

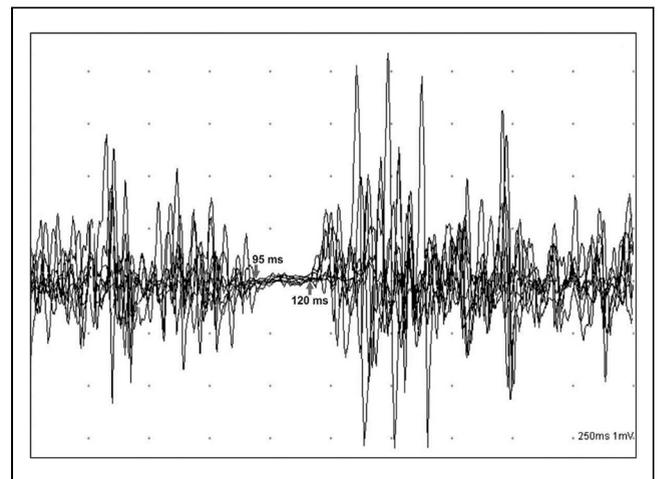
**BACKGROUND** Atrial fibrillation is (AF) the most common sustained cardiac rhythm disorder. Small nerve fibres carry autonomic modalities, somatic pain and temperature sensations. The aim of our study was to assess the role of small A-delta nerve fibres in the peripheral nerves of patients with AF.

**METHODS** The function of small nerve fibers was assessed by cutaneous silent period (CSP) elicited by electrical square pulse stimulation using stimulating ring electrodes on the index finger and recording electrodes over thenar muscles. The CSP onset, end latencies and suppression of muscle activity - duration over APB were measured. Patients either with polyneuropathy or drugs that contribute to peripheral nerve involvement were excluded.

**RESULTS** In the study 30 patients with paroxysmal AF and 30 age-matched healthy subjects were comprised. The mean CSP onset latency in patients with AF was significantly longer compared with the control group ( $86.67 \pm 8.19$  vs.  $68.05 \pm 7.81$ , CI 83.2-90.1 vs. 65.9-70.0), meanwhile CSP duration was markedly shorter in AF patients ( $45.10 \pm 10.96$  vs.  $60.95 \pm 10.14$ , CI 40.7-49.6 vs. 58.3-63.4). Nerve conduction study of the large motor and sensory fibers did not reveal any difference between patients and healthy subjects.

Fig. 1 Cutaneous silent period in patient with AF

**CONCLUSION** In our study, the main attention was focused on the extrinsic cardiac nervous system using a CSP measurement. The delay of CSP onset latency reflects the impairment of afferent volley of A-delta afferents, efferent motor axons and synaptic delay, while shortened CSP duration is related to the amount of activated axons and indicates the axonal lesion. Abnormality of CSP in AF patients supports the occurrence of small nerve fiber neuropathy. Our study of small nerve fibres may imply a new aspect in the etiology of AF.



**OTHER**

**CRT-500.06**

**Related Variables in the Screening for Prevalence of Prehypertension in Young Adults Students at Technical School**



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**BACKGROUND** Worldwide, several studies have been conducted about the association between hypertension in childhood and adolescence and socio-demographic factors: lifestyle, family history and anthropometry.

**OBJECTIVE** This study aims to identify the prevalence of prehypertension and related variables in young adults.

**METHODS** Cohort study. The variables were collected by questionnaire or measures. Univariate analysis was performed using the chi square and it was performed five multiple logistic regression models for the variables with  $p < 0.10$  in the univariate analysis. The students were from three courses, either college as vocational school, were evaluated: gender, age, course, skin color, income, education, lifestyle, history of hypertension, weight, waist circumference and prehypertension defined as VII Joint National Committee: systolic 120-139 and diastolic 80-89 mmHg.

**RESULTS** A total of 394 students were evaluated. There were 309 (78,43%) in the normal group (NG) and 85 (21,57%) in prehypertension group (PH) of students. It was found in NG and PG, respectively: females 254 (82.2%) and 44 (51.8%) ( $p < 0.001$ ); age (three age ranges: until 19 years, 20-25 and 25-30) more frequent in older ( $p = 0.001$ ); ethnicity (self declared) black 16 (5.2%) and 11 (12.9%) ( $p < 0.001$ ); 62 mother's hypertension (20.1%) and 28 (32.9%) ( $p = 0.024$ ); overweight 34 (11.0%) and 17 (20.0%) ( $p = 0.045$ ); obese 3 (1.0%) and 10 (11.8%) ( $p < 0.001$ ); increased abdominal circumference 37 (12.0%) and 19 (22.3%) ( $p = 0.024$ ). At least one of five multiple logistic regression models were associated with absence or presence of prehypertension (OR, 95% CI): females (4.026, 2.373 to 6.828), age (1.081, 1.004 to 1.164), hypertensive mother (1.838, 1.027 to 3.289) and greater waist circumference (1.067, 1.035 to 1.100).

**CONCLUSION** About a fifth of the students were considered to be in prehypertension group. Factors associated with prehypertension in this study: male, older, mother with hypertension and increased waist circumference.

**CRT-500.07**

**The Role of Small Nerve Fibers in the Development of Atrial Fibrillation**



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**CRT-500.09**

**Reliability of The Cardiac Output Measurements During Catheterization: Comparison of Various Commonly Used Formulae Calculating Assumed O<sub>2</sub> Consumption**



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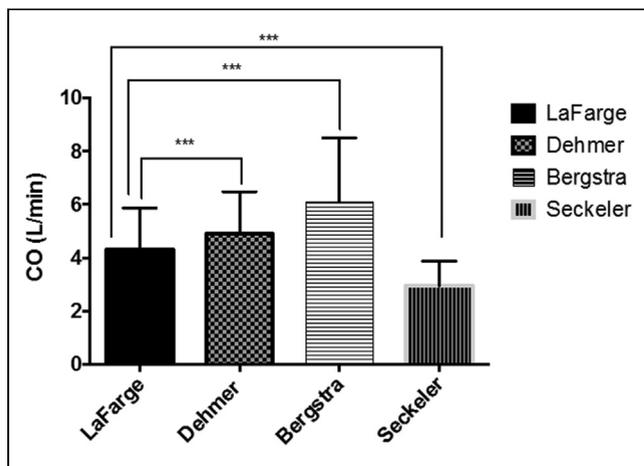
**BACKGROUND** Cardiac output (CO) measurement guides management of various medical conditions, including adult congenital heart diseases (ACHD) and pulmonary hypertension. It is mandatory to calculate patients' oxygen consumption (VO<sub>2</sub>), to measure CO. Ideally VO<sub>2</sub> consumption should be measured by using a metabolic apparatus;

however, due to complexity in their routine use, various formulae derived assumed  $VO_2$  are utilized. The most commonly used formula in catheter laboratories treating adult patients was reported by LaFarge. However, it was based on data from paediatric population, and their use in adult population is not validated. Additionally, limited information exploring agreement between these formulae is available. We sought to compare formulae derived CO measurement.

**MATERIALS AND METHODS** We sought to compare cardiac output measurement based upon four commonly used formulae, (1) LaFarge and Miettinen, (2) Dehmer, Firth & Hills, (3) Bergstra, Van Dijk, Jillege, and (4) Seckeler, Hirsch, Beekman formula in 112 ACHD patients who underwent diagnostic catheterization between 1<sup>st</sup> January 2015 to 31<sup>st</sup> March 2017.

**RESULTS** CO measured by various formulae is reported here with LaFarge:  $4.31 \pm 1.43$  L/min; Dehmer:  $4.91 \pm 1.50$  L/min; Bergstra:  $6.1 \pm 2.22$  L/min; and Seckeler:  $2.96 \pm 0.88$  L/min. LaFarge and Miettinen formula derived CO was significantly lower than Dehmer and Bergstra formulae ( $P < 0.0001$  for each), whereas significantly higher than the formula by Seckeler.

**CONCLUSION** There is no agreement between the formulae derived assumed  $VO_2$  and resultant CO. Such assumed formulae derived CO may be misleading. Every cardiologist should be cognizant of these limitations. Metabolic apparatus should be routinely used in catheter laboratories to obtain true  $VO_2$ , especially, when such a value is likely to influence major management decisions.



**CRT-500.10**  
**Should the Normal Range of Pulmonary Vascular Resistance Be Re-defined in Patients with Fontan Circulation?**



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**BACKGROUND** Creation of Fontan circulation helps separating pulmonary and systemic circuits in patients born with single ventricle. They have impaired exercise capacity, mainly due to limited augmentation of cardiac output (CO). Studies have suggested that pulmonary vasodilator therapies improve CO by augmenting venous return. Use of such therapy is significantly sparse, as many of these patients are reported to have normal pulmonary vascular resistance (PVR). However, the normal range of PVR was defined only in those with subpulmonic ventricle and pulsatile pulmonary flow. We sought to evaluate observed PVR in this cohort.

**MATERIALS AND METHODS** Retrospective review of data from a large tertiary ACHD centre.

**RESULTS** From a cohort of 4454 patients with complex ACHD conditions, 154 had Fontan circulation; of whom 70 patients with failing Fontan were investigated by cardiac catheterization. Thirty-four (48.6%) were male, mean age of  $30.1 \pm 6.2$  years (17-43), and mean body mass index of  $24.1 \pm 5.3$  kg/m<sup>2</sup> (16.6-47.7). Mean Fontan pressure was  $16 \pm 4$  mmHg (7-29), mean wedge capillary pressure was  $11 \pm 3$  mmHg (4-19), and mean trans-pulmonary gradient (TPG) was  $5 \pm 3$  mmHg (0-15). Mean CO was  $4.1 \pm 2.7$  L/min and calculated PVR was  $1.7 \pm 1.2$  Wood units (0.25-5.3). Although cardiac output was well maintained, Fontan pressure significantly correlated with capillary wedge pressure ( $R^2 = 0.75$ ,  $P < 0.0001$ ) and PVR ( $R^2 = 0.51$ ,  $P < 0.0001$ ). Rise in Fontan pressure was observed earlier than increase in PVR. Interestingly, 3/4<sup>th</sup> of patients with failing Fontan were noted to have normal PVR despite elevated Fontan pressure.

**CONCLUSIONS** In this large series of patients with Fontan circulation, rise in PVR above the normally accepted range was observed only after moderate rise in Fontan pressure. In patients with Fontan circulation, range of normally accepted PVR should be redefined.

