group 5 (34.4 ms, $p < 0.02$). The table shows mean ± SD in groups 1, 2 and 3.

	QRS	QTd (range)	QRSd	JTd
1.	202 ±22	112.5 ± 37(70–190)	58.5 ± 21.2	104 ±38.4
2.	192.2 ± 8	45.6 ± 10.1(35–60)*	24.4 ± 11.9*	43.8 ± 11.9*
3.	138.2 ± 21*	45.5 ± 14.3(30–75)*	19.5 ± 4.2*	39.5 ± 14.5*

*Significant p values < 0.001–0.005, compared with group 1.

QRS, QT and JT d was significantly greater in our patients with documented VTs suggesting that both depolarisation and repolarisation anomalies may be involved in the pathogenesis of VTs. Furthermore, having identified patients with a QRS ≥ 180 ms an additional QTd ≥ 70 ms or a QRSd ≥ 35 ms or a JTd ≥ 60 ms was 100% sensitive and specific for identification of VTs.

10:45

786-2 JTc and QTc Dispersion in Patients Following Surgical Repair of Tetralogy of Fallot

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Ventricular arrhythmias (VA) following repair of tetralogy of Fallot (TOF) are considered a probable mechanism of sudden death. Areas of myocardial damage following surgical repair are hypothesized to provide a substrate for abnormal ventricular depolarization. The aim of this study was to assess the usefulness of QTc dispersion (QTcd) and JTc dispersion (JTcd) as non invasive markers for risk stratification of VA. We reviewed the surface electrocardiograms (ECG) of 65 patients who had repair of TOF and developed complete right bundle branch block. Patients were divided in 2 groups. Patients in group 1 (n = 14) had inducible VA during electrophysiology studies and 11 of 14 patients also had clinical sustained VA. Group 2 consisted of patients with no documented episodes of sustained VA. QT, JT, QRS, QTc, JTc intervals as well as QTcd and JTcd were measured on standard ECGs. In group 1, ECGs were analysed was at a mean age of 14 ± 4 years. In group 2, ECGs were analysed at a mean age of 11 ± 6 years.

	AGE years	QTcd ms	JTcd ms	QRS ms
Group 1 n = 14	14 ± 4	83 ± 42**	83 ± 41**	160 ± 26*
Group 2 n = 51	11 ± 6	43 ± 29	42 ± 30	138 ± 23

*p < 0.05 **p < 0.01

A significant difference in JTcd and QTcd was found in comparing group 1 to group 2 ($p < 0.01$). Abnormal depolarization in the right ventricle following surgical repair of TOF is recognized to be associated with VA. Abnormalities of repolarization are documented in prior studies but have not been linked to VA. An increased JTcd in patients with VA suggests heterogenous repolarization. This may increase susceptibility to sustained VA triggered by premature ventricular beats. Following surgical repair of TOF, QTcd and JTcd may be appropriate non invasive markers of electrical instability predisposing to life threatening arrhythmias.

11:00

786-3 Prognosis of in Utero Congenital Complete Heart Block

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Isolated congenital complete heart block (CCHB) can be reliably diagnosed in utero. Little is known, however, about the prognostic value of the presenting fetal heart rate.

We observed the outcome of a consecutive series of 41 fetuses with CCHB diagnosed in utero between 1980–1995. Presenting fetal heart rate, change in fetal heart rate and development of fetal hydrops were documented. Anti-Ro antibody was present in 34 of the mothers, absent in 4 and not documented in 2.

Significant and progressive hydrops developed in 14 fetuses. Pregnancy was terminated in 2 of these and a further 6 died in utero. 3 fetuses were delivered prematurely because of worsening hydrops but they died in the neonatal period from problems of prematurity — despite effective pacing in 2.3 fetuses with hydrops recovered — 1 after a course of maternal sympathomimetic treatment. One non-hydropic fetus died suddenly in utero.

Heart rate at presentation ranged between 45–80 beats per minute (bpm). A fall in heart rate with advancing gestation was detected in 11/30 fetuses who had more than one examination: of these 6 died in utero. Presenting heart rate was related to outcome as shown in the table.

	no	Died	Pacemaker	Alive — no pacemaker
≤ 50 bpm	7	4	2	1
51–60 bpm	21	8*	8	5
> 60	13	1	3	9

*1 non-paced infant died from pertussis. Of the 28 survivors, 13 patients (46%) have required pacing (follow up 1–14 years).

Isolated CCHB does not always carry a good prognosis. Hydrops is a poor prognostic sign. A heart rate ≤ 50 bpm at presentation, or a falling heart rate, dictate the need for careful evaluation of cardiac function to guide possible intervention and a guarded prognosis.

11:15

786-4 Fetal Tachyarrhythmias: Intervention and Outcome

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A retrospective analysis of 35 fetal tachyarrhythmias (FT) detected during 2800 prenatal cardiac ultrasound scans from 1979–1995, to assess the efficacy of therapy, & immediate outcome.

23/35 patients had supraventricular tachycardia (SVT): mean gestational age (GA) was 30.5 (± 3.8) wks & heart rate was 253 (± 33). 22/23 received transplacental Digoxin (TPD) with slowing or conversion of the SVT in 61%. Hydrops improved in 26% & progressed in 35% until delivery at a mean of 5 weeks later. 1 death occurred in a refractory case with significant structural congenital heart disease (CHD) at 23 weeks GA. 10/35 patients had atrial flutter (AF) with variable A:V block. GA was 33.3 (± 2.6) weeks, & the mean ventricular rate was 222 (± 40). Rhythm slowing or conversion occurred spontaneously in 1 & with TPD in 1/8. Hydrops failed to improve AF & was progressive in 40% until delivery, a mean of 2.5 weeks later. 1 case of alternating brady-tachyarrhythmia, & 1 with chaotic atrial rhythm required no intervention.

In refractory cases, Verapamil (n = 3), Sotalol (n = 2), Flecainide (n = 4), & Amiodarone (n = 3) were used. Only Amiodarone proved consistently effective. Significant CHD was present in only 1 case (dysplastic RV and tricuspid valve). 33/35 patients survived with 1 intrauterine death, & 1 of chronic lung disease of prematurity. Comparing patients with early onset (GA < 31 weeks) and later onset (GA > 31 weeks) of arrhythmias: mean time to delivery was longer (> 8 weeks vs < 3 weeks), hydrops more often marked (50% vs 33%), and hypotension in the newborn period requiring inotropic support more common (33% vs 14%).

Conclusions: FT have a generally favourable outcome, with TPD often effective for SVT but not AF. Amiodarone is the most effective second line agent. Early onset of arrhythmia predicts a less favourable course.

11:30

786-5 Doppler Tissue Imaging to Localize Accessory Pathways in Pediatric Patients With Wolff-Parkinson-White Syndrome

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Doppler Tissue Imaging (DTI) is a new echocardiographic technique that allows direct measurement of myocardial velocities. In patients with Wolff-Parkinson-White syndrome (WPW), accessory pathways (AP) produce ventricular depolarization prior to the normal activation via the AV node. This early activation may cause systolic ventricular motion near the site where the AP enters the ventricle. We compared the earliest ventricular systolic motion seen by DTI with the location of AP found on electrophysiologic study (EPS).

Ten patients (pts) age 4.5–20 y with WPW underwent color M-mode DTI of the left ventricle (LV) in the parasternal short axis view prior to EPS. The time interval in milliseconds (ms), from the Q wave to initial septal (Q-S) and LV posterior wall (Q-LV) motion was measured for 3–5 beats. The location of the AP was determined by intracardiac mapping.

Q-S ranged from 4–117 ms and Q-LV ranged from 41–135 ms. In 4 pts Q-LV was less than Q-S, suggesting a left sided AP, and in 6 pts Q-LV was greater than Q-S, suggesting a right sided or paraseptal AP. EPS revealed 8 right paraseptal AP and 2 left posterior AP. DTI correctly predicted 6 of 8 paraseptal AP and 2 of 2 left sided AP. DTI incorrectly predicted a left AP in 2 pts found to have right posterior paraseptal AP by EPS. For the right paraseptal pathways, measured Q-S was significantly correlated with location of AP on the tricuspid valve annulus ($r = 0.79$). Thus, DTI analysis of initial systolic ventricular motion may provide a sensitive, noninvasive method for determining location of AP in pts with WPW.