ness (WT; external diameter — internal diameter/external diameter), number of layers, and measurements of the intima, media, and adventitia. All PH patients had ≥ 2 visible layers, whereas no control patient had a visible intima. Patients with PH had a thicker media (0.32 ± 0.17 vs 0.15 ± 0.01 mm, $p < 0.05$) and %WT (29 ± 3 vs 16 ± 2%, $p < 0.05$) than controls. We conclude that IVUS allows for direct visualization of structural changes in small pulmonary arteries in patients with PH and may complement hemodynamic and pulmonary wedge angiography evaluation in these patients.

4:15

808-2 Pulmonary Vascular Response to Oxygen and Inhaled Nitric Oxide

Andrew M. Atz, Ian Adatia, David L. Wessel. *Children's Hospital, Harvard Medical School, Boston, MA*

We compared the effects of known inhaled pulmonary vasodilators, oxygen (O2) and inhaled nitric oxide (iNO) in 58 pulmonary hypertensive patients at cardiac catheterization. In 42, measurements were made at room air (RA), 100% O2, return to RA, and with iNO (80 ppm at RA). Results (mean ± SEM):

n = 42 *p < 0.05	PAp mmHg	PVR U·m²	BP mmHg	SVR U·m²
RA	64.1 ± 3.5	17.8 ± 2.3	78 ± 2.3	23.6 ± 1.7
O2	55.7 ± 3.4	11.4 ± 1.6*	80 ± 2.4	24.8 ± 1.6
RA	61.9 ± 3.6	18.3 ± 2.4	78 ± 2.6	23.6 ± 1.8
NO + RA	52.0 ± 3.2*	12.0 ± 1.8*	78 ± 2.7	24.7 ± 1.8

A reduction in PVR of ≥ 20% was considered responsive. A response was seen in 35/42 with O2 alone. Of the 7 nonresponders, 3 showed a response to NO + RA. In the NO + RA group, response was seen in 29/42 with 9 of the 13 nonresponders responding to O2. An additional 16 patients were studied while on supplemental O2 (FiO2 0.4–0.95). The addition of iNO at 80 ppm caused additional pulmonary vasodilation in 14/16 (12 with > 20% reduction in PVR). Results (mean ± SEM):

n = 16	PAp (mmHg)	PVR (U·m²)
O2	62.2 ± 4.9	21.1 ± 3.8
NO + O2	53.6 ± 4.9	15.3 ± 3.4

Conclusion. iNO and O2 are both effective pulmonary vasodilators. Combination therapy can produce additional vasodilation. This suggests that the most comprehensive and specific testing of pulmonary vasoreactivity should include combination treatment with iNO and supplemental O2.

4:30

808-3 Aerolized Prostacyclin (aePGI$_2$) for Preoperative Evaluation and Postoperative Treatment of Pulmonary Hypertension (PHT)

Ingram Schulze-Neick, Frank Uhlemann, Jan Nürnberg, Felix Berger, Christian Opitz [1], Franz X. Kleber [1], Peter E. Lange. *German Heart Institute, Berlin, Germany;* [1] *Cardiovascular Catheterization Laboratory, Humboldt University, Berlin, Germany*

We examined the effects of aePGI$_2$ in patients with PHT as an alternative to inhaled nitric oxide (iNO). During routine catheterization for evaluation of pulmonary vasoreactivity, aePGI$_2$ versus iNO was compared in 9 cyanotic patients with congenital intracardiac shunting lesions and 3 patients with primary PHT, who inhaled aePGI$_2$ during spontaneous breathing (patient group A). AePGI$_2$ was used postoperatively in 3 patients in whom ongoing iNO-Therapy for PHT or right ventricular dysfunction had to be interrupted for technical reasons (patient group B). These ventilated patients had persisting PHT after termination of iNO therapy. No acute side effects occurred in either group. Group A: 5/12 patients showed no vasoreactivity. In 2/3 patients with primary PHT, a significant drop in pulmonary artery resistance occured (–33 ± 12%) with concommitant increase in cardiac output. In 5/9 patients with shunt lesions, the relative pulmonary vascular resistance (Rp:Rs) decreased with iNO from 96 ± 32% to 62 ± 33% systemic resistance, and to 52 ± 41% systemic resistance with aePGI$_2$ ($p < 0.02$). Thus, aePGI$_2$ showed a marked effect in 7/12 patients and in all patients where iNO was effective. Group B: Cardiac index (+21 ± 17%), systemic oxygenation and left atriel pressure improved; central venous pressure and pulmonary artery pressure (–45 ± 19%) decreased 2 min after application of aePGI$_2$ and remained low 20–30 min after application was stopped. AePGI$_2$ lowered pulmonary vascular resistance and improved cardiac function. This effect seemed to be selective, and was comparable to iNO. Therefore, aePGI$_2$ could represent a clinically useful alternative to iNO. Further studies will have to evaluate the benefits of either therapeutic strategy.

4:45

808-4 Hemodynamic and Histologic Evaluation of the Pulmonary Vascular Bed and Clinical Outcome in Children With Congenital Heart Disease (CHD): Is There a Gold Standard?

R.M.F. Berger, C.A. Wagenvoort, R.J. van Suylen, J. Hess. *Sophia Children's Hospital/University Hospital Rotterdam, The Netherlands*

Hemodynamic and histologic data of a selected group of 52 children (2.6 ± 4.4 yrs) with compex CHD were assessed to determine the relation between lung morphology and hemodynamic data, and outcome. Pulmonary artery pressures (PAP) showed significant correlations with medial hypertrophy and muscularisation of arterioles, however, the correlation coefficients were all < 0.5. Pulmonary bloodflow, shuntsize, the ratio of pulmonary (Rp) to systemic (Rs) vascular resistance were not correlated with histology. Concentric laminar intimal fibrosis (CLIF), dilatation lesions (DL), fibrinoid necrosis (FN) and plexiform lesions (PL) were not present in cases with diastolic PAP < 25 mmHg or Rp < 6 WU·m². In patients with diast. PAP > 25 mmHg or Rp > 6 WU·m² vascular changes could not be predicted accurately from hemodynamic data, including respons to vasodilators. *Short-term outcome:* 45 patients were operated, 16 died perioperatively, 12 in relation to acute pulmonary hypertensive crises (APHC). These children showed increased medial hypertrophy and muscularisation in relation to the survivors ($p = 0.003$). The occurence of APHC was not correlated with any of the hemodynamic parameters. *Mid-term outcome:* 17 patients died unrelated to operation, 16 without signs of pulmonary vascular disease (PVD). One patient, who showed "unfavourable" hemodynamics, but only medial hypertrophy of arteries and veins, died of progressive PVD. 19 patients were still alive (follow-up 57 ± 39 months). 6 showed signs of PVD. Original hemodynamic data did not differ significantly in patients with or without PVD. DL and PL were not associated with signs of PVD at follow up ($p = 0.257$). The presence of CLIF ($p = 0.027$) or FN ($p = 0.037$) was associated with PVD at follow up, however the predictive value was low.

Thus, although diast. PAP < 25 mmHg or Rp < 6 WU·m² predict favourable lung histology with respect to long term prognosis, they do not predict risk of APHC. In contrast, diast. PAP > 25 mmHg or Rp > 6 WU·m² do not predict lung histology. The value of both hemodynamic and histologic evaluation of pulmonary vasculature is limited with respect to mid-term (5 yr) prognosis in the individual child.

809 Pharmacology of Clotting and Platelets

Wednesday, March 27, 1996, 4:00 p.m.–5:00 p.m.
Orange County Convention Center, Room 208

4:00

809-1 The Extent and Mechanism of ACT Prolongation With GPIIb/IIIa Blockade

Sandeep Khurana, Darius Aliabadi, Mark Pica, Randall McPherson, Gerald C. Timmis, Robert D. Safian. *William Beaumont Hospital Royal Oak, MI*

To assess the degree of Activated Clotting Time (ACT) prolongation attributable to c7E3 Fab during PTCA, we measured the ACT with and without different concentrations (0, 2.5, 5 and 10 μg/mL) of c7E3 added to whole blood of pts before or after PTCA (25 pts; 100 ACT observations). The ACT increased by 6% for each 2.5 μg/mL c7E3 ($p < 0.001$ repeated measures ANOVA). A more detailed analysis (repeated measures ANCOVA with log ACT as covariate) revealed that the effect of c7E3 was more pronounced at higher baseline ACT ($p < 0.02$) suggesting synergy between heparin and c7E3. We also performed thromboelastography (TEG) on non heparinized whole blood (n = 12) to measure the effect of c7E3 on the dynamic elastic modulus (G, dynes.cm^{-2}), a measure of clot strength. On TEG, both the time to develop a "firm" clot and maximal clot strength (G) were dose dependently impaired by c7E3 as shown in the fig ($p < 0.001$).