11:45

702-6 Eisenmenger's Syndrome and Progressive Pulmonary Hypertension After Defect Closure: Prognosis and Cause of Death

Jane Somerville, Susan Stone. *Grown-Up Congenital Heart Unit, Royal Brompton Hospital, London, England*

The possibility of transplantation for patients with pulmonary hypertension makes it important to predict prognosis and causes of death. This studies the state (Ability Index [AI]), complications, age at and cause of death of 143 patients with Eisenmenger Syndrome (E Group) due to VSD (28%), one ventricle (9%), A-V canal (8%) and truncus (8%), 50 of whom died during observation and 19 patients whose defects (VSD, duct, truncus) were repaired in childhood and who died from progressive pulmonary hypertension (PPH Group). In E Group, 8 deaths occurred before age 16 years (child in UK); only 3 "natural" deaths, the other 5 early in relation to cardiac surgery. 42 of E Group died age 19–57 years, the majority in the 3rd decade from right heart failure (24%), sudden (26%), cerebral (14%), haemoptysis (14%), post cardiothoracic intervention (14%), and extracardiac surgery (7%). 22 (24%) of 93 alive in E Group survived to age 40–58 years with AI 2 (well, 13) and 3/4 (9). The PPH Group died age 2–35 years, 75% before 20 years. Prognosis in the PPH Group is worse than Eisenmengers where death in childhood is uncommon. In both groups frank heart failure and exertional presyncope are bad prognostic features but commoner in the PPH Group. In Eisenmengers, any surgery is dangerous. Large haemoptysis heralded disaster in 50% with symptoms when due to right pulmonary artery thrombosis. With the better prognosis of Eisenmenger cf. PPH, it is important to recognize those whose pulmonary vascular resistance may rise after surgery and will thus have an added risk for lung transplantation.

MONDAY AM

703 Advances in Contrast Echocardiography: Transvenous Applications

Monday, March 20, 1995, 10:30 a.m.–Noon
Ernest N. Morial Convention Center, Room 58

10:30

703-1 Detection of Coronary Stenoses and Quantification of Blood Flow Mismatch Using Myocardial Contrast Echocardiography During Coronary Hyperemia with FS-069, a New Intravenous Contrast Agent

Gustavo P. Camarano, Ananda R. Jayaweera, Suad Ismail, Norman C. Goodman, Sanjiv Kaul. *University of Virginia, Charlottesville, VA*

We hypothesized that coronary stenoses, which are not flow-limiting at rest, can be detected and the degree and spatial extent of blood flow mismatch can be quantified during pharmacologically-induced coronary hyperemia using FS-069, a new intravenous contrast agent.

In 15 open-chest dogs, myocardial contrast echo (MCE) was performed using 1–2 ml of FS-069 injected intravenously and myocardial blood flow (MBF) was measured using radiolabeled microspheres at baseline and during dipyridamole (0.56 mg/kg) induced coronary hyperemia. In the presence of this drug, a stenosis was placed either on the left anterior descending (LAD) (n = 9) or the left circumflex (LC) (n = 6) coronary arteries and MCE and MBF assessment were repeated at each stage. MCE images, where contrast disparity between LAD and LC beds was maximal, were digitally-subtracted from pre-contrast images and mean videointensities in the LAD and LC beds were measured.

There was a good correlation between the LAD/LC bed videointensity ratio and LAD/LC bed MBF ratio ($y = 0.2x + 0.71$, $r = 0.77$, $p = 0.0001$, SEE = 0.16, n = 45). In addition, the region showing hypoperfusion during LAD or LC stenosis was planimetered and expressed as a percent of the myocardial area in the short-axis slice. There was also a fair correlation between the hypoperfused bed size on MCE and the hypoperfused myocardium as determined by radiolabeled microspheres during LAD or LC stenosis ($y = 0.8x + 3.3$, $r = 0.65$, $p < 0.001$, SEE = 4.2, n = 15).

We conclude that it is possible to detect coronary stenoses that are not flow limiting at rest with MCE using intravenous injection of FS-069 during pharmacologically-induced coronary vasodilation. The magnitude of blood flow mismatch and the spatial extent of relative hypoperfusion can also be quantified using this approach. Thus it is possible not only to detect the presence of coronary stenoses, but also to quantify their severity and the quantum of myocardium they supply using MCE from venous injection of 1–2 ml of this new contrast agent.

10:45

703-2 Detection of Regional Perfusion Abnormalities During Adenosine Stress Echocardiography Using Intravenous Perfluoropropane-enhanced Sonicated Dextrose Albumin

Alan Kricsfeld, Thomas Porter, Karen Kilzer, Ubeydullah Deligonul, Feng Xie. *University of Nebraska Medical Center, Omaha, Nebraska*

Altering sonicated dextrose albumin microbubble gas diffusivity and solubility with perfluoropropane results in microbubbles which produce myocardial contrast (MC) following an intravenous injection. It is unknown whether the intensity of MC produced will accurately identify flow abnormalities due to coronary ischemia. Accordingly, we measured peak myocardial videointensity (PMVI) from the left anterior descending (LAD) and left circumflex (LCX) perfusion beds following intravenous 0.06 ml/kg injections of perfluoropropane enhanced sonicated dextrose albumin (PESDA). The same injection was given under resting conditions, and during peak adenosine stress (ADS) (140μ/kg/min) in 6 open-chest dogs who had either no stenosis or an angiographically significant stenosis in the proximal LCX coronary artery (using quantitative angiography). PMVI from the LCX perfusion beds during ADS was compared with actual flows measured with a Transonic Doppler cuff around the LCX, and quantitative angiographic (QA) severity. Wall thickening (WT) responses were also measured as the difference in LCX perfusion bed end-diastolic and end-systolic wall thickness.

Injections of PESDA were given during 10 different stenosis severities in the six dogs (range 0–87% diameter). There was a strong correlation between PMVI in the LCX bed and actual LCX flow by Doppler ($r = 0.75$, $p < 0.001$). In the absence of a >50% stenosis, the PVI ratio in the LCX perfusion bed during ADS compared to baseline PVI ranged from 1.8 to 2.1, while the Doppler flow ratios in the LCX ranged from 2.0 to 4.2. When a greater than 70% diameter stenosis was present in the LCX, the PVI ratio during ADS compared to baseline ranged from 0.8 to 1.4 ($P = 0.02$; compared to PVI ratio without >70% stenosis), while the simultaneous Doppler ratios also decreased to a range of 0.5 to 2.8. There was a close correlation between Doppler flow ratios in the LCX and PMVI ratios during ADS ($r = 0.75$; $p < 0.05$). WT increased during ADS by 20% or greater in three of the six dogs despite a greater than 70% diameter stenosis. These data indicate that the background-subtracted PMVI produced with intravenous PESDA can identify abnormalities in coronary perfusion during ADS. This ultrasound contrast agent, therefore, could be a non-invasive method of determining myocardial perfusion abnormalities during adenosine stress echocardiography in humans.

11:00

703-3 Second Harmonic Mode Imaging Improves Contrast Agent [SHU 508-A] Visualization in an In Vitro Model

Antonio F. Amico, Michael Zomack [1], Jörg Petrick [1], Reinhard Schlief [1], Sabino Iliceto, Paolo Rizzon. *Institute of Cardiology, University of Bari, Italy;* [1] *Clinical Research Diagnostic, Schering AG, Berlin, Germany*

Non invasive myocardial contrast echocardiography requires an ultrasound imaging mode capable of detecting small amounts of a crossing lungs contrast agent (CA) which reach the cardiac muscle. At present, commercial B-Mode imaging can not visualize CA in myocardium after intravenous injection and transpulmonary passage. Second harmonic Mode imaging (SHM) uses resonance properties of microbubbles to produce a gray scale image of CA. We tested an prototype ultrasound machine (Acuson) using SHM to detect CA SHU 508-A (Schering AG, Berlin) flow in an in vitro model at various concentrations. For each CA injection, videointensity in arbitrary Units (VU), in SHM and B-Mode was assessed:

SHU 508-A injected volume (ml)	VU B-Mode	VU SHM	VU % increase
0.125	19	32	68
0.25	31	51	67
0.5	53	69	30
1.0	69	87	26
2.0	82	102	24
4.0	100	115	15

SHM vs. B-Mode $p < 0.0001$

Conclusions: SHM is more sensitive than B-mode in detecting SHU 508-A, particularly at lower CA concentrations. SHM has a potential for the evaluation of low CA concentration in clinical setting and therefore in myocardial perfusion studies after CA intravenous injection.