

November 1998 Issue | Gerald M. Lemole, M.D.

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We define functional medicine as the field of health care focused on assessment and early intervention to improve physiological, cognitive/emotional, and physical function. We have made certain presumptions about how it is to be used in both assessment and intervention, and we are continuing to try to codify those uses to make them more clinically valuable in improving patient health outcome.

One reality in health care is that more than 80 percent of healthcare services are used by 10 percent of the population. Between 80 and 90 percent of the expenditures come from 10 percent of the subscribers of a health plan or program. When delivering messages to an audience regarding health care, one should focus the message on the high utilizers to get the most cost-effective return on your investment. One assumes that if you target the right message, the right program, or the right treatment to those individuals, you will get a return on the investment in the form of improved health, reduced use of services—extension of the health span, so to speak.

The public health philosophy, therefore, has been to distribute its message to everyone. Examples include lowering cholesterol, increasing fiber in the diet, and lowering salt consumption. Those messages are generally distributed to everyone, even though we know that although lower-cholesterol diets may be highly significant for some individuals, they have no relevance to cardiovascular function or health in others. The same can be said of salt restriction. For most people in a hypertensive population, we are beginning to see that salt restriction has little or no effect on lowering blood pressure. In those individuals who have sensitivity to salt, or sodium chloride, a salt-restricted diet does have benefit in lowering blood pressure. The challenge is to target the message to the right people.

A number of studies of high utilizers of health care have shown that socioeconomic factors relate to health behaviors, illness, and increased mortality, as also was described in a previous FMU. A recent paper published in the *Journal of the American Medical Association*, titled "Lower Socioeconomic Status and Increased Mortality. Early Childhood Roots and the Potential for Successful Interventions," reached some surprising conclusions. The study challenges the way we have thought about these high utilizers, individuals who may have disproportionately high health risks and therefore utilize more medical services.¹

Over the past several decades, health behaviors and lifestyle factors like cigarette smoking, being overweight, drinking excess alcohol, or leading a sedentary life have been cited repeatedly as major determinants of premature and preventable morbidity and mortality. In addition, it has been documented

that people of lower socioeconomic position are significantly more likely to lead sedentary lives, be overweight, smoke cigarettes, and drink more alcohol. Therefore, in the past we have formed what we believed was the logical hypothesis from this association. We assumed that the elevated mortality and morbidity (i.e., high utilization of health services) in this lower socioeconomic group was due primarily to the higher prevalence of poor health habits.

The *JAMA* article explored this hypothesis because previous efforts to explain the socioeconomic differences in mortality in a variety of subpopulations have found that strong differences remain, after controlling for major lifestyle risk factors in morbidity, in individuals of lower socioeconomic strata. Those differences are not, apparently, related merely to poor health habits and high risk factors, at least the risk factors we commonly associate with disease.

This paper uses some interesting evaluative tools. The results show that although lower income (net of demographic characteristics) leads to a significant increase in mortality risk, only a modest proportion of the relationship can be explained by traditionally acknowledged health risk factors like smoking, alcohol consumption, and sedentary lifestyle. The major explanation, therefore, must lie elsewhere. The study suggests that although differences in socioeconomic status and health behaviors are significant, those differences account for only some social inequalities and overall mortality. Public health policies and intervention, according to the author, that focus exclusively on the individual risk behaviors we commonly associate with disease, have limited potential for reducing the disparities and mortality in this group and move them from high utilizers to lower utilizers of medical services.

INTERVIEW TRANSCRIPT

Clinician of the Month:
Gerald M. Lemole, M.D.

This month as our Clinician of the Month, we are pleased to have Gerald Lemole, MD, a surgeon. Dr. Lemole will help us overcome the common assumption that surgeons think in terms of executing excellence in surgery but may not have an interest in the patient's whole-body physiology, pre- and postoperatively.

Gerald Lemole is a professor of surgery at Jefferson Medical College in Philadelphia. He received his medical degree at Temple University School of Medicine. He was formerly a professor of surgery at Baylor. He has been a visiting professor at the University of Dublin, the University of Istanbul, and in 1991 at the Fengtai Heart Institute in Beijing, China, where he was a Fellow. He is presently chief of cardiac surgery at Christiana Care Health Services in Newark, Delaware. He has published hundreds of papers and made presentations all over the world. Dr. Lemole is a respected authority in his field. He has also advanced into other areas and explored other options and alternatives.

JB: Welcome to *FMU*, Dr. Lemole. My first question, which will lead us into discussing surgery in the context of physiology of the whole individual, relates to a recent paper you and your colleagues

published. In the article, titled "Intravenous Insulin Infusion in Postoperative Coronary Artery Bypass Graft Surgery," you discuss the role of insulin infusion postoperatively. That article reminds us that response to surgery extends beyond the immediate event and the skill of the technician, and that other things are going on. Would you explain what led you to that focus in your work?

GL: Absolutely, Jeff. We collaborated with our colleague, Dr. DeCherney at the Diabetic Center, and felt that by modulating or regulating the insulin as rapidly as possible after surgery, we could decrease the complication rate (particularly strokes and infections) once we balanced the blood sugar levels. This was, indeed, proven to be the case. You see, surgeons are very practical. We always like to see a practical application to a theory or a scientific postulation. In fact, that's what we saw. After surgery, these patients have a tremendous amount of epinephrine release and stress, and their sugars go up. The sooner you can get it down to normal, the less chance you have of these other complications.

JB: That's a very interesting observation. In the less traumatic but stressful, time-urgent workaday world many people suffer from modest hypercortisolemia from activation of the adrenal glands. That situation creates a glycemic response, which then initiates an insulin response. We are referring here to altered signaling molecules that reflect changes in physiology, altered immunochemical function, and altered cell repair. It sounds as if, in the more traumatic situation of postoperative CABG, so to speak, modulating a hormone, in this case insulin, can have a positive impact on function.

GL: Well, you can look at surgery as being a six-hour telescoping of a stressful situation that somebody may have extended over periods of months. We think it's very important to try to modulate the stress reaction. We know you can do that with guided imagery, with positive feedback mechanisms, and working with the patients to keep their stress level down, because the mind can do tremendous things and change how the body reacts to a stressful situation.

JB: You have published some papers and given a number of presentations on the treatment of coronary artery disease with what might be termed "complementary medical therapies." Tell us about your experience and the pros and cons, as you have experienced them, of integrating these concepts into practice.

GL: About 16 years ago, some friends and I went to a functional medicine program you were giving in Washington. You presented some profound work that left us thinking about a lot of things, especially the omega-3 fatty acids, the 5 and 6 delta desaturases, and eicosapentaenoic acid, long before everybody was talking about this. It was a very important point in our lives, because we looked at that and saw how micronutrition and functional medicine could be practically applied to our experience in the surgical field. Over the years, I've used micronutrition in patients; for example, coenzyme Q10 for heart failure. I have one lady who was a transplant candidate 12 years ago who is still alive and well on coenzyme Q10. I personally turned down [for surgery] another person who had a 10 percent ejection fraction. We treated him for eight weeks on coenzyme Q10 and then restudied him. He then had an ejection fraction of 40 percent, so we operated on him and he did very well.

Two medical schools in New York City turned down another lady with cardiac cirrhosis because her liver enzymes were so out of whack. I would have turned her down, too, except that I gave her a chance, put her on some micronutrition—coenzyme Q10, some vitamins, and milk thistle. After about eight weeks, her enzymes came back to almost normal, and she sailed through the operation. We've been committed to the

idea that most people are deficient in some way or another, either because of the medications they're taking, their diet, or the stressful lifestyle they've chosen, and that supplementation is necessary, especially if they're going into a stressful situation like surgery. So, we feel that people with cardiac disease, especially, need basically five areas of supplementation. They need the B vitamins; coenzyme Q10; minerals like zinc, selenium, magnesium, and calcium; essential fatty acids; and antioxidants like A, C, and E. We've tried very hard to get these people on this regimen before and after surgery.

Bibliography

1. Williams RB. Lower socioeconomic status and increased mortality. Early childhood roots and the potential for successful interventions. *JAMA*. 1998;279(21):1745-1746.
2. Lantz PM, House JS, Lepkowski JM, Williams DR, Mero RP, Chen J. Socioeconomic factors, health behaviors, and mortality. Results from a nationally representative prospective study of US adults. *JAMA*. 1998;279(21):1703-1708.
3. Higley JD, Thompson WW, Champoux M, et al. Paternal and maternal genetic and environmental contributions to cerebrospinal fluid monoamine metabolites in Rhesus monkeys (*Macaca mulatta*). *Arch Gen Psychiatry*. 1993;50:615-623.
4. Meaney MJ, Bhatnagan S, Dioria J, et al. Molecular basis for the development of individual differences in the hypothalamic-pituitary-adrenal stress response. *Cell Mol Neurobiol*. 1993;13:321-347.
5. Grattasn LM, Oldach D, Perl TM, et al. Learning and memory difficulties after environmental exposure to waterways containing toxin-producing *Pfiesteria* or *Pfiesteria*-like dinoflagellates. *Lancet*. 1998;352(9127):532-539.
6. Nierenberg DW, Nordgren RE, Chang MB, et al. Delayed cerebellar disease and death after accidental exposure to dimethylmercury. *N Engl J Med*. 1998;338(23):1672-1676.
7. Kulig K. A tragic reminder about organic mercury. *N Engl J Med*. 1998;338(23):1692-1693.
8. Flora K, Hahn M, Rosen H, Benner K. Milk thistle (*Silybum marianum*) for the therapy of liver disease. *Am J Gastroenterol*. 1998;93(2):139-143.
9. Magliulo E, Gagliardi B, Fiori GP. Zur Wirking von Silymarin bei der Behandlung der akuten Virushepatitis. *Med Klin*. 1978;73:1060-1065.
10. Kiesewetter E, Leodolter I, Thaler H. Ergebnisse zweier Doppelblind-studien zur Wirksamkeit von Silymarin bei chronischer Hepatitis. *Leber Magen Darm*. 1977;7:318-323.
11. Martin GM, Wang E. Aging and longevity: towards the next millennium. *Mol Med Today*. 1998;4(4):145.
12. Fossel M. Telomerase and the aging cell. Implications for human health. *JAMA*. 1998;279(21):1732-1735.
13. Wallace DC, Melov S. Radicals r'aging. *Nature Genetics*. 1998;19(2):105-106.
14. Parkes TL, Elia AJ, Dickinson D, Hilliker AJ, Phillips JP, Boulianne GL. Extension of *Drosophila* lifespan by overexpression of human SOD1 in motorneurons. *Nature Genetics*. 1998;19(2):171-174.
15. Zastawny TH, Dabrowska M, Jaskolski T, et al. Comparison of oxidative base damage in mitochondrial and nuclear DNA. *Free Rad Biol Med*. 1998;24(5):722-725.
16. Hulley S, Grady D, Bush T, et al. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. *JAMA*. 1998;280(7):605-613.

17. Colditz GA. Relationship between estrogen levels, use of hormone replacement therapy and breast cancer. *J Natl Cancer Inst.* 1998;90:814-823.
18. Fenech M, Aitken C, Rinaldi J. Folate, vitamin B12, homocysteine status and DNA damage in young Australian adults. *Carcinogenesis.* 1998;19(7):1163-1171.
19. Fouty B, Frerman F, Reves R. Riboflavin to treat nucleoside analogue-induced lactic acidosis. *Lancet.* 1998;352(9124):291-292.
20. Palomaki A, Malminiemi K, Solakivi T, Malminiemi O. Ubiquinone supplementation during lovastatin treatment: effect on LDL oxidation ex vivo. *J Lipid Res.* 1998;39(7):1430-1437.
21. Fuchs J, Packer J, Zimmer G. Lipoic acid in health and disease: antioxidants in health and disease series/6. *Free Rad Biol Med.* 1998;24(9):1537-1538.
22. Taylor PR, Albanes D. Selenium, vitamin E, and prostate cancer—ready for prime time? *J Natl Cancer Inst.* 1998;90(16):1184-1185.
23. Yoshizawa K, Willett WC, Morris SJ, et al. Study of prediagnostic selenium level in toenails and the risk of advanced prostate cancer. *J Natl Cancer Inst.* 1998;90(16):1219-1224.
24. Ahmed S, Leo MA, Lieber CS. Interactions between alcohol and B-carotene in patients with alcoholic liver disease. *Am J Clin Nutr.* 1994;60(3):430-436.
25. Leo MA, Aleynik SI, Aleynik MK, Lieber CS. B-carotene beadlets potentiate hepatotoxicity of alcohol. *Am J Clin Nutr.* 1997;66(6):1461-1469.
26. Ridker PM, Hennekens CH, Roitman-Johnson B, Stampfer MJ, Allen J. Plasma concentration of soluble intercellular adhesion molecule-1 and risks of future myocardial infarction in apparently healthy men. *Lancet.* 1998;351:88-92.
27. Blann AD, McCollum. Circulating ICAM-1 in peripheral arterial disease as a predictor of adverse events. *Lancet.* 1998;351(9109):1135.
28. Albertini JP, Valensi P, Lormeau B, et al. Elevated concentrations of soluble E-selectin and vascular cell adhesion molecule-1 in NIDDM. *Diabetes Care.* 1998;21(6):1008-1013.
29. Diehm C, Trampisch HJK, Lange S, Schmidt C. Comparison of leg compression stocking and oral horse-chestnut seed extract therapy in patients with chronic venous insufficiency. *Lancet.* 1996;347:292-294.
30. McCarron DA. Diet and blood pressure—the paradigm shift. *Science.* 1998;281(5379):933-934.
31. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med.* 1997;336(16):1117-1124

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