INTERNAL MEDICINE ALERT

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INSIDE

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Treatment of Chronic Fatigue Syndrome

ABSTRACT & COMMENTARY

By Joseph E. Safdieh, MD

Assistant Professor of Neurology, Weill Medical College, Cornell University

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

This article originally appeared in the June issue of Neurology Alert. *At that time it was peer reviewed by M. Flint Beal, MD, Anne Parrish Titzel Professor, Department of Neurology and Neuroscience, Weill Cornell Medical Center, New York, NY. Dr. Beal reports no financial relationship to this field of study.*

Synopsis: *Chronic fatigue syndrome, as defined by clinical symptoms, may improve with a combination of behavior therapy and graded exercise.*

Source: White PD, et al. Comparison of adaptive pacing therapy, cognitive behavior therapy, graded exercise therapy, and specialist medical care for chronic fatigue syndrome (PACE): A randomized trial. *Lancet* 2011;377:823-836.

Chronic fatigue syndrome (CFS) is a syndrome defined by the presence of disabling fatigue in the absence of an alternative diagnosis. The prevalence of CFS is between 0.2% and 2.6% worldwide and the prognosis is poor if left untreated. Because CFS is a disorder with no scientifically established etiology, treatment often is attempted by treating the fatigue and providing supportive, symptomatic care. Prior small studies have demonstrated that cognitive behavioral therapy (CBT) and graded exercise therapy (GET) may be effective for treating CFS. However, no large trials were performed and CFS advocacy groups have reported that CBT and GET may be harmful. These groups have recommended adaptive pacing therapy (APT). The authors performed a randomized trial to compare the effectiveness of CBT, GET, and APT added to specialist medical care (SMC), and SMC alone.

SMC was provided by physicians with expertise in CFS. Subjects all received standard advice, such as instructions on avoiding extremes

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of activity and getting adequate rest. SMC physicians also were allowed to use pharmacotherapy to treat individual symptoms. CBT was done on the basis of the fear avoidance theory of CFS, which regards CFS as being reversible and associated with abnormal cognitive and behavioral responses. GET was performed based on the deconditioning and exercise intolerance theories of CFS and was accomplished by gradually escalating mild aerobic activity. APT was based on the theory that CFS is organic and irreversible and for whatever reason, results in a finite amount of available energy. APT focused on achieving optimum adaption to CFS, teaching subjects to maintain activity diaries and to avoid "overdoing it" by not undertaking activities that demand more than 70% of subjects' perceived energy limit.

Of the 3158 patients screened for eligibility, only 641 were able to be randomly allotted to treatment groups. One hundred sixty patients received APT+SMC, 161 received CBT+SMC, 160 received GET+SMC, and 160 received SMC alone. Due to the nature of the study design, subjects were not blinded to treatment type received. Baseline characteristics of the groups were generally similar, with overall mean age of 38, duration of illness of 32 months, 77% female and 92% white.

Compared with SMC alone, mean fatigue scores at 52 weeks were lower in the CBT and GET groups, but not in the APT group. Compared with SMC alone, mean physical function scores were higher in the CBT and GET groups, but not in the APT group. Serious adverse events were very rare (< 2%) in all groups. The authors concluded that CBT and GET can be safely added to SMC to moderately improve outcomes in CFS, but APT is not an effective addition.

■ COMMENTARY

CFS is a diagnosis that is defined purely on clinical symptoms. Many neurologists are quite dubious of whether CFS is a "real disease" or a manifestation of depression or another psychiatric or psychological process. Of course, fatigue can be caused by many symptoms, and before labeling a patient with CFS, primary medical and neurological causes should be excluded with appropriate testing. Whatever the cause of CSF is eventually determined to be, there are many patients who clearly experience disabling chronic fatigue and often end up in the care of a neurologist. Once alternative diagnoses, including sleep disorders, are excluded, my approach is not to focus on the "why" but on the "what to do." Pharmacotherapy is certainly an option. Stimulants, antidepressants, and amantadine all play a role. In this paper, we learn that recommending a submaximal escalating exercise program and CBT are useful additions to standard therapy. These therapies are safe and moderately effective. Patients with CFS should be encouraged to participate in basic aerobic exercise regimens and to consider CBT. Research into the etiology of this disorder is ongoing, but there are effective strategies that can be recommended even without understanding the underlying cause. ■

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Diagnosis of Thoracic Aorta Dissection

ABSTRACT & COMMENTARY

By Michael H. Crawford, MD

Professor of Medicine, University of California, San Francisco; Lucie Stern Chair in Cardiology, Chief of Clinical Cardiology, University of California, San Francisco Medical Center

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

This article originally appeared in the July issue of Clinical Cardiology Alert. *At that time it was peer reviewed by Ethan Weiss, MD, Associate Professor of Medicine, Division of Cardiology, University of California, San Francisco, CA. Dr. Weiss is an advisory board member for Bionovo.*

Synopsis: *Using an algorithm based on score and chest x-ray when appropriate, the overall sensitivity for the detection of aortic dissection was 96%.*

Source: Rogers AM, et al. Sensitivity of the aortic dissection detection risk score, a novel guideline-based tool for identification of acute aortic dissection at initial presentation. *Circulation* 2011;123:2213-2218.

THORACIC AORTIC DISSECTION IS NOTORIOUSLY DIFFICULT TO diagnose. Since the presenting symptoms are protean, it is not feasible to image everyone with symptoms that could be due to dissection. Thus, a risk assessment tool was devised by an expert committee, but it has never been validated clinically. These investigators applied the risk score to the International Registry of Acute Aortic Dissection database to test its utility for diagnosing aortic dissection. More than 2500 patients in the registry were categorized by 12 clinical markers: five predisposing conditions, three pain features, and four exam features. Those with no risk markers were scored 0; those with markers in at least one of the three categories were scored 1; and markers in two or three categories were scored 2 or 3, respectively. Score 0 was considered low risk; score 1 was intermediate risk; and 2 or 3 was high risk. A score of 0 was found in 4%; 1 in 37%; and 2 or 3 in 59%. Among the 108 low-risk score 0 patients, 72 had chest x-rays and 49% had a widened mediastinum. Using an algorithm based upon score and chest x-ray when appropriate, the overall sensitivity for the detection of aortic dissection was 96%. The most common of the 12 individual risk markers were abrupt onset of pain (79%); severe pain (73%); ripping or tearing pain (22%); new murmur of aortic regurgitation with pain (24%); and a pulse deficit or upper extremity blood pressure differences (20%). The authors concluded that this clinical risk marker score was highly sensitive for detecting aortic dissection.

■ COMMENTARY

This is an interesting study because clinical factors believed to be helpful in the diagnosis of aortic dissection were collated into a proposed risk score by a group of experts without any clinical testing. Of course this happens all the time and we often never know exactly how useful these scores will be. In this case, a large database was used to test the scores utility in retrospect. Although it did well (sensitivity 96%), a prospective study would give us more confidence in its utility. However, it is difficult to study a low incidence event like aortic dissection prospectively.

Inspection of the 12 individual markers shows that some were much more useful than others: Abrupt onset of pain and severe pain occurred in more than 70%. A new murmur of aortic regurgitation in conjunction with pain, ripping or tearing pain, and a pulse deficit or systolic blood pressure difference between limbs occurred in 20%-24%. These three features of the pain history and two physical examination findings seem more specific for aortic dissection than other less common findings such as known thoracic aortic aneurysm, known aortic valve disease, focal neurologic deficit, and hypotension or shock, which occurred in 11%-16%. The other three markers (Marfan Syndrome, family history of aortic disease, and recent aortic manipulation) occurred less than 5% of the time.

Unfortunately, this study cannot assess specificity because all the patients had aortic dissection. It is likely that specificity — and hence positive-predictive valve — will be lower than the sensitivity. This puts the aortic dissection score in the category of other highly sensitive tests with high negative predictive values such as d-dimer, troponin, and BNP. How much use of such a score will cut down on excessive imaging in the emergency department remains to be seen. ■

Pets in the Bedroom — Move Over Rover!

ABSTRACT & COMMENTARY

By Mary-Louise Scully, MD

Director, Travel and Tropical Medicine Center, Sansum Clinic, Santa Barbara, CA

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

This article originally appeared in the June 2011 issue of Travel Medicine Advisor. *At that time it was peer reviewed by Lin Chen, MD, Assistant Clinical Professor, Harvard Medical School; Director, Travel Medicine Center, Mt. Auburn Hospital, Cambridge, MA. Dr. Chen reports no financial relationship to this field of study.*

Synopsis: *The increasingly close and almost intimate relationships with our pets can lead to increased numbers of cases and the emergence of zoonotic diseases, including human plague* (Yersinia pestis).

Source: Chomel BB, Sun B. Zoonoses in the bedroom. *Emer Infect Dis* 2011;17:167-172.

THE NUMBERS OF HOUSEHOLDS WITH PETS ARE INCREASING IN many countries across the world. In addition, data obtained from media sources note a trend in the percentage of these pets sleeping in, or on, the owner's bed. To address whether this behavior is associated with the acquisition of zoonotic disease(s), the authors searched PubMed for peer-reviewed publications that demonstrated disease likely to have been acquired by sleeping with, sharing a bed with, kissing, or being licked by pets.

The results encompassed bacterial, parasitic, and viral associated zoonoses. The bacterial zoonoses included those with known animal associations such as *Yersinia pestis* (plague), *Bartonella* species (cat-scratch disease), *Pasturella* species, and *Capnocytophaga camimorsus*. In the case of a plague outbreak, one patient had the onset of his illness the morning after noting bites from his flea-infested cat who had shared his bed.[1] Another case-control study of plague survivors found 44% of survivors vs 10% of controls reported sleeping in the same bed with a pet

dog.[2] Although *Bartonella* infections are often associated with a scratch of a cat that harbors *Bartonella henselae*-infected fleas, a 9-year-old girl from Taiwan with multi-organ (hepatic, splenic, and renal) disease from *Bartonella*, became ill after sleeping with her cat at night.[3] In a study of *Pasturella multicida* meningitis, 27 (87%) of 31 infants exposed to animals had been exposed in various ways to oropharyngeal animal secretions through either licking or sniffing.[4] In addition, *Pasturella* wound infections have been reported when the animals had been observed licking the wounds prior to onset of illness.[5]

C. camimorsus is a gram-negative bacillus that is known for its presentation of a purpura fulminans-like sepsis, especially in asplenic, alcoholic, or steroid-dependant patients. Several cases in the literature exist for which the portal of entry was felt to be a direct result of a pet licking an ulcer or abraded skin of the patient. For example, a patient with chronic ulcerous eczema of the legs whose dog used to lick his legs, died of septic shock and renal failure caused by *C. camimorsus*.[6]

■ COMMENTARY

These are just some of the highlighted cases discussed in this article. I recently saw a patient with a post-surgical septic olecranon bursitis caused by *Staphylococcus intermedius*. The patient admitted his dog may have licked the wound or provided saliva exposure during their playful nightly wrestling on the floor. The most recent reference I found to include data on this evolving topic is from the Center for Food Security & Public Health from Iowa State University from January 2011 — an MRSA article with more than 180 references![7] We are very likely seeing the tip of the iceberg on this emerging issue.

In May 2011, shortly after the Chomel article was published, the CDC published two cases of human plague in *MMWR* from Oregon in 2010. These were the first cases reported from Oregon since 1995 and they were the only plague cases reported in the United States in 2010. The patients, ages 17 and 42, lived in the same household with a dog that was later found to be seropositive for *Y. pestis* by passive hemagglutination-inhibition assay. Although both patients had clinical illness compatible with human plague, including bilateral inguinal buboes, fever, and hypotension, plague was not suspected initially. One patient's blood culture specimen was later identified as positive, and the other patient had a positive serology. One of the patients admitted sleeping in the same bed with the dog during the 2 weeks prior to the onset of illness. Fortunately, both patients recovered after empiric therapy with doxycycline.

Pets are known to provide company and assuage the loneliness of countless human beings worldwide and as such, they are often considered "part of the family." Although transmission of zoonotic infections from pets is rare, it would seem prudent to ensure our pets are properly de-wormed and free of fleas, and defer from sharing the same bed with them to prevent serious and potentially fatal infections. ■

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Brief Report

Are You "Coated" with Bacteria?

By Carol A. Kemper, MD, FACP

Clinical Associate Professor of Medicine, Stanford University, Division of Infectious Diseases

Dr. Kemper does research for Abbott Laboratories and Merck.

This article originally appeared in the July issue of Infectious Disease Alert. *At that time it was peer reviewed by Timothy Jenkins, MD, Assistant Professor of Medicine, University of Colorado, Denver Health Medical Center. Dr. Jenkins reports no financial relationship to this field of study.*

Source: Burden M, et al. Newly cleaned physician uniforms and infrequently washed white coats have similar rates of bacterial contamination after an 8-hour workday. *J Hosp Med* 2011;6:177-182.

THE NATIONAL HEALTH SERVICE IN BRITAIN IN 2007 ELECTED to ban traditional white coats and other long-sleeved garments for physicians in the workplace (including long-sleeved blouses and shirts). Subsequently, Scotland adopted similar policies. This decision was based on limited

data suggesting that the cuffs and lower pockets of long-sleeved garments are more heavily colonized with bacteria than shorter garments.

These authors have succeeded in debunking this notion. One hundred residents and hospitalists working on the internal medicine service in hospital (in Colorado) were randomly assigned either to start the day fresh with a newly laundered standard short-sleeved uniform or to wear their own (presumably not recently laundered) white coats. The latter group was not informed of their randomization assignment till the day they showed up for work, giving them no chance to switch to an unused coat. Cultures were obtained throughout the workday, beginning before the coat was put on to 2.5, 5, and 8 hours later. Cultures were obtained from the breast pocket, sleeve cuff (of either the short uniform sleeve or the long sleeve), and the skin of the volar surface of the wrist area. Cultures were incubated for 18-22 hours, and colony counts (up to 200) were determined. In addition, colonies of *Staphylococcus* were tested for coagulase production and methicillin resistance.

At the end of the day, no differences were found between the colony counts cultured from the clean uniforms and that of the white coats (respectively, mean colony counts, 142 [range 83-213] vs 104 [range 80-127]; $P = 0.61$). No significant differences were found between the colony counts cultured from the sleeve cuffs of the short-sleeved uniforms vs. the longer sleeve cuffs of the white coats (mean colony counts, 37 vs 58), or between the pockets of either garment (mean colony counts, 75 vs 46). Colony counts were generally greater for the sleeve cuffs compared with the breast pocket of the long-sleeved coat (although the difference was small), whereas no difference in colony counts was observed between the short sleeve cuffs and breast pockets of the uniforms. No differences were found between the degree of bacterial colonization of the wrists for either those wearing a white coat or a short-sleeved uniform.

In addition, colonization with methicillin-resistant *Staphylococcus aureus* was similar for those wearing their own long white coats compared with the group assigned to wear clean uniforms (16% vs 20%).

Serial cultures obtained throughout the workday demonstrated that a freshly laundered uniform starts out nearly sterile. But within 2.5-3 hours, the uniform is colonized with 50% of the bacterial colonies found at 8 hours of wear.

■ COMMENTARY

These data demonstrate that, when worn by a resident or hospitalist on the hospital wards, bacterial colonization of a freshly laundered garment is remarkably fast, and within 1 workday is similar to that of an unwashed days-old long-sleeved white coat. There is no evidence that long sleeves vs. short sleeves is less likely to result in bacterial colonization of either the garment or the wearer's wrists. ■

Pharmacology Update

Fidaxomicin Tablets (Dificid™)

By William T. Elliott, MD, FACP, and James Chan, PharmD, PhD

Dr. Elliott is Chair, Formulary Committee, Northern California Kaiser Permanente; and Assistant Professor of Medicine, University of California, San Francisco. Dr. Chan is Pharmacy Quality and Outcomes Manager, Kaiser Permanente, Oakland, CA.

Drs. Elliott and Chan report no financial relationship to this field of study.

A NEW ORALLY ADMINISTERED MACROCYLIC ANTIBIOTIC HAS been approved by the FDA for the treatment of *Clostridium difficile* infection. Fidaxomicin is a fermentation product of *Dactylosporangium aurantiacum* and is marketed by Optimer Pharmaceuticals, Inc., as Dificid.

Indications

Fidaxomicin is indicated for the treatment of *C. difficile*-associated diarrhea (CDAD) in adults (18 years of age and older).[1]

Dosage

The recommended dose is 200 mg taken orally twice daily for 10 days.[1] The tablets may be taken with or without food.

Fidaxomicin is available as 200 mg tablets.

Potential Advantages

Fidaxomicin showed similar efficacy in treating *C. difficile* infection compared to vancomycin but with a significantly lower rate of recurrence.[2] Fidaxomicin also is more likely to preserve normal fecal flora than vancomyin.[3,4] It has minimal systemic absorption.

Potential Disadvantages

Fidaxomicin has greater systemic exposure (2-4 fold) in elderly patients (65 years and older).[1] Fidaxomicin does not provide any advantage in recurrence with the hypervirulent BI/NAP1/027 strains of *C. difficile*.[2] A single course of fidaxomicin is expected to cost approximately $2800.

Comments

Fidaxomicin is a minimally absorbed, macrocyclic antibacterial agent with demonstrated in vitro bactericidal and post-antibiotic activity against *C. difficile*. It generally lacks activity against gram-negative anaerobes and facultative aerobes and is less likely to affect the composition of the

intestinal flora.[3,5] The approval of fidaxomicin was based on two randomized, double-blinded, non-inferiority studies compared to vancomycin.[1,2] Adult subjects with CDAD were randomized to fidaxomicin (200 mg twice daily for 10 days) or vancomycin (125 mg four times a day for 10 days). CDAD was defined by > 3 unformed bowel movements or > 200 mL of unformed stool in the 24 hours before randomization and presence of either *C. difficile* toxin A or B in the stool within 48 hours of randomization. Enrolled subjects were required to have had only one prior CDAD episode in the past 3 months. Those with life-threatening disease or other complicated signs (e.g., significant dehydration, megacolon) were excluded. The primary efficacy endpoint was clinical response rate at the end of therapy (i.e., resolution of symptoms and no need for further therapy for *C. difficile*). Secondary endpoints included sustained response/recurrence of CDAD within 25 days/4 weeks post-therapy. Trial 1 included 596 subjects and Trial 2 had 509. Clinical responses were identical, 88% for fidaxomicin and 86% and 87% for vancomycin, respectively. However, sustained response was significantly better for fidaxomicin, 70% and 72% compared to 57% for vancomycin due to lower rates of proven or suspected CDAD during the follow-up period. The median time to resolution of diarrhea was shorter with fidaxomicin, 58 hours compared to 78 hours. The advantage of fidaxomicin over vancomycin in recurrence was mainly with non-B1/NAP1/027 strains.[2] There were no major differences in adverse events with gastrointestinal symptoms being most frequently reported including nausea, vomiting, and abdominal pain.

Clinical Implications

CDAD is a serious condition that is the most common antibiotic-associated diarrhea. It is becoming more common and more difficult to treat with a high recurrence rate. Oral vancomycin currently is the only FDA-approved therapy. Fidaxomicin provides an important alternative to vancomycin for the treatment of CDAD, particularly with non BI/NAP1/07 strains. ■

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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.

CME Instructions

To earn credit for this activity, follow these instructions:

1. Read and study the activity, using the provided references for further research.
2. Log on to www.cmecity.com to take a post-test; tests can be taken after each issue or collectively at the end of the semester. First-time users will have to register on the site using the 8-digit subscriber number printed on their mailing label, invoice or renewal notice.
3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.
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5. Once the completed evaluation is received, a credit letter will be e-mailed to you instantly. You will no longer have to wait to receive your credit letter! ■

CME Questions

1. Which of the following modalities has been demonstrated in a randomized trial to be effective in reducing fatigue in patients with chronic fatigue syndrome?

a. Adaptive pacing therapy
b. Biofeedback therapy
c. High intensity cardiac fitness program
d. Psychodynamic therapy
e. Graded exercise therapy

2. The sensitivity of a new clinical score for aortic dissection is:

a. 65%.
b. 75%.
c. 86%.
d. 96%.

3. All of the following organisms are associated with animal pet exposure *except***:**

a. *Capnocytophaga camimorsus*
b. *Yersinia pestis*
c. *Bartonella henselae*
d. *Vibrio vulnificus*
e. *Staphylococcus intermedius*

By Louis Kuritzky, MD, Clinical Assistant Professor, University of Florida, Gainesville

Dr. Kuritzky is an advisor for Endo, Kowa, Pricara, and Takeda.

Fracture Risk Stratification in Diabetics

Source: Schwartz AV, et al. Association of BMD and FRAX score with risk of fracture in older adults with type 2 diabetes. *JAMA* 2011;305:2184-2192.

It has recently been recognized that type 2 diabetes (DM2) increases risk for osteoporotic fracture, even though it has been demonstrated that DM2 is associated with a paradoxical increase in bone mineral density (BMD) compared to age-matched control populations. With a burgeoning prevalence of DM2 in the United States, almost 20% of the at-risk population for osteoporotic fracture has DM2, hence, clarification of risk stratification for this group is highly relevant.

The World Health Organization (WHO) and the U.S. National Osteoporosis Foundation (NOF) suggest that clinicians assess patient risk for osteoporotic fracture by means of the fracture risk algorithm (FRAX) score. FRAX, an online risk assessment tool (available free of charge at http://www.shef.ac.uk/FRAX/), allows input of patient characteristics including gender, ethnicity, body mass index, risk factors for osteoporosis, history of fracture, family history of fracture, and BMD to calculate a 10-year risk of any osteoporotic fracture as well as 10-year risk of hip fracture. Similar to the structure of the ATPIII lipid guidance, intervention is threshold-based: Anyone with a 10-year risk of hip fracture > 3%, or total fracture risk > 20%, should be considered for pharmacotherapeutics intervention.

Gathering data from three prospective observational studies (n = 9449 women, 7346 men), Schwartz et al studied the relationship between FRAX scores, BMD, and subsequent osteoporotic fractures. Of concern, for any given T-score or FRAX score, the rate of osteoporotic fractures was higher in DM2 subjects than controls. DM2 appears to be a risk factor for osteoporotic fracture, above and beyond what is predicted by BMD or FRAX. ■

Amantadine for Dysphagia in the Elderly

Source: Gokula M, et al. Does amantadine help elderly residents with symptomless dysphagia? *Ann Long-Term Care* 2011;19:37-40.

When amantadine (AMTD) was an appropriate first-line treatment for influenza, clinicians gained familiarity with its use. In the last decade, influenza resistance to the adamantanes (i.e., AMTD, rimantadine) has essentially eliminated their utility. The safety profile of AMTD is excellent however, heightening interest in clinical use for other syndromes.

Dysphagia in the elderly can be problematic, potentially leading to feeding difficulties and aspiration pneumonia. Probably the two most common scenarios in which we encounter dysphagia are Parkinson's disease and post-stroke, each of which is associated with reduced levels of dopamine. Since AMTD is a dopamine agonist, there is putative rationale for its potential use in dysphagia.

Gokula et al report their clinical experiences with AMTD in elderly patients with dysphagia. Based on positive responses in two test cases, they performed an uncontrolled case series (n = 12) among dysphagia subjects in a long-term care facility using an AMTD dose of either 50 mg or 100 mg/d orally. By 4 weeks, 11 of the 12 subjects demonstrated better swallowing, decreased cough, and weight gain. Additionally, fewer episodes of aspiration were seen.

Because AMTD is generally well tolerated, inexpensive, and there is little other resource for addressing dysphagia, clinicians may wish to consider a clinical trial. ■

Is Homocysteine a Culprit in Aging Skin?

Source: Namazi MR, Feily A. Homocysteine may accelerate skin aging: A new chapter in the biology of skin senescence? *J Am Acad Derm* 2011;64:1175-1178.

The association of homocysteine (HCST) with atherosclerosis is as strong and consistent as cholesterol, which prompted a flurry of clinical trials in the 1990s and early 2000s attempting to improve cardiovascular outcomes by lowering HCST levels (usually with pharmacologic doses of B vitamins). Unfortunately, HCST modulation did not result in cardiovascular risk reduction, to the point that interventions aimed at HCST have been largely abandoned.

HCST might, however, be a culprit in aging skin. Photoaging is attributed to up-regulation of cutaneous matrix metalloproteinases and down-regulation of collagen synthesis. Homocystinuria, an inborn error of metabolism characterized by marked elevation of HCST, is characterized by thin, transparent skin.

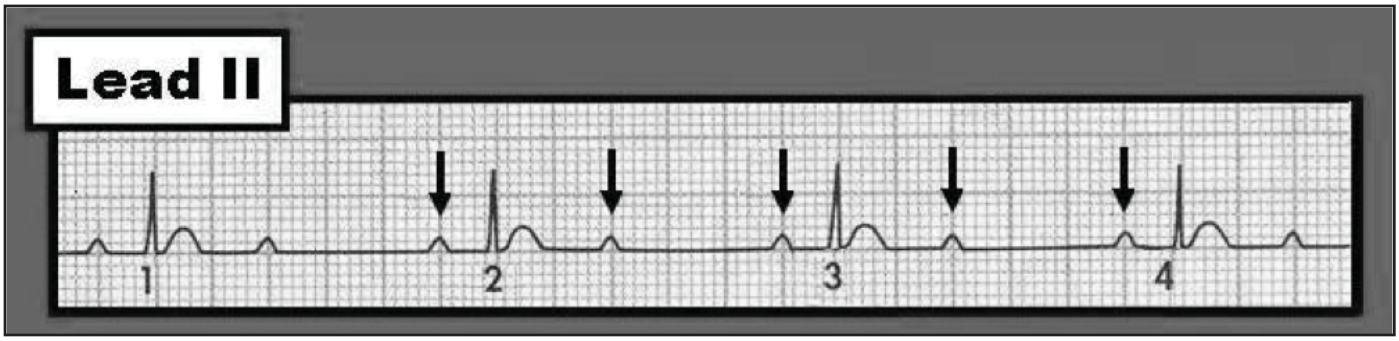
HCST negatively impacts the three primary structural elements of healthy skin: collagen, elastin, and proteoglycans. Not only does elevated HCST increase degradation of these components, it also inhibits their regeneration.

There have not yet been any clinical trials to examine whether HCST reduction favorably impacts skin aging. ■

2nd Degree AV Block, Mobitz Type II?

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.



Scenario: Interpret the rhythm strip shown above. Does it represent 2nd degree AV Block, Mobitz Type II? Clinically — why is it important to distinguish between Mobitz I and Mobitz II 2nd degree AV block?

Interpretation: The rhythm in the Figure is slow but regular. The QRS complex is narrow, and a regular atrial rate is seen at 100/minute (*arrows*). Every other P wave conducts (as evidenced by the fact that a P wave *does* precede each QRS complex with a fixed PR interval!).

Traditionally — the AV blocks are divided into three degrees based on severity of the conduction disturbance:

• **1st degree AV block** — in which *all* atrial impulses are conducted to the ventricles, albeit with delay (so that the PR interval exceeds 0.20 second).

• **2nd degree AV block** — in which *some* (but not all) atrial impulses are conducted to the ventricles.

• **3rd degree (or "complete") AV block** — in which *none* of the atrial impulses are conducted to the ventricles, despite having more than adequate opportunity for conduction to occur.

Second degree AV blocks are further classified into three types:

• **Mobitz I (AV Wenckebach)** — in which the PR interval progressively lengthens until a beat is dropped. This is by far the most common form of 2nd degree AV block. Mobitz I usually occurs at the level of the AV node. As a result, the QRS complex is typically narrow. Mobitz I is generally associated with inferior infarction; it often spontaneously resolves, and typically responds to atropine (which works on the AV node).

• **Mobitz II** — in which there is a *constant* PR interval for *consecutively* conducted beats until one or more beats are dropped. Because Mobitz II typically occurs *low* down in the conduction system — the QRS complex is generally wide. This less common form of 2nd degree AV block is generally associated with anterior infarction; it usually does not respond to atropine — and is important to recognize because pacing will probably be needed.

• **2-to-1 AV Block** — in which one *never* sees two consecutively conducted beats, so that you *cannot* tell if the PR interval is lengthening or not. As a result, it is impossible to know for sure whether this form of 2nd degree AV block represents Mobitz I or Mobitz II. This is precisely the situation seen in the Figure. We suspect this rhythm represents 2nd degree AV block, Mobitz Type I (Wenckebach) because: 1) Mobitz I is so much more common than Mobitz II; and 2) the QRS complex is narrow, as it almost always is with Mobitz I. Finding additional rhythm strips on this patient that clearly showed progressive lengthening of consecutively conducted QRS complexes before dropping a beat would strongly support our suspicion. Clinically the distinction is important because no treatment (other than perhaps atropine) is likely to be needed for Mobitz I (especially given that the ventricular rate in the above example is not overly slow at 50/minute). In contrast, pacing would probably be needed if the rhythm was Mobitz II. ■

In Future Issues:

Impact of Drug Class on Adherence to Antihypertensives