and TEL (5 echos). The rate of ECGs ordered was also not different. (P = 0.34)

Conclusion: Telemedicine interactive cardiology evaluations provide clinically accurate information, but important differences between TEL and FTF exist. Accurate telemedicine cardiology may be performed without echo in many pediatric patients.

1171-160 Telemedical Interpretation of Neonatal Echocardiograms: Impact on a General Pediatric Practice

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Background: Cardiology services can be difficult to provide in rural areas. This study was performed to evaluate the interpretation of remote site neonatal echocardiograms using real-time telemedicine.

Methods: Adult sonographers performed studies requested by general pediatricians at a site 350 miles from the pediatric cardiology center (hub). Preliminary images were recorded and transmitted from onbside to a pediatric echocardiologist at the hub using a T-1 system (1.5 megabits/sec). Additional real-time scans were then directed and monitored by the reviewer to complete each study.

Results: 115 T-1 echocardiograms were performed on 90 neonates. Median age was 2 days. 99% (89/90) of initial echocardiograms were performed urgently or emergently. Transmitted images provided adequate diagnostic information in all pts. T-1 diagnoses were confirmed in all pts with subsequent standard testing (N = 12). T-1 echo findings changed medical management or outpatient followup in 61% (55/90). An immediate change in management occurred in 26% (23/90). 6 babies were severely cyanotic and 1 was hypotensive due to CHD (Congenital Heart Disease). T-1 echo findings guided local stabilization and early transfer in 4. Unnecessary transfer was avoided in 2 cyanotic neonates whose cardiac malformations were not ductal dependent.

Conclusion: T-1 echo provides accurate diagnostic data in neonates. Rapid tele-diagnosis facilitates care of sick neonates with possible CHD in the primary care setting. Unnecessary long distance transfers can be avoided. Real-time, tele-echocardiography effectively extends tertiary pediatric cardiology into primary care settings.

1172 Renin Angiotensin System in Heart Failure

Wednesday, April 1, 1998, 9:00 a.m.–11:00 a.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 10:00 a.m.–11:00 a.m.

1172-53 Influence of Angiotensin on ANP Release and Renal Response to Volume Expansion in Moderate Heart Failure

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In heart failure, the release of ANP is impaired along with an activated renin-angiotensin system. We tested the hypothesis that ANP release is angiotensin II - dependent and studied the effect of ACE-inhibition (ramiprilat 50 mg/kg) and AT1-blockade (valsartan 0.3 mg/kg) on ANP release and on the renal response to acute volume expansion (5 ml/5 min hyperoncontic solution) in rats with shunt-induced moderate heart failure.

ANP plasma levels were increased in shunted rats (397 ± 39 pmol/l vs. 55 ± 8 in controls rats, $p < 0.001$) at baseline. After acute volume expansion, the ANP release was impaired in shunted rats (68 ± 4 vs. 135 ± 7 pmol/l, $p < 0.05$). Natriuresis and diuresis were blunted at baseline and after acute volume expansion (1250 ± 126 vs. 2753 ± 175 µl/60 min, $p < 0.001$). ACE-inhibition and AT1-antagonist did not influence hemodynamic parameters. Acute ACEI increased natriuresis and diuresis at baseline in heart failure rats compared to placebo treated rats (211 ± 50 vs. 95 ± 15 µl/20 min, $p < 0.05$). After acute volume expansion, ANP plasma levels significantly increased with ACEI and AT1-antagonist in shunted rats, inducing a further release of 190.9 ± 86.9 and 250.0 ± 48.5 vs. 67.7 ± 32.8 pmol/l with placebo ($p < 0.05$). N-terminal ANP increased similarly, confirming that the release of ANP was modified. Natriuresis and diuresis were enhanced by ACE-inhibition (2345 ± 332) and AT1-blockade (1860 ± 180 vs. 1250 ± 126 µl/60 min, $p < 0.01$). Our results demonstrate that acute angiotensin inhibition restores ANP release and renal responses after acute volume expansion in shunted rats. This suggests a new site of interaction between the angiotensin and the ANP system.

1172-54 Renin-Angiotensin System Inhibition Prevents Early but Not Late Remodeling of the Left Ventricle in Pacing-Induced Cardiomyopathy

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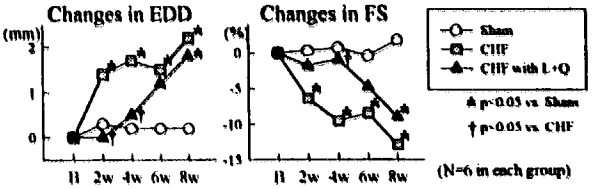
Background: The renin-angiotensin system (RAS) plays an important role in left ventricular (LV) remodeling in congestive heart failure (CHF); however, the results of RAS inhibition on LV remodeling have been conflicting. We speculate that the differences in results may be related to timing of interventions in CHF.

Methods: To study whether the effects of RAS inhibition may differ at various stages of development of CHF, we measured LV end-diastolic dimension (EDD, mm) and LV fractional shortening (FS, %) before (B) and after 2, 4, 6, 8 weeks of pacing-induced CHF (360 bpm for 8 weeks) in conscious rabbits. RAS inhibition was achieved by administration of both quinapril (Q, 10 mg/kg), an angiotensin-converting enzyme inhibitor and losartan (L, 50 mg/kg), an AT1 angiotensin II receptor blocker.

Results: Values were compared to Sham and untreated CHF animals.



Conclusion: Rapid ventricular pacing produces acute LV dilation and systolic dysfunction. RAS inhibition delays the onset of these changes in early CHF, but does not prevent LV remodeling in chronic CHF.

1172-55 Angiotensin II Type 1 Receptor Blockade: Myocardial Fibrosis, Stiffness, and Function After Infarction in the Rat

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Background: We examined the effects of specific angiotensin II type 1 (AT_1) receptor blockade on myocardial fibrosis, stiffness, and function after myocardial infarction (MI) in the rat.

Methods and Results: Rats were randomized to losartan or placebo 1 day after MI and treated for 8 weeks. In sham and MI rats, losartan decreased ($P < 0.05$) LV and RV weights, mean aortic and LV systolic pressures. LV end-diastolic pressure (EDP) was increased ($P < 0.05$) in MI vs sham rats. Losartan decreased ($P < 0.05$) LV-EDP in MI but not in sham rats. Maximal developed tension (DT) and peak rate of tension rise (+dT/dt) were decreased ($P < 0.05$) in MI vs sham (DT: 1.64 ± 0.46 vs 3.45 ± 1.11 g/mm^2; dT/dt: 15.9 ± 4.4 vs 34.5 ± 12.0 g/mm^2/sec). Losartan decreased DT and +dT/dt in sham rats and increased DT and +dT/dt in MI rats resulting in a significant interaction ($P = 0.043$ and $P = 0.045$, respectively). The peak rate of tension decline decreased ($P < 0.05$) in MI vs sham rats (-10.6 ± 3.8 vs -16.2 ± 5.4 g/mm^2/sec) with a trend to increase by losartan in MI rats (-2.5 ± 5.1 g/mm^2/sec). Interstitial fibrosis increased ($P < 0.05$) in MI vs sham and decreased ($P < 0.05$) with losartan. Myocardial stiffness increased ($P < 0.05$) after MI but normalized ($P < 0.05$) with losartan.

Conclusions: After MI, AT_1 receptor blockade has beneficial effects on myocardial contractility and normalizes myocardial stiffness with an associated reduction in myocardial fibrosis.

1172-56 Angiotensin Converting Enzyme Inhibition Increases Cardiac Adenylycyclase Expression in Congestive Heart Failure

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Background: Heart failure is associated with decreased adenylycyclase (AC) activity and downregulation of AC mRNA, particularly type VI (AC_{VI}). The effect of ACE inhibition on AC, a pivotal regulating element in transmembrane, signaling, is unknown.

Methods: We studied 23 pigs (51 ± 6 kg): 11 controls: 6 received no treatment (CON) and 5 received lisinopril (LIS, 0.5 mg/kg/d iv for 10d) and 12 paced from the LV (220 bpm, 20d): 6 received no treatment (HF) and 6 received lisinopril for the final 10 days (HF/L). Heart failure was documented (pacers off) by decreased LV fractional shortening ($p < 0.001$) and increased left atrial pressure ($p < 0.001$). We measured LV AC activity, and mRNA content of AC_{VI}, the predominant AC isoform in pig heart.