SOUTHERN MEDICAL JOURNAL

Rapid Response

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REFERENCE ARTICLE

ACUPUNCTURE FOR FIBROMYLAGIA: A SYSTEMATIC REVIEW OF RANDOMIZED CLINICAL TRIALS.

Mayhew E, Ernst E. *Rheumatology* 2007; 46:801–804.

Response from

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Is There a Role for Acupuncture in Fibromylagia?

In a recent systematic review of the use of acupuncture for fibromyalgia,[1] the authors conclude that "acupuncture cannot be recommended for fibromyalgia." I take issue with this broad conclusion on several counts. First, they found only five eligible studies and of those, their own table reports positive results in four, including outcomes such as decreases in pain and tender points, improved Fibromyalgia Impact Questionnaire, and other condition-related parameters compared with controls. Secondly, a number of controls actually involved needling "sham" acupuncture points located some distance away from the true meridian points. If one has ever treated a fibromyalgia patient, he or she knows they are hypersensitive to pain ("allodynia"), and it might be suspected that such sham needling was anything but innocuous. This addresses a major issue in acupuncture research in general, that of the control condition, which has more recently been dealt with by retractable, nonpenetrating sham needles.

Another question I would raise about this report is although we have a clinical definition of fibromyalgia, we do not know the mechanism nor the pathophysiology of it. Furthermore, since we have only theoretical understanding of the potential mechanism of acupuncture, what is the meaning of a study that brings these two unknown states together?[2] Are there potentially genomic, metabolic, or psychological subtypes of fibromyalgia patients and might such a stratification differentiate those for whom acupuncture might be more or less effective? For example, a previous review found that some fibromyalgia patients don't tolerate the needling of acupuncture, experiencing it as too painful and thus excluding acupuncture as a potential therapy.[3]

In addition to these problems, the authors dismissed even positive studies because the benefits diminished after a few weeks or months. I found this peculiar. Fibromyalgia is a chronic disease. If a patient with another chronic disease such as diabetes had recurrent problems after stopping their medication, no one would be surprised. In fibromyalgia patients, the standard of acupuncture practice is to explore with the patient after an initial series of 6 to 10 treatments if there is any subjective improvement, and if there is, to continue such treatments on a time scale and schedule that maintains improvement.[2]

In my medical and acupuncture practice, I have had the occasion to treat many fibromyalgia patients over the past 20 years. Some have improved dramatically, others less so, and others, not at all. This brings us back to the basic question of what fibromyalgia really is as a clinical entity and how it should best be managed. A variety of medications, physical modalities, exercises, trigger point injections, dry-needling, manipulative therapies, and mind-body methods have been utilized for this condition with greater or lesser success.[4] An interdisciplinary mix of approaches seems to be the most pragmatic solution, including acupuncture if desired by the patient and if it provides added benefit *for them as an individual.* The often crippling chronic fatigue and immune deficiency that frequently accompanies fibromyalgia must also be addressed, a matter that was minimally mentioned in any of the studies.

We all have seen the distress of the fibromyalgia and/or chronic fatigue, immune deficient patient who has been diagnosed as mentally ill or psychosomatic by numerous providers just because no lab test was definitive and no one treatment was helpful. Compassionate care would suggest that we try any and all safe therapies, individualizing them in a real world clinical context to provide evidence of effectiveness. Acupuncture was at least considered a safe therapy in this review, so it ought to be considered for the desperate patient with few other real options. Another problem is that randomized controlled trials limit the possibility of studying a mix of modalities, which is the usual approach in fibromyalgia (eg, amitriptyline or an SSRI, graded aerobic exercise, stress management, nutritional support, massage, etc.) along with acupuncture (with or without electrostimulation).

My conclusion is thus diametrically opposite to Mayhew and Ernst. *The evidence from several randomized controlled trials is promising for efficacy of acupuncture in fibromyalgia, and while more and better studies are needed, physicians should consider offering acupuncture to fibromyalgia patients as part of an integrative and holistic approach.*

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Accepted June 28, 2007.

0038-4348/0−2000/10000-1183

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REFERENCE ARTICLE

DOES THIS PATIENT HAVE DEMENTIA?

Holsinger T, Deveau J, Boustani M, et al. *JAMA* 2007; 297:2391-2404.

Response from

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The Diagnosis of Dementia

The clinical diagnosis of probable dementia, although daunting, is really quite simple and should not be too time consuming provided the examiner is focused, organized, and methodical. The criteria for this diagnosis are clearly stated in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*.[1]

The clinical diagnosis can be established once there is evidence of memory impairment and cognitive deficit severe enough to interfere with the patient's occupation or daily activities. Cognitive deficits include anomia, agnosia, aphasia, apraxia, visuospatial impairment, and impaired executive functions. The impairment must be a deterioration from a previously higher level of functioning.

Memory impairment is the sine qua non condition for the diagnosis of dementia. The two main distinguishing features of the memory impairment associated with dementia are, first, that it is global and not selective, affecting trivial as well as important matters. This is different from the memory impairment seen in normal aging, benign forgetfulness, or the impairment sometimes associated with depression, which tends to be selective: the patient forgetting only what is trivial or what he or she considers trivial. Second, the memory impairment interferes with the patient's daily activities and/or occupation. The patient may try to use memory aids, making lists, for instance, but often forgets to use these lists.

Anomia is the inability to name objects in the absence of visual impairment. It is tested by asking the patient to name various objects, such as a wrist watch, the hands of the watch, or the wrist strap. The patient sees and recognizes the object but is not able to recall the name of that object. Often the patient paraphrases the name of the object. Dr. Alois Alzheimer describes a patient in his original paper that could not name the milk "jug"; instead, she stated that this was the milk "pourer." Patients may describe the hands of the watch as the "things that tell the time." Objects with which the patient is not very familiar are the first ones to be affected (ie, a stethoscope for a nonphysician).

Agnosia is also the inability to recognize objects in the absence of visual impairment. Unlike anomia, where the main problem is finding the correct word, patients with agnosia are not able to recognize various objects because they are not able to integrate various stimuli and identify the object in question by comparing this information with that stored in the memory. Agnosia may have serious consequences if the patient lives alone and is not able to recognize that his or her food has rotted, for example.

Aphasia is an impairment in language: the patient is not able to recognize a word (sensory aphasia) or is not able to state the word correctly (expressive or motor aphasia). An important caveat, especially important these days, is the "accent." If the patient is not familiar with the examiner's accent, she may not be able to recognize some words. Aphasia also should be differentiated from dysarthria when the patient has difficulties pronouncing the word.

Apraxia is an inability to perform physical tasks in the absence of motor, sensory, or cerebellar deficits. Apraxia also can be tested by asking the patient to perform common tasks, such as buttoning or unbuttoning her shirt or taking her coat off and putting it back on. These tasks, often taken for granted, are the result of the successful, almost instantaneous, integration of various changing tactile and sometimes visual stimuli. Patients with dementia may not be able to process all this information in a timely manner. The clock drawing test is often used to test visuospatial skills.

Impaired executive function is the inability of the patient to plan, initiate, sequence, monitor, and alter behavior in response to changing circumstances. Losing one's way while driving and not being able to find the way back, repeated road traffic accidents when the patient is at fault, running out of gas while driving, the inability to prepare a meal, and the inability to balance one's checkbook (provided the patient was able to balance it) are examples of impaired executive functions. Impaired executive functions could have serious consequences if the patient lives alone and is not able to adjust the thermostat, decide what clothes to put on, know what information to give on the phone, or whether or not to let a stranger in the house. Patients with impaired executive functions also may not be able to take care of their finances and are at risk of squandering their resources by making judgmental errors.

The diagnosis of dementia therefore should be based on the answer to two questions: 1) Is there a memory impairment? and 2) Is there evidence of cognitive deficit? If both answers are affirmative, then the diagnosis is probable dementia. If there is evidence of memory impairment, but no cognitive deficit, the patient may have mild cognitive impairment.

The term mild cognitive impairment (MCI) is used to describe memory impairment interfering with daily activities or professional life but with intact cognitive function. The memory impairment should be objectively identified and preferably corroborated by an informant. The importance of diagnosing MCI is that in many patients it is a precursor of dementia, the annual conversion rate being between 6% and 25%.[2]

Once the diagnosis of dementia is made, the subtype of dementia should be identified. The various dementias have different presentations, courses, and associated features which are beyond the scope of this article. This differentiation, however, is more than an academic exercise, as it has therapeutic implications: patients with dementia with Lewy bodies, for instance, tend to respond better than patients with Alzheimer disease to acetylcholinesterase inhibitors and tend to be very sensitive to psychotropic medication.

The diagnosis of dementia should also include the severity of the condition, as this too has important therapeutic implications. For example, the management strategy of a patient with very early Alzheimer disease, where the main concern is impaired executive function, is quite different from the management strategy of the moderately cognitively impaired

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Accepted June 14, 2007.

0038-4348/0−2000/10000-1184

patient, where safety is an important issue. This is also quite different from the management strategy of the bed-ridden patient.

A number of scales to assess memory and cognitive functions are available. Their sensitivity, specificity, merits, and limitations have been reviewed in the paper by Holsinger et al[3] As most of these tests have a number of limitations, it can be debated whether they could be used to screen for and even diagnose dementia.

The term "screening" is indeed controversial. Screening implies the detection of a disease during its asymptomatic stage. Once a patient starts to have memory impairment, symptoms are present and therefore the administration of these tests is not for screening, but rather for diagnostic purposes, albeit of a disease in its very early stages. The main use of these tests, therefore, is neither to screen nor to make a diagnosis, but rather to establish a baseline against which the patient's progress can be evaluated. This is quite important now as there are a number of medications that can help patients with dementia, and it is important to detect subtle changes that would otherwise be missed.

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REFERENCE ARTICLE

CARDIOVASCULAR DISEASE AND SUBSEQUENT KIDNEY DISEASE.

Elsayad EF, Tighiouart H, Griffith J, et al. *Arch Int Med* 2007; 167:1130-1136.

Response from

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Cardiovascular Disease is an Independent Risk Factor for Chronic Kidney Disease

Chronic kidney disease (CKD) is a significant problem in the United States, and its incidence is increasing at an alarming rate. Nearly 20 million Americans are affected by CKD and some 20 million more are at risk of developing it.[1,2] While a diagnosis of CKD can be made by recognizing the presence of kidney damage for ≥3 months as defined by structural or functional abnormalities with or without decreased glomerular filtration rate (GFR), most patients are found to have CKD because of a GFR <60 mL/min/1.73 m^2.[1]

Cardiovascular (CV) disease is extremely prevalent in CKD, even more so than in the general population. Nearly half of all patients with CKD die of CV events, particularly heart failure and acute myocardial infarction.[3] In fact, most individuals at risk for end-stage renal disease die of CV disease before they ever require dialysis. The National Kidney Foundation (NKF) task force on CV disease in CKD suggests that patients with CKD be considered the highest risk group for subsequent CV events.[4] While we know that the timely diagnosis and appropriate treatment of CKD can delay kidney disease progression as well as decrease adverse CV outcomes, it has been unclear if this relationship is bidirectional. That is, does the presence of underlying CV disease contribute to the development and/or progression of CKD?

To answer this question, a recent study pooled data from the Atherosclerosis Risk in Communities Study and the Cardiovascular Health Study; two longitudinal, community-based studies in which 13,826 people had kidney function measured at regular intervals over nine years of follow-up.[5] Of these, 1,787 (12.9%) had evidence of underlying CV disease and 12,039 did not. Recruited between 1987 and 1990, subjects were at least 45 years of age (mean age 57) with follow-up information available for 78% of participants. Baseline CV disease was defined by stroke, angina, intermittent claudication, transient ischemic attack, coronary angioplasty or bypass and recognized or silent MI. Study outcomes included decline of kidney function primarily defined by an increase in serum creatinine level of at least 0.4 mg/dL between the first (mean baseline 0.9 mg/dL) and last visits, and development of kidney disease defined by the same increase in serum creatinine in which the baseline level was less than 1.4 mg/dL in men and less than 1.2 mg/dL in women. Secondarily, kidney function decline and disease were defined by reductions in estimated glomerular filtration rate (eGFR) of at least 15 mL/min with the final eGFR less than 60 mL/min. No measurement of albuminuria was used.

Using these models, 3.8% of all patients developed a decline in kidney function and 2.3% developed frank renal disease; occurring in 3.3% of those without underlying CV disease and 7.2% where underlying CV disease was present. Using the serum creatinine level, underlying CV disease was associated with an increased risk of kidney function decline (odds ratio 1.70; 95% CI, 1.36–2.13) and the development of CKD (odds ratio 1.75; CI, 1.32–2.32). In one of the cohorts, serum creatinine had to be adjusted for patients who were using angiotensin-converting enzyme (ACE) inhibitor therapy as their use in those with underlying CV disease was associated with delayed progression to CKD. For eGFR, the odds ratio was 1.28; 95% CI, 1.13 to 1.45) for kidney function decline and 1.54; 95% CI, 1.26 to 1.89) for development of kidney disease.

This is the first community-based study to find that the presence of underlying CV disease is independently associated with both the decline of kidney function and the development of kidney disease. Individuals with a decline in kidney function had higher baseline serum creatinine levels, and were more likely to be older, have hypertension and diabetes, and be African American. The most reasonable explanation for all of this is that atherosclerotic disease does not limit itself to the cardiac vasculature. It affects the renal vasculature as well and this coexistence results in CKD, especially in these high-risk populations.

So what does this mean to the practicing physician? CV disease is an independent risk factor for kidney function decline and the development of kidney disease. Healthcare practitioners should remain vigilant for the development and progression of kidney disease when caring for individuals with multiple CV disease risk factors or those with underlying CV disease. These atherosclerotic risk factors need to be aggressively approached. These individuals should be considered for use of an ACE inhibitor or angiotensin type 1 receptor blocker, B-blocker, statin, or aspirin; as they are at high risk for the development of CKD. Future studies

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Accepted June 26, 2007.

0038-4348/0−2000/10000-1185

using levels of proteinuria are needed to provide an earlier view into the systemic vasculature, as small increases in urinary protein excretion often provide an earlier marker for generalized endothelial dysfunction than increases in serum creatinine. For now, clinicians treating individuals with multiple cardiac risk factors should recognize that CV disease is independently associated with decline of kidney function and the development of CKD. Utilizing evidence-based strategies in these high-risk individuals should reduce the development of CKD.

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Season's Greetings

May All the Joys of the Season be with You Now and in the Coming Year

from the Editor and Staff of the Southern Medical Journal