A Short PR Interval on a Routine ECG

1. Is there any significance to a short PR interval on a routine ECG? Are further investigations indicated?

Question submitted by: Dr. Ramsamooj, Winnipeg, Manitoba

The PR interval on ECG represents the timing from the onset of atrial activation to the onset of ventricular activation and a short PR interval is defined as less than 120 ms. The term pre-excitation refers to the scenario when the ventricle is activated from two foci–normal activation through the AV node as well as early activation through an accessory pathway connecting the atria to ventricles.

While the term pre-excitation has been recently broadened to include all scenarios in which the ventricle is activated early, it classically refers to the characteristics of the baseline ECG in patients with Wolf-Parkinson-White syndrome. Since accessory pathways do not have the same slowing effect on conduction that the AV node does, an impulse travelling through the accessory pathway results in ventricular activation earlier than through the AV node, and this pre-excitation is represented by a short PR interval and widened QRS secondary to a delta wave. Wolf-Parkinson-White refers to a syndrome of pre-excitation on baseline ECG and a history of paroxysmal palpitations or supra-ventricular tachyarrhythmias. Since patients with pre-excitation are at increased risk of sudden cardiac death, due to the presence of an accessory pathway, consultation with an electrophysiologist is warranted for consideration of an electrophysiology study and ablation (in selected patients) irrespective of a patient's symptoms.

In contrast, Lown-Ganong-Levine is a syndrome of a short PR interval, normal QRS complex, and lack of delta wave on baseline ECG. It is due to either an accessory pathway adjacent to the AV node or enhanced AV nodal conduction. While at increased risk of supra-ventricular tachycardias, they do not have the same increased risk of sudden cardiac death and only require consideration for referral if they have a history of palpitations or syncope.

Resource
1. Podrid PJ: Lown-Ganong-Levine Syndrome and Enhanced Atrioventricular Nodal Conduction. Uptodate.com Accessed: February 2012.

Answered by:
Dr. Richard Vandegriend and Dr. Brett Heilbron

Significance of a PFO on an Echocardiography

2. What is the clinical significance of a patent foramen ovale (PFO) seen incidentally on echocardiography in an asymptomatic patient?

Question submitted by: Dr. Roshan Dheda, Bradford, Ontario

Doing screening tests on asymptomatic patients has its disadvantages; uncovering minor anomalies of questionable clinical significance, like a patent foramen ovale, is one of them. A patent foramen ovale (PFO) is the non-fused remnant connection between right and left atrium, absolutely mandatory for normal fetal development in utero, but a nuisance thereafter. PFO is present in 10 to 25% of the population, and, as an isolated finding, should not raise eyebrows of concern. However, as postulated back in 1877, some strokes of unknown etiology may be the result of a paradoxical embolism traversing through a PFO. Although a clear causative relationship has not been established between PFO and stroke, the risk of stroke is higher in patients who have a PFO in addition to other risk factors, such as diabetes, hypertension, or an atrial septal aneurysm. But, as for the asymptomatic patient with no risk factors and an otherwise structurally normal heart, only calming reassurance is needed.

Reference
1. Kutty S, Sengupta PP, Khandheria BK: Patent Foramen Ovale: The Known and the To Be Known. J Am Coll Cardiol. 2012; 59(19):1665-1671.
2. Homma S, Sacco RL, Di Tullio, *et al*: Effect of Medical Treatment in Stroke Patients with Patent Foramen Ovale: Patent Foramen Ovale in Cryptogenic Stroke Study. Circulation 2002; 105(22):2625–2631.
3. Handke M: Patent Foramen Ovale and Cryptogenic Stroke in Older Patients. N Engl J Med 2007; 3579(22):2262–2268.

Answered by:
Dr. Theodore K. Fenske

CHD Risk in Japan vs. the West

3. Why is the risk of CVD lower in Japan?

Question submitted by: Dr. Shiraz Shariff, Brampton, Ontario

I would like to change the question a little bit to discuss the risk differences for coronary heart disease (CHD) since that is where we have most of the information.

Probably the best attempt at explaining the differences in CHD risk between industrialized Western societies and Japan comes from a study reported in 2003 in the Journal of Human Hypertension. In this study, several groups of middle-aged Japanese men and women living in Japan were compared to a similar group of ethnic Japanese who had been living in Hawaii for three or four generations. This latter group shared the same genetic milieu, but were presumably living a Western lifestyle. Ethnic Japanese living in Japan had significantly lower risk of coronary heart disease events, and stroke. The question is — how do the risk factors differ, and do they help explain the difference in risk?

Japanese persons living in Japan had much higher smoking rates (especially among men), higher blood pressure (both systolic and diastolic), and substantially lower treatment rates for hypertension than those living in Hawaii. On the other hand, body mass index, serum total and low-density lipoprotein (LDL) cholesterol, hemoglobin A1c, and fibrinogen were significantly lower, and high density lipoprotein (HDL) cholesterol was higher in Japan than in Hawaii.

There were also important dietary differences. Total fat, saturated fatty acid intake, and dietary lipid scores were lower in Japan than Hawaii. In contrast, polyunsaturated fat/saturated fatty acid ratios and omega-3 fatty acid intake were higher in Japan than in Hawaii. Alcohol consumption among men was higher in Japan than in Hawaii.

Over all, the differences in CHD risk factors were much smaller in women than in the men. It appears that the lower CHD risk factors, especially lipid factors, counter the adverse risk from smoking and hypertension, especially among Japanese men.

Answered by:
Dr. Wayne Warnica

Benefit to Adding a Nighttime Antihypertensive

4. Is there any benefit to adding a nighttime antihypertensive?

Question submitted by: Julie Connolly, Sudbury, Ontario

There may well be a benefit to adding a nighttime antihypertensive. The Heart Outcomes Prevention Evaluation (HOPE) study found that a dose of ramipril, 10 mg at bedtime resulted in marked reductions in morbidity and mortality.[1] More recently, an observational study in patients with resistant hypertension showed that those receiving one or more of their antihypertensive drugs at bedtime had lower ambulatory blood pressure at nighttime and for the following 24 hours.[2] Surprisingly, they also showed lower values of glucose, LDL cholesterol, and urinary albumin excretion. Whether these differences in surrogate endpoints will translate into reduced clinical complications awaits further data.

References
1. Yusuf S, Sleight P, Pogue J, *et al*: Effects of an Angiotensin-converting-enzyme Inhibitor, Ramipril, on Cardiovascular Events in High-risk Patients. The Heart Outcomes Prevention Evaluation Study Investigators. N Engl J Med 2000; 342(3):145–153.
2. Hermida RC, Ayala DE, Mojon A, *et al*: Effects of Time of Antihypertensive Treatment on Ambulatory Blood Pressure and Clinical Characteristics of Subjects with Resistant Hypertension Am J Hypertens; 2010; 23(4):432–439.

Answered by:
Dr. Thomas W. Wilson

Using hs-CRP in CV Risk Stratification

5. How should hs-CRP be used in Cardiovascular risk stratification?

Question submitted by: Larry Bobyn, Kelowna, British Columbia

Clinical and laboratory studies have increasingly shown that inflammation plays a central role in the initiation, progression, and destabilization of atherosclerotic vascular plaques. When the body's inflammatory response is turned on, the production of complement proteins escalates. The best known acute-phase complement protein is the C-reactive protein, or CRP, which was first identified during the Great Depression, around the same time that Sir Alexander Fleming discovered penicillin. With our current technological advances, it has been demonstrated that even the tiniest elevations in CRP levels measured as high sensitivity CRP (hs-CRP), reflect low-grade, grumbling inflammation and are strongly correlated with an increased risk of developing coronary artery disease. The Justification for the Use of Statins in Prevention: An Intervention Trial Evaluating Rosuvastatin (JUPITER) demonstrated that statin therapy can reduce the risk of vascular events in otherwise healthy adults who have elevated hs-CRP levels. Recognizing its additive prognostic value, the Canadian Working Group included hs-CRP measurement in their recommendations to further stratify patients at risk who fall into the intermediate or moderate risk category, defined as 10 to 20% with a 10 year risk of a vascular event. Men older than 50-years-of-age and women older than 60-years, who are at moderate risk for CVD (determined by Framingham Risk Score) and whose level of LDL-C is less than 3.5 mmol/L, are candidates for hs-CRP measurement. Pharmacological therapy is indicated if the hs-CRP is higher than 2 mg/L in such patients, irrespective of LDL-C level (class IIa, level B).

Resources
1. Ridker PM, Danielson E, Fonseca FA, *et al*: JUPITER Study Group. Rosuvastatin to Prevent Vascular Events in Men and Women with Elevated C-reactive Protein. N Engl J Med 2008; 359(21):2195–2207.
2. Genest J, McPherson R, Frohlich J, *et al*: Canadian Cardiovascular Society/Canadian Guidelines for the Diagnosis and Treatment of Dyslipidemia and Prevention of Cardiovascular Disease in the Adult — 2009 Recommendations. Can J Cardiol 2009; 25(10):567–579.

Answered by:
Dr. Theodore K. Fenske

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