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Internal Medicine

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ABSTRACT & COMMENTARY

Jogging and Long-term Mortality

By Harold L. Karpman, MD, FACC, FACP

Clinical Professor of Medicine, UCLA School of Medicine

Dr. Karpman reports no financial relationships relevant to this field of study.

SYNOPSIS: Joggers who perform light and moderate jogging programs have lower mortality than sedentary non-joggers, whereas strenuous joggers have a mortality rate not statistically different from that of the sedentary group.

SOURCE: Pate RR, et al. Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 1995;273:402-407.

Over the years, hundreds of articles have been published about the positive effects of physical activity on the rate of occurrence of cardiovascular disease and its mortality. In 1965, the President's Council on Physical Fitness published a recommended program and, since then, numerous studies have strongly supported the finding of an inverse relationship between the degree of regular exercise and mortality, demonstrating that physically active men and women have an approximately 30% lower risk of death compared with inactive people.[1-4] In 1969, the first running race in Europe (the Eremitage Race) took place in Denmark. Unfortunately, one of the 2344 participants, a 46-year-old naval officer, died of a myocardial infarction during the event. The race has continued despite the event organizers' initial concern that the 7.6 mile course was too strenuous and possibly even dangerous for the general population, but, of course, since that time, numerous reports of death during jogging have been published.[5-10]

The Copenhagen City Heart Study is composed of a random sample of 19,329 Caucasian men and women between 20 and 93 years of age drawn from the Copenhagen Population Register as of January 1,

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[INSIDE]

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1976. This study revealed that the relative intensity of walking and cycling, and not the duration, was of most importance in relation to all-cause and coronary heart disease mortality, and that the increase in survival among joggers was on average 6.2 years in men and 5.6 years in women.[10,11]

Schnohr and his colleagues re-analyzed the data of 5048 men and women (1098 healthy joggers between 20 and 81 years of age and 3950 healthy non-joggers) who were followed for 12 years.[12] The joggers were categorized into three groups: light, moderate, and strenuous joggers. The detailed analysis revealed that the optimal frequency of jogging was 2-3 times per week, resulting in a total of 1-2.4 hours of jogging per week; the optimal pace was slow or average; and the lowest hazard ratio for mortality was found in light joggers, followed by moderate joggers and strenuous joggers.

The results suggested a U-shaped association between all-cause mortality and the dose of jogging, as calibrated by pace, quantity, and frequency of jogging. Light and moderate joggers had lower mortality than sedentary non-joggers, whereas strenuous joggers had a mortality rate not statistically different from that of the sedentary group.

■ COMMENTARY

There seems to be little question that exercise improves health, decreases mortality, and contributes to longevity. With respect to running, a study of 55,000 adults between ages 18 and 100 years of age who were followed for a mean of 15 years reported that runners, as compared to non-runners, had 30% and 45% lower risks of all-cause and cardiovascular mortality, respectively, with a mean improvement in life expectancy of 3 years.[13]

However, the Schnohr study revealed that although joggers as a group appeared to live longer than sedentary non-joggers, light and moderate joggers have lower mortality rates than sedentary non-joggers, whereas strenuous joggers have a mortality rate that is not statistically different from that of the sedentary group. The U-shaped association suggests the existence of an upper limit of jogging exercise that is optimal for health benefits if the goal of jogging exercise is to decrease the risk of death and improve life expectancy.

Going for a leisurely jog a few times per week at a moderate pace appears to be good strategy; however, it must be recognized that higher doses of running are not only unnecessary but may actually diminish some of the longevity benefits conferred by lower doses of running. In the Schnohr study, the dose of running that was most favorable for reducing mortality was jogging 1-2.4 hours per week performed on no more than three running days per week at a slower average pace. This goal appears to be quite practical, achievable, and sustainable for most men and women.

In summary, in a large random sample of men and women, joggers appear to live longer than sedentary non-joggers.[12] Moderate joggers had a lower mortality rate than sedentary non-joggers, whereas strenuous joggers had a mortality rate that was not statistically different from that of the sedentary group. The U-shaped association suggests the existence of an upper limit of benefit for exercise dosing that is optimal for health benefits. The results of the Schnohr study suggested that higher doses of running are not only unnecessary but may actually diminish some of the remarkable longevity benefits conferred by lower doses of running activities. It should be recognized though, that the Schnohr study was an observational and not a randomized controlled study and, hopefully, these results will be confirmed by a full-scale randomized, controlled study at some time in the future. ■

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ABSTRACT & COMMENTARY

Are Atrial Premature Complexes Benign?

By Michael H. Crawford, MD

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Dr. Crawford reports no financial relationships relevant to this field of study. This article originally appeared in the April 2015 issue of *Clinical Cardiology Alert*.

SYNOPSIS: The authors concluded that in a general population free of AF or cardiovascular disease, the presence of APCs on a routine ECG is associated with AF and cardiovascular death.

SOURCE: Murakoshi N, Xu D, et al. Prognostic impact of supraventricular premature complexes in community-based health checkups: The Ibaraki Prefectural Health Study. *Eur Heart J* 2015;36:170-178.

Atrial premature complexes (APCs) are commonly observed on routine ECGs and believed to be harbingers of atrial fibrillation, especially in patients with cardiovascular disease. However, little is known about the long-term prognosis of APCs in the general population. Thus, these investigators from Japan analyzed the database of a large community-based cohort from 1993 to 2008 to determine the risks of APCs seen on the subjects' baseline ECGs. There were 63,197 subjects without heart disease or atrial fibrillation (AF) who were followed for at least 1 year (20,492 men and 42,705 women, mean age 58 years at baseline). The primary endpoint was mortality and the secondary endpoint was AF. The mean follow-up was 14 years, but if censured by AF occurrence on the yearly follow-up exam, it was 6 years. In addition to analyzing the raw data, the data were adjusted for age and other potential confounders such as blood pressure, body mass index, alcohol use, and other ECG findings. Also, a propensity-matched analysis was done matching subjects with APCs to those without.

Results: APCs were observed in 6%, and these subjects were more likely to be older and have other risk factors for AF and mortality. APCs were significantly associated with death from stroke, cardiovascular death, and all-cause mortality in women, but only cardiovascular death in men. AF occurred in 1 per 1000 person years, and APCs were a significant predictor of AF (hazard ratio [HR], 4.87 men and 3.87 women). In the propensity score-matched subjects, APCs were significantly associated with AF and cardiovascular death in all subjects and stroke death in women, but not all-cause mortality. The authors concluded that in a general population

free of AF or cardiovascular disease, the presence of APCs on a routine ECG is associated with AF and cardiovascular death.

■ COMMENTARY

This study affirms what has been seen in smaller studies of higher-risk patients, that APCs predict future AF. Why APCs would predict cardiovascular death in a general population is not clear from this study. It could be simply that by being associated with AF, you are more likely to have a stroke or develop heart failure. On the other hand, APCs may be markers of underlying cardiovascular disease. This makes sense since in the baseline data, APC subjects were older and had more risk factors for cardiovascular disease. That APCs are strong predictors of AF is not surprising. Pathophysiology studies show that APCs originating in the pulmonary vein orifices can trigger AF. Also, when the number of APCs per ECG was evaluated, more APCs increased the risk of AF. This is remarkable given that we are talking about a routine ECG, approximately 15 seconds of monitoring. Since APCs are frequently seen on ambulatory ECG monitoring done for a variety of reasons, one wonders if there is some threshold for APCs 24 hours above which the risk of AF and cardiovascular disease increases significantly in a general population.

In addition to the limitations of an ECG as a monitoring device for APCs, it was also the way AF was confirmed. Thus, asymptomatic intermittent AF was unlikely to be detected. Also, the subjects were not extensively evaluated for cardiovascular disease on the yearly exams, so some subclinical disease may have been present and unaccounted for in the propensity analysis. In addition, there were twice as many women as men in the study. The authors don't offer an explanation for this, but since subjects with known cardiovascular disease were excluded in this older middle-aged population, many men may have been excluded. The main clinical message of this study is that patients with APCs on a routine ECG should undergo screening for heart disease and asymptomatic intermitten AF. ■

ABSTRACT & COMMENTARY

Angiotensin Receptor Blockade, Renal Function, and Outcomes in Chronic Heart Failure

By Van Selby, MD

Assistant Professor of Medicine, UCSF Cardiology Division, Advanced Heart Failure Section

Dr. Selby reports no financial relationships relevant to this field of study. This article appeared in the April 2015 issue of *Clinical Cardiology Alert*.

SYNOPSIS: The authors concluded that compared with 50 mg losartan, 150 mg losartan is associated with an increased risk of early WRF, but this appears to be a benign event.

SOURCE: Kiernan MS, et al. Early and late effects of high- versus low-dose angiotensin receptor blockade on renal function and outcomes in patients with chronic heart failure. *JACC Heart Failure* 2015;3:214-223.

Renin-angiotensin-aldosterone (RAAS) blockade is an important component of guideline-recommended therapy for heart failure with reduced ejection fraction (HFrEF). ACE inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) often cause a reduction in the glomerular filtration rate (GFR). The relationship between ACEI or ARB dose and changes in renal function and the long-term implications of these changes is not well-described. To address this issue, Kiernan and colleagues performed a secondary analysis of the Heart Failure Endpoint evaluation of Angiotensin II Antagonist Losartan (HEAAL) study. HEAAL randomized 3834 patients with HFrEF to either 150 mg or 50 mg losartan daily. Patients with serum creatinine > 2.49 mg/dL, potassium > 5.7 mmol/L, or renal artery stenosis were excluded.

Compared to the 50 mg dose, patients receiving 150 mg losartan had a greater reduction in GFR over

time (mean difference -3.79 mL/min/1.73 m^2, $P < 0.0001$). The difference was driven by early worsening renal function (WRF), defined as an increase in creatinine > 0.3 mg/dL. After the first 4 months of therapy, there was no significant difference in GFR between the two doses ($P = 0.016$). WRF in the first 4 months was not associated with an increased risk of death or hospitalization for heart failure (HF) (HR [hazard ratio], 1.09, $P = 0.20$). Overall, losartan 150 mg was associated with reduced risk of death or hospitalization for HF (HR, 0.85, $P < 0.0001$). Among patients with chronic kidney disease (CKD) at baseline, there was no significant difference in change in GFR between the two doses. The authors concluded that compared with 50 mg losartan, 150 mg losartan is associated with an increased risk of early WRF, but this appears to be a benign event. Losartan 150 mg daily retains its net clinical benefit and is associated with reduced risk of death or hospitalization for HF in patients with HFrEF.

■ COMMENTARY

It is well known that initiation of ACEI or ARBs can cause a rise in serum creatinine, and WRF is a common reason for ACEI/ARB discontinuation or dose reduction. Although WRF, in general, has been associated with adverse outcomes, the clinical significance of this early WRF, following ACE or ARB initiation, is less clear. This is among the first studies to examine the dose effect of ACEI or ARB therapy on renal function in patients with HFrEF, as well as the association with long-term outcomes.

The acute rise in serum creatinine following ARB initiation is likely related to alterations in hemodynamics, related to the role angiotensin II plays in regulating renal blood flow. Longer-term declines in renal function, on the other hand, generally reflect disease progression, and not surprisingly are associated with worse outcomes. Thus, when patients with HFrEF develop WRF, it is crucial the clinician carefully evaluate the cause of the decline before adjusting the patient's medication. Current ACC/AHA guidelines recommend an angiotensin receptor blocker for patients with HFrEF who cannot tolerate ACE inhibitors. For losartan, the target dose is 150 mg daily, and given the clear long-term benefit associated with this higher dose, it is important to consider whether dose modification or discontinuation is truly necessary when patients develop WRF.

The findings among patients with baseline CKD are important to note. In the HEAAL study, patients with baseline mild-to-moderate CKD saw smaller average changes in GFR following ARB initiation compared to patients without baseline CKD. Prior studies have shown that CKD patients are less likely to receive target doses of RAAS inhibitors compared to patients with HFrEF and normal renal function, likely related to concern for WRF. These findings support the same target dose of 150 mg in patients with mild-to-moderate CKD (remember that patients with serum creatinine > 2.49 mg/dL were excluded from HEAAL). Of course it is critical that patients be monitored closely for worsening renal failure or hyperkalemia after ACEI or ARB initiation.

This was a retrospective, secondary analysis with important limitations. Study investigators were not blind to changes in renal function, and it is possible that treatments were modified in response to changes in serum creatinine or potassium. The study also was not powered to detect differences in clinical events according to presence of WRF. The authors could not determine what was considered a "safe" increase in creatinine following ARB initiation, and what degree of early WRF should prompt drug discontinuation.

Despite these limitations, the findings suggest the early decline in GFR associated with initiation of high-dose losartan does not increase adverse outcomes, and the net clinical benefit favors targeting the 150 mg dose in patients with HFrEF. It is important to consider the cause of WRF in all HF patients, and strive to maintain the guideline-recommended dose of ACEI or ARBs whenever possible, rather than discontinuing or reducing the dose as an automatic reaction to a rise in serum creatinine. ■

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Clinical Briefs

By Louis Kuritzky, MD

Reducing Drug-induced Xerostomia with Sorbet

SOURCE: Crogan NL. *Annals Long-Term Care* 2015;23:17-21.

Xerostomia, or dry mouth, is common in senior citizens, partially because of disorders that are directly associated with xerostomia (e.g., Sjogren syndrome, HIV, hepatitis C, diabetes) and, additionally, because numerous pharmacologic treatments seniors receive produce "drying" effects: anticholinergics (e.g., antimuscarinic OAB drugs, tricyclic antidepressants), sympathomimetics (e.g., milnacipran, atomoxetine), or diuretics. Although for some geriatric patients, xerostomia is merely an irksome symptom, for others it leads to impaired nutrition as well. Pharmacologic treatments (e.g., cholinergic agonists), while having some degree of efficacy, have their own adverse-effect profile. Is there a simpler, kinder way to address the problem?

As part of a quality improvment program, Crogan performed a study in cognitively intact nursing home residents (n = 22) who scored positively on a xerostomia index and were consuming medications known to induce xerostomia. They directly measured the food intake and wasted food that had been provided in the facility dining room from these subjects, comparing results from the 7 days prior to intervention to results after 6 weeks of intervention.

The active intervention was provision of 2 ounces of sugar-free lemon-lime sorbet prior to lunch and dinner for 6 weeks. Measured outcomes included fluid intake during meals — with decreased fluid intake suggesting less dry mouth — calorie intake, and body weight.

Pre-meal sorbet was associated with a (mean) 22% increase in food intake, and 81% of participants either maintained or gained weight. Provision of pre-meal sorbet may improve nutrition among seniors treated with medications that produce xerostomia. ■

The Ongoing Search for Cognitive Impairment Biomarkers

SOURCE: Wang T, et al. *J Clin Psychiatry* 2015; 76:135-141.

Messenger RNA (mRNA) markers are used for identification of a variety of pathologic processes, most recently malignant melanoma. Although the labels "BACE1" and "miRAN107" likely hold little meaning for most clinicians, investigators have found that diminished levels of these particular biomarkers may help identify persons with cognitive impairment, specifically in Alzheimer's disease.

The low levels of such biomarkers in cerebrospinal fluid (CSF) as a correlate of dementia — as well as identification of their diminution being associated with tissue deposition of amyloid — strengthens the case, considering their etiologic role in dementing disorders. Because of the relative inaccessibility of CSF, however, it is fortunate that biomarker plasma levels of BACE1 and miRAN107 reflect CSF levels.

In a study of elderly patients with Alzheimer's disease (n = 97), mild cognitive impairment (n = 116), or normal cognitive function (n = 81), investigators identified a nearly four-fold lower plasma level of BACE1 and miRAN107 in mild cognitive impairment (as well as Alzheimer's disease) compared to healthy controls. The authors are hopeful that these biomarkers may ultimately help us identify mild cognitive impairment early, as well as discriminate Alzheimer's disease from other forms of dementia. ■

Bipolar Disorder is Associated with New-onset CVD

SOURCE: Goldstein BI, et al. *J Clin Psychiatry* 2015; 76:163-169.

Although perhaps not widely recognized, bipolar disorder (BPD) is associated with an excessive risk of cardiovascular disease (CVD). Not only is CVD more prevalent, but it occurs as much as a decade earlier than comparators without BPD. Some of this risk is attributed to utilization of pharmacotherapies that are known to be diabetogenic (e.g., mood stabilizers), but this is insufficient to fully explain the observed increased CVD risk.

To better clarify risk of CVD in BPD patients, an analysis was performed of persons in the NESARC epidemiologic survey (National Epidemiologic Survey on Alcohol and Related Conditions). The population studied included persons with BPD (n = 1439), major depressive disorder (n = 4396), and controls (n = 26,266). The incidence of CVD was compared in two survey "waves," the first performed in 2001-2002, and the next performed in 2004-2005.

Even within this very short window of observation, the odds ratio for new onset CVD amongst persons with BPD was over 2.5 compared to controls. In contrast, persons with Major Depressive Disorder did not have an incidence of CVD that differed meaningfully from controls.

These concerning data show a nearly 3-fold increase risk of new onset CVD in persons with BPD, despite being controlled for commonly recognized risk factors (e.g., age, smoking, HTN, obesity). Equally alarming is the observation that the mean age of CVD onset in BPD patients was 14-17 years earlier than controls! Clinicians should heighten their vigilance for addressing CVD risk factors amongst persons with BPD. ■

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CME QUESTIONS

1. Joggers who perform light or moderate jogging programs:

a. have a lower mortality rate than sedentary non-joggers.

b. have the same mortality rate as do sedentary non-joggers.

c. have the same mortality rate as do strenuous joggers.

d. have a higher mortality rate than do sedentary non-joggers.

2. In a general population, APCs are associated with which of the following?

A. Atrial fibrillation

B. Stroke death

C. Cardiovascular death

D. A & C

3. Which of the following is correct concerning worsening renal function after initializing high-dose angiotensin receptor blocker therapy for heart failure?

A. It usually self corrects within 4 months

B. It predicts early mortality

C. It predicts early rehospitalization

D. It usually worsens over time

CME OBJECTIVES

Upon completion of this educational activity, participants should be able to:

- describe new findings in the differential diagnosis and treatment of various diseases;
- describe the advantages, disadvantages and controversies surrounding the latest advances in the diagnosis and treatment of disease;
- identify cost-effective treatment regimens;
- explain the advantages and disadvantages of new disease screening procedures.

[IN FUTURE ISSUES]

Steroids for Severe Communityacquired Pneumonia

Internal Medicine [ALERT]

ECG Review

By Ken Grauer, MD

Professor Emeritus in Family Medicine, College of Medicine, University of Florida

Dr. Grauer is the sole proprietor of KG-EKG Press, and publisher of an ECG pocket brain book.

A Regular SVT with Marked ST Depression

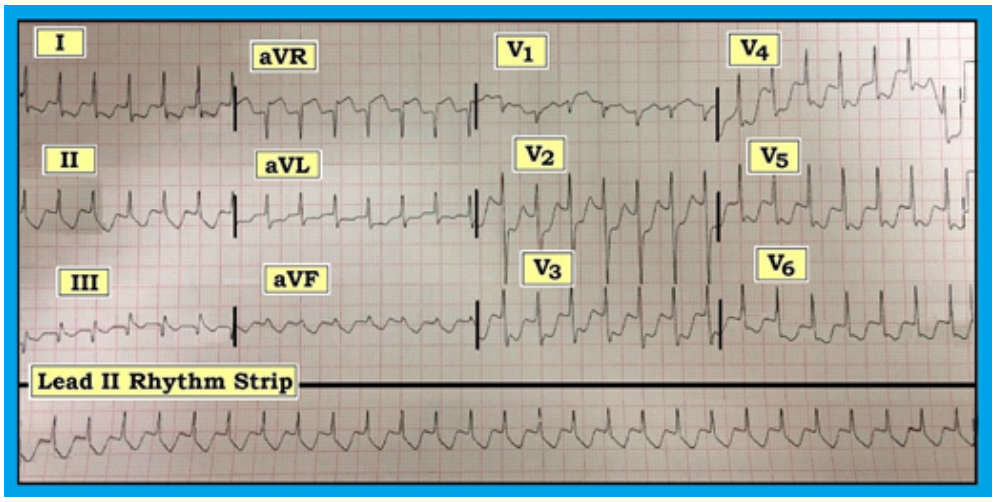

Figure: 12-lead ECG obtained from a 60-year old man with chest pain.

The patient is a previously healthy 60-year-old man who presented with palpitations and new-onset chest pain. He was on no medications and had no prior history of heart disease. His blood pressure was 70 systolic at the time the 12-lead ECG in the *Figure* was obtained.

- What is the rhythm in the *Figure*?
- Why is there so much ST depression?
- What are your diagnostic considerations? Clinically, what would you do?

Interpretation: The rhythm is a regular SVT (supraventricular tachycardia) at a rate of 180/minute. The QRS complex is narrow in all 12 leads. Normal *P* waves are not seen.

- The principal entities to consider in the differential diagnosis of a regular SVT rhythm when normal sinus *P* waves are not seen are: 1.) Sinus tachycardia (with *P* waves hidden within the ST-T wave); 2.) Atrial flutter; and 3.) PSVT (paraoxysmal supraventricular tachycardia — also known as AVNRT = atrioventricular nodal reentrant tachycardia). Although there are other possibilities (i.e., automatic atrial or junctional tachycardias), the vast majority of regular SVT rhythms will turn out to be one of the three entities listed above.
- The rate of the SVT in this case (= 180/minute) makes it highly likely that the rhythm is PSVT. This is because sinus tachycardia is rarely this fast, and atrial flutter with 2:1 AV conduction almost always presents with a ventricular rate range between 140-160/minute.
- Perhaps the most striking finding on this tracing, however, is the marked and diffuse ST depression seen in virtually all leads (with exception of ST elevation in lead aVR).

Impression: As discussed above, the most likely etiology of this regular SVT without sinus *P* waves is PSVT given the rate of 180/minute. The marked and diffuse ST depression (with ST elevation in lead aVR) should prompt consideration of severe coronary disease in this 60-year-old man with new-onset chest pain. Given his low blood pressure, immediate cardioversion (rather than trial of medication) would seem warranted.

- Follow-up was available on this clinical case. The patient was not cardioverted. Instead, Adenosine was given with successful conversion to sinus rhythm. Cardiac catheterization was performed and revealed insignificant nonobstructive disease. An important lesson learned from this case is that even marked and worrisome diffuse ST depression as seen here is not always the result of severe coronary disease when the patient presents with sustained tachycardia. ■

Note: Further discussion of this tracing is available on an ECG video found at this site: http://tinyurl.com/nex6xcn