Respiratory Rate Change during Balloon Valvuloplasty

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(Key words: aortic stenosis, pulmonary stenosis, respiration)

Balloon valvuloplasty has become widely used to treat congenital pulmonary and aortic valve stenosis since 1982[1], and anesthesia is often maintained under spontaneous breathing without tracheal intubation. Suárez de Lezo et al.[2] reported that pulmonary and aortic vavle valloon inflations induce drastic changes in arterial and ventricular pressures. These hemodynamic changes may induce arterial oxygen desaturation. The decreases in carotid sinus pressure and/or pressure loading in cardiopulmonary mechanoreceptors are known to increase respiratory rate[3–7]. And also hypoxia causes tachypnea[8–10]. Thus respiration may be affected strongly by arterial and cardiopulmonary baroreflexes, and chemoreflex during balloon valvuloplasty under spontaneous breathing. In this study we evaluated the change in respiratory rate during balloon valvuloplasty.

Case Report

This study was approved by the Human Studies Committee of Kyoto Prefectural University of Medicine, and informed consent was obtained from each patient. Characteristics of seven patients before ballooning were presented in table 1. Patient 1 and 2 had no intracardiac shunt with pulmonary stenosis, and both patients 3 and 4 had atrial septal defect and both patients 5 and 6 had foramen ovale for intracardiac shunt with pulmonary stenosis. Patient 7 had aortic stenosis. All patient had no complication except for these cardiac anomalies. Premedication was given with intramuscular atropine sulfate (0.01 $mg \cdot kg^{-1}$) and hydroxydine (1 $mg \cdot kg^{-1}$), and with chloral hydrate suppository (50 $mg \cdot kg^{-1}$) 30 min before induction of anesthesia. Anesthesia was induced with ketamine (2 $mg \cdot kg^{-1}$ i.v.) and diazepam (0.1 $mg \cdot kg^{-1}$ i.v.). Ketamine (30–50 $\mu g \cdot kg^{-1} \cdot min^{-1}$) was used to maintain anesthesia under spontaneous breathing. Heparin (1 $mg \cdot kg^{-1}$) was injected for anticoagulation.

End-tidal CO_2 pressure with capnograph ($P_{ET}CO_2$, mmHg) and respiratory rate (f, breaths$\cdot min^{-1}$) was continuously monitored (Nellcor N-1000). Respiratory rate was counted from the changes in breath by breath $P_{ET}CO_2$. The tip of 3 Fr catheter (Atom Indwelling feeding tube, Tokyo) was positioned in the vestibule of nose and

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Table 1. Characteristics of each patient and control values of respiratory rate, mean arterial pressure and mean ventricular pressure

Patient No.	Age/ Gender	Disease	Intracardiac shunt	f, breaths·min^{-1}	MAP, mmHg	MRVP or MLVP mmHg
1	2/F	PS	none	27.6 ± 5.3	84.6 ± 3.2	14.8 ± 1.7 (MRVP)
2	4/M	PS	none	29.8 ± 0.7	86.8 ± 0.9	–
3	4/F	PS	Atrial Septal Defect	19.7 ± 0.5	72.1 ± 2.4	17.0 ± 0.6 (MRVP)
4	1/F	PS	Atrial Septal Defect	33.4 ± 1.1	64.2 ± 1.4	–
5	1/M	PS	Foramen Ovale	33.0 ± 0.5	70.2 ± 0.6	22.4 ± 0.2 (MRVP)
6	1/M	PS	Foramen Ovale	30.5 ± 0.6	68.8 ± 1.0	–
7	2/M	AS	none	25.3 ± 1.3	53.1 ± 0.5	37.3 ± 0.9 (MLVP)

Values are the mean ± SEM of each inflation. PS, pulmonary stenosis; AS, aortic stenosis; f, respiratory rate; MAP, mean arterial pressure; MRVP, mean right ventricular pressure; MLVP, mean left ventricular pressure.

the sample gas for measuring P_{ETCO_2} was continuously suctioned through the catheter with 50 ml·min^{-1} flow rate. The P_{ETCO_2} expiratory plateau phase was confirmed for proper position of the catheter since the expiratory plateau indicated that mixed alveolar gas reached the CO_2 sensor.

Oxygen saturation by pulse oxymeter (Sp_{O_2}, %) was also monitored continuously using a Nellcor N-1000. The pulse oxymeter sensor was positioned over the second finger.

The right femoral artery was cannulated for monitoring systemic arterial pressure. In patient 1–6, a Swan-Ganz catheter was inserted into the left femoral vein and advanced to the right ventricle for monitoring right ventricular pressure. To reduce the pulmonary valve stenosis, a balloon catheter (TORAY, Tokyo) was inserted from the right femoral vein until the tip was located in the proximal main pulmonary artery. In patient 7, the balloon catheter was inserted from the left femoral artery until the tip was located in the left ventricle, and left ventricular pressure was monitored. In patient 2, 4 and 6, right ventricular pressure could not be measured due to technical difficulty. Each value was recorded on an analogue data recorder (Teac MR-30, Tokyo) and analyzed after the procedure by retracing the recorded data on a chart recorder (San-ei Recti-Horiz-8K, Tokyo).

Before the balloon inflation, 100% oxygen gas (3 l·min^{-1} with face mask) was given and the Sp_{O_2} was confirmed to 100%. Balloon inflation was repeated several times in each patient. Each inflation-inflation cycle was more than 1 min to achieve stabilization of each value. Balloon cavity was filled with iopamidol, a contrast medium, to confirm the balloon inflation with X-ray fluoroscopy. Complete expansion of the pulmonary valve was assured under X-ray fluoroscopy. The duration of each inflation was measured as the time from the beginning of inflation to the complete deflation.

Control values of mean arterial pres-

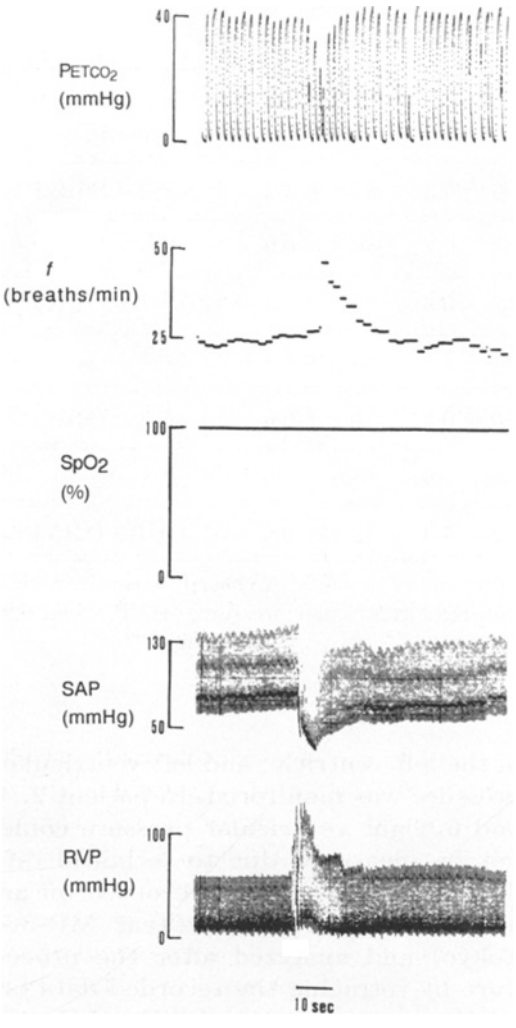

Fig. 1. Typical pattern of end tidal CO_2 pressure ($P_{ET}CO_2$), respiratory rate (f), oxygen saturation by pulse oxymeter (Sp_{O_2}), systemic arterial pressure and right ventricular pressure in patient 1 with no intracardiac shunt.

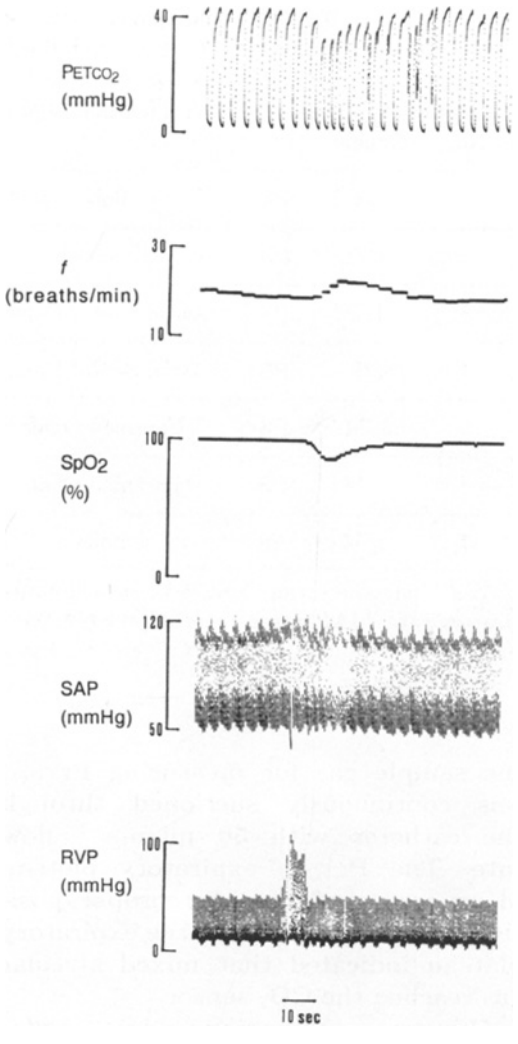

Fig. 2. Typical pattern of the same values in figure 1 in patient 3 with intracardiac shunt.

sure (MAP), mean right or left ventricular pressure (MRVP or MLVP) and f just before each inflation are shown in table 1. Figure 1 shows the typical pattern of each value in patient 1 with pulmonary stenosis with no intracardiac shunt, and figure 2. shows the typical pattern of each value in patient 3 with pulmonary stenosis with intracardiac shunt. Table 2 summarized the results on seven patients. The peak changes in MAP (ΔMAP), MRVP or MLVP (ΔMRVP or ΔMLVP), f (Δf) and Sp_{O_2} (ΔSp_{O_2}) from the control value during the inflation are shown. The number and the time duration of the inflation are also shown in table 2. Figure 3 shows the relationship between Δf and ΔMAP. Δf was correlated with ΔMAP ($R = -0.70$) and the regression equation was $\Delta f = -0.57 \times \Delta MAP + 1.87$. However, ΔMRVP, ΔMLVP and ΔSp_{O_2} showed no significant correlations with Δf.

Table 2. Changes of respiratory rate, mean arterial pressure, mean ventricular pressure and oxygen saturation during balloon inflation

Patient No.	Δf, breaths·min^{-1}	ΔMAP, mmHg	ΔMRVP or ΔMLVP mmHg	ΔSp_{O_2}, %	Inflation time, sec	n
1	18.0 ± 5.4	−45.5 ± 2.7	28.4 ± 3.1 (ΔMRVP)	0	6.6 ± 1.0	5
2	5.8 ± 0.9	−10.7 ± 1.4	–	− 9.4 ± 1.2	2.4 ± 1.7	9
3	4.7 ± 1.0	− 8.1 ± 1.8	28.0 ± 1.0 (ΔMRVP)	− 5.8 ± 0.8	6.1 ± 0.5	9
4	11.1 ± 2.3	−15.5 ± 1.0	–	−22.7 ± 3.9	11.8 ± 1.2	10
5	11.9 ± 1.1	−23.2 ± 1.0	25.9 ± 1.2 (ΔMRVP)	− 4.9 ± 0.9	8.6 ± 0.8	10
6	15.2 ± 1.9	−18.1 ± 1.4	–	− 8.3 ± 0.8	14.2 ± 1.1	6
7	23.4 ± 6.3	−28.6 ± 1.6	32.1 ± 2.2 (ΔMLVP)	–	8.8 ± 1.7	5

Values are the means ± SEM of each inflation. Δf, respiratory rate change; ΔMAP, mean arterial pressure change; ΔMRVP, mean right ventricular pressure change; ΔMLVP, mean left ventricular pressure change; ΔSp_{O_2}, oxygen saturation change by pulse oxymeter; n, No. of inflations.

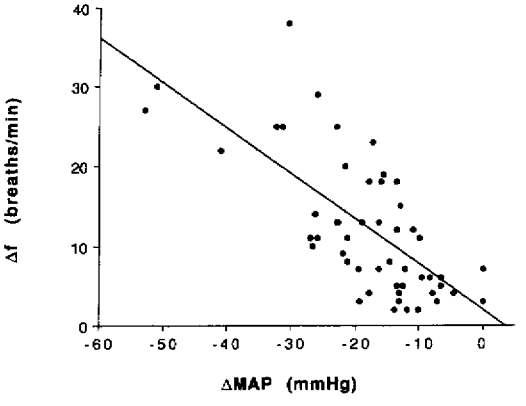

Fig. 3. Relationship between changes of f (Δf) and changes of mean arterial pressure (ΔMAP) in each inflation. $\Delta f = -0.57 \times \Delta MAP + 1.87$, $r = -0.70$ ($P < 0.001$).

Discussion

In the present study, the balloon inflation increased ventricular pressure and decreased systemic arterial pressure by obstructing cardiac output from the ventricle in the patients having pulmonary stenosis without intracardiac shunt or aortic stenosis. On the other hand, in the patients having pulmonary stenosis with intracardiac shunt, the balloon inflation increased right ventricular pressure to the same degree, but the decrease in systemic arterial pressure was attenuated by the increase in intracardiac right-to-left shunt flow[2]. These stepwise dicreases in MAP were accompanied with the increase in *f*, and there was a significant correlation between Δ*f* and ΔMAP. Fifty mmHg decrease in MAP caused 30 breaths·min^{-1} increase in *f*. Brunner et al.[3] reported that a decrease in pressure in isolated carotid sinus from 200 to 50 mmHg increases *f* from 4.8 to 9.7 breaths·min^{-1} in dogs, and the decrease in MAP affects *f* through arterial baroreflex. Chapman et al.[11] reported that the reduction of cerabral blood flow in goats increases *f*. We suggest that in balloon valvuloplasty the increase in *f* accompanied with the decrease in MAP may be caused by the response of arterial baroreflex and the reduction of cerebral blood flow.

In patient 3 as shown in figure 2, f increased without the decrease in MAP and with the decrease in Sp_{O_2} (Δf was 5 breaths·min^{-1}). Right ventricular pressure loading was reported to increase respiration[5,6], and Uchida[12] observed that there are mechanoreceptors present in the right ventricle, Lloyd[7] showed that an increase in left atrial pressure induces the increase in Δf to 4 breaths·min^{-1} with little decrease in systemic arterial pressure. These investigators suggested that cardiopulmonary pressure loading increases f. On the other hand, hypoxia in aortic and carotid bodies was reported to cause stimulation of respiration through chemoreceptors[9,10], and also brain hypoxia caused tachypnea[8]. Both cardiopulmonary pressure loading and chemoreflex by arterial oxygen desaturation may increase f in balloon valvuloplasty, but were relatively minor effects on f compared with the effect of the decrease in MAP.

In summary, we evaluated the change in f during balloon valvuloplasty, and suggest that f increases proportionally with the decrease in MAP in addition to a few increase in f by cardiopulmonary pressure loading and arterial oxygen desaturation.

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