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# Correspondence

## Hyperbaric Oxygen Therapy in Multiple Sclerosis

Articles by Neubauer et al.<sup>1</sup> and Maxfield<sup>2</sup> review the history of the use of hyperbaric oxygen therapy (HBOT) to prevent progression in multiple sclerosis (MS). Unfortunately, the emphasis seemed to be on the controversy rather than the potential. In medicine, a controversy is often a consequence of resistance by scientific and medical authorities to changing established ways of thinking in the presence of evidence that challenges the status quo.

Many hyperbaric physicians report good results with HBOT in patients with MS: some patients don't deteriorate, and some even improve. This result is virtually unheard of in MS. The data that Neubauer et al. present in Tables 1 and 2 is remarkable. The usual teaching is that MS only gets worse. Most insurers do not even cover physical therapy for MS patients because there is no expectation that they can recover functional abilities. Reports of recovery go against everything we have been taught in medical school and residency. Traditional teaching holds that the injured brain does not recover, and retraining uninjured brain to compensate is the best that one can hope for. But we need to search for better answers.

The benefits of HBOT in neuro-rehabilitation can revolutionize old thinking and old treatments. Within 8 months of opening my hyperbaric chamber treating exclusively patients with neurological disease, I have watched patients with traumatic brain injury, multiple sclerosis, stroke, and cerebral palsy make recoveries that none of their other physicians would have expected. The results also support new

thinking about stroke: that there is an ischemic penumbra of recoverable brain tissue that has been injured but can be rendered metabolically active again, with functional recovery to follow.

Controversy should inspire inquiry and reevaluation of standard theories, not stonewalling. I am going to stay excited about the potential of neurorehabilitation with HBOT for patients with MS.

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<sup>1</sup> Neubauer RA, Neubauer V, Gottlieb SF. The controversy over hyperbaric oxygenation therapy for multiple sclerosis. *J Am Phys Surg* 2005;10:112-115.

<sup>2</sup> Maxfield WS. Hyperbaric oxygen therapy for multiple sclerosis: my experience. *J Am Phys Surg* 2005;10:116.

## The End of Welfare

The excellent article by Dr. Alphonse Crespo<sup>1</sup> is a brief but surprisingly thorough discussion on the merits of an individual's direct impact on the needs of another individual in a society—good, bad or ugly. Rather than holding individuals accountable, “modern (mostly western) society” has constructed groups to become distant parties enslaved to politically correct societal needs for the purposes of wealth redistribution. The brokers, of course, must be fed so the redistribution schemes ultimately become very inefficient. In the case of Medicaid, almost no tax-sourced funds get to the primary providers. Rather, monies are largely consumed by the brokers: government and hospital administrative employees, and social-service personnel, among the more obvious examples.

As a physician I am paying large sums in taxes that are paid to states for the purpose of funding programs such as Medicaid. My services to qualifying patients are not reasonably compensated, and I am forbidden to ask the patient for direct payment. So, in effect, I pay twice. It is not surprising that so few physicians are seeing Medicaid patients. Is it any surprise that Medicare is coming under the same pressure?

A reasonable alternative to the welfare state is Health Saving Accounts (HSAs). They must become more available so that productive “baby-boomers” will not be dispossessed of their hard-earned retirement assets. If lifelong wards of the state wish to continue their lifestyle of dependency uninterrupted, then they will surely suffer from the inevitable cutbacks in services. Given a chance, personal responsibility (HSAs) will trump the moral bankruptcy of the socialist systems. Medicare and Medicaid will eventually collapse.

Which is preferable? Guaranteed economic disaster for retirees trapped in government dependency? Or a chance to choose freedom and self-reliance?

**Ted Yaeger, M.D.**  
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<sup>1</sup> Crespo A. The end of welfare, and its effect on the poor. *J Am Phys Surg* 2005;10:122-123.

## On Homosexuality

Dr. Nathaniel Lehrman’s article “Homosexuality: Some Neglected Consequences”<sup>1</sup> is worthy of serious consideration. There are indeed public health consequences of choosing homosexual behavior.

A recent nationwide survey conducted by the Centers for Disease Control and Prevention showed that among homosexual men between the ages of 15-44 years, 73% of them had more than one sexual partner over the course of one year, whereas 78% of straight men had only one partner. Data were similar among surveyed lesbians in the same

age cohort; only 25% of these women had only one sexual partner over the course of a year, while 69% of heterosexual women reported only one partner.

With this impressive degree of promiscuity, is it any wonder that the gay and lesbian community experiences rampant sexually transmitted infections?

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<sup>1</sup> Lehrman, N. Homosexuality: some neglected considerations. *J Am Phys Surg* 2005;10:80-82

## The Scientific Basis for LDL Cholesterol-Lowering Therapy

The recent paper by Anthony Colpo<sup>1</sup> is critically flawed from a scientific standpoint.

The author states: “The belief that low-density lipoprotein (LDL) cholesterol causes atherosclerosis and subsequent heart disease is a fundamental precept of modern medicine.”

Generally, normal levels of LDL-cholesterol, in and of themselves, do not cause atherosclerosis; rather it is important for normal physiological functions. There is no disputing that hypercholesterolemia, most commonly associated with an elevation of plasma LDL, is an important contributing factor in atherogenesis; its correction can reduce the risk of coronary heart disease (CHD).

Colpo states that “proliferation of [atherosclerotic] plaques may occur, not because of simple elevations in blood cholesterol, but because of unfavorable physiological conditions that damage or weaken the structure of the arterial wall,” a statement that is illogical and unscientific because a high blood level of cholesterol itself is one of the most unfavorable physiological conditions that can lead to damaging the structure of the arterial wall.

He then goes on to conclude that “all of these factors have been shown to exert an atherogenic effect unrelated to serum

cholesterol elevation.” Reaching such a conclusion suggests a basic misunderstanding of the pathogenesis of cardiovascular diseases. The earliest recognizable lesion of atherosclerosis is the fatty streaks within the innermost layer of the artery wall. They precede the development of intermediate lesions, and develop into the more complex occlusive lesions that may impede blood flow by projecting into the arterial lumen. The occlusive lesions of atherosclerosis can be clinically reversed in many cases after aggressive treatment with various lipid-lowering drugs.<sup>2,3</sup>

Colpo realized that “the number of deaths from CHD has indeed decreased since the late 1960s,” but argued that “total incidence of CHD has not declined.” He further blamed the medical community, since it has “failed to help people avoid CHD in the first place.” Strangely, Colpo has ignored the fact that the world’s population has doubled since 1960 and is aging. The risk for CHD increases steeply with advancing age.<sup>4</sup> The claim that the “war on cholesterol” has “delivered no benefit to public health” is not only irresponsible, but false.

Of further concern, Colpo has repeatedly mixed up the terms LDL, high-density lipoprotein (HDL), and cholesterol. He states that, “the concept that LDL is ‘bad’ cholesterol is a simplistic and scientifically untenable hypothesis.” LDL is not cholesterol. He also states that, “HDL cholesterol, on the other hand, is the ‘heart-friendly’ lipoprotein.”

Cholesterol is not a lipoprotein. Cholesterol is a lipid present in cell membrane and travels in the blood in distinct particles containing both lipid and lipoproteins. LDL provides cholesterol to cells through LDL receptors, whereas HDL removes excess cholesterol through reverse cholesterol transport,<sup>5</sup> thus maintaining cholesterol homeostasis.

The link between lipids and CHD has been firmly established by epidemiologic studies and long-term outcomes trials.<sup>4</sup> Despite the emergence of new markers of

CHD such as C-reactive protein, LDL cholesterol currently should remain the primary target for reduction of risk of CHD.<sup>4,6</sup>

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<sup>1</sup> Colpo A. LDL cholesterol: “bad” cholesterol, or bad science? *J Am Phys Surg* 2005;10:83-89.

<sup>2</sup> Ross R. The pathogenesis of atherosclerosis: a perspective for the 1990s. *Nature* 1993;362:801-809.

<sup>3</sup> Lusis AJ. Atherosclerosis. *Nature* 2000; 407:233-241.

<sup>4</sup> National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Final report. *Circulation* 2002;106:3143-3421.

<sup>5</sup> Ashen MD, Blumenthal RS. Low HDL cholesterol levels. *N Eng J Med* 2005;353: 1252-1260.

<sup>6</sup> Goldstein JL, Brown MS. The cholesterol quartet. *Science* 2001;292:1310-1312.

**In Reply:** Wu and Schauss insist that elevated total and LDL cholesterol levels are “an important contributing factor in atherogenesis,” but fail to address the numerous contradictions inherent in the lipid hypothesis that I raised in my article. Why have scores of studies found a complete lack of correlation between serum LDL and total cholesterol levels and extent of atherosclerosis? Why have numerous tightly controlled clinical dietary cholesterol-lowering trials failed to produce any reduction in CHD, despite the fact that cholesterol levels were indeed lowered in the treatment groups?<sup>1-6</sup> Why have controlled clinical studies observed more atherosclerotic regression, less decrease in minimal luminal diameter, fewer cardio-

vascular events, and significant declines in mortality among patients taking fish oil, despite the fact that it *raised* serum LDL cholesterol levels? Where is their discussion of the fact that statins exert anti-inflammatory, anti-atherogenic effects even when their cholesterol-lowering capabilities are disabled? To claim that elevated LDL or total cholesterol promotes atherosclerosis in the face of such observations defies logic.

Fatty streaks are considered to be an early manifestation in the development of atherosclerotic plaque. Fatty streaks are actually not fat, but congregations of cells under the endothelium, primarily made up of macrophages but possibly also containing lymphocytes, leukocytes, platelets, and smooth muscle cells.<sup>7</sup> The macrophages have a foamy appearance due to high concentrations of cholesterol and phospholipids in their cytoplasm, but a high cellular lipid content does not prove that cholesterol is the causative factor. In fact, the extensive involvement of white blood cells reinforces the concept that atherosclerosis is not the product of simple blood cholesterol elevation, but an inflammatory immune response to arterial injury.

Lipid-lowering drugs can indeed impede and even reverse atherosclerosis, but there is a paucity of evidence for the claim that lipid-lowering is the responsible factor. As my article carefully detailed, an overwhelming body of evidence indicates that it is in fact the pleiotropic effects of statins that explain their beneficial cardiovascular effects. Wu and Schauss offer no discussion of this abundant evidence.

Had Wu and Schauss read the studies I cited, which highlight the glaring disparity between CHD mortality and CHD incidence, they would know that these studies used *age-adjusted* data, and thus already account for the increased proportion of the population aged over 65.

Wu and Schauss’s claim that I do not know the difference between cholesterol and its carrier lipoproteins is rather bizarre considering I clearly elucidate the difference in the first page of my article (see

fifth paragraph). The terminology employed throughout the rest of my paper merely reflects popular use of the terms LDL and HDL as abbreviated references to LDL cholesterol and HDL cholesterol.

Apart from alluding to the simplistic and distinctly unscientific “passive osmosis” theory, Wu and Schauss offer no physiologically plausible mechanism by which elevated cholesterol can instigate atherosclerosis. Despite absence of evidence of a causative role for LDL cholesterol or total cholesterol in CHD, and the significant, clinically proven benefits of such interventions as omega-3 supplementation, antioxidant-rich diets based on unrefined foods, and exercise, Wu and Schauss nonetheless recommend that “LDL cholesterol currently should remain the primary target for reduction of risk of CHD.”

Such blind devotion to current dogma was the very stimulus for writing my paper in the first place.

**Anthony Colpo**

<sup>1</sup> Rose GA, Thomson WB, Williams RT. Corn oil in treatment of ischaemic heart disease. *BMJ* 1965;1:1531-1533.

<sup>2</sup> Research Committee to the Medical Research Council. Low-fat diet in myocardial infarction: a controlled trial. *Lancet* 1965;2:501-504.

<sup>3</sup> National Diet Heart Study. Final report. *Circulation* 1968;37:1-428.

<sup>4</sup> Medical Research Council. Controlled trial of soya-bean oil in myocardial infarction. *Lancet* 1968;2:693-699.

<sup>5</sup> Woodhill JM, Palmer AJ, Leelarthaeapin B, et al. Low fat, low cholesterol diet in secondary prevention of coronary heart disease. *Adv Exp Med Biol* 1978;109:317-330.

<sup>6</sup> Frantz ID Jr, Dawson EA, Ashman PL, et al. Test of effect of lipid lowering by diet on cardiovascular risk. The Minnesota coronary survey. *Arteriosclerosis*, 1989;9: 129-135.

<sup>7</sup> Stary HC, Chandler AB, Glagov S, et al. A definition of initial, fatty streak, and intermediate lesions of atherosclerosis. A report from the Committee on Vascular Lesions of the Council on Arteriosclerosis, American Heart Association. *Arterioscler Thromb* 1994;14:840-856.