IS U.S. HEALTH REALLY THE BEST IN THE WORLD?

[The following information is derived from the above titled article in the Journal of the American Medical Association, Vol. 284, No. 4, 483-5, 26 July 2000, by Barbara Starfield, MD, MPH.]

JAMA: “Information concerning the deficiencies of US medical care has been accumulating. The high cost of the health care system is considered to be a deficit, but seems to be tolerated under the assumption that better health results from more expensive care, despite evidence from a few studies indicating that as many as 20% to 30% of patients receive contraindicated care.”

BP: The “deficit” reference to the high cost of health care in the United States is so strong that many, if not most, Americans have been priced out of the market. Most readers of this newsletter are health care professionals. Even so, without insurance would you be able to afford major health care expenses? Criticism of HMOs is easy and popular, but few Americans would have health care without them. Alternative solutions affordable to most Americans had better soon be found.

JAMA: “In addition, with the release of the Institute of Medicine (IOM) report “To Err is Human,” millions of Americans learned, for the first time, that an estimated 44,000 to 98,000 among them die each year as a result of medical errors.”

BP: The findings of 44,000-98,000 deaths per year from medical errors and that 20-30% of patients receive contraindicated medical care are astounding! We claim to have the best, and most expensive, health care system in the world. Yet, the documentation places the United States far from the best. Where have we gone wrong?
JAMA: "The fact is that the US population does not have anywhere near the best health in the world. Of 13 countries in a recent comparison, the United States ranks an average of 12th (second from the bottom) for 16 available health indicators. Rankings of the United States on the separate indicators are: 
*13th (last) for low-birth-weight percentages. 
*13th for neonatal mortality and infant mortality overall. 
*11th for postneonatal mortality. 
*13th for years of potential life lost (excluding external causes). 
*11th for life expectancy at 1 year for females, 12th for males. 
*10th for life expectancy at 15 years for females, 12th for males. 
*10th for life expectancy at 40 years for females, 9th for males. 
*7th for life expectancy at 65 years for females, 7th for males. 
*3rd for life expectancy at 80 years for females, 3rd for males. 
*10th for age-adjusted mortality.

The poor performance of the United States was recently confirmed by the World Health Organization, which used different indicators. This report ranked the United States as 15th among 25 industrialized countries.

The perception is that the American public "behaves badly" by smoking, drinking, and perpetrating violence. The data show otherwise, at least relatively. The proportion of females who smoke ranges from 14% in Japan to 41% in Denmark; in the United States it is 24% (5th best). For males, the range is from 26% in Sweden to 61% in Japan; it is 28% in the United States (3rd best).

The data for alcoholic beverage consumption are similar; the United States ranks fifth best. Thus, although tobacco use and alcohol use in excess are clearly harmful to health, they do not account for the relatively poor position of the United States on these health indicators. The data on years of potential life lost exclude external causes associated with deaths due to motor vehicle collisions and violence, and it is still the worst among the 13 countries. The United States has relatively low consumption of animal fats (5th lowest in men aged 55-64 in 20 industrialized countries) and the third lowest mean cholesterol concentrations among men aged 50 to 70 years among 13 industrialized countries."

BP: Is the medical profession feeding the country a bill of goods? The country has been told that if we stop smoking, exercise regularly, and limit intake of fats and cholesterol we will be healthier and will live longer. The data from these two reports do not support that hypothesis.

JAMA: "The real explanation for relatively poor health in the United States is undoubtedly complex and multifactorial. From a health system viewpoint, it is possible that the historic failure to build a strong primary care infrastructure could play some role. Although better access to care, including universal health insurance, is widely considered to be the solution, there is evidence that the major benefit of access accrues only when it facilitates receipt of primary care. The health care system also may contribute to poor health through its adverse effects. For example, US estimates of the combined effect of errors and adverse effects that occur because of iatrogenic damage not associated with recognizable error include: 
*12,000 deaths/yr from unnecessary surgery. 
*7,000 deaths/yr from medication errors in hospitals. 
*20,000 deaths/yr for other errors in hospitals. 
*80,000 deaths/yr from nosocomial infections in hospitals. 
*106,000 deaths/yr from nonerror, adverse effects of medications.
These total to 225,000 deaths per year from iatrogenic causes. Three caveats should be noted. First, most of the data are derived from studies in hospitalized patients. Second, these estimates are for deaths only and do not include adverse effects that are associated with disability or discomfort. Third, the estimates of death due to error are lower than those in the IOM report. If the higher estimates are used, the deaths due to iatrogenic causes would range from 230,000 to 284,000. In any case, 225,000 deaths per year constitutes the third leading cause of death in the United States, after deaths from heart disease and cancer.

One analysis overcomes some of these limitations by estimating adverse effects in outpatient care and including adverse effects other than death. It concluded that between 4% and 18% of consecutive patients experience adverse effects in outpatient settings, with 116 million extra physician visits, 77 million extra prescriptions, 17 million emergency department visits, 8 million hospitalizations, 3 million long-term admissions, 199,000 additional deaths, and $77 billion in extra costs."

BP: Whoa!! This is truly shocking information. It is also a scathing indictment of “Standard of Care” concept of medical/dental care. How good is current medical/dental care in the United States in the face of this information?

JAMA: “Recognition of the harmful effects of health care interventions, and the likely possibility that they account for a substantial proportion of the excess deaths in the United States compared with other comparably industrialized nations, sheds new light on imperatives for research and health policy. Alternative explanations for these realities deserve intensive exploration.”

BP: Amen! First, eliminate reliance upon the Standard of Care concept, from the professions, their Boards, and the Courts. Second, pay attention to the “alternative explanations” offered by physicians, dentists, and other health professionals that are currently not considered to be in the “main stream” of health care. If the health care professions do not act forthwith, than the United States Congress must.

For biological dentistry, the implications of the information in this JAMA article are profound, indeed. We already know that current research is showing a strong relationship between oral health, especially periodontally, and various systemic illnesses. Much of the ill health found in the United States might eventually be traced to oral conditions.

Currently, the “Standard of Care” in the United States separates medical from dental. This posture is obviously outdated and contradictory to human health. The dental profession must take its rightful role as a specialty within medical health care.

Clearly, the “Standard of Care” concept for the direction of the medical/dental profession has failed to provide the American public with the standard of health care it deserves (and so dearly pays for). The information from the JAMA article provides the proof. It is time to change, preferably to a standard based on valid clinical and scientific evidence. “Evidence Based” medical/dental care must replace the current “Tradition Based” Standard of Care!

*************

DOCTORS’ GROUP OPPOSES
MANDATED VACCINES

The Association of American Physicians and Surgeons (AAPS), formed in 1943, is a professional association of physicians dedicated to the sanctity of the patient-physician relationship. At its Annual Meeting held on 25-28 October 2000 in St. Louis. AAPS resolved: That AAPS calls for a moratorium on vaccine mandates and for physicians to insist upon truly informed consent for the use of vaccines. AAPS focused on various aspects of informed
consent and freedom of choice and provided a “Fact Sheet on Mandatory Vaccines.” They stated that according to government statistics, children under age 14 are three times more likely to suffer adverse effects, including death, from hepatitis B vaccine than to catch the disease itself.

AAPS stated that it does not oppose vaccines and has never taken an anti-vaccine position. However, it noted that the vaccine approval process is flawed and there are possible conflicts of interest in the process.

AAPS concluded: “Mandatory vaccines violate the medical ethic of informed consent. A case could also be made that mandates for vaccines by school districts and legislatures is the de facto practice of medicine without a license.”

[www.aapsonline.org]

***************

SCIENCE

In Vitro Toxicity of Mercury, Cadmium, and Arsenic to Platelet Aggregation: Influence of Adenylate Cyclase and Phosphodiesterase Activity.

Kumar, SV; Bhattacharya, S.


ABSTRACT: In vitro effect of mercury (Hg\textsuperscript{2+}), cadmium (Cd\textsuperscript{2+}), and arsenic (As\textsuperscript{3+}) on adenylate cyclase (AC) and phosphodiesterase (PDE) activity in relation to platelet aggregation (PA) was studied in rats. Cd\textsuperscript{2+} significantly elevated cAMP (p<0.005) in dose-dependent (5, 10 and 20 pmoles) manner while Hg\textsuperscript{2+} and As\textsuperscript{3+} significantly reduced the cAMP level (p<0.01) and p<0.005 respectively).

Our studies further reveal that Hg\textsuperscript{2+} and As\textsuperscript{3+} inhibit AC and stimulate PDE activity with a concomitant increase in the rate of PA. On the other hand, Cd\textsuperscript{2+} stimulates AC and inhibits PDE activity with a decrease in the rate of PA. The present investigation suggests that cellular cAMP is a regulatory molecule in the event of PA and the disruption of its homeostasis is directly correlated to xenobiotic effects on PA. It is concluded that other than divalent heavy metal cations, As\textsuperscript{3+} appears to be one of the most toxic xenobiotics to platelet function.

***************

Steady-State Transfer and Depletion Kinetics of Mercury From Amalgam Fillings.

Halbach, S; Welzl, G; Kremers, L; Willruth, H; Mehl, A; Wack, FX; Hickel, R; Greim, H.


ABSTRACT: In 29 volunteers with a low amalgam load, the number of amalgam-covered tooth surfaces and the occlusal area of the fillings were determined. Before and at select times after removal of all amalgams, concentrations of total mercury were measured by cold-vapor atomic absorption in plasma and erythrocytes as well as in urine together with the excretion rate.

Absorbed daily doses were estimated from intraoral Hg emission by two separate methods. The transfer of Hg from the fillings via the oral cavity and blood to urinary excretion was evaluated according to the most representative combination of parameters. This consisted of occlusal area (1), absorbed dose (2), Hg concentration in plasma (3) and urinary excretion (4).

Pairwise correlation coefficients were 0.49 for parameters 1 vs. 2, and 0.75 each for parameters 2 vs. 3 and 3 vs. 4. Within 9 days after removal of the fillings, a transient increase in Hg levels was observed in plasma only; in the group without a rubber dam, concentrations increased significantly above pre-removal values at days 1 and 3, whereas they decreased significantly below pre-removal values at day 30 in the rubber-dam group and at day 100 in both groups.

Excretion rates decreased significantly at day 100 in the protected group. Peak plasma-Hg was 0.6 ng/ml on average at day 1 and
decreased with halftimes of 3 and 43 days in subjects protected by a rubber dam.
The results indicated that concentrations of total mercury in plasma responded rapidly to
changes in the amalgam status and reflected the actual absorption most reliably. Notably,
plasma-Hg levels were sensitive enough to detect a transient attenuation of the additional
exposure after using a rubber dam during the removal of only a few fillings. However, being
small in magnitude and lasting 100 days at best, the rubber dam effect had minor toxicological
relevance.

*******

Augmentation of Mercury-Induced Nephrotoxicity by Endotoxin in the Mouse.
Rumbeiha, WK; Fitzgerald, SD; Braselton, WE; Roth, RA; Pestka, JJ; Kaneene, JB.
Toxicology, 151(1-3):103-16, 26 Oct 2000.
ABSTRACT: Endotoxin (lipopolysaccharide; LPS) and mercury are compounds of food
safety concern. Endotoxin is a product of cell walls of gram negative bacteria. Humans are
constantly exposed to LPS through infection plus translocation into circulation from the
gastrointestinal tract. Food is the major source of mercury in humans. [BP: According to the
scientists and WHO, dental amalgam is!]
The toxic interaction between LPS and mercury has not been well investigated. In a previous
study, we demonstrated that LPS potentiated mercury-induced nephrotoxicity in the rat.
Whether this observation was species specific was not clear. In this study we tested the
hypothesis that LPS enhances mercuric chloride (HgCl₂)-induced nephrotoxicity in mice.
In a 2x2 factorial design, mice received either Escherichia coli 0128:B12 endotoxin (2.0
mg/kg body weight) or 200 mul of 0.9%
sodium chloride (saline), and this was followed
4 h later by either mercury (1.75 mg mercuric
chloride per kg body weight) or 200 mul of
saline. Mice were monitored for 48 h.

Monitored end-points included body and renal
weights, urine volume, renal histology and
ultrastructural pathology, serum urea nitrogen
and creatinine, selected serum and urine
cytokines, and renal mercury concentrations.
Endotoxin by itself was not nephrotoxic at the
dose used in this study. Overall, mice given
LPS plus mercury were the most severely
affected. Mice given LPS and mercury also had
significantly greater renal mercury
concentration than those given mercury alone
(P<= 0.05). In conclusion, LPS potentiates
mercury-induced nephrotoxicity in the mouse.

*******

Mutagenicity of Mercury Chloride and
Mechanisms of Cellular Defense: The Role of
Metal-Binding Proteins.
Schurz, F; Sabater-Vilar, M; Fink-Gremmels, J.
ABSTRACT: The mechanisms of toxicity and,
particularly, the potential carcinogenicity of
inorganic mercury are still under debate.
Results of mutagenicity and genotoxicity
testing with mercury have been inconsistent:
mercury induces DNA single-strand breaks at
low concentrations in mammalian cells but has
not proved mutagenic in several bacterial
mutagenicity assays.
We investigated the mutagenicity of subtoxic
concentrations of inorganic mercury and the
role of metal-binding proteins and free radicals
in this process. A mutagenicity assay using
NIH 3T3 cells, transfected with a vector
containing lacZ' as a reporter for mutational
events, was applied.
In this model, inorganic mercury significantly
increased the mutation frequency in the lacZ'
gene, even at the lowest concentration tested.
The mutation frequency was greatest at an Hg²⁺
concentration of 0.5 μM.
To identify the mechanisms involved, different
cellular responses to non-cytotoxic
concentrations of HgCl₂ were measured. Hg²⁺
increased the intracellular amount of reactive oxygen species. This induction of oxidative stress was observed, although the intracellular glutathione (GSH) and metallothionein (MT) concentrations were increased significantly. Mercury-induced MT expression was even more pronounced after GSH depletion. Correspondingly, radical formation was more evident in the presence of the GSH-depleting agent L-buthioneine-[S.,R.]-sulfoximine. These findings suggest that the observed mutations might be a consequence of oxidative processes, rather than due to a direct interaction of mercury with nuclear DNA. The results also indicate that the auto-induction of MT by Hg^{2+} fails to prevent these mutational events.

******

Concentrations of Arsenic, Cadmium, Copper, Lead, Mercury, and Zinc in Human Placentas from Two Cities in Ukraine.
Zadorozhnaja, TD; Little, RE; Miller, RK; Mendel, NA; Taylor, RJ; Presley, BJ; Gladen, BC.

ABSTRACT: Ukraine is a highly industrialized country with major environmental problems and deteriorating reproductive health. Heavy metals are known reproductive toxins. A study was undertaken to determine whether they were present at sufficient concentrations to be playing a major role in these health problems. Placental concentrations of arsenic, cadmium, copper, lead, mercury and zinc were determined in 200 women from the general population of two urban areas of Ukraine, Kyiv an Dniprodzerzhinsk.
Arsenic was detected in only 5% of the samples, lead in 22%, and mercury in 28%. Cadmium was detected in almost all samples, with a median of 5.2 ng/g. Concentrations of lead, mercury, and cadmium were low compared to those reported elsewhere, while zinc and copper concentrations were comparable.

The Effect of Ethylenediamine-Tetraacetic Acid on Candida Albicans.
Sen, BH; Akdeniz, BG; Denizci, AA.

ABSTRACT: Objectives: The aim of this study was to evaluate the antifungal effect of ethylenediamine-tetraacetic acid (EDTA) on Candida albicans, comparing it with that of various disinfectants and common antifungal agents.

Study design: two clinical oral isolates and 1 standard strain of C albicans were included in this study. Main contents of the test solutions were sodium hypochlorite, EDTA, chlorhexidine, hexetidine, benzalkonium chloride, povidone-iodine, nystatin, and ketoconazole. The agar diffusion method was used to determine the antifungal effects of the solutions. Zones of inhibition were recorded and the results were analyzed statistically by using a 2-way analysis of variance.

Results: EDTA demonstrated the highest antifungal activity in comparison with routine antifungal drugs and all other solutions (P< .0001). Oral cavity isolate was more resistant to the test solutions (P< .0001).

Conclusion: The selection of irrigating and disinfecting solution in root canals of patients with a particularly high incidence of oral candidiasis gains extreme importance. EDTA may be strongly recommended during endodontic therapy of these patients.

********

Mercury Accumulation and Accelerated Progression of Carotid Atherosclerosis: A Population-Based Prospective 4-Year Follow Up Study in Men in Eastern Finland.
Salonen, JT; Seppänen, I; Lakka, TA; Salonen,
R; Kaplan, GA. Atherosclerosis, 148(2):265-73, 1 Feb 1999. ABSTRACT: Basic research and our previous studies have suggested that mercury exposure enhances lipid peroxidation and the risk of myocardial infarction, but there are no studies concerning the association between mercury accumulation and atherosclerosis. We therefore investigated whether high hair mercury content is associated with accelerated progression of carotid atherosclerosis, determined by ultrasonographic assessment of common carotid intima-media thickness (IMT), in a prospective study among 1014 men aged 42-60 years. In a linear regression model adjusting for other atherosclerotic risk factors, high hair mercury content was one of the strongest predictors of the 4-year increase in the mean IMT (P2.81 μg/g [fifths] had an IMT increase of 0.105, 0.102, 0.113, 0.017 and 0.140 mm/4 years, respectively [P= 0.041 for heterogeneity between groups]). The IMT increase was 0.034 mm/4 years (31%) greater in the highest fifth than in the other fifths (P< 0.05 for the difference). These findings suggest that mercury accumulation in the human body is associated with accelerated progression of carotid atherosclerosis.

******

Urinary Mercury Levels in Patients With Autoantibodies to U3-RNP.
Arnett, FC; Fritzler, MJ; Ahn, C; Holian, A. J Rheumatol, 27(2):405-10, Feb 2000. ABSTRACT: Objective: Autoantibodies to the U3 nucleolar ribonucleoprotein (RNP) fibrillarin occur in some patients with systemic sclerosis (SSc) or other connective tissue diseases and can be induced in certain mouse strains by injections of mercuric chloride, perhaps due to antigenic alteration of fibrillarin by mercury (Hg). Thus, potential occult exposure to Hg was explored in patients with SSc.

Methods: Urinary Hg levels were measured by cold vapor atomic absorption in 13 patients with antifibrillarin antibodies (11 with SSc), 39 SSc patients without antifibrillarin antibodies, and 32 healthy controls.
Results: Mean urinary Hg levels were significantly elevated in the antifibrillarin antibody positive patients compared to those in other patients with SSc and controls. After correction for urinary creatinine levels, mean urinary Hg levels remained significantly different than in the other 2 groups, although Hg levels in all were still within the normal or “unexposed” range. When patients and controls with low urinary creatinine levels were excluded from analysis, there were no significant differences in mean urinary Hg levels among the 3 groups.
Conclusion: These findings suggest that further epidemiological and basic research studies of mercury are warranted in patients with SSc, especially those expressing antifibrillarin antibodies.

Dental Restorative Biomaterials Induce Glutathione Depletion in Cultured Human Gingival Fibroblast: Protective Effect of N-Acetyl Cysteine.
Stanislawski, L; Soheili-Majd, E; Perianin, A; Goldberg, M. J Biomed Mater Res, 51(3):469-74, Sep 2000. ABSTRACT: Eight biomaterials eluted from four different types of dental restorative biomaterials, that is, from glass ionomer cement (GIC: Ketac-fil and Fuji II), resin-modified glass ionomer cement (RM-GIC: Fuji II LC and Photac-fil), composite (Z100 MP and Tetric- flow), and componer (Compoglass F and F-2000), were studied for their cytotoxic properties in relation to glutathione (GSH) content in cultured human gingival fibroblasts. Z100 MP, Tetric-flow, and Compoglass F were less cytotoxic than the others, with a toxic concentration of 50% (TC 50) > 24% (of eluate), as determined by MTT test. F-2300, Tetric-flow, and the other
biomaterials were relatively more cytotoxic (TC 50 = 9.16%).

With the exception of Z100 MP, all the biomaterials induced a depletion of cellular glutathione (GSH) that was variable depending upon the biomaterial eluates. The strongest GSH depletion was with F-2000, Fuji II, and Photac-fil. GSH depletion, with Compoglass and F-2000, was rapid-detectable after one h of cell treatment and complete within 3 h - whereas a longer period of incubation was required for the other biomaterials. Interestingly, the drug cytotoxic effects induced by all the biomaterials were prevented by cell treatment with the antioxidant N-acetylcysteine (NAC).

This study provides evidence that the cytotoxic property of dental restorative biomaterials is associated with depletion of the glutathione level in gingival fibroblasts. While the molecular mechanisms of this phenomenon require further investigations, our data suggest that NAC may be useful in preventing the cellular damage induced by dental restorative biomaterials.

******

A Study of Metallic Mercury Polluting a Room After Being Spilled From a Sphygmomanometer.
Ye, H; Katsumata, M; Minami, M.

ABSTRACT: Mercury spilled from a mercurial sphygmomanometer on a hot carpet can vaporize and pollute the environment. We observed the vaporization of mercury in model experiments. Mercury (0.15 g) was heated on a hot carpet and the near-by air was sampled with a midget impinger. The evaporated mercury levels were 5.0, 6.3, 8.1 and 10.0 mg/m³ at 20, 40, 60 and 80 minutes, respectively at a height of 30 cm from the carpet. The result indicated that even if a small quantity of mercury remained on the hot carpet, it could evaporate and pollute the indoor air. Little is known about the influence on human health of low mercury exposure, especially on children. In order not to pollute the air, we need to pay attention to mercury.

*******

FORUM
IAOMT 2001 MID-YEAR MEETING

SITE: Las Vegas, Nevada.
HOTEL: Tropicana Hotel; 3801 Las Vegas Blvd., S. Las Vegas, NV 89109. T: 702-739-2222 or 800-634-4000. Specify IAOMT. Room rate: $119/night Friday/Saturday, $59/night Thursday; s/d.


WELCOME RECEPTION (Cash bar): Thursday, 8 March 2001, 7:30pm.


*******

AMERICAN ACADEMY OF
HEAD, NECK & FACIAL PAIN
HOTEL: Double Tree La Posada Resort; 4949 E. Lincoln Dr., Scottsdale, AZ 85253. T: 602-952-0420. $179/night, s/d.


PROGRAM: Soft Tissue Injuries Resulting from Motor Vehicle Accidents: Biomechanics, Diagnosis and Testimony Preparation: Session I. Faculty: Wesley E. Shankland, II, DDS, PhD; Christopher R. Brown, DDS, MPS.