with diabetes. The assessment of vascular stiffness using AI in patients with hypertension must also take into account the additional presence of diabetes.

### Table: Hypertensive, Diabetic and Control Groups

<table>
<thead>
<tr>
<th>Age (Yrs)</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
<th>PP (mmHg)</th>
<th>HR (bpm)</th>
<th>AR (%)</th>
<th>PWV (m/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>57.4 ± 2</td>
<td>129.5 ± 2</td>
<td>76.9 ± 1</td>
<td>52.7 ± 2</td>
<td>65.2 ± 3</td>
<td>25.8 ± 3</td>
</tr>
<tr>
<td>Diabetic</td>
<td>58.5 ± 2</td>
<td>141.8 ± 3*</td>
<td>79.5 ± 2</td>
<td>62.2 ± 3*</td>
<td>72.4 ± 2*</td>
<td>27.3 ± 3*</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>57.2 ± 2</td>
<td>170.5 ± 3*</td>
<td>95.5 ± 2**</td>
<td>74.5 ± 3**</td>
<td>65.9 ± 2</td>
<td>34.1 ± 1*</td>
</tr>
</tbody>
</table>

*P<0.05, **P<0.01.

Key Words: Hypertension, Vascular Stiffness, Application Tonometry

### P.99

**THE EFFECT OF STEPWISE CHANGES IN PACED HEART RATE ON CARDIAC OUTPUT, ARTERIAL BLOOD PRESSURE AND ARTERIAL PROPERTIES AS MEASURED BY SYSTEMIC ARTERIAL COMPLIANCE AND PULSE WAVE VELOCITY**

**Christoph D. Gatzka, Anthony M. Dart. Alfred and Baker Medical Unit, Alfred Hospital and Baker Medical Research Institute, Melbourne, VIC, Australia.**

**Background:** Recent research has examined the effect of an increased heart rate on arterial stiffness. The results were partially contradictory and in particular it remains unresolved how pulse pressure amplification can change without a change in arterial properties.

**Methods:** We measured brachial and finger blood pressure (BP, Dinamap & Finapres), carotid-femoral (CF) and femoral-dorsalis pedis (FD) pulse wave velocity and cardiac output (CO, echo) at paced heart rates (in random order) of 60, 75, 90, 105 and 120 bpm in 13 middle-aged otherwise healthy, unmedicated males with an implanted dual chamber cardiac pacemaker (sinus-sinus or AV-block). Patients were examined twice at 75 bpm at the start and end to assess reproducibility of all methods.

**Results:** Doubling heart rate (60-120) had no effect on cardiac output with appropriate stepwise changes of stroke volume (reduction to half at double heart rate, p<0.001). Brachial and mean arterial pressure increased stepwise modestly by up to 10 mm Hg (p<0.01). Diastolic pressure increased more than systolic pressure both at the finger and at the brachial artery (p<0.001). Hence, pulse pressure decreased by 14 mmHg at the brachial artery (p<0.001) and by only 4 mmHg at the finger (n.s.). Systemic arterial compliance (stroke volume/pulse pressure) decreased gradually by up to 41% with increases in heart rate (p<0.01) and both CF and FD pulse wave velocity increased gradually by 6 and 22%, respectively (p<0.05,p<0.01).

**Conclusions:** We conclude that increasing heart rate by dual chamber pacing over a wide range (doubling) results in small, but measurable increases in arterial stiffness both centrally (systemic arterial compliance, CF) and peripherally (FD). This increase in arterial stiffness could be related to an increase in mean arterial pressure and not heart rate per se since the amount of change in pressure and pulse wave velocity is similar, but less than a quarter of the change in heart rate. Hence, previously observed changes in pressure amplification and earlier return of the reflected wave with an increase in heart rate are likely secondary to this observed pressure-related increase in arterial stiffness.

Key Words: Heart Rate, Systemic Arterial Compliance, Pulse Wave Velocity

### P.100

**RELATIONSHIP BETWEEN ARTERIAL DISTENSIBILITY AND LEFT VENTRICULAR FUNCTION IN THE TIMING OF KOROTKOFF SOUNDS (QKD INTERNAL). AN AMBULATORY PRESSURE MONITORING AND ECHOCARDIOGRAPHIC STUDY**

**Philippe Abassade, Yves Baudouy. Cardiology, Saint Michel, Paris, France.**

Timing of Korotkoff sounds (QKd) is the time interval between Q wave ECG and pressure diastolic sound, determined by a routine ambulatory pressure monitoring (AMP) (Diassys Integra, Novacor). It is known to be an arterial distensibility index. However, this interval time includes pre-ejection time (PET) which is related to left ventricular (LV) function.

The aim of the study is to define the relative influences of LV function and arterial distensibility in QKD interval.

Sixty consecutive patients, with or without cardiac disease, out or in-hospital, were included. Patients with left bundle branch block, pace maker, or atrial fibrillation were excluded. The echocardiography-Doppler (ED) study collected LV function index: Shortening Fraction (SF), Ejection Fraction (EF), stroke index (SI), (PET), ejection time (ET). Pulse Wave velocity (PWV) was determined by ED in two points of the descending thoracic aorta. The AMP study collected usual pressure data: systolic (Ps), diastolic, mean (Pm), pulse pressure (Pp), simultaneously with PWV and over 24h. An automatic assessment of the QKD interval simultaneously with PWV was performed by the monitoring device. Another arterial distensibility index was calculated from ED and AMP: Burton index as SI / Pp. Values are mean plusminus SD, relationships between various parameters were fitted by a linear function.

QKd is correlated with PWV (n = 53, r = 0.007, r = 0.37), with Burton index (n = 50, p = 0.001, r = 0.47).

QKd is correlated with CV function index like FE (n = 55, r = 0.001, r = 0.46), Psystolic diameter (n = 54, r = 0.75 , SF / ET (n = 35, r = 0.001, r = 0.55), ET / PET (n = 35, p < 0.001, r = 0.60)

No correlation was found between QKd and EF when PET are withdrawn from QKd (n = 35, r = 0.21, r = 0.22).

QKD interval is a composite index which reflects not only arterial distensibility but also LV function.

Key Words: Arterial Distensibility, Left Ventricular Function, Echocardiography