CONTINUOUS KETAMINE INFUSION FOR ONE-LUNG ANAESTHESIA

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ABSTRACT

The mechanism which normally affects distribution of blood flow through unventilated areas of the lung is hypoxic pulmonary vasoconstriction; this acts to divert the blood to well ventilated alveoli, resulting in a better ratio of ventilation to perfusion.

Several reports have focused attention on the reduction or abolition of this reflex in the unventilated lung by most of the volatile anaesthetic agents used in clinical practice. This response was not abolished by the intravenous anaesthetic agents.

One hundred and ten patients undergoing elective pulmonary resection were studied to evaluate the effect of a continuous infusion of ketamine during one-lung anaesthesia, by observing the changes in Pa_{O_2} as a reflection of shunt. Ketamine was chosen as the intravenous agent for its positive inotropic and chronotropic action. Additionally, by providing both analgesia and hypnosis, we were able to admininster inspired oxygen concentrations of 50–100 per cent without concern that the patient might have recall for events during operation.

We have demonstrated that in all cases a Pa_{O_2} in excess of 9.31 kPa (70 torr) was achieved with ketamine and FI_{O_2} 1.0 as well as an increase in shunt fraction from 25.9 per cent (FI_{O_2} 0.5) to 36.0 per cent (FI_{O_2} 1.0).

We feel that ketamine provides a satisfactory alternative to the volatile agents for one-lung anaesthesia in patients where relative hypoxaemia might be unacceptable during operation.

ONE LUNG VENTILATION with a high inspired oxygen fraction, a volatile anaesthetic agent and a muscle relaxant is commonly used for endobronchial anaesthesia, despite the fact that this technique has been associated with significant arterial hypoxaemia (Pa_{O_2} less than 9.33 kPa (70 torr)) in 15–25 per cent of cases.[1–3] This figure agrees with our own review of 100 cases of pulmonary resection for carcinoma anaesthetized with halothane and FI_{O_2} 1.0 during one-lung anaesthesia, where the Pa_{O_2} was less than 9.33 kPa (70 torr) in 20 per cent. The main factors contributing to this arterial hypoxaemia are: (1) Excessive pulmonary shunting in the unventilated, non-dependent lung.[4–6] (2) The development of atelectatic areas in the ventilated dependent lung, secondary to a reduction in functional residual capacity and increase in airway closure, leading to a relative mismatching of ventilation and blood flow with an increase in venous admixture.[6–8] (3) A reduction in cardiac output due to anaesthetic agents,[9–11] mechanical ventilation,[6,12,13] and manipulations during operation.

The major cause of hypoxaemia during one-lung anaesthesia is persistence of blood flow through the unventilated lung. Hypoxic pulmonary vasoconstriction is normally an important mechanism for reducing the amount of blood flowing through unventilated regions of the lung; this diverts the blood to well ventilated alveoli.[14–16]

Several reports, both in experimental animals and, most recently, in man,[4,5,17–19] have focused attention on the reduction or abolition of this reflex in the unventilated lung by most of the volatile anaesthetic agents used in clinical practice. This response was not abolished by the intravenous anaesthetic agents.[20,21] The use of continuous ketamine by "microdrip technique" after induction with diazepam and ketamine has been reported for most types of surgical procedure in all risk categories.[22,23] Vaughan and Stephen[24] reported on 19 thoracotomies, including six pulmonary resections, using a nitrous oxide/oxygen/curare technique with intermittent ketamine, given arbitrarily every 40 to 60 minutes. They did not indicate whether endobronchial techniques were employed, nor did they report on arterial blood gas values.

This study was undertaken to evaluate the effect of a continuous infusion of ketamine during one-lung anaesthesia by observing the changes in Pa_{O_2} and shunt fraction.

Ketamine was chosen as the intravenous agent for its positive inotropic and chronotropic action, as we wished to minimize the effect of a reduction in cardiac output on the shunt.[11] Additionally, by providing both analgesia and hyp-

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TABLE I

Patients	
Male	71
Female	39
Average age	62.2 years (range 19–91)
Diagnosis	
Carcinoma	76
Other malignant tumors	16
Benign tumors	9
Other	9
Surgical procedures	
Wedge resection	21
Lobectomy	65
Pneumonectomy	24

nosis, we were able to administer $F_{I_{O_2}}$ 0.5 and 1.0 as required without concern that the patient might have recall for events during operation.

Materials and Methods

One hundred and ten patients undergoing elective pulmonary resection were studied (Table I). Patients were interviewed extensively by the authors before operation and were excluded from the study if there was a history of uncontrolled hypertension, previous psychiatric disability or delirium on emergence from anaesthesia. Patients were examined specifically for evidence of ulnar collateral flow. A flow-directed balloon-tipped thermodilution pulmonary artery catheter was placed in those with a history of myocardial disease.

Patients were sedated on the evening before operation with diazepam 10–15 mg and premedicated 90 minutes before induction of anaesthesia with diazepam 10–25 mg by mouth and atropine 0.4–0.6 mg intramuscularly to overcome the excessive salivation associated with ketamine anaesthesia.

In the operating room adequate intravenuous access was obtained and anaesthesia was induced with droperidol 5 mg, diazepam 10–20 mg and ketamine 2 $mg \cdot kg^{-1}$. Relaxation was obtained with d-tubocurarine 30–45 mg and the trachea was intubated with a previously selected Carlens or Robertshaw double lumen bronchial tube. Anaesthesia was maintained with a continuous infusion of ketamine 2 $mg \cdot kg^{-1}$/hr, using an IVAC 530 micro-infusion pump* and curare was given in 9 mg increments as required. Ventilation was controlled with an Engstrom ventilator delivering 50 per cent nitrous oxide with oxygen at a rate of 12 breaths per minute with tidal volume set initally at 10–12 $ml \cdot kg^{-1}$ body weight and adjusted to maintain Pa_{CO_2} at 4.0–4.67 kPa (25–35 torr). During one-lung anaesthesia, full ventilation and expansion of the dependent lung was maintained by using the same tidal volume for both lungs. Arterial blood samples and, in 30 cases, mixed venous blood samples were obtained once the patient's state had stabilized, after ventilation for ten minutes in each of the following four situations:

Ventilation of both lungs at $F_{I_{O_2}}$ 0.5 and 1.0

Ventilation of one lung at $F_{I_{O_2}}$ 0.5 and 1.0

Heparinized blood samples were analyzed for pH, Pa_{O_2}, Pa_{CO_2} BE and SBE using an ABL-1 machine. Saturation was measured directly with an IL 182 co-oximeter.†

Shunt fraction was calculated using the original shurt equation:

$$Qs/Qt = (CcO_2 - CaO_2)/(CcO_2 - CvO_2)$$

utilizing Severinghaus' correction factors.

Pa_{O_2} values greater than 19.95 kPa (150 torr) were corrected for linearity, using the formula:

Actual Pa_{O_2} = indicated Pa_{O_2} + (Pa_{O_2} indicated $- 150 \times 0.1$)

Throughout each case, normothermia, normovolaemia and haematocrit were maintained as close as possible to the preoperative levels.

Results

During one-lung anaesthesia, none of the 110 patients had an arterial oxygen tension of less than 9.33 kPa (70 torr) on 100 per cent oxygen, the mean being 17.33 kPa (130 torr). With an inspired oxygen of 50 per cent the mean value for the arterial oxygen tension was 11.2 kPa (84 torr). Of these, five patients representing 4.5 per cent had an arterial oxygen tension of less than 9.33 kPa (70 torr) and then improved to a tension greater than 13.33 kPa (100 torr) on the introduction of 100 per cent oxygen (Table II).

The increase in the alveolar arterial oxygen gradient between ventilation of both lungs and then one lung indicates the degree of increased venous admixture occurring during one-lung anaesthesia. A significant increase of the $D(A\text{-}a)O_2$ mean to 28.33 kPa (213 torr) when 100 per cent oxygen is inspired compared to a mean of 10.77 kPa (81 torr) with 50 per cent oxygen reflects the influence of 100 per cent oxygen on absorption atelectasis in the dependent lung (Table

*IVAC Corp., San Diego, California.

†Instrumentation Laboratories, Lexington, Massachusetts.

TABLE II

MEAN Pa_{O_2} IN 110 PATIENTS

		kPa	(±S.D.)	torr	(±S.D.)
Both lungs	FI_{O_2} 0.5	21.12	(±12.53)	158.4	(±94)
	FI_{O_2} 1.0	43.73	(±12.53)	328.0	(±94)
One lung	FI_{O_2} 0.5	11.20	(±2.80)	84.0	(±21)
	FI_{O_2} 1.0	17.33	(±3.87)	130.0	(±29)
		MEAN D(A-a)O_2			
Both lungs	FI_{O_2} 0.5	21.89	(±4.80)	164.2	(±36)
	FI_{O_2} 1.0	47.12	(±14.53)	354.4	(±109)
One lung	FI_{O_2} 0.5	32.73	(±9.07)	245.5	(±68)
	FI_{O_2} 1.0	75.59	(±17.60)	567.0	(±132)

TABLE III

MEAN SHUNT FRACTION (Qs/Qt) PER CENT ±SD IN 30 PATIENTS

	FI_{O_2} 0.5	FI_{O_2} 1.0
Both lungs	13.6 (±4)	14.4 (±9)
One lung	25.9 (±6)	36.0 (±9)

II). This contention is borne out by the increase in calculated shunt from 25.9 per cent at FI_{O_2} 0.5 to 36.0 per cent at FI_{O_2} 1.0 (Table III).

DISCUSSION

Hypoxaemia during selective collapse of one lung is unacceptable where the technique is employed for surgical convenience only; while it may be anticipated in the elderly, the obese, or in those with pre-existing abnormal pulmonary function, it remains imposssible to predict which patient will be unable to tolerate hypoxaemia during endobronchial anaesthesia or, for that matter, during collapse of the lung by packs and retractors. Special situations do exist, however, where isolation of the dependent lung is mandatory, as in operations for bronchopleural fistula with empyema, traumatic rupture of the bronchus, or inflating solitary pulmonary bullae.

In these circumstances, ketamine appears to offer considerable advantages.

A decrease in the oxygen tension of alveolar gas results in a contraction of smooth muscle in the walls of the small arterioles in the hypoxic region: hypoxic pulmonary vasoconstriction. This mechanism is obscure; it also occurs in the isolated denervated lung, and may be induced experimentally by reducing the oxygen tension of pulmonary artery blood.[15,17,25] Furthermore, this pressor response is potentiated by a hypercapnic acidosis.[17,26] It may be induced experimentally by ventilating one side with an hypoxic mixture while perfusing the other with fully saturated blood. The effect is to direct the blood flow away from the hypoxic regions of the lung to well oxygenated areas. More blood is delivered throught the relative dead space areas of the dependent lung because of gravity dependent perfusion.[27,28] This results in a decrease in dead space, reduces the D(A-a)O_2 gradient and diminishes venous admixture. The end result is an improvement in arterial oxygen tension.[26]

The abolition of this reflex by volatile anaesthetic agents permits the return of pulmonary blood to the unventilated region, producing a true intrapulmonary right to left shunt. Here, increasing the inspired oxygen concentration will have no effect on the saturation of this portion of blood.

Following pneumonectomy or clamping of the pulmonary artery on the collapsed side, there is considerable improvement in the Pa_{O_2} and diminution in calculated shunt[29] demonstrating the persistence of pulmonary blood flow through the unventilated lung. This has been documented by flow studies with radioactive isotopes.[1,17]

Some degree of ventilation/perfusion inequality exists in the dependent lung and, in some instances, can make a considerable contribution to venous admixture and arterial desaturation.

The induction of anaesthesia, muscle paralysis and mechanical ventilation alters the normal ventilation/perfusion relationships with an increased D(A-a)O_2 gradient.[3] This is accentuated by the lateral position with a downward displacement of the mediastinum and a cephalad movement of the diaphram, resulting, sometimes, in a reduction in functional residual capa-

city and early airway closure, producing areas of hypoventilation.[6,12,27,28]

In this situation, increasing the inspired oxygen concentration can result in improved Pa_{O_2} by minimizing the ventilation to perfusion inequality in the hypoventilated area of the dependent lung. However, the higher blood solubility of the anaesthetic agents and the high inspired oxygen fraction may lead to absorption atelectasis and the development of areas of true shunt.[39,31]

In 95.5 per cent of the patients we demonstrated an acceptable level of Pa_{O_2} with ketamine and inspired oxygen of 50 per cent as well as an increase in shunt fraction when the inspired oxygen is increased from 50 to 100 per cent.

Undoubtedly, some of the improvement that occurs in Pa_{O_2} and calculated shunt with ketamine is due to greater cardiovascular stability. A comparison between the effects of ketamine and halothane on the haemodynamic parameters undertaken by Reves, *et al.*[11] demonstrated an increase in cardiac index, blood pressure and heart rate with ketamine and a decrease or an equivocal effect of halothane on those parameters. Overall, the negative effect of halothane leads to a fall in the ejection fraction, lowering the cardiac output and a proportional decrease in venous oxygen saturation with arterial hypoxaemia.[9,10,21]

The correlation between venous admixture and cardiac output is well recognized. Prys-Roberts[32] showed that the decrease in Pa_{O_2} paralleled the reduction in cardiac output. According to the Fick principle, the cardiac output is inversely proportional to the arterio-venous oxygen difference. A significant degree of hypotension will have the same result by its effect in cardiac output and shunt.

The rise in arterial blood pressure that accompanies the induction of anaesthesia with ketamine was not observed in this study. Diazepam has been shown to control the hypertension, tachycardia and increase in cardiac output to a large extent.[33] In addition, our technique requires a large dose of d-tubocurarine following the induction with ketamine. This does not result in a significant fall in blood pressure. If blood pressure starts to rise during operation it can be controlled by 9 mg increments of curare. On two occasions administration of chlorpromazine 5 mg returned blood pressure to control values; consequently, we have not excluded controlled hypertensive patients.

A great initial concern was the possibility of a high incidence of emergence reaction disturbing to the patient and creating resistance from the nursing personnel in the intensive care unit. While the probability of prolonged somnolence was not a consideration in this group, the majority were able to respond to commands within two hours of operation. Corssen and Domino[34] have suggested that the emergence phenomena may be related to stimuli during the awakening phase. The time between arousal and full orientation, in their study, was 8.5 minutes. An analysis of the dreams recalled by ketamine subjects was related to this period. They indicated that emergence phenomena were less likely to occur if external stimuli could be minimized during emergence from the anaesthetic.

There were no halucinatory phenomena, emergence delirium, or dreams in this group. We attribute our success in eliminating emergence penomena to the use of droperidol and diazepam immediately before induction, as proposed by Hatano[22,23] and Sadove[35]; and diazepam before discontinuing the ketamine. In addition, we feel that the time during induction with ketamine is extremely important, as a bad experience at this moment may precipitate anxiety which persists into the postoperative period. Therefore we maintain close contact with the patient during the slow induction with ketamine and during recovery, stroking and reassuring, saying the operation is over; it has been successful; they are coming around from the effects of the anaesthetic and need not be afraid. We make the patients aware that they are about to be transferred from the operating table to the intensive care bed and accompany them back to the intensive care unit, where the nurses have been taught to follow the same procedures. No questions are asked of the patient. Only reassurance is provided. Normal routine is followed in post-operative monitoring.

The advantages of the microdrip technique with ketamine, as described by Hatano, *et al.*,[22] provide for a greater degree of control over dosage and less total drug is administered. It is suggested that the incidence of emergence phenomena may be related to the total dose of ketamine employed per unit of time.

The use of 50 per cent nitrous oxide with ketamine enhances its analgesic effect, reduces the dose of ketamine and shortens the recovery period. It also provides more adequate visceral anaesthesia, thus reducing reflex phenomena;[34] though when we have used ketamine as the sole anaesthetic, the only effect appeared to be an

increase in blood pressure which could be managed by increasing the infusion rate of ketamine for that period.

Conclusion

We feel that ketamine provides a satisfactory alternative to halothane for one-lung anaesthesia in patients where the preoperative arterial oxygen values are low, for those who are haemodynamically unstable, or where relative hypoxaemia during operation might be unacceptable, as in patients with anaemia, cerebrovascular or myocardial insufficiency, as well as in those special situations where there is no safe alternative to the use of endobronchial anaesthesia.

In addition to its contribution to arterial desaturation during one-lung anaesthesia, the general use of halothane is now controversial for multiple reasons: the dilemma of hypersensitivity, operating room pollution and, most recently, its role as an immuno-suppressant[36,37] which is of particular concern in surgery where the spread of malignant tumours is important. Ketamine is not implicated in any of these areas.[38]

While no conclusion can be drawn from this study as to the mechanism for the superior arterial oxygenation during one-lung anaesthesia with ketamine in man, our animal studies indicate the role of ketamine in the preservation of the hypoxic vasoconstrictor reflex.[39]

While the use of continuous intravenous ketamine in thoracic anaesthesia needs to be further investigated, it appears to offer a safe, simple and economical anaesthetic technique.

REFERENCES

1. Tarhan, S. & Lundborg, R.O. Effects of increased expiratory pressure on blood gas tension and pulmonary shunting during thoractomy with use of the Carlen Catheter. Canad. Anaesth. Soc. J. *17*: 4–11 (1970).
2. Thomson, D.F. & Campbell, D. Changes in arterial oxygen tension during one-lung anaesthesia. Brit. J. Anaesth. *45*: 611–616 (1973).
3. Torda, T.A., McCulloch, C.H., O'Brien, H.D., *et al.* Pulmonary venous admixture during one-lung anesthesia. Anaesthesia *20*: 272–279 (1974).
4. Benumof, J.L. & Wahrenbrock, E.A. Local effects of anesthetic on regional hypoxic pulmonary vasoconstriction. Anesthesiology *43* (5): 525–532 (1975).
5. Bjertnaes, L.L. Hypoxia-induced pulmonary vasoconstriction in man: inhibition due to diethyl ether and halothan anesthesia. Acta Anaesth. Scand. *22*: 570–588 (1978).
6. Rehder, K. Pulmonary gas exchange during anesthesia and ventilation. Refresher Course in Anesthesiology. The American Society of Anesthesiologists, Inc., edited by S.G. Hershey *6*: 97–110 (1978).
7. Don, H.F., Wahba, W.M. & Craig, D.H. Airway closure gas trapping and the functional residual capacity during anesthesia. Anesthesiology *36*: 533–539 (1972).
8. Stone, J.G., Khambatta, H.J., Donham, R.T., *et al.* Pulmonary shunting during anaesthesia in man. Canad. Anaesth. Soc. J. *22*: 647–652 (1975).
9. Mahaffey, J.E., Aldinger, E.E., Sprouse, J.H., *et al.* The cardiovascular effects of halothane. Anesthesiology *22*: 982–986 (1961).
10. Morrow, D.H. & Morrow, A.G. The effects of halothane on myocardial contractile force and vascular resistance – direct observations made in patients during cardiopulmonary bypass. Anesthesiology *22*: 537–541 (1961).
11. Reves, J.G., Samuelson, P.N. & Younes, H.J. Anesthetic considerations for coronary artery surgery. Anesth. Rev. *4/8*: 19–31 (1977).
12. Froese, A.B. & Bryan, A.C. Effect of anesthesia and paralysis on diaphragmatic mechanism in man. Anesthesiology *41*: 242–255 (1974).
13. McCarthy, G.S. & Hedenstierna, G. Arterial oxygenation during artificial ventilation. Acta Anaesth. Scand. *22*: 563–569 (1978).
14. Euler, U.S. von & Liljestrand, G. Observations on the pulmonary arterial blood pressure in the cat. Acta Physiol. Scand. *12*: 301–320 (1946).
15. Haugea, A. Ventilation hypoxia and pulmonary vascular resistance. The relative effects of pulmonary arterial and alveolar Po_2. Acta Physiol. Scand. *76*: 121–130 (1969).
16. Hughes, J.M.B. Lung gas tensions and active regulation of ventilation-perfusion ratios in health and disease. Br. J. Dis. Chest *69*: 153–160 (1975).
17. Bjertnaes, L.J., Hauge, A., Nakken, K.F., *et al.* Hypoxic pulmonary vasoconstriction: Inhibition due to anaesthesia. Acta Physiol. Scand. *96*: 282–285 (1976).
18. Sykes, M.K., Hill, A.E.G., Loh, L., *et al.* Evaluation of a new method for the continuous measurement of the distribution of the blood flow between the two lungs. Brit. J. Anaesth. *49*: 285–292 (1977).
19. Sykes, M.K., Davies, D.M. & Chakrabarti, M.K. The effects of halothane on vascular resistance in the isolated perfused cat lung. Brit. J. Anaesth. *45*: (2) 655–665 (1973).
20. Berjenates, L.J. Hypoxia induced vasoconstriction in isolated perfused lungs exposed to injectable or inhalation anaesthetics. Acta Anaesth. Scand. *21*: 133–147 (1977).
21. Marshall, B.E., Hoffman, D.A., Neufeld, G.R., *et al.* Influence of induction agent on pulmonary venous admixture during halothane: oxygen anaesthesia with controlled respiration in man. Canad. Anaesth. Soc. J. *21*: 461–466 (1974).
22. Hatano, S., Keane, D.M., Boggs, R.E., *et al* Diazepam-ketamine anaesthesia for open-heart surgery. A "micro-mini" drip administration technique. Canad. Anaesth. Soc. J. *23*: 648–656 (1976).
23. Hatano, S., Nishiwada, M. & Matsumura, M. Ketamine-diazepam anesthesia for abdominal surgery. Anaesthesist *27*: 172–182 (1978).

24. VAUGHN, R.W. & STEPHEN, C.R. Abdonimal and thoracic surgery in adults with ketamine, nitrous oxide, and d-tubocurarine. Anesth. Analg. *53*: 271–280 (1974).
25. LOH, L., SEED, R.F. & SYKES, M.K. The cardiorespiratory effects of halothane, trichloroethylene and nitrous oxide on the dog. Brit. J. Anaesth. *45*: 125 (1973).
26. BERGOVSKY, E.H. Mechanisms underlying vasomotor regulation of regional pulmonary blood flow in normal and disease states. Am. J. Med. *57*: 378–394 (1974).
27. WEST, J.B. Blood flow to the lung and gas exchange. Anesthesiology *41*: 124–138 (1974).
28. WEST, J.B. Ventilation/Blood Flow and Gas Exchange. Third Edition, Oxford, Blackwell Scientific Publication (1977).
29. TARHAN, S. & LUNDBORG, R.O. Blood gas and pH studies during use of the Carlens catheter. Canad. Anaesth. Soc. J. *15*: 458–463 (1968).
30. NUNN, J.F., WILLIAMS, I.P., JONES, J.G., *et al.* Detection and reversal of pulmonary absorption collapse. Brit. J. Anaesth. *50*: 91–100 (1978).
31. SHAPIRO, A.R. & PETERS, R.M. Interpretation of alveolar-arterial oxygen tension difference. Surg. Gynec. Obst. *144*: 547–552 (1977).
32. PRYS-ROBERTS, C. & GREENBAUM, R. The influence of circulatory factors on arterial oxygenation during anaesthesia in man. Anaesthesia *22*: 257–263 (1967).
33. ALLEN, G.D. & EVERETT, G.B. Modification of cardiovascular effects of ketamine by diazepam. Anesth. Prog. *21*: 8–11 (1974).
34. CORSSEN, G. & DOMINO, E.F. Dissociative anesthesia: further pharmacologic studies and first clinical experience with phencyclidine derivative CI-581. Anesth. Analg. *45*: 29–40 (1966).
35. SADOVE, M.D., HANTOS, S., REDLIN, T., *et al.* Clinical study of droperidol in the prevention of side-effects of ketamine anesthesia. Anesth. Analg. *50*: 526–532 (1971).
36. CULLEN, B.F., HUME, R.B. & CHRETIEN, P.B. Phagocytosis during general anesthesia in man. Anesth. Analg. *54*: 501–504 (1975).
37. LEVIN, N., SILVERTON, J. & BRECHNER, V.L. A comparison of the effects of halothane and ketamine anesthesia on phytohemagglutinin induced transformation of human lymphocytes *in vivo*. Abstracts of Scientific Papers, American Society of Anesthesiologists, Inc., p. 99 (1972).
38. WIEBER, J., GUGLER, R., *et al.* Pharmakokinetics of ketamine in man. Anaesthetist *24*: 260–263 (1975).
39. LUMB, P.D., SILVAY, G., WEINREICH, A. & SHIANG, H. A comparison of the continuous ketamine infusion and halothane on oxygenation during one-lung anaesthesia in dogs. Canad. Anaesth. Soc. J. *26*: 394–401 (1979).

RÉSUMÉ

La vasoconstriction pulmonaire réflexe à l'hypoxie constitue le mécanisme normal qui vient modifier la distribution du volume sanguin dans les régions non ventilées du poumon. Ceci permet de dévier le flot sanguin vers les alvéoles bien ventilées et d'améliorer le rapport ventilation-perfusion.

Plusieurs travaux ont faut état de l'abolition de la diminution de ce réflexe, dans les régions du poumon non ventilées, par la plupart des agents volatils d'usage courant, alors que les anesthésiques intraveineux ne l'abolissent pas.

Nous avons étudié 110 opérés subissant une pneumonectomie élective, dans le but d'évaluer l'influence d'une perfusion continue de kétamine au cours d'anesthésies à un poumon. Les modifications de la Pa_{O_2} étaient utilisées comme reflets du shunt. Le choix de la kétamine comme agent d'anesthésie a été basé sur ses propriétés inotropes et chronotropes. En plus de fournir à la fois l'analgésie et l'amnésie, cet agent nous permettait d'administrer de l'oxygène à des concentrations de 50 à 100 pour cent sans crainte de mémorisation de la chirurgie par le patient. Dans tous les cas, nous avons observé une Pa_{O_2} supérieure à 9.31 kPa (70 torr) avec la perfusion de kétamine et une FI_{O_2} de 1.0, de même qu'une élévation du shunt qui passait de 25.9 à 36 pour cent en faisant passer la FI_{O_2} de 0.5 à 1.0.

En conclusion, nous considérons la kétamine comme une bonne alternative aux anesthésiques volatils au cours d'anesthésie à un seul poumon chez les patients susceptibles de mal tolérer un certain degré d'hypoxémie en cours de chirurgie.