AUTISM: IS MERCURY A FACTOR?
A growing number of parents of autistic children think that it is! This was the topic of the hearing conducted by the Government Reform Committee of the U.S. House of Representatives on 18 July 2000. The hearing was entitled "Mercury in Medicine - Are We Taking Unnecessary Risks?" Bio-Probe, Inc. has obtained transcripts of some of the testimony presented to the committee [www.house.gov/reform/hearings/healthcare]. The thrust of the committee was on the use of mercury (thimerosal) in vaccines. Testimony was provided by parents of autistic children, physicians and scientists. The information presented was overwhelming! One physician provided documentation on her treatment of autistic children with mercury chelators. The treatment resulted in a marked improvement in the symptoms of autism and a large provoked increase in urine mercury. Other witnesses provided documentation on the appearance of autism following vaccinations containing thimerosal. Others detailed the amount of mercury (thimerosal) received from vaccinations, which far exceeded existing mercury exposure standards (for adults). The USEPA standard is 0.1 micrograms per kilogram of body weight per day. This equates to 7.0 micrograms for the 70 kilogram (154 pound) adult.

It was detailed that children could receive as
much as 237.5 micrograms of mercury from vaccines, in doses of up to 25.0 micrograms. Thus, administration of a single vaccine to a child would greatly exceed the USEPA adult standard.

It was also pointed out that prior to 1970 the prevalence of autism was 1 in 2000; in 1970 it was 1 in 1000; in 1996 the NIH estimated it to be 1 in 500; in 2000, the prevalence of autism is now estimated as 1 in 150. Parallels were drawn between vaccine thimerosal and autism. Evaluation of the amount of mercury eliminated in the urine from a series of administrations of a mercury chelator is intriguing. A significant portion of the known dose of vaccine mercury was eliminated. With the knowledge that mercury is very difficult to eliminate from the body, especially from the central nervous system, this would not be expected. This information suggests a pre-existing body burden of mercury prior to vaccination.

The fact that mercury from amalgam dental fillings transfers through the placenta to the body tissues of unborn babies and to newborns through mothers' milk is well established by published human and animal studies. [see BPNL, 14(4), July 1998]

An introduction to the subject of dental mercury was heard by the Committee on Government Reform. A brief presentation was given by H. Vasken Aposhian, Ph.D. of the Univ.of Arizona, who presented documentation that dental amalgam is the largest contributor of mercury to the body burden of non-occupationally exposed individuals.

There are several organizations dedicated to the investigation of autism. These include Cure Autism Now (CAN), Defeat Autism Now (DAN), and the Autism Research Institute (ARI). All of these seem convinced that mercury is a primary cause, if not the primary cause of autism.

ARI [www.autism.com/ari/mercurylong] has provided a 70 page document entitled "Autism: A Unique Type of Mercury Poisoning." This document details the correlations between autism and mercury poisoning, symptom by symptom. Twenty-two of these pages are published scientific references, establishing the connection between mercury and autism. The Synopsis states: "The parallels between the two diseases are so thorough as to suggest, based on total Hg injected into U.S. children, that many cases of autism are a form of mercury poisoning."

In the section Diagnosing Mercury Poisoning in Autism, the document states:

1. Observation of impairments in many but not all of the following domains: (a) movement / motor disorder, (b) sensory abnormalities, (c) psychological and behavioral disturbances, (d) neurological and cognitive deficits, (e) impairments in language, hearing, and vision, and (f) miscellaneous physical presentations such as rashes or unusual reflexes.

2. Known exposure to Hg (a) at a level that has been documented as causing impairment in similar individuals under similar circumstances, and (b) at approximately the same time as the symptoms emerge, with allowances given for the latency period. It should be noted that the dose which is considered "toxic" vs. "safe" is unresolved among toxicologists; some researchers feel that any amount of exposure is "unsafe."

3. Detectable levels of mercury in urine, blood, or hair. Importantly, because mercury can clear from biologic samples before the patient feels symptoms or is tested, the lack of detectable mercury is not cause for ruling out mercury poisoning; and conversely, detectable levels have been observed in unaffected individuals.

4. Improvement in symptoms after chelation. While many patients' symptoms resolve with chelation, some clearly poisoned individuals do
not improve. Other exposed subjects have also been known to improve without intervention. This final point is interesting. Already, positive results from mercury chelation for the treatment of autism have been found. All autism groups agree that a genetic factor is also involved, as it is with most pathologic conditions. Moreover, it is possible that a pre-existing body burden of mercury (as from the mercury fillings of mothers) could predispose a child to adverse effects from vaccine mercury.

Given the severity of the affliction of autism, the impact on human life, the increased prevalence exhibited, and the success of mercury chelation in treatment, a full scale investigation of a mercury/autism connection is absolutely imperative. No effort should be spared.

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OKLAHOMA DENTAL ASSOCIATION TOUTS POSTERIOR COMPOSITES!
The Oklahoma Dental Association (ODA) placed an insert advertisement in the 18 September 2000 edition of Newsweek.
The ad headlined “No More Visible Fillings” and “You Have To Take Care of Things If You Want Them To Last.” It concluded with: “For the look that will last. ODA - Oklahoma Dental Association. This message brought to you in partnership with the ADA - American Dental Association.”

Some of the ODA statements are fascinating. For example: “Dentistry now has the technology and procedures to improve the looks of your teeth, while helping you maintain your good oral health. Ask your dentist about the benefits of white fillings. [The bold emphasis was theirs.] Although they are often used on the front teeth where a natural appearance is important, they may also be used on back teeth in areas where the filling may be visible. This procedure preserves the beauty of your smile and restores the health and soundness of the tooth.”

What is this?? A state dental association publicly advocating the use of posterior composites, and stating that they will “restore the health and soundness of the tooth.” This is in specific reference to “back” teeth.

What happened to charges against mercury-free dentists for placing an “inferior” material in posterior teeth, thereby violating standards of care? Now that a state association, in partnership with the ADA, publicly advocates placing composites in posterior teeth, do we now have a new standard of care?

This new policy should be very helpful in confrontations with state dental boards.

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ORTHODONTICS WITHOUT METAL
[Associated Press, 20 September 2000.]
A California company (Align Technology Inc.) has developed a 3-D computer-imaging system to provide orthodontic treatment without braces.
The system - called “Invisalign” - maps out a treatment plan with 3-D computer images. Technicians then prepare a series of clear, removable retainer-like “aligners” that move teeth. The company claims that the orthodontic treatment is with few hassles, little pain and no obtrusive wires or brackets. In most cases, the treatment is completed with 20 removable aligners for 2-3 week periods (ca. 50 weeks).

Not unexpectedly, the article stated that “orthodontists are skeptical of the procedure.” One quoted orthodontist stated that “there is also potential for abuse here” and other orthodontists are concerned about relinquishing so much control over treatment. The success of the treatment depends a great deal on the patient’s dedication to wearing the aligners, and since the reliance is heavily on computers and lab work, in-office visits are less important. These drawbacks notwithstanding, this new approach to orthodontic therapy is interesting.
Although not mentioned in the article, the elimination of nickel-containing orthodontic wires will be viewed as important to some dentists and patients, especially if other metals are present in the patient’s mouth. The procedure brings to mind the Krozat approach to orthodontic therapy which, of course, has encountered strong opposition from traditional orthodontists and from dental boards. However, Align Technology Inc. apparently has very strong commercial and financial backing. The company president, the daughter of a former U.S. Senator, stated: “Orthodontics has been in the horse-and-buggy age for a long time now. We are the industry’s automobile.” They may prove to be much stronger opponents than a handful of alternative dentists. This may be interesting! [Align Technology, Inc. 442 Potrero Ave., Sunnyvale, CA 94085. 800-468-4742.]

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NEW LOCAL ANESTHETIC NO ANILINEs!

In 1997, we reported that all local anesthetics approved for use in the United States broke down into anilines in the body. [BPNL, 13(6):2-3, Nov 1997] This has been confirmed by animal and human studies, and even confirmed by the FDA itself in 1993. In tissue studies, the FDA determined that 67% of lidocaine converts to aniline. Anilines are commonly known as “coal tar derivatives” and have been found to be cancer causing dating back to the 19th Century. Anilines are the main carcinogens found in tobacco. An injection of only 1 cc of 2% lidocaine provides a dose of aniline equal to smoking 84,000 cigarettes.

The problem in eliminating the use of these local anesthetics has been the lack of a suitable alternative. Now, this is no longer true. A local anesthetic that does not break down into anilines is now available. [Septocaine (articaine HCl 4%); Distributed by: Septodont, Inc., 245 Quigley Blvd., Suite C, New Castle, DE 19720.]

Rather than believe only the claim of the manufacturer, Bio-Probe checked the formula with a prominent biochemist. We were told that the chemical formula does not even resemble anilines, nor could it possibly break down into anilines.

This is certainly good news for biological dentists. Although the product might be more expensive than other local anesthetics, at least there is no concern for the “aniline” factor. As with any other amide-type local anesthetics, there is still a toxicity potential with overdose.

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SCIENCE

Attention Deficit Hyperactivity Disorder, Infantile Autism, and Elevated Blood-Lead: A Possible Relationship.


ABSTRACT: This case involves a 4 ½ year old boy diagnosed with autism, attention deficit hyperactivity disorder (ADHD), and an elevated blood-lead with the chelating agent succimer. The parents reported a decrease in repetitive behaviors while on succimer with a regression to previous symptoms when medication was discontinued. Also seen was a decrease of hyperactive behavior while being treated with succimer.

This article explores the interaction and possible causal relationship of an elevated blood-lead, autism, and ADHD as well as treatment of the behavioral symptoms.

Bio-Probe Comment: Succimer (DMSA) is also well known as a chelator of mercury.

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Disturbing Behavior: Neurotoxic Effects in Children.

May, M.


ABSTRACT: An epidemic of neurobehavioral
problems is sweeping through children today. According to In Harm's Way: Toxic Threats to Child Development, a May 2000 report published by the Greater Boston Physicians for Social Responsibility, 12 million American children suffer from learning, developmental, or behavioral disabilities. Specifically, these disabilities may include attention-deficit/hyperactivity disorder (ADHD), autism, learning disabilities, mental retardation, and other neurobehavioral problems. And the prevalence of these disabilities may be increasing.

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The Environment as an Etiologic Factor in Autism: A New Direction for Research.
London, EA.
ABSTRACT: Autism is one of a group of developmental disorders that have devastating lifelong effects on its victims. Despite the severity of the disease and the fact that it is relatively common (15 in 10,000), there is still little understanding of its etiology. Although believed to be highly genetic, no abnormal genes have been found.
Recent findings in autism and in related disorders point to the possibility that the disease is caused by a gene-environment interaction. Epidemiologic studies indicate that the number of cases of autism is increasing dramatically each year. It is not clear whether this is due to a real increase in the disease or whether this is an artifact of ascertainment.
A new theory regarding the etiology of autism suggests that it may be a disease of very early fetal development (approximately day 20-24 of gestation). This theory has initiated new lines of investigation into developmental genes. Environmental exposures during pregnancy could cause or contribute to autism based on the neurobiology of these genes.

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The Developing Brain and the Environment: An Introduction.
Weiss, B; Landrigan, PJ.
ABSTRACT (Excerpts): We have come to understand that chemicals in the environment can cause a wide range of developmental disabilities in children, and that anatomic malformations are only the most obvious. Current concerns especially focus on the concept that certain chemicals can cause clinical and subclinical deficits in neurobehavioral development through injury to the fetal brain. The implications of small shifts in intelligence quotient score and a slightly increased tendency to aggression are not so easily conveyed or grasped as a picture of deformed limbs. However, recognition of the importance of such changes is gathering momentum and is documented in this monograph.
A prime motivating force is the realization that we know the causes of fewer than 25% of neurodevelopmental disabilities. These disabilities, including dyslexia, attention deficit hyperactivity disorder (ADHD), intellectual retardation, and autism, affect an estimated 3-8% of the 4 million babies born each year in the United States.
For most neurodevelopmental disabilities, the cause remains unknown. A diverse assortment of toxic chemicals in the environment is capable of causing neurodevelopmental disabilities. Organic mercury compounds are among the most potent developmental neurotoxicants. In the words of pediatrician Herbert L. Needleman: "We are conducting a vast toxicologic experiment in our society, in which our children and our children's children are the experimental subjects."
The American Academy of Pediatrics has just published its Handbook of Pediatric
Environmental Health, the “Green Book,” which is available to pediatricians throughout the Americas. Children’s environmental health has climbed to a critical position as we launch the new millennium. This monograph marks a significant milestone in the evolution of this emerging discipline.

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Mobilization of Mercury by DMPS in Occupationally Exposed Workers and in Model Experiments on Rats: Evaluation of Body Burden.


ABSTRACT: The aim of the study was to evaluate the efficacy of DMPS (sodium-2,3-dimercapto-1-propane sulfonate) (Dimaval) administration for mobilizing mercury from the body in occupationally exposed people and experimental animals.

Two doses of DMPS were administered at a 24-h interval to: (a) groups of people occupationally exposed to mercury - workers of the chloralkali industry (n= 43), and dentists (n= 12), (b) non-exposed individuals (n= 20), and (c) rats chronically exposed to mercury vapour at the concentration of 0.8 mg/m³ Hg vapour (6 h/day, 5 days/week) for 15 weeks.

In an out-patient mobilizing test, the urinary excretion of mercury 48 h after the administration of the first dose reached 1513 micrograms in the group of industrial workers, 132.6 micrograms in dentists, and 3.78 micrograms in controls.

In rats, two consecutive doses of DMPS decreased kidney content of mercury by about 30% and 50% after oral and intraperitoneal administration, respectively. Kidney mercury burden was calculated on the basis of the data from animal and human studies of the mobilization of mercury via urine after DMPS treatment: 61, 2800 and 28,000 ng/g in controls, dentists and workers respectively.

It was estimated that two doses of DMPS mobilized 17-20% (after oral administration) and 25-30% (after intramuscular administration) of kidney mercury burden, both in the control and exposed subjects.

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Skeletal Muscle Abnormalities Associated With Occupational Exposure to Mercury Vapours.


ABSTRACT: There is scarce information on the possible effects of chronic exposure to mercury on skeletal muscle. Dental personnel are frequently exposed to inhalation of metallic mercury vapours.

The skeletal muscle of five technicians and one dentist (females, age 36-55) was studied. All of them presented symptoms of chronic mercury poisoning. Needle biopsy was taken from the quadriceps femoris muscle and samples were prepared for light microscope histochemistry and for transmission electron microscopy.

Selective atrophy of type IIB muscle fibres was found in patients, and in one of them there was fibre grouping. Most of the muscles showed increased fibre area per capillary. Atrophy was confirmed by the ultrastructural study, demonstrating increase of intermyofibrillar spaces, loss of myofibrils or complete disappearance in some fibres, and sarcolemmal foldings. Splitting of the fibres was also found. Some capillaries were altered, showing endothelial infoldings into the lumen, thickened basement membrane and partial or total occlusion.

The alterations found in muscle may be secondary to nerve damage, to ischemia caused by capillary lesion and/or to a direct effect of mercury on muscle fibre proteins.
Bio-Probe Comment: These findings have presented dramatic results heretofore not addressed. The effect of mercury on the cardiovascular muscle must now be considered and investigated.

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Mercuric Chloride Damages Cellular DNA by a Non-apoptotic Mechanism.
Ben-Ozer, EY; Rosenspire, AJ; McCabe, MJ; Worth, RG; Kindzelskii, A; Warra, N; Petty, H. Mutat Research, 470(1):19-27, 10 Oct 2000.

ABSTRACT: Mercury is a xenobiotic metal that is well known to adversely affect the immune system, however, little is known as to the molecular mechanism. Recently, it has been suggested that mercury may induce immune dysfunction by triggering apoptosis in immune cells. Here, we studied the effects of Hg\(^{2+}\) (HgCl\(_2\)) on U-937 cells, a human cell line with monocytic characteristics. We found that these cells continued to proliferate when exposed to low doses of mercury between 1 and 5 microM. Using the single cell gel electrophoresis (SCGE) or ‘comet’ assay, we found that mercury damaged DNA at these levels. Between 1 and 50 microM Hg\(^{2+}\), comet formation was concentration-dependent with the greatest number of comets formed at 5 microM mercury.

However, the appearance of mercury-induced comets was qualitatively different from those of control cells treated with anti-fas antibody, suggesting that although mercury might damage DNA, apoptosis was not involved. This was confirmed by the finding that cells treated with 5 microM mercury were negative for annexin-V binding, an independent assay for apoptosis. These data support the notion that DNA damage in surviving cells is a more sensitive indicator of environmental insult than is apoptosis, and suggests that low concentrations of ionic mercury may be mutagenic.

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Amyotrophic Lateral Sclerosis After Accidental Injection of Mercury.
Schwarz, S; Husstedt, IW; Bertram, HP; Kuchelmeister, K. J Neurol Neurosurg Psychiatry. 60(6):698, Jun 1996.

LETTER TO EDITOR (References excluded): Mercury has been widely discussed in the pathogenesis of amyotrophic lateral sclerosis. We describe a case of amyotrophic lateral sclerosis after accidental injection of mercury. While shaking a mercury thermometer, a female nurse plunged it into her left hand. Elemental mercury infiltrated into the soft tissues of her palm. The diffusely distributed mercury particles could not be removed completely by surgical means. Two years later, blood mercury concentrations, analyzed with atomic absorption spectroscopy, were raised (15 microg/l, reference < 5 microg/l), but declined soon afterwards to normal values.

Three and a half years after mercury infiltration, the 38 year old woman was admitted with progressive weakness of the legs which had begun a few weeks before. She had moderate weakness of the musculature - most pronounced in the lower limbs - slight cerebellar ataxia, and fasciculations in the thighs. Deep tendon reflexes were hyperactive at all her limbs. Babinski’s sign was positive on the left. Cranial nerves and sensation were normal. Electromyography showed pathological spontaneous activity in muscles of both legs. Gastrocnemius muscle biopsy suggested neurogenic muscle atrophy. Mercury and lead concentrations in blood, urine, and hair were not raised. Medical history was unremarkable.

With a presumptive diagnosis of amyotrophic lateral sclerosis, we performed a chelation treatment with dimercaptosuccinic acid. Urinary mercury excretion increased (peak
value 41.6 microg/24 h, reference < 10 microg/24 h) without clinical improvement. Muscular atrophy, cramps, and fasciculations become prominent in all limbs. Six months later, a severe bulbar syndrome developed. Four years after the onset of neurological symptoms, the patient is tetraplegic. Communication is possible via eye movements. There is no respiratory insufficiency, sensory deficits, or dementia.

Mercury accumulates within the CNS, entering either by crossing the blood-brain barrier or via retrograde axonal transport. In our patient, mercury from the deposit in the hand could have slowly accumulated in the CNS over time. This would explain the dissociation between exposure to mercury and onset of clinical symptoms. Initial trauma and repeated surgical interventions may have enabled retrograde axonal transport.

Chelation treatment, as in this patient, has never been successful in amyotrophic lateral sclerosis. Such treatment - to be of any use - should be initiated shortly after exposure to mercury to prevent an accumulation and irreversible damage within the CNS.

In previous reports of motor neuron diseases after mercury incorporation, clinical symptoms developed shortly after the intake of large amounts of mercury and included typical signs of mercury intoxication. Our patient illustrates the possibility that, if present over a longer period of time, relatively small amounts of mercury may cause sporadic amyotrophic lateral sclerosis without other signs of mercury intoxication.

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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, $59/night Thursday (US); s/d.
MEETING REGISTRATION: IAOMT, P.O. Box 608531, Orlando, FL 32860-8531. T: (407) 298-2450; F: (407) 298-3075. email: mziff@iaomt.org. Registration (US$): Members: $445, non-members: $545; additional persons with registrant: $135. Includes continental breakfast and lunch on Friday and Saturday.
WELCOME RECEPTION (Cash bar): Thursday, 8 March 2001, 7:30pm.

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