changes are best prevented or treated.[6] Lung hyperinflation, that is, controlled ventilation with high tidal volumes (HVT) or intermittent hyperinflation (HI) of the lungs, has been shown to be of benefit under some conditions, but its effectiveness in high-risk patients has not been clearly established. Oxygenation and compliance after HI are only transiently improved.[7] Ventilation with HVT has not been studied under appropriately controlled clinical conditions.[8–10] Although improvement has been observed, in one study it had detrimental effects on gas exchange.[11] We therefore undertook a randomized control trial, in patients at high risk for physiological shunting, to compare the effects of HVT and conventional tidal volume (CVT) ventilation on pulmonary gas exchange, and to examine the further effect of manual HI.

Methods

The protocol for this study was approved by Departmental and Institutional Ethics Review Committees.

The subjects were 24 adult ASA physical status I and II women having elective major gynaecological surgery through lower abdominal incisions. Various degrees of intra-abdominal packing, retraction, and supine Trendelenburg (head down) positioning were common to all.

Routine preanaesthetic workup included history, physical examination and chest radiograph. Exclusion criteria were recent symptomatic respiratory tract disease or currently smoking more than 20 cigarettes per day. One Group B subject who had copious bronchial secretions and required frequent tracheal suctioning was excluded from data analysis. Moderate obesity, a history of symptomatic lung disease or smoking less than 20 cigarettes per day were identified as respiratory risk factors.

A teflon 20-gauge radial artery cannula was inserted, with the patient's consent, after infiltration with local anaesthesia. Premedication was with lorazepam or midazolam, and all had total intravenous general anaesthesia with tracheal intubation, in three cases combined with epidural analgesia. Induction of anaesthesia was with propofol 2 $mg \cdot kg^{-1}$, fentanyl 2–3 $\mu g \cdot kg^{-1}$, and succinylcholine 1–2 $mg \cdot kg^{-1}$. After tracheal intubation, maintenance was by a continuous infusion of propofol, 4–10 $mg \cdot kg^{-1} \cdot hr^{-1}$ and supplemental narcotic. Muscle paralysis was maintained with either alcuronium (0.3 $mg \cdot kg^{-1}$) or atracurium (0.5 $mg \cdot kg^{-1}$), and controlled intermittent positive pressure ventilation (IPPV) with an Ohmeda 7000 volume-controlled ventilator at a rate of 10 min^{-1} and an I:E ratio of 1:2 via a circle system with a soda lime absorber. The fresh gas mixture was air/O_2 with an inspiratory O_2 concentration (FiO_2) of 0.5. Tidal volume (VT) during CVT ventilation was adjusted so that end tidal CO_2 ($FETCO_2$) was between 4.5 and 5.0% (VT of 7–8 $ml \cdot kg^{-1}$). During HVT ventilation a 300 ml dead space was added to maintain $FETCO_2$ constant.

Patients were randomized by year of birth (odd or even numbered) into two groups, A and B. While lying supine before induction of anaesthesia all breathed air/O_2 (FiO_2 = 0.5) for five minutes through a tight-fitting mask connected to the anaesthesia circle system (AC condition). After tracheal intubation and several manual inflations of the lungs Group A patients were given CVT ventilation and switched after 30 min to HVT. For Group B patients the order was reversed. Following the second 30-min study the lungs of all patients were manually inflated three to four times to total lung capacity (TLC), standardized as a peak pressure of 30 cm H_2O held for 10–15 sec, then returned to the previous mode of ventilation. In the majority of cases the packs and retractors had also been removed by that time. The endotracheal tube was suctioned only if secretions were audible.

The FiO_2 was measured by a galvanic fuel cell oxygen analyzer, $FETCO_2$ by an infra-red analyzer, and VT by a turbine vane flow transducer (Ohmeda 5250 RGM Monitor). After five minutes of AC breathing and 30 min at each tidal volume, and five minutes after HI, an arterial blood sample was drawn anaerobically from the radial artery cannula, placed on ice, and analyzed within three minutes with a Nova Biomedical Stat Profile blood gas analyzer at 37° C. The analyzer was programmed to execute a two-point calibration at two hourly intervals and a one-point calibration before each sample. Heart rate (HR) and blood pressure (BP) were measured by a Dinamap Automatic BP Monitor at three-minute intervals.

The calculation of alveolar oxygen tension was based on the formula adopted by Pappenheimer *et al.*:[12]

$$P_AO_2 = FiO_2(P_B - P_AH_2O) - PaCO_2[FiO_2 + (1 - FiO_2)/RQ]$$

Since Singapore is at sea level, the barometric pressure P_B was assumed to be 760 mmHg and alveolar water vapour

partial pressure (PAH_2O) to be 47 mmHg. Day to day variation in barometric pressure in Singapore is less than one percent.* If FIO_2 and RQ are assigned constant values of 0.5 and 0.9† respectively the Pappemheimer formula reduces to:

$$PAO_2 = 357 - (PaCO_2 \times 1.06)$$

Pulmonary gas exchange was assessed by calculating the indices of gas exchange based on FIO_2, temperature corrected PAO_2, and calculated PaO_2:[13]

1 the alveolar-arterial oxygen tension difference, (A-a)DO_2
2 the arterial/alveolar oxygen tension ratio, PaO_2/PAO_2
3 the arterial oxygen tension/inspired oxygen fraction ratio, PaO_2/FIO_2
4 the respiratory index (RI), (A-a) DO_2/PaO_2.

Only the results for (A-a)DO_2 are reported since the conclusions are identical for all four indices.

Group A and B intra-group comparisons were by ANOVA and Fisher's Least Significant Difference test, with inter-group comparisons by unpaired t tests (Statgraphics Statistical Graphics System).

Results

Demographic and comparative data for Groups A and B are shown in Table I. There were no significant differences in age, weight, ASA physical status, or respiratory risk factors. The power of the experiment to detect a difference of 20% in age or weight was greater than 0.8.

During CVT ventilation average VT was 7.5 ± 0.9 ml·kg^{-1} and 12.7 ± 1.8 during HVT, an increase of 70%. Peak AWP was similarly increased. There were no differences among the four study conditions in pH, $PaCO_2$, BP, or temperature (Table II).

When the pooled data for Groups A and B, factored by type of ventilation, were analyzed (Table II), (A-a)DO_2 was significantly increased from AC with both CVT and HVT, but they were not different from each other. While (A-a)DO_2 after HI was significantly less than during CVT ($P < 0.05$), it was not different from the results during AC or HVT.

Comparison of Groups A and B (Figure 1) revealed the importance of the order in which the two tidal volumes were introduced. Changes in (A-a)DO_2 were significant by ANOVA in Group A, but not in Group B, and the difference between the two groups was due entirely to the 30 min values. The (A-a)DO_2 after 30 min of ventilation with HVT (Group B) was less ($P < 0.01$) than after 30 min of ventilation with CVT (Group A).

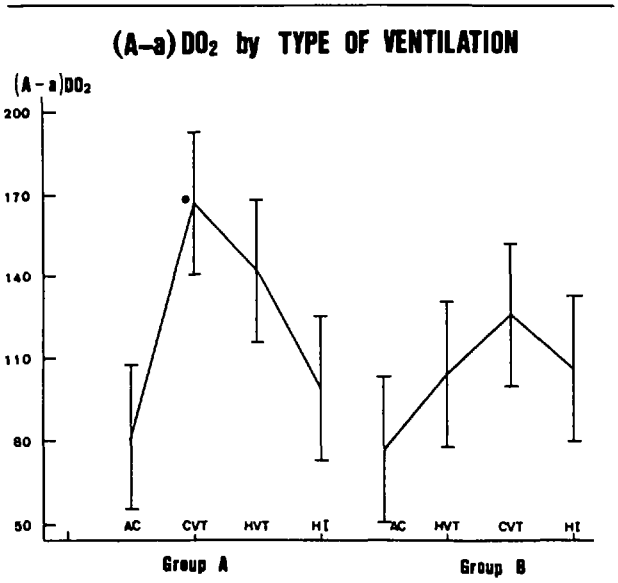

FIGURE 1 (A-a)DO_2 by type of ventilation for Groups A and B. Inter-group comparisons by t tests for unpaired data. *Significantly different from the corresponding time period of Group B, $P < 0.01$.

TABLE I Demographic data (mean ± SD)

	Group A	*Group B*
Age yr	46.7 ± 11.8 (21–69)	43.8 ± 9.7 (26–64)
Weight kg	61.3 ± 10.4 (47–84)	59.5 ± 10.6 (43–73)
Resp risk factors	4/12	4/12
ASA status II	4/12	5/12

The effect of time on (A-a)DO_2, using pooled data for Groups A and B, is examined in Table III. The four groups in this table were significantly different by ANOVA, and multiple group comparisons (LSD test) identified two homogeneous groupings, that is, combinations of groups which shared a common mean. One such grouping was AC and HI, the other the 30 min and 60 min groups. This means that there was no deterioration with time from 30 to 60 min and no significant difference between AC and HI. The power of this experiment to detect a 25% change in (A-a)DO_2 between 30 and 60 min or a 50% change between AC and HI was greater than 0.9.

Discussion

In this sample of patients there was deterioration of gas exchange when the initial mode of ventilation after induction of anaesthesia was CVT (Group A), but not

*Personal communication, The Meteorological Service of Singapore.

†Value for RQ from metabolic studies of another sample of patients during anaesthesia by a similar technique in this Department.

TABLE II Results by type of ventilation: pooled data for 24 subjects (mean ± SEM). Units of measure: gas tensions–mmHg, BP–mmHg. Statistical comparison by ANOVA and LSD tests (with 95% confidence)

	AC	*CV_T*	*HV_T*	*HI*
Tidal vol mL · kg^{-1}		7.5 ± 0.9	12.7 ± 1.8	
Peak AWP cm H_2O		19.3 ± 4.4	27.1 ± 5.0	
$PaCO_2$	40 ± 1	38 ± 1	39 ± 1	37 ± 1
PaO_2	235 ± 6	170 ± 11	193 ± 11	215 ± 10
Syst BP	126 ± 2	129 ± 14	119 ± 5	113 ± 3
Diast BP	70 ± 2	66 ± 3	68 ± 3	68 ± 2
Temp	37.0 ± 0.1	36.1 ± 0.1	36.1 ± 0.1	36.0 ± 0.1
(A-a)DO_2	79 ± 6	146 ± 11*	123 ± 11*	103 ± 9†

*Significantly different from AC, $P < 0.05$.
†Significantly different from CVT, $P < 0.05$.

TABLE III Time-related changes: pooled data for 24 subjects (mean ± SEM). Units of measure: gas tensions–mmHg, BP–mmHg. Statistical comparison by ANOVA and LSD tests (with 95% confidence)

	AC	*30 min*	*60 min*	*HI*
Tidal vol mL · kg^{-1}		10.2 ± 0.7	10.2 ± 0.6	
$PaCO_2$	40 ± 1	38 ± 1	39 ± 1	37 ± 1
Syst BP	126 ± 2	122 ± 5	127 ± 16	113 ± 3
Diast BP	70 ± 2	68 ± 3	67 ± 2	68 ± 2
(A-a)DO_2*	79 ± 6	136 ± 12	134 ± 10	103 ± 9

*Two homogeneous groupings of factors, AC with HI and 30 min with 60 min, were identified by the LSD test. Homogeneous groupings are combinations of factors which share a common mean.

when it was HVT (Group B). However, there was no difference between the two modes when randomized pooled data were compared. This suggests that HVT was more effective in preventing than in reversing the gas exchange defect in this clinical model. Manual HI (combined in most cases with removal of packs and retractors) restored (A-a)DO_2 to values which were not different from awake control, evidence that the defect was reversible and that surgical manipulations, packing, retraction and positioning were key causative factors. As discussed below we believe that this is a model of compression atelectasis with venous shunting, and that the dependence of gas exchange on volume of ventilation can be explained by accepted physiological principles.

Computer models[14–16] and clinical studies[17,18] have shown that the oxygen tension-based indices of gas exchange do not reliably predict $\dot{Q}S/\dot{Q}T$ when FIO_2 varies, $\dot{Q}S/\dot{Q}T$ is large, or cardiac output is low. However, in the present study FIO_2 was held constant, severe shunting with O_2 desaturation was not observed, nor were there changes in HR or BP. For these reasons we believe that under the conditions of these investigations changes in the gas exchange indices reflect changes in $\dot{Q}S/\dot{Q}T$.

Many investigators have confirmed that physiological shunting increases during general anaesthesia in older or obese patients, cigarette smokers, and those with pre-existing lung disease, but only minimally in healthy, young, non-smokers.[19] The FRC and total pulmonary compliance are reduced with onset of general anaesthesia. Changes in the recoil pressures of the lung and particularly the chest wall appear to be primary mechanisms,[20] while altered mechanics of the diaphragm in the horizontal anaesthetized patient have a contributory role.[21] Dueck[3] has shown that physiological shunt increases when FRC is reduced below awake closing capacity (CC). Hedenstierna *et al.*[22–24] have demonstrated increased shunting and dependent atelectasis in anaesthetized patients, in both the supine and lateral positions, by CT scanning.

Manual sighs (i.e., HI) and large tidal volumes were first advocated by Bendixen *et al.*[9] to improve oxygenation during anaesthesia. Manual sighs have been shown to restore oxygenation and compliance temporarily, but must be repeated frequently.[7–9] High VT ventilation was studied in both dogs[25] and humans.[9] Anaesthetized dogs required a VT of 25 ml · kg^{-1} ($PaCO_2$ = 21 mmHg) to prevent deterioration of $\dot{Q}S/\dot{Q}T$, indicating that the model has no clinical relevance. In a clinical study, by the same investigators, PaO_2 and compliance decreased during low

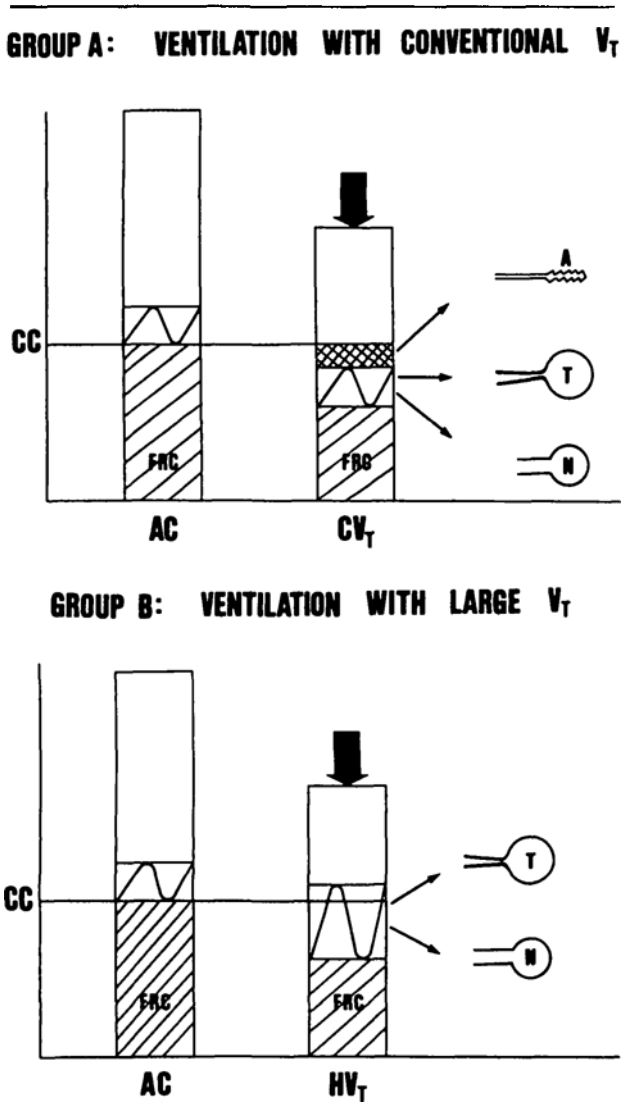

FIGURE 2 Proposed model for ventilation and lung volumes during lower abdominal surgery in the Trendelenburg position. The horizontal line is closing capacity (CC). Volume A = atelectasis, T = the ventilated volume in which airway closure and gas trapping occurs, and N = the volume of normally ventilated and perfused alveoli.

tidal volume ventilation, but not when VT was normal or increased. Both compliance and oxygenation were restored, at least temporarily, by manual sighs.

Visick *et al.*[10] compared small tidal volumes (5 $ml \cdot kg^{-1}$) with and without CPP (10 cm H_2O) with large tidal volumes (15 $ml \cdot kg^{-1}$) in supine anaesthetized patients. With large VT there was increased compliance and decreased VD/VT, but no difference in mean (A-a)DO_2. Only when large VT or CPP replaced small VT was (A-a)DO_2 improved, suggesting that the prior volume history of the lungs was important. In a subsequent study[26] they measured FRC, closing volume (CV), and O_2 exchange at VT of 5 and 10 $ml \cdot kg^{-1}$. Their results support the hypothesis that the relationship between inspiratory lung volume and closing capacity (CC) is a determinant of gas exchange. Oxygenation improved only when increasing VT moved end inspiratory lung volume from below to above CC. However, their comparison was with hypoinflation, that is VT less than awake control, and therefore the clinical application of their results is uncertain.

The studies cited above, though seminal works in our understanding of ventilatory impairment during anaesthesia, have been inconclusive in establishing the effectiveness of large volume ventilation in clinical practice.

Although we have not examined the pathophysiology of impaired gas exchange in our patients, we believe it can be reasonably ascribed to increased intra-abdominal pressure, cephalad shift of the diaphragm, compression atelectasis, and increased venous shunting. We have excluded patients with copious bronchial secretions or acute obstructive airways disease, and heavy smokers, who might have been at risk for obstructive atelectasis. Based on this assumption of causation, the studies reviewed above allow us to propose an hypothesis for the beneficial effects of large volume ventilation in our clinical model (Figure 2, A and B).

In supine sedated subjects, breathing spontaneously, FRC is at or below CC, depending on age and other factors.[27,28] A crucial assumption, that the constant relationship between CC and awake TLC is not altered by anaesthesia, has been confirmed in clinical experiments.[29]

We propose that the lung compression in this clinical model reduced FRC to well below CC. If CC was above the inspiratory lung volume during CVT ventilation, three populations of alveoli would result (Figure 2A). In lung volume A, between CC and VT, airways would be closed throughout the respiratory cycle. After gas absorption, or simply as a result of the same forces that cause airway closure, this lung volume would become atelectatic, corresponding to the areas of dependent atelectasis described by Hedenstierna *et al.* In the absence of hypoxic pulmonary vasoconstriction this would become a region of true shunting, with a zero $\dot{V}_A/\dot{Q}$.

Two populations of alveoli would inhabit the tidal volume range: one in which airway closure and gas trapping occurred (volume T), and another with normal $\dot{V}_A/\dot{Q}$ (volume N). The former would have had a low $\dot{V}_A/\dot{Q}$ contributing to the physiological shunt.

We assume that in Group B, whose lungs were ventilated preemptively with HVT, CC and FRC were the same as in Group A. With CC in the VT range, airways which closed during expiration would be reopened or "recruited" during subsequent inspirations. This might be facilitated by the increase in peak AWP. Volume T would

contribute to $\dot{V}_A/\dot{Q}$ mismatch, but with less hypoxaemia than would occur if the same volume of lung were completely unventilated.

This model explains the protective effect of HVT when instituted preemptively, its diminished effect following CVT, and the improvement after HI. After atelectasis formation, ventilation must overcome higher resistance to re-expand atelectatic lung units. This requires a slow TLC inflation, such as a standardized manual HI, but collapse probably recurs on return to conventional ventilation. We did not study PEEP, but anticipate that it might also have a beneficial effect by expanding FRC.

The present studies have shown that, in patients at risk for compression atelectasis and physiological shunting, impairment of O_2 exchange can be prevented by preemptively instituting HVT ventilation with VT of 12 to 15 $ml \cdot kg^{-1}$. Severe hypocapnia can be prevented by reducing fresh gas flow when using a partial rebreathing circuit or by adding dead space when using a circle system. We have not examined the effects of continuous positive airway pressure or PEEP, but will do so in a subsequent study. Effective measures to reverse established compression atelectasis are more problematic. In our patients HI plus reduction of intra-abdominal pressure restored oxygenation to near normal for at least 5 min, demonstrating that the gas exchange defect was reversible. Persistence of the post-HI improvement or supplementary measures to prevent recurrence of atelectasis, such as HI followed by HVT ventilation, has yet to be studied.

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