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CARDIOVASCULAR AND RENAL EFFECTS OF INDOMETHACIN IN NEWBORN DOGS. Mary P. Bedard, Leonard I. Kleinman, Uma R. Kotagal and Theresa A. Disney. University of Cincinnati College of Medicine, Dept. of Pediatrics, Cincinnati.

The cardiovascular effects of indomethacin were studied acutely in 9 newborn dogs (2-32 days) using the radioactive microsphere reference organ technique. Indomethacin, 0.3 mg/kg intravenously, produced a 22% drop in cardiac output (CO) from 224±31* to 175±26 ml/kg/min ($p<.05$). Renal blood flow fell slightly from 1.6±.2* to 1.3±.2 ml/gm/min ($p<.05$). Renal inner cortical (IC) and outer cortical (OC) blood flow both fell ($p<.01$), but did so proportionately with no resultant change in IC/OC ratio. There were no changes in blood flow to the GI tract, heart or lungs. Of particular interest is that although in animals over 7 days (n=5) indomethacin produced no change in cerebral blood flow (CBF), in 2-day-old puppies (n=3) it produced a 47% fall in CBF ($p<.05$). Indomethacin produced no significant changes in glomerular filtration rate or potassium excretion, but there was a transient drop in tubular sodium reabsorption ($p<.01$) which rapidly returned to control values after 1½ hours.

In summary, indomethacin in a dose currently being used clinically causes small, but statistically significant decreases in CO and renal blood flow, with minimal changes in renal function. However, in very young animals, this dose of indomethacin results in a marked drop in cerebral blood flow not found in older animals.

*mean±SE

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VENTRICULAR TACHYCARDIA IN CHILDHOOD
USE OF APRINDINE HYDROCHLORIDE

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Six cases of ventricular tachycardia seen at Children's Hospital since 1970 are presented. Diagnosis was made by classical electrocardiographic criteria and/or pacing studies. Three patients, aged 3/12-4 years, had paroxysmal ventricular tachycardia with signs and symptoms of heart failure. Two patients had probable associated myocarditis. Ventricular rates were 220-280/min. One case responded to quinidine, one converted to sinus rhythm during pacing studies. A third patient, age 16 months, was unresponsive to digoxin, procainamide, propranolol and d/c cardioversions. Aprindine HCL administered IV, with cardioversion, converted the patient to sinus rhythm. Three cases of nonparoxysmal ventricular tachycardia presented with rates of less than 130/min. Two were asymptomatic, one had dizziness. Ventricular tachycardia was suppressed by exercise in all. We recommend no treatment for nonparoxysmal ventricular tachycardia in absence of symptoms. One patient with paroxysmal ventricular tachycardia is in sinus rhythm on no drugs, one is on propranolol and the third is maintained on Aprindine. Aprindine HCL is useful in treatment of life threatening ventricular arrhythmias.

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IS THERE AN ANATOMICAL BASIS FOR SOME CASES OF SO CALLED "PERSISTENT FETAL CIRCULATION"? W.A. Blanc, A. C. Moessinger, Depts. of Path. & Ped., Columbia Univ., New York

Anatomical measurements of the circumference of the ascending aorta (AA), aortic isthmus (AI), left carotid (LC), ductus arteriosus (DA), and of the size of the foramen ovale were determined in 37 full term newborn infants without congenital heart disease who expired within 3 days of birth. Using the DA/LC ratio as a standard anatomical index of ductal size, we found that it did not vary significantly during the 3 first days of life (1.33 + 0.32). In contrast, 8 patients with pulmonary hypertension (diaphragmatic hernia) had a significantly higher ductal index: 2.15 + 0.66, $p<0.01$. Assuming that this larger ductal size could be the result of increased pulmonary resistance, we analyzed the anatomical findings in a group of patients with a DA/LC ratio 2 SD. above the norm. Most of these patients were found to have one of the three following associated malformations: 1-lung hypoplasia, 2-narrowing of the aortic isthmus (AA/AI ratio 2 SD. above the norm), or 3-stenosis of the foramen ovale. It is speculated that these malformations are related to the development of pulmonary hypertension in utero with larger ductal size at birth and a delayed fall in pulmonary vascular resistance in the early neonatal period. Several clinico-pathological correlations supporting this view were made in patients with documented persistence of the fetal circulation. The ductal index has frequently shed some light on the disease process by suggesting an initially undetected lesion and should be part of every neonatal autopsy.

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EFFECT OF OXYGEN ON THE SYSTEMIC VASCULAR BED IN PATIENTS WITH TETRALOGY OF FALLOT. ROBERT BOXER, EHUD KRONGRAD, ALLAN HORDOF, CARL STEEG WELTON GERSONY. College of Physicians and Surgeons, Columbia University, Department of Pediatrics, New York City, 10032

The mechanism by which oxygen (O_2) administration improves systemic arterial saturation (SA sat) in patients (pts) with Tetralogy of Fallot (ToF) has not been previously elucidated. In order to explain this phenomenon, hemodynamic measurements were made before and after 90-100% O_2 administration to 16 pts, age 4 mos. - 8 yrs. (M= 3.4 yrs.).

During O_2 administration, mean SA sat increased from 76.5±1.7%(M± SEM) to 90.9±1.9%($p<.001$), mean SA pressure increased from 68.3±3.1 mmHg to 76.5±4.1mmHg($p<.02$), mean systemic flow index decreased from 4.6±0.4L/min/m^2 to 3.3±0.2L/min/m^2 ($p<.01$), and mean systemic vascular resistance (SVR) increased from 15.6±1.2 Units/m^2 to 24.4±1.3 Units/m^2 ($p<.001$). Mean pulmonary flow index increased from 2.4±0.4L/min/m^2 to 2.7±0.6L/min/m^2 ($p>.05$). The mean % right to left (R→L)shunt decreased from 51.4±3.6% in room air to 35.5±4.6% in O_2($p<.001$). In room air, 4 pts had bidirectional shunts. However, with O_2, an additional 7 pts developed bidirectional shunts. In all pts, the increment in oxygen saturation was significantly greater than could be due to dissolved O_2 alone.

It is concluded that O_2 administration in pts with ToF increases SA sat by inducing systemic vasoconstriction. The resultant increased SVR causes a decrease in the R→L shunt and improved oxygenation. Thus, in ToF, the beneficial effect of O_2 results primarily from its effect on the systemic vascular bed.

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EFFECTS OF ALVEOLAR HYPOXIA AND INTRAVENOUS TOLAZOLINE ON PULMONARY HEMODYNAMICS AND LUNG LYMPH FLOW OF AWAKE WEEK-OLD LAMBS. Michael A. Bressack and Richard D. Bland. University of California, San Francisco, Cardiovascular Research Institute, Department of Pediatrics, San Francisco, Ca.

To study the effects of alveolar hypoxia on lung fluid dynamics of newborn lambs, we measured mean pulmonary arterial (Ppa) and left atrial ($\bar{P}$la) pressures, cardiac output ($\dot{Q}$b), steady-state lung lymph flow ($\dot{Q}$l), and lymph (L) and plasma (P) protein concentrations of 8 awake 1-2 week old lambs breathing air for 2 h, then 10-12% O_2 for 2-4 h. The lambs were at least 2 days postoperative during studies. Summary of results (mean):

		Pressures		---- Proteins ----			Flow Rates	
	PaO_2	Ppa	$\bar{P}$la	Lymph	Plasma	L:P	Qb	Ql
	torr	-- torr --		---- g/dl ----			l/min	ml/h^x
Baseline	76	17	2	3.52	5.77	.61	2.4	.10
Hypoxia	31*	32*	1	2.86*	5.73	.50*	2.9*	.17*

x: per g dry bloodless lung *: significant difference, $p<.05$

Contrary to results of previous studies of mature sheep (Circ Res 40:269, 1977), $\dot{Q}$l of lambs increased and L:P decreased when pulmonary vascular resistance (PVR) increased 66%, suggesting that alveolar hypoxia increased pulmonary transvascular filtration pressure and resultant fluid exchange,with no change in microvascular permeability to protein. To 6 lambs we infused tolazoline intravenously (2 mg/kg rapidly, then 4 mg/kg·h^{-1} for 3-4h) during steady-state hypoxia. As PVR diminished 16%, $\dot{Q}$l decreased 23%, showing that tolazoline decreased pulmonary transvascular filtration pressure and net fluid flux into the lung of hypoxic lambs.

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VIABLE ATRIAL BAFFLE FOR HEMODYNAMIC CORRECTION OF TRANSPOSITION OF THE GREAT VESSELS. Ramon V. Canent, Keith W. Ashcraft, Thomas M. Holder, R. Gowdamarajan.

Since Mustard reported pericardial baffling for correction of transposition of the great vessels, this has became the procedure of choice. Caval and pulmonary venous obstructions, tricuspid regurgitation, encroachment of atrial chambers and sudden death have occurred following this surgical technique.

The purpose of this report is to present a modified baffling procedure which uses the viable atrial wall to prevent late baffle contracture and it's dreaded obstruction. A flap of pericardium preferably pedicled is used to close and form the anterior atrial wall providing a large chamber to accomodate pulmonary venous return.

The procedure was performed in 5 infants 11 to 15 months old. Except for one patient who died suddenly 16 hours post-op, 4 surviving patients had smooth post-surgical courses. One patient who had an associated VSD closed, developed complete heart block but continues to maintain a ventricular rate of 68 to 72/minute.

Cardiac catheterization was performed in 3 patients 6 to 8 months after their surgery. Pulmonary capillary wedge, vena caval, atrial chambers and pulmonary venous pressures showed no obstruction. The atrial pressures tended to be slightly higher than normal. Selective right and left pulmonary arteriograms were filmed and demonstrated well the unobstructive pulmonary venous return.

Atrial wall baffle has the advantage that viable tissue is used and unlikely to constrict systemic and pulmonary drainage.