A Quantitative Risk Assessment Study by G. M. Richardson and associates, specifically investigating patient exposure to Bisphenol-a, has just been published [see Science section in this issue]. This study utilized established risk assessment procedures and published data. Besides its use in dental composites, BIS-GMA is also used to line food and beverage cans and is found in the atmosphere as an industrial pollutant. The study determined that human exposure to Bisphenol-a from all three sources was 65 times lower than the acceptable level (TDI) established by the Canadian government. The exposure from dental composites was 140 times lower than the Canadian standard. One must not consider this proof that dental composites are harmless to ALL patients. There
is obviously still the possibility of patients being allergic or hyper reactive to the material, especially if they have previous experience of intolerance to plastics (petrochemicals). However, this formal risk assessment is strongly indicative that dental composites do NOT present a health risk to most patients. This is in direct contrast to patient exposure to mercury from dental amalgam fillings, which has been shown to far exceed established standards in the United States and Canada.

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

**MERCURY AND LOU GEHRIG DISEASE**

Amyotrophic Lateral Sclerosis (Lou Gehrig Disease, ALS): "The etiology is unknown." "Though some patients have lived for >5 yr, the prognosis is grave and death usually occurs in 2 to 5 yr. Motor neurons degenerate in the spinal cord, medulla, and motor cortex. Atrophy occurs in the fibers of the muscles.", and "No specific treatment is known." [The Merck Manual, 12th Edition. Ed: Alpert, E; et al. Merck Sharp & Dohme Research Laboratories; Rahway, NJ, 1972.]

ALS is fatal, has no known cause and no known treatment! A reference text addresses the subject of ALS thoroughly [Kasarskis, EJ; Ehmann, WK; Markesberry, WR. Trace Metals in Human Neurogenerative Diseases. In: Essential and Toxic Trace Elements in Human Health and Disease: An Update Pg. 299-310, Wiley-Liss, Inc. 1993 (see BPNL, 9(5):2-3, Sep 1993)].

The authors state that "it has been shown that 50% of spinal motor neurons will have degenerated before the typical features of the disease are noticed. Therefore, the exposure to a harmful neurotoxin could have occurred many years preceding the clinical onset of the disease." They also point out that there is no evidence of viral involvement, and that a genetic factor may render motor neurons more susceptible to secondary insults, such as environmental toxins.

These authors further state that scientists are now focused on exposure to environmental toxins, especially lead and mercury, and that epidemiologic evidence indicates that long-term exposure to heavy metal is more common among ALS victims compared to controls. They further point out that an ALS-like syndrome has been linked to chronic intoxication with mercury and lead. These, and other, details indicate that long term exposure to dental amalgam derived mercury should receive the strongest of consideration in the investigation of ALS. It is well established that mercury can cause irreversible damage to the nervous system. It hardly seems responsible to wait until 50% of the motor neurons have been destroyed before consideration is given. Scientifically, it has been well established that mercury accumulates in motor neurons, even when the exposure is to very low doses [Cited studies abstracted in “Science” section below.] (Arvidson, B, 1992; Pamphlett & Waley, 1996). It has also been demonstrated that mercury causes oxidative damage to motor neurons (Pamphlett, R; et al, 1998). These studies are only a representation of the many published studies demonstrating these factors.

Scientists in Japan have been particularly interested in the possible connection between ALS and exposure to mercury. A statistically significant positive correlation between ALS and hair mercury has been reported (Mano, et al; 1989). A deficiency of selenium in relation to mercury exposure has also been investigated by these scientists (Mano, et al; 1990). This factor has also been emphasized by other scientists. A research team at the University of Kentucky measured levels of trace elements in the brain tissue of ALS victims (Khare, et al; 1990). They stated: "The changes observed in Hg concentration and the interactions of Hg and Se are worthy of special comment and may possibly be relevant to the pathogenesis of ALS."
As early as 1978, individual cases of mercury intoxication presenting an ALS-like syndrome had been described (Barber, TE; 1978; Adams, CR, et al; 1983). There has even been a published case of ALS reversal after amalgam removal (Redhe & Pleva; 1994). Several other of these reversals have been reported but, unfortunately, documentation is lacking. All of this does not prove, beyond a shadow of a doubt, that exposure to mercury is one of the environmental toxins that can cause ALS. It does, however, definitely provide compelling evidence for consideration.

If the subject were a pathology that was minor, ignoring this evidence might be more forgivable. ALS, however, is a condition that is considered to be always fatal, and has no known cause or treatment. Even a 1% chance of helping the victims of ALS is better than what they are currently offered! Given the state of the documented evidence and the seriousness of the condition, it is imperative that the medical community consider the possible connection of exposure to mercury, especially that from amalgam dental fillings, to ALS!

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

CALIFORNIA DENTAL BOARD
URGES WARNINGS ON MERCURY

A landmark decision was issued on Friday, 3 December 1999! Upon urging from attorney Charles G. Brown of Consumers for Dental Choice, the California Board of Dental Examiners relented on dental amalgam!

By unanimous vote, the Board advised dentists to warn patients about exposure to mercury from amalgam dental fillings!

Failure to take this stand may have placed the Board in jeopardy for violation of California’s hazardous materials law (Proposition 65). Although the Board does not have the legal authority to order dentists to comply and the law technically applies only to dental offices with 10 or more employees, some feel that all dentists may be liable under law if their patients (and staff) are not properly advised.

The Board found itself in the midst of the controversy after its president had stated during a license revocation hearing earlier this year, that amalgam-free dentistry does not fit the current practice of dentistry. The president, attempting to clarify his statement, explained that mercury-free dentistry is fine as long as the dentists offer their patients the option of mercury amalgam fillings, even if that comes from referral to other dentists. However, it was pointed out that amalgam-using dentists are not required to offer amalgam-free options, which constitutes a double standard.

The Board will inform the dentists of California in its quarterly newsletter and will also state that it does not officially favor any filling material, a position that the opposition had requested to reassure dentists who choose to use materials other than mercury amalgam. This position should give comfort not only to mercury-free dentists, but to a large segment of cosmetic dentists and dentists who feel that gold restorations are in the patients’ best interest.

A spokesperson for the California Dental Association said that silver amalgam fillings have been used for 150 years and there is no concrete research that shows there should be a ban on amalgam or that it is toxic. These weak and standard arguments are now withering under the weight of accumulating research to the contrary and public demand for informed consent.

Articles on this action appeared on Saturday, 4 December 1999 in the Los Angeles Times (Pg. A3) and in the San Diego Union-Tribune (Pg. A4). Attorneys Charles G. Brown and James S. Turner deserve accolades for their efforts in bringing about this tremendous breakthrough. All readers are urged to financially support the efforts of Consumers for Dental Choice (1424 16th Street, N.W., Suite 105, Washington, DC 20036. T: 202-462-8800; F: 202-265-6564). Your future depends on it!
SCIENCE

Preliminary Estimates of Adult Exposure to Bisphenol-A From Dental Materials, Food and Ambient Air.

Richardson, GM; Clark, KE; Williams, DR.
In: Environmental Toxicology and Risk Assessment: Standardization of Biomarkers for Endocrine Disruption and Environmental Assessment: Eighth Volume, ASTM STP 1364.
ABSTRACT: Bisphenol-a is an estrogenic compound which is a degradation product of the polymer bisphenol-a glycidyl methacrylate (BISGMA) used in dental materials. Human exposure to bisphenol-a may also arise from epoxy resins used as coatings in food and beverage cans. Bisphenol-a is an industrial pollutant and has been detected in atmospheric fallout and surface waters, which may also contribute to exposure. Therefore, a study was undertaken to estimate potential exposure from composite resin dental materials, from canned foods and beverages and from ambient air. Applying probabilistic exposure assessment methods, and using available data and information on the release of bisphenol-a from dental composites, on bisphenol-a concentrations in canned foods, canned beverages and ambient air, total adult exposure was estimated for the Canadian population.
Calculations indicated that mean total exposure from all sources was about 65 times lower than a tolerable daily intake (TDI) for bisphenol-a derived by Health Canada to address potential estrogenic effects of this compound. Maximum exposure was also less than this TDI.
On average, composite resin dental materials and canned foods contributed approximately equally to total exposure whereas canned beer, canned soft drinks and ambient air were relatively insignificant sources of exposure.

Mercury Intoxication Simulating Amyotrophic Lateral Sclerosis.

Adams, CR; Ziegler, DK; Lin, JT.
ABSTRACT: A 54-year old man had a syndrome resembling amyotrophic lateral sclerosis after a brief but intense exposure to elemental mercury. The syndrome resolved as his urinary mercury levels fell.
Mercury toxicity must be considered not only in individuals with recent anterior horn cell dysfunction but also with otherwise unexplained peripheral neuropathy, tremor, ataxia, and a gamut of psychiatric symptoms including confusion and depression.

Inorganic Mercury is Transported From Muscular Nerve Terminals to Spinal and Brainstem Motoneurons.

Arvidson, B.
ABSTRACT: The distribution of mercury within the brainstem and spinal cord of mice was investigated with the autometallographic technique after intramuscular administration of a single dose of mercuric mercury (HgCl2). Deposits of mercury were localized to motor neurons of the spinal cord and to brainstem motor nuclei; i.e., neurons with their peripheral projections outside the blood-brain barrier. Unilateral ligation of the hypoglossal nerve prior to the injection of HgCl2 prevented the accumulation of mercury deposits in the ipsilateral hypoglossal nucleus.
The selective accumulation of mercury in spinal and brainstem motoneurons is most probably due to a leakage of metal-protein complexes from capillaries in muscle into myoneural junctions, followed by uptake into nerve terminals and retrograde axonal transport. The possible link between this process and the development of motor neuron degeneration in ALS is discussed.
Inorganic Mercury Intoxication Reminiscent of Amyotrophic Lateral Sclerosis.
Barber, TE.
ABSTRACT: Two employees in a mercuric oxide manufacturing plant developed neurologic changes not previously reported from the exposure to inorganic mercury or elemental mercury vapor. The symptoms, physical findings and laboratory studies resembled those found in amyotrophic lateral sclerosis (ALS) and organic mercury intoxication.
Nineteen employees are reported who precipitously developed signs and symptoms which may be regarded to be the early onset of a symptom complex of mercury intoxication that would likely have progressed to the ALS-like syndrome if the progression had not been interrupted by removal of the individuals from exposure to mercury. All symptoms, signs, and laboratory findings returned completely to normal after approximately three months in a mercury free work environment.

Trace Element Imbalances in Amyotrophic Lateral Sclerosis.
Khare, SS; Ehmann, WD; Kasarskis, EJ; Markesbery, WR.
ABSTRACT: Concentrations of 15 elements were determined by instrumental neutron activation analysis in brain, spinal cord, blood cells, serum and nails of Amyotrophic Lateral Sclerosis (ALS) patients and appropriately matched control subjects.
Several significant imbalances were detected in trace element levels in ALS samples compared to control samples. Some of these changes are probably secondary to the loss of tissue mass, especially in spinal cord. However, the widespread changes observed in Hg and Se levels in ALS tissues deserve special attention. The significance of these alterations in trace element levels in relation to the pathogenesis of ALS is discussed.

Mercury in Hair of Patients With ALS.
Mano, Y; Takayanagi, T; Ishitani, A; Hirota, T.
ABSTRACT: In middle of Kii peninsula, one of the biggest mercury mines in Japan had been present until about 10 years ago. The mercury contents in water and fish are reported to be higher in this district. So we investigated the mercury in hair of patients and normal controls. In this study the subjects are 23 cases of ALS including 15 cases in Nara and Mie and 8 cases in other prefectures except in Kii peninsula, 14 cases with ataxia, 11 cases with other degenerative diseases like Parkinson’s disease and Alzheimer’s disease, 25 cases of cerebrovascular disease as compared to 26 normal controls.
The hair are taken from 3 areas on head of patients and normal controls. They are washed in 2% sodium lauryl sulfate and stirred in distilled water several times, and they are soaked in acetone and dried in filter paper. They are inserted in fire and vaporized mercury are measured (Zeeman Effect Mercury Analyzer) in ppm.
The hair mercury concentration is 2.81 ppm in ALS in total, 3.62 ppm in ALS in Nara and Mie and 1.39 ppm in outside of Kii Peninsula, 2.34 ppm in ataxia, 1.83 ppm in other degenerative diseases, 1.66 ppm in cerebrovascular disease and 1.44 ppm in normal controls. Statistically it is significant (p <0.05) between that in ALS in Nara and Mie and that in normal controls. 6 cases (40%) with ALS in Nara and Mie have the value above the mean +2 standard deviation of controls.

Amyotrophic Lateral Sclerosis and Mercury: Preliminary Report.
Mano, Y; Takayanagi, T; Abe, T; Takizawa, Y.
ABSTRACT: The mercury and selenium content in the hair of 13 ALS cases was studied by neutron activation analysis. The total mercury content of the hair was 3.70 +/- 2.73 ppm (mean +/- standard deviation) in the ALS patients as a whole, 4.46 +/- 3.16 ppm in the ALS patients from the middle of Kii Peninsula, and 2.49 +/- 1.38 in the ALS patients from other regions. As the comparison, mercury content was 2.43 +/- 0.79 in the patients with Parkinsonism, and 2.10 +/- 1.13 ppm in the patients with multiple sclerosis (MS).

The selenium content of the hair was 0.36 +/- 0.35 ppm for all ALS patients as a whole, 0.45 +/- 0.25 ppm in the ALS patients from the middle of the Kii Peninsula, and 0.21 +/- 0.47 ppm in the ALS from other regions. It is well known that the selenium decreases the toxicity of mercury in the human body. From these data mercury with low content of selenium might be one of the environmental factors which are thought to be involved in producing of ALS.

********

Motor Neuron Uptake of Low Dose Inorganic Mercury.
Pamphlett, R; Waley, P.
ABSTRACT: In animals, inorganic mercury can bypass the blood brain barrier and enter motor neurons. We sought to determine the lowest injected dose of mercury that could be detected in mouse motor neurons. Mice were injected intraperitoneally with mercuric chloride in doses from 0.05 mcg/g body weight and studied between 5 days and 18 months after injection. After formalin fixation, 7 micrometer sections of cerebrum, cerebellum, brain stem, spinal cord and kidney were stained with silver nitrate autometallography.

Five days after injection, mercury granules were detected at doses from 0.2 mcg/g upwards in the cell bodies of spinal and brain stem motor neurons, more granules were also seen in 5% of posterior root ganglion neurons. At doses from 0.05 mcg/g upwards mercury was detected 5 days later in renal tubule cells. Mercury was still present in motor neurons 6-11 months after injection, but by this time mercury had been cleared from kidneys.

Low doses of inorganic mercury are therefore selectively taken up and retained by motor neurons, making this neurotoxin a good candidate for cause of sporadic motor neuron disease.

********

Oxidative Damage to Nucleic Acids in Motor Neurons Containing Mercury.
Pamphlett, R; Slater, M; Thomas, S.
ABSTRACT: Heavy metals have been implicated in the pathogenesis of sporadic motor neuron disease (MND). We were interested to see if inorganic mercury leads to oxidative damage in motor neurons since free radicals have been suspected to be involved in MND, so a method to examine oxidatively damaged DNA in situ was used to examine individual motor neurons. Mice were exposed to 500 mcg/m3 of mercury vapor for 2h. Two, five, or ten days later sections from formalin fixed, paraffin embedded blocks of cervical spinal cord were incubated in avidin-FITC. Sections were examined under a fluorescence microscope and photographs of pairs of mercury exposed and control spinal motor neurons were analyzed semi-quantitatively for the amount of fluorescence using an image analysis program. Avidin fluorescence was seen in the perikaryon of both control and mercury exposed motor neurons. In each control-mercury pair (four pairs per group) significantly more perikaryal fluorescence was seen in the mercury-containing than in the control motor neurons (Mann Whitney testing). Mercury with the motor neuron perikaryon therefore leads to increased avidin binding, an indicator of oxidative damage to DNA.

The findings support the hypothesis that an
environmental toxin such as mercury can enter and damage motor neurons.

********

Recovery from Amyotrophic Lateral Sclerosis and from Allergy after Removal of Dental Amalgam Fillings.
Redhe, O; Pleva, J.
ABSTRACT: In the debate about the possible risks with mercury released from dental amalgam fillings (DA), an important point is whether DA can cause serious, pernicious diseases. The following two patient cases are a contribution to this debate. They are translated from the Swedish original [truncated to the one ALS case; see BPNL, 10(3):6-7, May 1994 for full abstract], which contains an evaluation of 100 cases of poisoning and immunological effects in dental amalgam patients, documented in clinical practice, including recovery from most symptoms after amalgam removal.
The patient had suffered for a long period from neurological problems. In 1984, following a complete neurologic evaluation, a diagnosis of amyotrophic lateral sclerosis (ALS) was made at the department of neurology of the University Hospital in Umea, Sweden. No further visit to the clinic was proposed, as the disease is pernicious and there is no known therapy for ALS.
From his prior experience, the dentist recognized the symptoms as those familiar in the patient group with health problems attributable to dental amalgam fillings. Patient history revealed the onset or exacerbation of neurologic symptoms following placement of amalgam dental fillings. The patient had 34 tooth surfaces filled with amalgam, most of which were shallow and of moderate extent. There were no fillings of materials other than amalgam and no teeth had root canal therapy. Oral hygiene was exemplary and periodontal status was unremarkable. There were minor occlusal discrepancies.
With the consent of the patient, all DA fillings were removed and replaced with alternative material. Treatment was completed in March 1984. Removal of the DA in the first tooth that had originally given post-operative problems resulted in an exacerbation of symptoms, with a continued recurrence of exacerbation following each subsequent replacement.
Following the replacement of the last DA, the patient’s entire condition rapidly improved. Six weeks following the replacement, the patient was able to go up stairs without experiencing back pain. Pains in the mouth also receded and the sore throat, present during the whole history of the disorder, recovered. Five months after completion of the DA removal, the patient returned to the same University Hospital at Umea for a week-long follow-up investigation, after which the following notation was placed in her record: “The neurologic status is completely without comment. Hence, the patient does not show any motor neuron disease of type ALS. She has been informed that she is in neurological respect fully healthy.”
The EMG report further noted: “In comparison to the previous investigation the visible changes are considerably smaller. Now, only a medium denervation of short toe extensors can be seen. The conductivity is normal. An explanation of the syndrome was probably mercury angiopath in the spinal cord.”
At this time, nine years have elapsed since removal of the DA fillings, and the patient continues to enjoy good health. This case is supportive of experimental findings of adverse effects of mercury exposure on motor neuron function.

********

Chronic Low-Dose Maternal Exposure to Methymercury Enhances Epileptogenicity in Developing Rats.
Szasz, A; Barna, B; Szupera, Z; De Visscher, G; Galbacz, Z; Kirsch-Volders, M; Szente, M.
ABSTRACT: Effects of continuous low-dose
maternal methylmercury intoxication on the induction and propagation of ictal epileptiform activity induced by 3-aminopyridine, were investigated on the neocortex of 4-weeks-old offspring rats. Epileptogenicity was significantly increased in offspring of mercury-treated animals compared to those of controls, characterized by more frequent occurrence of periodic ictal activity, a facilitated propagation of epileptiform discharges and a strong tendency to generalization. The latency of first ictal event was slightly shorter and the average duration of individual ictal periods slightly longer in treated animals. However, the amplitude of seizure discharges was significantly smaller in treated animals than in controls.

We conclude, that the synaptic and membrane mechanisms responsible for initiation and propagation of paroxysmal activity were probably facilitated, while the efficacy of cortical inhibition, in preventing initiation and spread of epileptiform discharges was reduced by mercury treatment in the developing nervous system. The smaller amplitude of paroxysmal discharges could be a sign of a remarkable loss of cortical neurons.

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

FORUM

IAOMT 2000 MID-YEAR MEETING
DATE: Friday-Saturday, 31 March-1 April, 2000.
SITE: Orlando, Florida.
HOTEL: Sheraton World Resort Orlando; 10100 International Drive, Orlando, FL 32821-8095 (adjacent to Sea World). T: (407) 352-1100, (800) 341-4292 [inside Florida], (800) 327-0363 [outside Florida]; F: (407) 352-3679. Specify ORAL MED room rate: $119.00/night/1-2; $134.00/night/3-4. Suites $295-$495. [Book early or rent car!] 15 minutes from airport; transportation available to nearby attractions.
MEETING REGISTRATION: IAOMT, P.O. Box 608531, Orlando, FL. 32860-8531. T: (407) 298-2450; F: (407) 298-3075. email: mziff@iaomt.org. Members: $375; non-members: $475; additional persons: $125 ea. [Registration includes light breakfast and lunch on Friday and Saturday, if registered prior to 03/28/2000. Cancellation Fee of 15% after 03/15/2000.]
MEETING HOST: Dr. Janet Stopka.
WELCOME NO-HOST RECEPTION: Thursday, 30 March 2000, 7:30pm.
PROGRAM: Friday morning Clinical Theme: “Root Canals: Yes or No?”
Michael F. Ziff, DDS: The Science on Endodontics.
J. C. Pendergrass, PhD: ALT Findings on Root Canals.
Root Canal Therapy Panel: IAOMT Committee; Chair Stephen M. Koral, DMD.
Friday Afternoon Speakers:
IAOMT Clinical Practice Committee: “Tips for the Biological Practice.”
Thomas E. Baldwin, DDS: “Biological Periodontal Therapy: Update.”
Saturday Speakers:
David C. Kennedy, DDS: “Dental and Skeletal Effects of Fluoride.”
Boyd E. Haley, PhD: “A Review of Mercury and Alzheimer’s Disease.”
Louis W. Chang, PhD: “The Toxic Effects of Mercury.”
James E. Hardy, DMD: “Bio-Electromagnetism and Dental Metals.”

********

American Academy of Head, Neck and Facial Pain

7th Annual Mid-Winter Symposium
SITE: Scottsdale, Arizona.
HOTEL: The Radisson Resort. T: 480-991-3800. $185/night.
PROGRAM: Yoshiaki Omura, MD: “Diagnosis of Acute and Chronic Disease with a Cost Effective and accurate Treatment Protocol.”