Ventilatory Alterations Associated with Operation for Tetralogy of Fallot

Honorato F. Nicodemus, M.D., and John J. Downes, M.D.†*

Minute volumes ($\dot{V}_E$), physiologic deadspace–tidal volume ratios (V_{DP}/V_T) and arterial blood gas tensions were determined in children with Fallot's tetralogy prior to, one day, and two weeks, after shunt procedures and total correction. Preoperatively, patients with functioning shunts had higher Pa_{O_2} values than those without. Both groups had $\dot{V}_E$ values higher than those predicted by the Radford nomogram and mild compensated respiratory alkalosis.

Twenty-four hours after operation, in the total-correction group (TC) Pa_{O_2} during breathing of air fell from the preoperative value of 48 to 42 mm Hg; in the shunt-procedure group (SP) it rose from 35 to 50 mm Hg. Two weeks after operation, Pa_{O_2} in the TC group had risen to 72 mm Hg but in the SP group it had fallen to 38 mm Hg. V_{DP}/V_T ratio decreased from 0.32 preoperatively to 0.28 24 hours after operation in the TC group but rose from 0.35 to 0.41 in the SP group. $\dot{V}_E$ remained approximately twice that predicted by the Radford nomogram in both groups throughout the postoperative period. Although $\dot{V}_E$ remained high, Pa_{CO_2} rose in both groups 24 hours after operation. In the SP group Pa_{CO_2} remained above preoperative levels two weeks later despite comparable hypoxemia.

TETRALOGY OF FALLOT, the most common form of cyanotic congenital heart disease in infants and children, results in severe diminution of pulmonary blood flow, due to pulmonary outflow tract obstruction, and a large venous-to-arterial shunt through a ventricular septal defect. In infants and small children, creation of an anastomosis between the aorta or subclavian artery and a pulmonary artery improves pulmonary blood flow. This permits delay of definitive surgical correction until the patient is older and better able to survive cardiopulmonary bypass and total correction. Children with concurrent pulmonary disease, such as bronchial asthma, and on rare occasion those without known pulmonary disorders, may develop acute respiratory failure with extreme hypoxemia and hypercapnia in the immediate postoperative period.[1, 2] This has also been observed following total correction.

We undertook this study to determine the minute volume, physiologic deadspace, and blood gas tension changes associated with operation in infants and children with tetralogy who were free of known pulmonary disorders. One of these children underwent cavopulmonary arterial anastomosis (Glenn), and six, a systemic-to-pulmonary arterial anastomosis (Blalock–Taussig or Waterston).[3] An additional seven patients, each with a functioning systemic arterial-to-pulmonary arterial anastomosis created five to 12 years previously, underwent total correction of tetralogy during cardiopulmonary bypass.

Clinical Material and Methods

The patients were divided into two groups. The first consisted of seven children with a mean age of 12 years (range: 7 to 17 years) who underwent total correction of tetralogy with cardiopulmonary bypass (TC). The other group consisted of seven children with a mean age of 7 years (range 6 months to 12 years) who underwent creation of a surgical shunt

* Fellow in Pediatric Anesthesiology, Children's Hospital; Instructor in Anesthesia, University of Pennsylvania School of Medicine.

† Senior Anesthesiologist, Children's Hospital; Assistant Professor of Anesthesia and Pediatrics, University of Pennsylvania School of Medicine.

Received from the Division of Anesthesiology, Children's Hospital of Philadelphia, and Department of Anesthesia, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania. Accepted for publication May 13, 1969. Supported by Grants HD-01412, FR 5506, and HD-00201 from the National Institutes of Health, Bethesda, Md. Presented at the Annual Meeting of the American Society of Anesthesiologists, Washington, D. C., 1968.

TABLE 1. Arterial Blood-gas Tensions and

Patient	Sex	Age (years)	Weight (kg)	Weight Percentile for Age	Preoperative								
					$P_{a_{O_2}}$ (mm Hg) $F_{I_{O_2}}$.21	$P_{a_{CO_2}}$ (mm Hg)	$\dot{V}_E$ (l/min)	$\dot{V}_E/\dot{V}_{E_p}$*	f (breaths/min)	V_T/kg (ml)	V_{D_P}/kg (ml)	V_{D_P}/V_T
Total-correction group												
1	M	13	29	< 3	52	32	5.11	1.5	15	12	3	0.28
2	F	10	18	< 3	64	39	3.47	1.4	25	8	3	0.43
3	F	17	46	<10	45	37	9.74	2.1	22	10	4	0.37
4	M	13	35	<10	45	26	8.44	2.1	20	12	5	0.41
5	M	11	27	< 3	42	29	6.06	1.5	16	14	5	0.38
6	M	11	25	< 3	52	27	7.56	2.2	17	18	2	0.14
7	M	7	22	50	36	25	8.64	2.3	33	10	3	0.27
Mean		12	29	—	48	29	—	1.8	21	12	4	0.32
N		7	7	—	7	7	—	7	7	7	7	7
SD		—	9.3	—	9	5	—	—	6	3	1	—
Shunt-procedure group												
8	F	5	18	25	27	35	5.65	2.2	39	8	2	0.24
9	M	11	45	90	—	30	8.75	1.6	24	8	3	0.39
10	M	12	44	75	35	36	11.14	2.3	19	13	4	0.31
11	F	4	15	25	35	29	3.51	1.3	22	11	4	0.42
12	M	½	6	<10	34	29	4.64	3.6	50	15	***	***
13	F	11	43	90	44	34	10.42	2.3	24	10	3	0.34
14	F	4	13	3	**	—	—	—	—	—	—	—
Mean		7	26	—	35	29	—	2.1	29	11	3	0.35
N		7	7	—	5	6	—	6	6	6	5	5
SD		—	16.9		6	3	—	—	11	2	2	—

* $\dot{V}_{E_o}/\dot{V}_{E_p}$ = ratio of observed $\dot{V}_E$ to the $\dot{V}_E$ predicted according to body weight by the Radford nomogram[7] corrected to BTPS.
** Cyanotic episode occurred during sampling; data not representative.
*** Measurement error.

(SP). Studies were performed the day prior to operation, within 24 hours after operation, and two to three weeks later. In each study the patient breathed air ($F_{I_{O_2}}$ 0.21) through a low-deadspace, low-resistance, nonrebreathing valve and Rendell-Baker mask. Total apparatus deadspace was 8 ml. When ventilation appeared stable, expired gas was collected for three minutes in a meteorologic balloon and the frequency of breathing recorded. An arterial blood sample was obtained during the last minute of gas sampling from the femoral or brachial artery after previous local infiltration with one per cent lidocaine (Xylocaine®). These techniques result in a preoperative minute volume that averages 25 per cent greater than that predicted by the Radford nomogram in an acyanotic child with uncomplicated aortic coarctation.

Arterial blood was analyzed in duplicate for *p*H and $P_{a_{CO_2}}$ by the Astrup method[4] with a Radiometer AME-1 apparatus, and $P_{a_{O_2}}$ was determined with a platinum microtip electrode (Radiometer-Beckman). Base excess (B.E.) was calculated from the Siggaard-Andersen nomogram.[5] Total expired gas volume was measured in a water-sealed spirometer, and minute volume ($\dot{V}_E$) and tidal volume (V_T) determined. An aliquot of expired gas was analyzed in duplicate for carbon dioxide concentration with an infrared analyzer previously calibrated with gases of known carbon dioxide concentrations. The mean expired carbon dioxide tension was then calculated. All blood and gas values were corrected to body temperature and saturation (BTPS). Using the Bohr–Enghoff[6] equation, physiologic deadspace (V_{D_P}) and physiologic deadspace-to-tidal volume ratio (V_{D_P}/V_T) were calculated. The patient's predicted minute volume was obtained from the Radford nomogram[7] at BTPS. Anesthesia for the SP group consisted of cyclopropane and oxygen in a circle absorber system, whereas for the TC group, halothane–oxygen–*d*-tubocurarine was used in conjunction with a Sarnes pump and disk oxygenator for cardiopulmonary bypass.

Ventilation in Children with Tetralogy of Fallot

24 Hours Postoperative								2(3) Weeks Postoperative							
Pa_{O_2} (mm Hg) $F_{I_{O_2}}$ .21	Pa_{CO_2} (mm Hg)	$\dot{V}_E$ (l/min)	$\dot{V}_E/\dot{V}_{E_P}$*	f (breaths/min)	V_T/kg (ml)	V_{D_P}/kg (ml)	V_{D_P}/V_T	Pa_{O_2} (mm Hg) $F_{I_{O_2}}$ .21	Pa_{CO_2} (mm Hg)	$\dot{V}_E$ (l/min)	$\dot{V}_E/\dot{V}_{E_P}$*	f (breaths/min	V_T/kg (ml)	V_{D_P}/kg (ml)	V_{D_P}/V_T
35	40	3.20	0.9	22	5	2	0.41	80	40	6.07	1.8	47	4	2	0.43
—	34	4.84	1.9	31	9	3	0.35	66	33	6.26	2.5	27	13	4	0.30
—	27	5.97	1.3	29	4	1	0.18	62	29	7.40	1.6	24	7	1	0.10
50	34	7.32	1.8	26	8	2	0.29	87	36	9.45	2.3	37	7	3	0.42
—	25	4.52	1.1	29	8	1	0.18	73	36	5.70	1.4	25	8	2	0.20
40	32	8.14	2.3	34	10	3	0.32	72	30	6.52	1.9	19	13	3	0.19
45	36	5.98	1.6	34	8	2	0.24	69	35	8.01	2.1	50	7	3	0.41
42	32	—	1.5	28	7	2	0.28	72	34	—	1.9	32	8	2	0.29
4	7	—	7	7	7	7	7	7	7	—	7	7	7	7	7
6	5	—	—	6	2	1	—	8	4	—	—	12	8	0.5	—
59	41	1.89	0.7	***	***	***	0.43	36	36	5.48	2.1	38	8	2	0.31
—	35	8.47	1.5	36	5	4	0.52	35	34	10.30	1.9	30	8	3	0.45
48	45	10.63	2.2	31	8	4	0.59	38	37	12.00	2.2	23	12	5	0.45
45	34	4.32	1.6	43	7	2	0.35	48	30	4.30	1.6	31	9	5	0.50
54	31	2.86	2.2	45	10	2	0.41	36	30	2.63	2.0	45	10	5	0.46
45	36	8.92	2.0	38	5	2	0.37	43	37	10.00	2.0	30	8	3	0.35
—	30	4.32	2.0	29	11	2	0.23	35	25	5.87	2.7	35	13	3	0.17
50	36	—	1.7	37	8	3	0.41	38	32	—	2.1	33	10	4	0.38
5	7	—	7	6	6	6	7	7	7	—	7	7	7	7	7
6	5	—	0.5	6	2	1	—	5	4	—	—	7	2	1	—

Results

Ages, weights, blood gas tensions, and ventilation data are given in table 1.

Prior to operation, while breathing air ($F_{I_{O_2}}$ 0.21), patients in the SP group had a mean Pa_{O_2} of 35 mm Hg, compared with a mean of 48 mm Hg in the TC group ($P < 0.01$). The mean preoperative Pa_{CO_2} was 29 mm Hg in each group.

Twenty-four hours following operation, each patient in the SP group at $F_{I_{O_2}}$ 0.21 had an increase in Pa_{O_2} with a mean of 50 mm Hg, and an increase in Pa_{CO_2} to 36 mm Hg, ($P < 0.01$). However, two to three weeks following operation, the mean Pa_{O_2} had decreased to 38 mm Hg while Pa_{CO_2} decreased to 32 mm Hg, not different from the preoperative levels despite functioning shunts. In the TC group, 24 hours following operation, Pa_{O_2} had decreased to a mean of 42 mm Hg associated with a slight rise in Pa_{CO_2} to 32 mm Hg. Three weeks following operation, Pa_{O_2} increased in each patient to a mean of 72 mm Hg, although none of the values were within our normal range of 95–105 mm Hg for children of the same age. Pa_{CO_2} rose to 34 mm Hg, from the preoperative level ($P < 0.01$).

Acid–Base Status

Preoperative arterial acid–base values, presented in table 2, indicate that the children in both groups had mild, compensated hypocapnia. Mean arterial pH (pH_a) values in both groups were normal. One child in each patient group (patients 2 and 13) had a pH_a value below the normal fasting lower limit of 7.35, associated with a Pa_{CO_2} within normal limits (34–40 mm Hg). Patients with normal fasting pH_a values (7.35–7.45) had mild hypocapnia (Pa_{CO_2} 27–32 mm Hg) and mild base deficits (B.E. −4.5 to −7.5 mEq/l). A child in the TC group (patient 6) had a slightly alkaline pH of 7.46. These findings are similar to those reported by Bing and associates[3]; the B.E. of −4.9 mEq/l confirms their assumption that there is increased elimination of bicarbonate.

TABLE 2. Preoperative Arterial Acid-Base Values in Tetralogy of Fallot

Patient	pH$_a$	Pa_{CO_2} (mm Hg)	B.E. (mEq/l)
Total-correction group			
1	7.39	32	−4.5
2	7.34	39	−4.0
3	7.38	27	−7.5
4	7.44	26	−6.0
5	7.45	29	−4.5
6	7.46	27	−2.5
7	7.34	25	−5.2
Mean	7.41	29	−4.9
N	7	7	7
SD	.04	5	1.6
Shunt-procedure group			
8	7.44	25	−4.5
9	7.38	30	−5.5
10	7.44	30	−3.0
11	7.40	29	−5.0
12	7.35	29	−8.0
13	7.32	34	−8.0
14*	—	—	—
Mean	7.38	29	−5.6
N	6	6	6
SD	.05	3	1.8

* Cyanotic episode during sampling.

In the TC group, the preoperative and two-week-postoperative Pa_{CO_2} values bore a direct linear relation to Pa_{O_2} during breathing of air ($r = 0.80$, $P < 0.01$) (fig. 1). A significant relationship between Pa_{CO_2} and Pa_{O_2} did not exist in the SP group. The standard error of the estimate (σ xy) of the regression in figure 1 provides the limits for Pa_{CO_2} associated with a given Pa_{O_2} in patients with tetralogy free of pulmonary complication having functioning systemic-to-pulmonary arterial shunts.

VENTILATION

Prior to operation, the observed $\dot{V}E$ in each patient group averaged approximately twice the resting $\dot{V}E$ predicted by the Radford nomogram. Both elevated respiratory rates and elevated tidal volumes contributed to this hyperventilation. The mean physiologic dead-space (V_{DP}) and V_{DP}/V_T values of both groups were within normal limits. However, two patients in the TC group and one in the SP group had V_{DP}/V_T ratios above 0.40 (patients 2, 4, and 11).

Twenty-four hours following operation, $\dot{V}E$ remained elevated in both groups, averaging 1.5 to 1.7 times predicted values. The V_{DP}/V_T ratios increased in four of the five patients studied in the SP group. The mean V_{DP}/V_T ratio rose from 0.35 to 0.41; mean V_{DP}/kg did not change, although individual responses varied. By contrast, five of the seven patients studied in the TC group demonstrated decreases in V_{DP}/kg and V_{DP}/V_T ratios. The mean V_{DP}/kg fell from 4 to 2 ml, and the mean V_{DP}/V_T ratio decreased from 0.32 to 0.28.

Three weeks postoperatively, the mean $\dot{V}E$ remained elevated at approximately twice predicted levels in both groups. Mean V_{DP} and V_{DP}/V_T ratio remained below preoperative values in the TC group, in contrast to a slight rise in the SP group. V_T/kg in the SP group tended to approach control levels after three weeks.

Discussion

The SP group demonstrated minimal increases in Pa_{O_2} two to three weeks after operation. The mean Pa_{O_2} rose from 35 mm Hg preoperatively to 38 mm Hg ($P > 0.10$), an increase in oxygen saturation of only 5 per cent. Had the hemoglobin stayed at the mean preoperative level of 18.3 gm/100 ml, the increase in Pa_{O_2} would account for a 1.2 ml/100 ml increase in arterial oxygen content. However, this was not the case because the mean hemoglobin concentration fell to 12 gm/100 ml two to three weeks after creation of the shunt. The fall in hemoglobin partly explains the disappearance of cyanosis in most of these patients. The improvement in arterial oxygen saturation observed in our study is substantially less than the 14 per cent increase found by Vinitskaia and Donetskii[9] in arterialized capillary blood from their patients at an unspecified time after operation. They also state that the shunt lumens they created ranged from 7 to 11 mm, in contrast to those in our patients, which were approximately 4 to 5 mm in diameter.[3]

Arterial oxygenation had not reached its maximum by the second postoperative week. Our clinical observations indicate that cyanosis diminishes over a period of several months. One possible explanation for the delay in im-

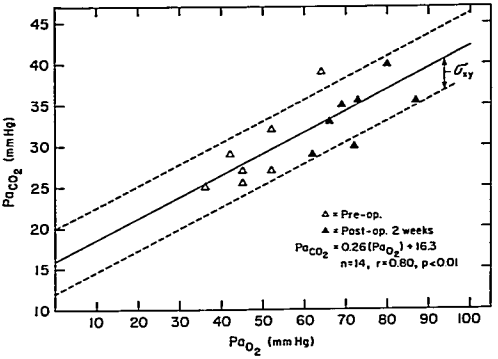

FIG. 1. Relationship between Pa_{CO_2} and Pa_{O_2} before, and two to three weeks after total correction, F_{IO_2} 0.21. Arrow represents one standard error of the estimate (σ xy).

provement is ventilation–perfusion inequality. Following subclavian-to-pulmonary artery anastomosis in dogs, 74 per cent of the blood flowed to the ipsilateral lung, which at best would receive only 55 per cent of the ventilation.[10] Lees *et al.*[11] found considerable ventilation–perfusion inequality prior to operation in children with tetralogy of Fallot. Perhaps this persists despite the functioning shunt.

Another plausible explanation for persisting cyanosis is the decrease in collateral flow into the pulmonary capillary.[12,13] After creation of the anastomosis, pulmonary blood flow may fall because of the disappearance of the collateral circulation, which is dependent on a systemic vascular resistance well in excess of pulmonary vascular resistance. With the shunt created, systemic vascular resistance falls and blood flow through collateral vessels decreases. The flow through the shunt may be less than the collateral flow, thereby temporarily decreasing pulmonary blood flow.[13] Gradual improvement in the distribution of pulmonary blood flow and alveolar ventilation must occur, since those with functioning anastomoses created years previously had a mean Pa_{O_2} of 48 mm Hg just before total correction. A similar mean Pa_{O_2} has been reported by Strong and colleagues[14] prior to total correction, representing a value intermediate between that existing three weeks after the shunt and that some months later when clinical cyanosis is minimal.

In the TC group, one day after operation, the low mean Pa_{O_2} of 42 mm Hg can be attributed to the ventilation–perfusion inequality and atelectasis that follow cardiopulmonary bypass.[15,16] Collateral blood flow thru the lungs may also cause a persistent ventilation–perfusion abnormality. Complete disappearance of mild hypoxemia may never occur, as shown by the data of Sorensen and Severinghaus.[17] One to seven years after total correction, they observed values from 62 to 94 mm Hg (mean 81 mm Hg) in five subjects.

The preoperative V_{DP}/V_T ratios in the SP group were not elevated except in patient 11 (V_{DP}/V_T 0.42). On the first postoperative day, four of the five patients studied had increased V_{DP}/V_T ratios, and two (patients 9 and 10) had quite abnormal values (V_{DP}/V_T 0.52 and 0.59). We have observed in three children with normal lungs on the first postoperative day an average increase in V_{DP}/V_T of 0.09 after thoracotomy for repair of coarctation of the aorta.* Thus, the increased V_{DP}/V_T ratio in the SP group can be attributed, in part, to the effect of thoracotomy itself. After three weeks, V_{DP}/V_T ratios decreased in five of the seven patients. One might expect a further fall in V_{DP}/V_T ratio over the years to that seen preoperatively in the TC group. Although the mean V_{DP}/V_T ratio following median stern-

* Nicodemus, H. F. and Downes, J. J.: unpublished data.

otomy and cardiopulmonary bypass with total correction showed a decrease, individual responses varied, reflecting the many factors that can influence physiologic deadspace.[18,19]

In unsedated children with uncomplicated coarctation of the aorta, we observed an average $\dot{V}_E$ 1.25 times that predicted by the Radford nomogram prior to operation. We interpret this to be a ventilatory response to the anxiety provoked in a child by the procedure. Such an increase in $\dot{V}_E$ accounts for approximately a fourth of the elevation of $\dot{V}_E$ observed before and after surgery in both groups of unsedated patients with tetralogy. If one extends the regression line of $P_{a_{CO_2}}$ on $P_{a_{O_2}}$ (fig. 1) to a normal child's $P_{a_{O_2}}$ of 95 mm Hg, the $P_{a_{CO_2}}$ would be 41 mm Hg, a value only slightly above the normal children's range of 32 to 40 mm Hg. Therefore, in the SP group and preoperatively in the TC group, it seems reasonable to attribute the elevated $\dot{V}_E$ primarily to ventilatory response to hypoxemia. Postoperatively, in the SP group, the elevated $\dot{V}_E$ may represent a response to the rise in V_D/V_T ratio and to the increased metabolic rate that occurs during recovery.[20] $\dot{V}_E$ remained at twice predicted levels three weeks postoperatively in the TC group, perhaps due to residual hypoxemia. Similarly, three weeks following repair of aortic coarctation we found an increase in mean $\dot{V}_E$ to levels nearly twice these predicted. This may be related in part to the increased oxygen consumption and energy expenditure that has been observed in some patients during convalescence from major surgery,[21] and may be exaggerated in tetralogy of Fallot. Bing *et al.*[12] found that patients with cyanotic congenital heart disease had metabolic rates as low as −48 per cent[8], and that after creation of a Blalock shunt metabolic rate increased, indicating that depressed metabolic rate is reversible. The increase in $\dot{V}_E$, however, must be persistent since three of five adult subjects studied by Sorensen and Severinghaus[17] years after total correction had elevated resting $\dot{V}_E$ values (7–8 l/min/m²), with $P_{a_{CO_2}}$ values between 36 and 41 mm Hg. At present we have no reasonable explanation for the persistent hyperventilation following total correction.

Conclusions

Only a modest improvement in arterial hypoxemia immediately follows a shunt procedure; the maximal rise in $P_{a_{O_2}}$ requires more than three weeks to develop. Even after total correction a normal $P_{a_{O_2}}$ cannot be achieved in two weeks and perhaps may never be reached in some individuals.

Physiologic deadspace tends to increase in the immediate postoperative period in children who have undergone shunt procedures. This can be attributed in part to the effect of thoracotomy itself. The physiologic deadspace decreases after total correction.

The elevated minute volume found preoperatively in both groups can be attributed to the presence of arterial hypoxemia. This hyperventilation persists postoperatively in the SP group, probably because of hypoxemia, increased physiologic deadspace, and the increased metabolic rate which occurs during recovery. In the TC group, the continuing elevation of minute volume might be related to an increased metabolic rate and to the mild hypoxemia that persists.

Postoperative pulmonary complications or depression of minute volume to predicted normal levels predisposes these patients to respiratory failure. This applies particularly in the immediate postoperative period of a child who has undergone a shunt procedure.

The authors gratefully acknowledge the technical assistance of Mr. Robert Carson.

References

1. Waldhausen, J. A., Friedman, S., Nicodemus, H. F., Miller, W. W., Rashkind, W., and Johnson, J.: Absence of the pulmonary valve in patients with tetralogy of Fallot: Surgical management, J. Thorac. Cardiov. Surg. 57: 669, 1969.
2. Downes, J. J., Nicodemus, H. F., Pierce, W. S., and Waldhausen, J. A.: Acute respiratory failure in infants following cardiovascular surgery, J. Thorac. Cardiov. Surg. (to be published).
3. Waldhausen, J. A., Friedman, S., Tyers, G. F. O., Rashkind, W., Petry, E., and Miller, W. W.: Ascending aorta–right pulmonary artery anastomosis: Clinical experience with thirty-five patients with cyanotic congenital heart disease, Circulation 38: 463, 1968.

4. Astrup, P., Siggaard-Andersen, O., Jorgensen, K., and Engel, K.: The acid–base metabolism, a new approach, Lancet 1: 1035, 1960.
5. Siggaard-Andersen, O.: The *p*H, log P_{CO_2} blood acid–base nomogram revised, Scand. J. Clin. Lab. Invest. 14: 598, 1962.
6. Comroe, J. H., Forster, R. E., Dubois, A. B., Briscoe, W. A., and Carlsen, E.: The Lung. Second ed. Chicago, Year Book Publishers, 1962, p. 339.
7. Radford, E. P., Ferris, B. G., Kriete, B. C.: Clinical use of a nomogram to estimate proper ventilation during artificial respiration, New Engl. J. Med. 251: 877, 1954.
8. Bing, R. J., Vandam, L. D., Handelsman, J. C., Campbell, J. A., Spencer, R., and Griswold, H. E.: Physiological studies in congenital heart disease with cyanosis VI. Adaptation to anoxia in congenital heart disease with cyanosis, Bull. Johns Hopkins Hosp. 81: 439, 1948.
9. Vinitskaia, R. S., and Donetskii, D. A.: Oxygenation of the arterial blood at rest and under physical loading in patients with Fallot's tetrad following establishment of interarterial subclavicular–pulmonary anastomoses, Grudn. Khir. 7(3): 61, 1965.
10. Fort, L., Morrow, A. G., Pierce, G. E., Saigusa, M., and McLaughlin, L. S.: The distribution of pulmonary blood flow after subclavian–pulmonary anastomosis, J. Thorac. Cardiov. Surg. 50: 671, 1965.
11. Lees, M. H., Burnell, P. H., Morgan, C. L., and Ross, B. B.: Ventilation–perfusion relationships in children with heart disease and diminished pulmonary blood flow, Pediatrics 42: 778, 1968.
12. Bing, R. J., Vandam, L. D., and Gray, F. D., Jr.: Physiological studies in congenital heart disease II. Results of preoperative studies in patients with tetralogy of Fallot, Bull. Johns Hopkins Hosp. 80: 121, 1947.
13. Vandam, L. D., Bing, R. J., and Gray, F. D., Jr.: Physiological studies in congenital heart disease IV. Measurements of circulation in five selected cases, Bull. Johns Hopkins Hosp. 80: 192, 1947.
14. Strong, M. J., Keats, A. S., and Cooley, D. A.: Arterial gas tensions under anesthesia in tetralogy of Fallot, Brit. J. Anaesth. 39: 472, 1967.
15. Moffitt, E. A., Sessler, A. D., Lundborg, R. O., and McGoon, D. C.: Acid–base balance and oxygenation in prolonged perfusion: Effects of diluted heparinized and acid-citrate-dextrose blood, Anesth. Analg. 46: 153, 1967.
16. Geha, A. S., Sessler, A. D., and Kirklin, J. W.: Alveolar–arterial oxygen gradients after open intracardiac surgery, J. Thorac. Cardiov. Surg. 51: 609, 1966.
17. Sorensen, S. C., and Severinghaus, J. W.: Respiratory insensitivity to acute hypoxia persisting after correction of tetralogy of Fallot, J. Appl. Physiol. 25: 221, 1968.
18. Sykes, M. K.: Aspects of dead space in anaesthetic practice and artificial pulmonary ventilation. *In* Evans, F. T., and Gray, T. C., (eds.): Modern Trends in Anesthesia. 3, Aspects of Metabolism and Pulmonary Ventilation. New York, Appleton-Century-Crofts, 1967.
19. Severinghaus, J. W., and Stupfel, M.: Alveolar dead space as an index of distribution of blood flow in pulmonary capillaries, J. Appl. Physiol. 10: 335, 1957.
20. Liubchenko, L. N.: The changes occurring in some external respiration indices in patients with Fallot's tetralogy following operations of aorto-pulmonary and cava-pulmonary anastomosis, Grudn. Khir 7(4): 51, 1965.
21. Kinney, J. M., and Roe, C. F.: Caloric equivalent of fever: Patterns of post-operative response, Ann. Surg. 156: 610, 1962.

Obstetrics

MATERNAL HYPOTENSION Fetal acid-base status and neonatal clinical condition were assessed in 34 consecutive elective cesarean sections. The effects of: 1) spinal analgesia with prevention of hypotension by forced hydration, 2) spinal analgesia with treatment of hypotension by forced hydration, and 3) thiopental–nitrous oxide–succinylcholine anesthesia were compared. Spinal analgesia with prophylactic hydration produced more favorable biochemical and clinical conditions than spinal analgesia with therapeutic hydration. The thiopental–nitrous oxide–succinylcholine sequence was associated with a higher incidence of neonatal depression than either of the spinal analgesia groups; this, however, was not accompanied by an increase in acidosis of the neonate. (*Cosmi, E. V., and Marx, G. F.: Acid–Base Status of the Fetus and Clinical Conditions of the Newborn Following Cesarean Section, Amer. J. Obstet. Gynec. 102: 378 (Oct.) 1968.*)