Circulation/Cardiology

P 165

Pulmonary Hypertension and Treatment with Magnesium-Aspartate-Hydrochlorid

H. Ringe, H. J. Feickert, H. C. Kallfelz

Department of Pediatric Cardiology Medizinische Hochschule

Hannover (Germany)

In some cases severe pulmonary hypertension (PHT)in childhood is caused by adult respiratory distress syndrome (ARDS) or congenital heart disease with left-to-right shunt. Several therapeutic trials have been undertaken to reduce PHT: deep sedation, muscle relaxation, prostacyclin, calcium-antagonist, NO and ECMO.

There is good theoretical and experimental evidence to support that magnesium reduces PHT.

Abu Osba et al treated 7 of 9 new-borns with persistent pulmonary hypertension successfully with magnesium sulphate

Two children with VSD, left to right shunt and severe PHT and three children with ARDS were treated with magnesium-aspartate-hydrochlorid. 1-2 mmol / kg magnesium were given slowly intravenously. Serum magnesium concentration was maintained between 2-4 mmol / l ionized and 3-7mmol / l total magnesium by continuous intravenous infusion. After six hours four patients had a 50% reduced oxygenation index. Two of them died later with severe Sepsis. One patient with ARDS did not improve.

P 166

PURULENT PERICARDITIS: CLINICAL FORMS

Dang Phuong Kiet and Nguyen Xuan Thu

Examining 6 cases of purulent pericarditis with various clinical forms treated by surgery, the authors drew the following experiences for their diagnosis.

I. Clinical factors.

Purulent pericarditis appeared like a cardiac tamponade in a septicemia due to staphylococci with classical symptoms: severe dyspnea, tachycardia, faint heart-sound, big liver, prominent cervical vein ; rentgenography of the chest showing enlargement of the cardiac silhouette, a diminution of ventricular pulsations, a clear lung field. By an emergency operation, 500ml of diluted blood were drained.

Purulent pericarditis and pleural effusion appeared at the same time but at first the symptoms of purulent pericarditis were masked by the predominant symptoms of pleural effusion. After the pleura was drained, its pus was no more, the general state was relatively stabilized but there still were big liver, dyspnea, enlargement of the cardiac silhouette while central venous pressure increased.

Purulent pericarditis appeared late. In the first stage (about 2 weeks) there was no suspected sign. Later on gradually appeared such symptoms as dyspnea (during serum transfusion for instance). Central veinous pressure also raised. The heart chest diametre increased at first (up to 60-65%) then decreased (down to below 50%) but the liver kept on swelling together with the particular changes of electrocardiogramme. Now the pericardium had no more pus but get fibrous (up to 3mm) thus constricting the heart and its main arteries (like Pick syndrome).

2. Diagnostic values of electrocardiograms :

Common signs of ECG related of these purulent pericarditis were: a diminution of voltage, a widespread elevation of the ST segment, the TF wave flattened and inverted. However, what should be stressed was : the diagnostic values of an electrocardiogram for purulent pericarditis was mainly in the dynamics of their signs: in the first week, the voltage diminished corresponding to a pericardium containing pus, while the ST segment went up then seemed parallel to the fibrosis of the epicardium, the liver swelled, the central veinous pressure increased, the heart/chest dimension ratio decreased, the ST segment went down, the T wave became more flat and inverted.

P 167

NEBULIZED NITROPRUSSIDE (NP): A HIGHLY EFFECTIVE, READILY AVAILABLE AGENT TO SELECTIVELY REVERSE HYPOXIA-INDUCED PULMONARY HYPERTENSION. William Meadow, Brian Rudinsky, Anthony Bell, Robert Hipps Department of Pediatrics, University of Chicago, Chicago IL USA

Selective reduction of pulmonary artery pressure (PAP) in the context of pulmonary hypertension is oft desired but rarely achieved. Inhaled nitric oxide (NO) has been shown to produce this desirable effect, but is relatively difficult to administer or monitor. We wondered whether NP, chemically related to NO but more stable in solution, would produce similar physiologic effects when administered in the convenient modality of nebulization.

Methods: 9 piglets were anesthetized, mechanically ventilated, and surgically instrumented. Systemic blood pressure (BP), PAP, and cardiac output (CO) were monitored continuously. After post-operative stabilization, 0.9% NaCl nebulization was begun, and pulmonary hypertension was induced by reducing FiO2 from 0.30 to 0.07. The piglets were monitored for 15 minutes during this hypoxic phase. Next, without altering FiO2 or ventilator settings, NP (10 mg/ml, dissolved in 0.9% NaCl, flow 4 lpm) was substitued for 0.9% NaCl in the nebulizer circuit. NP was nebulized for 15 mins.

Results: During hypoxia, PaO2 fell from 150 to 29 mm Hg. PAP rose during hypoxia from 14 to 31 torr ($p< 0.01$), while BP and CO did not change significantly. PAP fell during nebulized NP in each piglet, (mean ΔPAP = 31 to 21 torr; $p< 0.01$; mean reduction of hypoxia-induced rise in PAP = 61%; range: 36 to 78%; $p < 0.01$). PVR/SVR fell by 28% during NP nebulization ($p< 0.01$), while BP and CO did not fall significantly (90 to 86 torr; 653 to 636 mL/kg-min). The reduction in PAP began within 2 minutes of the onset of nebulized NP, and appeared to reach a plateau by 15 minutes. No tachyphylaxis to nebulized NP was noted. Nebulized NP did not significantly affect PAP, BP, or CO under normoxic conditions.

Conclusions: **1)** Like NO, NP selectively reduced hypoxia-induced pulmonary hypertension without altering systemic BP. **2)** Unlike NO, NP can be administered by nebulizer, a technique familiar to virtually all health-care providers, and potentially adaptable to both intubated and non-intubated patients. **3)** Nebulized NP may be beneficial in clinical contexts where inhaled NO is impractical.

P 168

BALLOON DILATION OF CRITICAL VALVAR PULMONARY STENOSIS IN THE FIRST MONTH OF LIFE

Eicken A., Sebening W., Balling G., Bühlmeyer K.:
Deutsches Herzzentrum, München,Deutschland

Between 1986 and 1995 23 neonates, aged 2 - 23 days (median 5), weight 2,38 - 4kg (median 3,28) with critical valvar pulmonary stenosis were scheduled for balloon dilation (PSVP). 19 children (83%) were on PGE1 and 13 (57%) needed mechanical ventilation. After stepwise dilation a final balloon : pulmonary valve (PaV) ratio of 114% (25-150) was achieved. There was a significant correlation ($p<0,01$) between an adequately sized balloon and freedom of reintervention. Two valves could not be passed, four neonates underwent surgical procedures (brock n = 3, commissurotomy n = 1), two children (10%) died of sepsis. 17/23 patients (73%) were successfully palliated by PSVP in the first month of life. The RV : systemic pressure value fell from 132% (75-231) to 58% (40-87). Complications included 2 transient dysrhythmias, 1 transient hypoxia, 3 vessel occlusions, 1 right ventricular outflow tract perforation. In 16/17 patients follow up data is available. The residual systolic peak doppler gradient over the PaV on the last out patient visit (5-103 months after PSVP) was 10-41 mmHg (median 20). Four children needed repeated PSVP 26 to 72 months after the initial intervention.

Conclusion: PSVP of critically ill newborns is possible. The risk of mortality is relatively low. PSVP in neonates with an adequately sized balloon is a challenging alternative to surgical treatment .

P 169

POST HYPOXIC-ISCHEMIC REPERFUSION INJURY OF THE NEWBORN HEART REDUCED BY DEFEROXAMINE. Majidah Shadid, Paul Steendijk, Ralph Moison, Enno T van de Velde, Jan Baan, Howard M Berger, Frank van Bel. Depts. of Peds. & Cardiol. University Hospital, Leiden. The Netherlands.

Post hypoxic-ischemic (HI) reperfusion induces the formation of non protein bound iron (NPBI), leading to production of the reactive hydroxyl radical. It was investigated if the iron-chelator deferoxamine (DFO) could reduce free radical production and improve neonatal myocardial performance after HI. Severe HI was produced in 13 newborn lambs and changes from pre-HI values were measured at 15, 60 and 120 min post-HI for (mean) aortic pressure (mean Pao), cardiac output (CO) and stroke work (SW). Left ventricular (LV) contractility and CO were assessed by measuring LV pressure (tip-manometer) and volume (conductance catheter), using inferior caval vein occlusion to obtain slope (Ees) and intercept of the end systolic PV relationship (V10). NPBI, reduced and oxidized vitamine C ratio (VCred/ox) and lipid peroxidation (MDA) were measured from sinus coronarius blood. 7 Lambs received DFO (10 mg/kg i.v.) immediately post-HI, control lambs (CONT) received a placebo.

Results: Mean Pao was stable, CO and SW decreased up to 60 and 40% respectively in CONT as compared to pre-HI. In both DFO-groups CO and SW remained within the normal range. Ees and V10 decreased in all groups post HI, but did not differ between groups. NPBI and MDA were higher at 15 min post HI ($p<0.05$), VCred/ox was lower at 15 min post-HI ($p<0.05$) in CONT as compared to DFO-group, indicating more oxidative stress in CONT.

Conclusions: DFO reduced the oxidative stress of the heart and prevented CO and SW to drop, suggesting a positive effect of iron chelation on the myocardium after HI.

P 170

ON HUMAN HEART "VENTRICULAR BLOCK" FUNCTION (HEART PERFORMANCE MONITORING DURING AND AFTER DELIVERY)

Gebel G.Ya.,Kruglov A.G.,Golostenova L.M.,,Dasaev A.N., Utkin V.N., Bagdatjev V.E., Ignatov N.G.*, Suvorov S.G.**
State Scientific Center-Institute of Biomedical Problems
* "Medicine for you" Corporation,** Stock Company Ltd "VEZHA"

Moscow, Russia

From 1987 we published reports with analysis of performance of the heart as a whole organ.While analysing the heart performance as a whole, we recognize three block in it located intrapericardially: 1."atrial"block - left (LA) and rigth (RA) atriums; 2."aorta-pulmonary" block-aorta bulb (A) and pulmonary artery(PA); 3.ventricular "three-chambered" block (VB) consisting of:

- left (LV) and right (RV) myocardial chambers, both with blood outflow into "aorta-pulmonary" block vessels, and
- spongy (venous) myocardial chamber with the blood outflow through coronary sinus (CS) and Thebesian vein (TV) into "atrial" block. The following conceps are introduced for VB functions assessment "common" systole and "common" diastole of "three-chambered" VB.

The process of normal "common" VB systole:

- begins with blood ejection from VB spongy chamber into the "atrial" block;
- continues with blood outflow from RV and LV into "aorta-pulmonary" block with venous minimums - x-collapses -formation in "atrial" block;
- completes with "three-chambered" VB general emptying.

At this period the following blood volumes are transferring:

- two-from RV and LV(their stroke volumes)into"aorto-pulmonary"block;
- two-from "spongy" chamber into "atrial" block;
- two-from systemic and pulmonary veins into "atrial" block during the process of so called "systolic" membrane suction (at pulling atrio- ventricular valves into RV and LV chambers as blood ejects out of them). It appears to be the regulation of blood inflow to "atrial" block by blood outflow from "three-chambered" VB into "aorto-pulmonary" block.

The preliminary reports are published in:
9th Europ. Congr.Anaesthesiol., 1994,218,476,513,514;
Intensive Care Medicine,1995,v.20,suppl.1,s141

P 171

CHANGES IN LEFT VENTRICULAR FUNCTION IN SHOCKED NEWBORNS

Fink C[1], Simma B[2], Fritz M[1], Trawöger R[2], Geiger R[1], Hammerer I[1]

Department of Pediatric Cardiology[1] and Intensive Care Unit[2] Children's Hospital, Anichstrasse 35, 6020 Innsbruck, Austria

The role of heart rate and stroke volume in regulating cardiac output (CO) in newborns is still controversial. The purpose of this study was to assess the change of left ventricular function due to fluid administration in shocked infants and to determine if CO is regulated by heart rate (HR) and/or stroke volume (SV).

Material and Methods: 13 newborns with a mean gestational age of 38 (35-41) weeks, mean birth weight of 2915 (2200-3850)g were examined at mean age of 37 (2-264) hours. Indication for administration of 20ml/kg body weight of Ringer's lactate was blood pressure <10th percentile related to age and weigth. Left ventricular diastolic (LVDD) and systolic (LVDS) diameter, aortic diameter (AoD), aortic velocity-time integral (VTIAo) were determined by M-mode, two dimensional and Doppler echocardiography. Shortening fraction (SF=LVDD-LVDS/LVDD), stroke volume (SV=VTIAoxAoD), cardiac output (CO=SVxHR) and cardiac index (CI=CO/min/kgBW) were calculated.

Results: Changes in blood pressure (31±5 vs. 37±5mmHg, p<.005) stroke volume (9.7±2.2 vs. 11.2±1.8mL, p<.005), cardiac output (793±309 vs. 900±290mL/min, p<.005) and cardiac index (282±120 vs. 321±120mL/min/kgBW, p<.005) were statistically significant. Changes in heart rate, LVDD, LVDS, and SF did not differ significantly.

Conclusion: Volume replacement in hypovolemic newborns lead to improvement of left ventricular cardiac output by increasing stroke volume and not by increasing heart rate.