Air Ventilation during Pulmonary Artery Banding Operation

Toshimichi TAKAHASHI, Yoko TOSAKI*, Ikuko SAKUMA and Yasuhiro SHIMADA

(Key words: Anesthetic management, Air ventilation, Pulmonary artery banding)

In pediatric cardiac anesthesia, we usually use high F_{IO_2} to avoid hypoxia due to surgical and/or anesthetic mainpulation. However, it is well known that high F_{IO_2} increases the pulmonary blood flow because of inhibition of hypoxic pulmonary vasoconstriction[1–3]. Thus, theoretically, ventilation with higher F_{IO_2} might be disadvantageous to the patients who have left to right (L-R) shunt. We evaluated the effects of air ventilation and 100% O_2 ventilation on pulmonary and systemic circulation during anesthesia in infants who underwent PA-banding for reducing high pulmonary blood flow due to atrial septal defect (ASD) and/or ventricular septal defect (VSD).

Case Presentation

Case 1: 58-day-old, male

A 30-year-old primipara was admitted to the hospital to undergo an emergency Caesarean section because of the monoumbilical artery and agenesis of left kidney of the fetus. She delivered a male neonate with 40-week-gestation, weight 2146g. Five days later the delivery, two dimensional echocardiography revealed VSD, patent foramen ovale (PFO), severe tricuspid valve regurgitation, and pulmonary hypertension. Digitalization was started. After 24 days, tachycardia, tachypnea, and retraction of the abdomen and chest wall appeared. He gradually manifested heart failure with cyanosis. Dopamine at a dose of 7.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ was started. One month after, however, heart failure did not subside, and Pa_{CO_2} was increased to 60 torr. Immediate endotracheal intubation and artificial ventilation were started and the emergency operation of PA-banding was proposed on the next day.

Preoperative physical examination revealed hepatomegaly and systolic murmur of grade 3/6 of Levin. Hematological examination revealed hemoglobin of 13.5 $g \cdot dl^{-1}$. The chest X-ray film showed marked cardiomegaly with a cardiothoracic ratio of 66%. Arterial blood gas (ABG) analysis at intermittent mandatory ventilation (IMV) with F_{IO_2} of 0.21 and respiratory rate (RR) of 40 $breath \cdot min^{-1}$ was pH 7.302, Pa_{CO_2} 39.3 torr, Pa_{O_2} 42.5 torr, Base Excess (BE) −6.3 $mEq \cdot l^{-1}$, HCO_3^- 19.4 $mEq \cdot l^{-1}$. Electrocardiogram (ECG) showed bi-

Department of Anesthesiology, Nagoya University School of Medicine, Nagoya, Japan

*Department of Anesthesia, Nagoya First Red-Cross Hospital, Nakamura-ku, Nagoya, Japan

Address reprint reqeusts to Dr. Takahashi: Department of Anesthesiology, Nagoya University School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya, 466 Japan

J Anesth 7:229–233, 1993

lateral ventricular hypertrophy. Body weight was 3762g, and he was 58 days old.

The blood pressure (BP) during the anesthesia was maintained at 60–70/30–40 mmHg. Surgery was done uneventfully.

Case 2: Two month-old, female

A female neonate as a twin sister was delivered after an uneventful 39-week-gestation and weight 2,282g at birth. Two days after the delivery, a heart murmur was noticed, and echocardiogram revealed muscular VSD and ASD. Because of respiratory insufficiency, she was transported to our hospital for PA banding at one month-old. After admission, digitalization and tube feeding were started. However, because of persistent tachypnea and tachycardia, she was intubated and artificially entilated. The ABG analysis at F_{IO_2} 0.21, IMV 30 beats·min^{-1} and PEEP 2 cmH$_2$O was pH 7.553, Pa_{CO_2} 40.8 torr, Pa_{O_2} 46.8 torr, HCO_3^- 35.8 mEq·l^{-1}, B.E. 13.5 mEq·l^{-1}. ECG showed left ventricular dominant biventricular hypertrophy with left axis deviation. A chest X-ray film revealed cardiomegaly with cardiothoracic ratio of 60%. The lung field was congestive. At the first PA banding, banding was too tight resulting in Pa_{O_2} 29 torr at F_{IO_2} 0.21. Seven days after the operation, the reoperation was performed.

Preoperative physical examination revealed the edema of eye lids and hepatomegaly. Laboratory data showed hypoproteinemia and elevated transaminases. Five mcg·kg^{-1}·min^{-1} of dopamine was administered because of aggravation of heart failure. Her body weight was 3,900g, and she was two months old.

BP during the second anesthesia was maintained between 80 and 100 mmHg of systolic pressure. Surgery was performed uneventfully.

Anesthetic Management

In both cases, anesthesia was induced with fentanyl 10 μg·kg^{-1} and pancuronium bromide 0.2 mg·kg^{-1} intravenously. After induction of anesthesia, the radial artery and the femoral vein were cannulated for monitoring systemic arterial pressure (Psa) and central venous pressure (Pcv). Anesthesia was maintained with intermittent injection of fentanyl and pancuronium bromide. Respiration was manually controlled with air.

We analyzed the blood gases from three pressure lines (Psa, Pcv, and Ppa), and Qp/Qs was calculated according to the conventional formula,

$$Qp/Qs = \frac{Sa_{O_2} - Scv_{O_2}}{Sa_{O_2} - Spa_{O_2}}$$

where Sa_{O_2} is the O_2 saturation of the systemic artery, Scv_{O_2} is the O_2 saturation of the central venous, and Spa_{O_2} is the O_2 saturation of the pulmonary artery. Sa_{O_2} was measured after PA was encircled with the tape, 100% O_2 instead of air was used for ventilation for 5 minutes, and then we calculated Qp/Qs. Then F_{IO_2} was returned to 21% again, and Qp/Qs was calculated.

The results of the measured variables are shown in table 1 and 2. In the case 1, 100% O_2 ventilation decreased Psa by 75% of the air ventilation. Qp/Qs during air ventilation was 6.9, and that during pure O_2 ventilation was 7.9. The ABG analysis revealed no progressive acidosis. Sa_{O_2} during air ventilation was kept greater than 95%. In the same way the comparison of Sa_{O_2} between air ventilation and 100% O_2 ventilation in the case 2 indicates that 100% O_2 ventilation decreased Psa by 93% of air ventilation. And the Qp/Qs during air ventilation was lower than that during 100% O_2 ventilation. The ABG analysis revealed no progressive acidosis (table 1).

The O_2 extraction ratio or utiliza-

Table 1. Hemodynamic data and blood gas data of the patients

		Case 1		Case 2	
F_{IO_2}		0.21	1.0	0.21	1.0
Psa	(mmHg)	45/31	34/28	79/52	74/50
Ppa	(mmHg)	35/17	29/15	39/18	45/20
Pcv	(mmHg)	10/6	7	11	11
Qp/Qs		6.9	7.9	3.1	4.9
Artery Blood Gas Analysis					
pH		7.49	7.47	7.40	7.45
Pa_{CO_2}	(torr)	38.1	37.4	31.8	37.1
Pa_{O_2}	(torr)	69.8	249.9	84.6	323.1
BE	$(mEq \cdot l^{-1})$	6.8	5.6	−3.4	0.8
HCO_3^-	$(mEq \cdot l^{-1})$	29.4	25.2	19.6	25.9

Table 2. Blood gas data and oxygen extraction ratio of the patients

		Case 1		Case 2	
F_{IO_2}		0.21	1.0	0.21	1.0
Psa_{O_2}	(torr)	69.8	249.9	84.6	323.1
Ssa_{O_2}	(%)	95.2	99.7	96.2	99.7
Csa_{O_2}	$(ml \cdot dl^{-1})$	20.1	21.5	15.0	16.2
Ppa_{O_2}	(torr)	52.4	82.3	53.2	130.7
Spa_{O_2}	(%)	89.7	97.3	86.2	98.4
Cpa_{O_2}	$(ml \cdot dl^{-1})$	18.9	20.5	13.3	15.4
Pcv_{O_2}	(torr)	37.6	47.1	36.2	49.1
Scv_{O_2}	(%)	56.2	74.2	63.4	79.2
Ccv_{O_2}	$(ml \cdot dl^{-1})$	11.8	15.6	9.8	12.3
$\frac{C(a-v)_{O_2}}{Ca_{O_2}}$		0.41	0.27	0.34	0.24

Main symbols: P, pressure; S, saturation; C, content.
Modifiers: sa, systemic artery; pa, pulmonary artery; cv, central vein
$C(a\text{-}v)O_2$ means Csa_{O_2} minus Ccv_{O_2}; Ca_{O_2} means Csa_{O_2}

tion coefficient ($\dot{V}o_2/\dot{D}_{O_2}$) is the fraction of delivered O_2 that is actually consumed. This can be expressed as $C(a\text{-}v)O_2$ divided by Ca_{O_2}. In both cases the O_2 extraction ratio with 100% O_2 ventilation decreased compared with air ventilation (table 2).

Discussion

In our study, Qp/Qs was reduced by the air ventilation, and simultaneously Psa was increased. This result is in contrast to the clinical investigation by Beekman et al[4]. His study demonstrated an increase in systemic vascular resistance (SVR) and arterial pressure by hyperoxia. No patient investigated by him, however, had an intracardiac shunt because of operative repair. Many textbook of cardiac anesthesia for the pediatric patients did not refer to the oxygen concentration of ventilation during PA-banding. Residents in anesthesia are apt to use high oxygen concentration since low oxygen saturation is common in many patients undergoing PA-banding. However, under the 100% O_2 ventilation, L-R shunt was increased by the decrease in pulmonary vascular resistance (PVR). Especially, in the patients with immature left ventricular function, the increase in L-R shunt may result in a marked decrease in systemic blood pressure. On the contrary, under the air ventilation large pulmonary muscular arteries were constricted by the reflex of autonomic nervous system, resulting in an increase in systemic blood flow and decreased L-R shunting.

The effect of air ventilation on the small pulmonary muscular arteries is influenced directly by the oxygen tensions of both the alveolar gas and the pulmonary artery blood, leading to hypoxic pulmonary vasoconstriction (HPV)[2,5–9]

The lung of banding side is collapsed during surgical procedure, resulting in an increase in PVR of the collapsed area. This increase in PVR is reportedly not caused by twisting or kinking of pulmonary vessels but by HPV mainly[10]. Thus, improvement in Qp/Qs may be expected by decreasing F_{IO_2}.

The major drawback for the use of air is inadequate oxygenation to the body. During 21% O_2 ventilation, does this critical level of O_2 delivery satisfy O_2 demand of the body? The O_2 extraction ratio or utilization coefficient ($\dot{V}O_2/\dot{D}_{O_2}$) is the fraction of delivered O_2 that is actually consumed. This can be expressed as $C(a\text{-}v)O_2$ divided by Ca_{O_2}, since the effect of the cardiac output is canceled out in the above equation. Normal values are around 0.25. The relationship between $\dot{D}_{O_2}$ and $\dot{V}O_2$ has been the subjects of numerous studies. Shibutani et al. showed that an increase of O_2 consumption ($\dot{V}O_2$) at O_2 delivery ($\dot{D}_{O_2}$) less than 330 $ml \cdot min^{-1} \cdot M^2$ develops tissue O_2 deprivation[11]. The patients with the congenital heart disease (CHD) have varying degrees of hypoxemia. These pathophysiologic mechanisms of the patients with CHD may alter the diffusion mechanism of the O_2 delivery. For example, oxyhemoglobin dissociation curve of the patients with CHD patients, primordial environment where the O_2 tension required by the mitchondoria, and capillary networks of the tissue may differ from the normal patients[12,13]. However, these has been no information about the difference between CHD patients and normal patients. In our study the O_2 extraction ratio during air ventilation was higher than that during 100% O_2 ventilation.

In our cases the O_2 saturation detected by the pulse oximeter (Sp_{O_2}) was more than 70%, and systemic acidosis was not observed during anesthesia. Thus, O_2 delivery was sufficient in maintaining the systemic circulatory status.

In summary, air ventilation may be preferable to high O_2 ventilation in patients with L-R shunt during anesthesia for PA banding.

(Received Jun. 22, 1992, accepted for publication Jul. 9, 1992)

References

1. Dugard A, Naimark A: Effect of hypoxia on distribution of pulmonary blood flow. J Appl Physiol 23:663–671, 1967
2. Melot C, Naeije R, Hallemans R, et al: Hypoxic pulmonary vasoconstriction and pulmonary gas exchange in normal man. Respir Physiol 68:11–27, 1987
3. Von Euler US, Liljestrand G: Observations on the pulmonary arterial blood pressure in the cat. Acta Physiol Scand 12:301–320, 1946
4. Beekman RH, Rocchini AP, Rosenthal A: Cardiovascular effects of breathing 95 percent oxygen in chil-

dren with congenital heart disease. Am J Cardiol 17:106–111, 1983
5. Fishman AP: Hypoxia on the pulmonary circulation how and where it acts. Crit Res 38:221–231, 1976
6. West JB, Wagner PD: Pulmonary gas exchange. In: Bioenginering aspects of the lung. Vol. 3, edited by J.B. West. New York, Dekker, pp. 361–458, 1977
7. Marshall C, Marchall B: Site and sensitivity for stimulation of hypoxic pulmonary vasoconstriction. J Appl Physiol 55:711–716, 1983
8. Hales CA: The site and mechanism of oxygen sensing for the pulmonary vessels. Chest 88:235S–240S, 1985
9. Staub NC: Site of hypoxsic pulmonary vasoconstriction. Chest 88:240S–245S, 1985
10. Miller FL, Chen L, Malmkvist G, et al: Mechanical factors do not influence blood flow distribution in atelectasis. Anesthesiology 70:481–488, 1989
11. Shibutani K, Komatsu T, Kubal K, et al: Critical level of oxygen delivery in anesthetized man. Crit Care Med 11:640–643, 1883
12. Granger HJ, Goodman AH, Cook BK: Metabolic models of microcirculatory regulation. Fed Proc 34:2025–2030, 1975
13. Robin ED: Of men and mitchondria: Coping with hypoxic dysoxia. The 1980 J. Burns Amberson Lecture. Am Rev Resp Dis 122:517–531, 1980