The Gulf War Illness and Sheepherders' Disease; Acetylcholine Deficiency Disorders?

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OUTLINE

BACKGROUND

The Rand Corporation issued a report on Gulf War Illness in 1999; it was funded by the Department of Defense and made no mention of vaccines or petrochemicals. The role of vaccines in suppressing cell-mediated immunity and causing infection have been well-documented by several authors (see below), but have been covered up by US Defense Department and British Ministry of Defense. This follows papers in the Lancet, 16 January 1997? On the Gulf War Illnesses by Catherine Unwin and colleagues (1) and Khalifda Ismail and colleagues that concluded that "there is no unique Gulf War syndrome"; such great oversimplification is completely unwarranted; at least 3 Gulf War illnesses exist, either alone or in combination.

The first of these syndromes (approx. 80%) is due to the marked depression of cell-mediated immunity by more than 3 vaccines given simultaneously and many soldiers received 17 simultaneously (3); many of these were hurriedly and poorly attenuated, and hence potent infectious agents, but with a prolonged latent period.

In my many inquires of patients and their physicians over 4 years (1993-96) until at least 12 months ago, neither the many Dept. of Defense, Veteran's Administration or physicians in private practice asked the patients about vaccines (a "hush" topic); nor were any relevant immune function tests performed or any test of cell mediated immunity(3). I called these to the attention of the American Department of Defense on many occasions (reply to first letter enclosed) and the British Ministry of Defense in 1996. I have published that tests of cell mediated immunity have a characteristic profile in 80 per cent of those so diagnosed, and this pattern is restricted to vaccine recipients not exposed to petrochemicals or organophosphate insecticides(3).

About 20-25% of all U.S. troops and of British and Canadian troops in the area, developed symptoms. However, none of the French, Algerian, or Moroccan troops, all of whom refused the vaccines, developed symptoms of this illness. Some of the troops from the Czech Republic and Slovakian Republic who received these vaccines developed identical symptoms. However, the incidences of those receiving the vaccines in Slovakia and the Czech Republic and those developing the highly infectious Gulf War syndrome with its characteristic symptoms are unknown.

Some of my patients who received the 17 vaccines, many poorly if at all attenuated, developed full blown illness even though the war ended 2-3 days before they were to be sent over, and they remained on U.S. soil for the whole duration of the conflict, never setting foot in the Gulf(3).

The role of the vaccines is complicated by the pyridostigmine administration, the latter caused severe vomiting and diarrhea for as long as it was taken, and most of the American troops date the onset of illness from the day after they began taking "the pill".

Unless and until they are treated successfully (with immunotherapy), these veterans do not remember that there was an interval of 6-18 months before other symptoms appeared, including in some, a blindness of about 15 months duration, for which ophthalmologists could find no cause, a finding suggestive of anthrax.

The second of the Gulf War syndromes is apparently associated with, and presumably due to, ORGANOPHOSPHATE TOXICITY. Seventy-seven per cent of British Troops refused the vaccines (4). But the British troops were housed in tents painted with organophosphates to protect against mites and other insects (5). In addition to the symptoms of the 1st group, they developed other symptoms including tightness in the chest, coughing, dyspnea, and pulmonary edema (the cough and breathing difficulties were originally ascribed by the US Defense Dept. to fine sand particles (6), bizarre behavior, sweating,

salivation, and blurred and/or dark vision. (This agent inhibits blood cholinesterase; it was originally developed as a biologic warfare agent! And is the cause so called Sheepherders' Disease, caused the pyridostigmine used as insecticides in NE England, references attached. Requests to the US Defense Department for information on whether any American troops were housed in such tents, what percent of these received the vaccines and what percent did not, remain "classified information". The American troops with this syndrome whom I have talked to said they slept on the ground and wore petrochemical insecticide collars. It is noteworthy that the French troops (who, again, refused the vaccines), under separate command, refused such pesticides (7).

However, M. Hooper, Professor Emeritus of Medicinal Chemistry from the University of Sunderland, has obtained some data by spending much time visiting with officials of British Gulf War Veterans Associations (7). The same agent is used in some areas of the U.S. as an insecticide and in Northern England for "dipping" sheep to protect against insect infestation. The "sheepherders syndrome", indistinguishable in symptoms from those described above (8) is now defined as recognized medical entity by the Ministry of Health(9); is no longer considered psychological, and sheep dipping is no longer mandatory. Many of the victims have myocardial disease, a finding characteristic of many patients with Coxsackie virus -induced myalgic enkephalitis, and in an experimental model thereof in inbred mice by mouse Coxsackie virus B69. Furthermore, the selective immunosuppression observed in CFIDs and in organophosphate toxicity is also induced by Coxsackie B virus infection (20).

The third Gulf War illness is related to being drenched in petrochemical-soaked clothing for 3 days, after explosion of oil wells by Allied missiles (10). The percentage of troops who suffered from such environmental exposure is unknown, and those who previously had (or developed from such exposure) petrochemical hypersensitivity

therefore had additional symptoms (11), some are now universal reactors to all chemicals. Some Czech troops had both vaccine exposure and petrochemical exposure of the type outlined if not described above.

Some of those with petrochemical hypersensitivity who also received the vaccines had symptoms of Myalgic Enkephalitis plus neurologic symptoms similar to multiple sclerosis. The situation is further complicated by the so far lack of data on differences in symptoms between those that received vaccinations plus the "pill" versus those that received vaccinations without the pill; and also between data on those who were and those who were not exposed to massive doses of organophophosphates (the French were not); and between those who were exposed to the vaccines with, and those who received vaccines without, petrochemical exposure, etc.

The article by Dr. Ismael and Dr. Wessel, one of the co-authors referred to above, implies that there is no characteristic illness; they appear unaware, since the illness of those who received the vaccines was highly infectious; 80% of spouses and 60% of children living in the same household developed the same symptoms, and 40% of DOGS and/or CATS in the HOUSEHOLD DEVELOPED NEUROLOGIC SYMPTOMS, again within 6-18 months after exposure to an affected veteran. Perhaps the other 20% who did not infect household contacts suffered from type 2 or 3, rather than type 1 GWI.

I have also seen silent carriers of the infectious agent(s) e.g., a healthy Canadian nurse who apparently transmitted disease to her mother (12). The mother developed the disease 6 months after exposure to her daughter for 3 months beginning in Nov. 1993. SEVERE SYMPTOMS DEVELOPED in March 1994. The carrier, a nurse who had worked in a Saudi Hospital, had been on vacation in Canada at her parents' house during these 3 months, before returning to the Gulf Hospital. Symptoms persist to this day, undiminished in severity; the patient is confined to the house, as she is now a "UNIVERSAL

REACTOR" to all chemicals. Her daughter remains well, but the daughter's child, age 25 months, was hospitalized in the same Hospital with a mysterious illness.

As for the extensive epidemiological studies by Unwin referred to above, Servicemen who served in the Gulf, those who served in Bosnia, and those in service but who were not combatants in either were compared. The findings that servicemen involved in active conflict have a higher incidence and more severe disease than non-combatants is not surprising. But they have used a cannon to kill a mouse and, deliberately or by accident, ignored the most significant finding in the mass of data amassed from the respondents, the approximately 4250 veterans in each group who were sent questionnaires for this cross-sectional epidemiological survey who did not respond or refused to respond. In a second questionnaire sent to 7515 Gulf War veterans, 4583 did not respond, and 201 refused to participate. Another questionnaire was then sent to each of the 4583 non-responders: only 1460 responded, 2914 did not and 302 refused to respond. This indicates a high degree of mistrust by these veterans of any data requested by the U.S. Dept. of Defense, Veterans Administration, or even the NIH, and of similar U.K. agencies such as the Royal Defense Medical College, a fact not surprising in that the dread exposures and dread results were covered up for so long. Furthermore, some of the veterans may have been too ill to respond. Severe cognitive function and complete exhaustion occur in many.

Indeed, immunologic testing has still not been performed. In addition, in the absence of abnormalities on physical examination or routine blood chemistries, a psychiatric diagnosis of post-traumatic stress disorder was often made and many veterans died at an early age with no diagnosis save post-traumatic stress syndrome.

An eminent psychiatrist, Professor of Medicine at UCLA, examined many of these patients and came to the conclusion that their illness(es) were not psychiatric, psychological, or psychosomatic in

origin, that post-traumatic stress syndrome accounted for no more than 10% of cases and that the remainder were purely organic, usually neurologic in nature. He was warned that unless he conformed to the hospital diagnostic policy in this area, he would be fired, tenured or not (3).

In any event, troops in Bosnia who received any one of the Biologic warfare vaccines cited (anthrax, plague, and etc.) in U.S also Brucellosis, tularemia, botulism toxin and others, some not approved by FDA; requests for informed consents denied) were only 2.9 % of the total, whereas they comprised 58.4% of the Gulf War contingent. It is therefore not surprising that in the Gulf War veteran population, the incidence and severity of illness is considerably higher than in the Bosnia veterans. Also noteworthy is that the authors found no interaction between pyridostigmine bromide, another deadly agent, and multiple vaccinations. Since I have seen similar incidence and severity of dread symptoms in a small cohort of U.S. troops immunized but who did not get to the Gulf, it would be of considerable interest, and shed considerable light on etiology, if British troops who were vaccinated with the same vaccines but did not get to the Gulf War were similarly studied.

Again, if this is done I urge appropriate immunologic studies. The Chronic Fatigue - Immune Dysregulation Syndrome (CFIDS) was originally dismissed as a psychiatric or stress disorder, although subsequently SPECT Scans, Beam Scans, and MRIs showed a characteristic abnormality (13). A characteristic profile also observed in 2 of the 5 CD8+ lymphocyte subsets is also diagnostic (14). (Only the entire CD8+ population is measured in England; this value is worthless for the purpose of diagnosing CFIDS, Gulf War syndrome, petrochemical toxicity, etc.)

That the commentary (15) should by S.E. Strauss of the NIH is ironic, as is his conclusion that there is no Gulf War Syndrome, and that the symptoms are largely the result of post-traumatic stress. Dr. Strauss

maintained for many years that the symptoms of CFIDS were psychological in origin, excluded anyone with depression from CFIDS group studies (16) although CFIDS depression differs from ordinary depression in that it is worse at 1-3 PM, (17) when cell-mediated immunity is at its lowest, (18, 19) and is made worse rather than better by tranquilizers, benzodiazepines, or an early morning walk (20). These findings clearly differentiate CFIDS depression, present in 50% of such patients, from ordinary depression (20). Furthermore, the abnormalities in 24-hr cortisol in ordinary depression and CFIDS depression are diametrically reversed. And Gulf War veterans have as little respect for the CDC criteria for CFIDS as they do for any CDC, Veterans Administration, or Defense Department data on Gulf War Illness.

The symptoms of GWI are those of CFIDS, but far more severe (21). In addition, and have neurologic symptoms (vide infra). Hypothyroidism, with or without decreases inT3w or T4 (22) is often present.

Introduction

The U. S. HOUSE and Senate subcommittees on Veterans' Health began hearings i early Sept, 2007 on the Gulf War Illness. This review analyzes the medical literatu on this poorly understood illness and on another disorder, Sheepherders Disease, with identical symptoms, but a very different etiology. Evidence for a common pathogenesis is provided and two hypotheses advanced to explain the similarity ir symptoms.

Mercury

The adverse effects of mercury on the brain have been known for 3 centuries. Remember the Mad Hatter at Alice's tea party? The incidence of 'madness" was very high in London hat cleaners who used mercury solutions to clean hats. The amounts of MERCURY in our bodies depend on both Genetic and Environmental factors. About 15% of Caucasians have genes that cause defective catabolism and excretion of excess mercury. Environmental sources include a. large fish in the diet, e.g., tuna, the town of Minowada Japan (1) was wiped out by fish-derived mercury; the Inhabitants were almost all tuna fishermen, and their diet and that of their family members consisted mainly of fish they had caught.);The inhabitants of a fishing village in China suffered a similar fate (2) Furthermore, maternal ingestion of large amounts of mercury rich fish during Pregnancy to an extent that raises blood mercury levels leads to impaired child cognition when tested et 3 years(3)_b) cutaneous absorption from showers with high mercury water content in the water source used; in agriculture, for example pineapple plantations in Hawaii d) CREMATORIA EMISSIONS, fluorescent light bulbs, mascara, etc.) VACCINES CONTAINING THIMEROSAL (ETHYL MERCURY) as a bacteriostatic agent. The interrelationship between mercury and autism is illustrated by the sky high incidence of autism in San Jose, CA and Richmond CA; the former was built on top of a mercury mine, the latter has many crematoria.

Inorganic mercury is a dangerous neurotoxin (e.g. 4.5). Adequate toxicity studies were never performed. Eli Lilly, the manufacturer, evaluated toxicity in only 22 subjects, all adults, none infants or children, before applying to for FDA approval.

The amount of cumulative mercury given to children by age 5 in 1991 exceeded the safety level 8=49 fold. And the amount THEY RECEIVE NOW IS EVEN GREATER BECAUSE of the HEPATITIS B VACCINE, MANDATORY WITHIN 24 HOURS of BIRTH, and the flu vaccine, "highly recommended" at 6 months.

The Scandinavian countries banned thimersol-containing vaccines in 1992+; these were REPLACED by SINGLE-DOSE, MERCURY-FREE VACINNES. Damage to U.S. infants continued until recently. Eight states banned thimerosal between 2001-2008; similar legislation is being debated in 5 others. A bill, HR 5887, to make this ban nationwide, cosponsored by Weldon, R., FL., an M. D. and Maloney, D. NY, was introduced into Congress early in the current session, and into the Senate by Senator Hegel, (R, NE); it has wide bipartisan support.

As a result, the amount of mercury in most vaccines has been greatly reduced, but the total cumulative dose U.S. infants receive is far above the level the EPA considers safe. And in many it has been replaced by aluminum, a dreadful neurotoxin (13). Aluminum also increases the permeability of the blood-brain barrier (14).

An 18 month investigation by a U.S. Senate subcommittee reported on Oct. 1 2007 that the FDA effects of thimersol thereby needlessly exposing millions of infants to this neurotoxin. Footnote 1

A federal court awarded parents of a child harmed by simultaneous injection of multiple vaccines $1 million, CDC, and others (pharmaceutical companies) were guilty of deliberate minimizing the adverse last April; Five thousand additional cases are pending.

Squalene and Anthrax Vaccine

Squalene is a low molecular weight substance often incorporated into Freund's adjuvant ant which causes proliferation of antibody- producing immune cells and boosts humoral (antibody) response to any antigen given simultaneously; however, it suppresses Cell Mediated Immunity (CMI). It is rarely given to humans, since it may cause over production of antibody and predispose to infection with viruses, fungi and strange bacteria, e.g. Bartonella, mycobacteria, etc. because of the impaired CMI. Theoretically it can lead to cancer of the antibody producing cells, multiple myeloma. Indeed, Freund himself died of myeloma as did several other noted immunologists in the late 1940 and 50s before its dangers were known.

As a member of a committee appointed by the National Academy of Sciences 20 tears ago I had the opportunity to examine the records of technicians working with our biologic warfare agents at Fort Dietrick, MD. All had been given 'Protective substances" but none of their records mentioned Freund's adjuvant or squalene.

These were given to enhance the anti body response responded to anthrax. These were not mentioned in my conversations with the then Asst. Secretary of Defense in charge of the Gulf War Illness nor in the available re records of veterans with that illness, yet many knew they received it be cause they developed "bumps" at the injection sites, and had antibodies to squalene (47). They also developed high Immunoglobulin G levels, a universal finding (plus 10 others) of Immune Dysregulation tion (see discussion).

Anthrax is a biological warfare agent. It can cause blindness; the anthrax vaccine used to protect against anthrax, but the vaccine was ineffective (48-50) and dangerous, (51-53 and footnote 2) It caused severe illness in many who received it during the first Gulf War. Nonetheless since 9/11 it has been given to more than 1.4 million of our troops, often despite their strong objections. (Footnote 2)

Sheepherders Disease and Organophosphates

About 20-25% of the UK troops in the Gulf developed symptoms very similar to hose of GWI. They received no anthrax or squalene nor multiple vaccinations. However they used organophosphate insecticides. A syndrome with very similar with very similar symptoms occurred in many SHEEPHERDERS in northern England, e.g. ( ), where the sheep dip insecticide were organophosphates ( ). This illness was recognized as a disease entity by the British Ministry of Health in 1988 ( ); Organophosphates were placed in the pajamas of 700,000 troops in The Gulf in Gulf War and in the underwear of troops in the Gulf during Gulf War II.

Is there a Gulf War Illness?

Dod refuses to admit there is a Gulf War Syndrome, but there have been many reports of clinical and laboratory findings characteristic of this illness. e.g. refs 61-67.

Discussion

I reiterate, presence in the Gulf was not necessary for troops to develop the Gulf War Illness. Many of the troops received three different kinds of injections just before being sent to the Gulf; some never reached the Gulf because the war ended a few days later o because they were assigned to submarines 1500 miles from the Gulf or for other reason As noted elsewhere (11) U.S. troops that never reached the Gulf developed the illness. Other troops exposed to the same environmental toxins –i.e. the French, Israeli and Moroccan troops, did not develop the illness; none of them received the 3 injections tha were mandatory for American troops, namely, 1. Anthrax vaccine. 2. Freund's adjuvant; (some batches contained squalene; this causes a marked increases humoral immunity (antibodies) but causes a concomitant decrease in Cell Mediated Immunity, i.e. that par of the immune system that protects against viral fungal and parasitic infection and also contains the suppressor T lymphocytes which suppress autoantibody formation. Forty four (44) patients with GWI have recurrent infections and a variety of autoimmune disorders. They have abnormalities in both humoral and cell mediated immunity (46) and in cytokine profiles (47). Their symptoms are those of mercury toxicity ( ) and they ofter have neurologic symptoms resembling atypical ALS, atypical, Myasthenia Gravis, or oth neuroimmunologic disorders ( ) associated with autoantibodies to brain constituents.

The symptoms in Sheepherders' Disease which is due to organophosphates insecticides previously used as pesticides for sheep are very similar (62-66). One good question; Why don't all individuals exposed to these toxins develop disease? Why only 25%? I suggest that clinical symptoms occur only in those with a genetically determine defect in this case inability to excrete toxic heavy metals. The type of disorder perhaps depends on which triggering agent, and the severity is perhaps to total duration of exposure; it would be most interesting if the predisposition gene is closely linked to genes at one of the Immune Response loci HLA-A,-B,-C or D, CTL 4, and/or Ig allotype linked genes Gm, Am, or Km.

LITERATURE REVIEW

The Gulf War Illness has been ascribed by various investigators to the mercury in the standard vaccines, to anthrax vaccine, and to pyridostigmine.

Mercury is a dangerous neurotoxin (26-32); it is also an Immunosuppressant (33). It can cause mitochondrial dysfunction (34). It is now present in only “trace quantities” on most vaccines, but has been replaced by aluminum, which greatly enhances the adverse effects of mercury (35). If akxo lmcfd Mercury also causes increased apoptosis of suppressor T lymphocytes (37 -38), causing a marked increase in autoimmune diseases (39), especially neuroimmunologic Disorders (40-43), t depletes suppressor (44-45).

Anthrax was given, together with vaccines against plague bacilli, to protect against these biologic warfare agents (45) protect against these biologic warfare agent (46-53). Anthrax vaccine was often given in Freund’s adjuvant to enhance the immune response to anthrax vaccine; however, this caused a concomitant decrease in cell mediated immunity (46-48). The anthrax vaccine is neither safe nor effective (49-57). Some batches of the Freund’s adjuvant contained squalene. The Defense Department denies this bit antibodies to squaleme have been found in Gulf War era troops who were given the anthrax vaccine (58).

UK troops on the Gulf did receive anthrax, but their tents were covered with orthophosphate pesticides, substances known to cause Sheepherders’ disease (59-65) the symptoms is very similar to those of GWI; Organophosphates, like mercury, can cause a myriad of neurologic problems and infections with unusual organisms, e.g., Coxsacchi. As noted above, mercury toxicity is associated with a marked increase in neuroimmunologic disorders. Apparently this is also true of orthophosphate toxicity (64). The symptoms of mercury, and of organophosphate toxicity resemble those of pyridostigmine toxicity this compound increases production of acetylcholine (ACH) esterase, the enzyme that destroys ACH.

The mechanisms whereby anthrax-squalene cause neurotoxicity is unknown; I postulate that squalene decreases ACH production, known and postulated, are listed in Table1 and (65), the common denominator appears to be reduction in acetylcholine or in uptake by its receptors.

Mechanisms

The mechanism whereby mercury disrupts neurologic function is unclear but there seem to be multiple effects (62-3), including neurotoxicity (64) and blockage of receptors for several neurotransmitters (65- 66)

Apparently organophosphates block the acetyl chorine receptor (67-68) could mercury exert its deleterious effect by blocking receptors for neurotransmitters by immune complexes of mercury-anti-mercury receptors? Some evidence to support this is already available in that mercury potentates the deleterious effects of organophosphates on neurotransmitters (69-75).

HYPOTHESIS 1

I suggest that the clinical symptoms of GWI are due to deposition of antigen-antibody complexes on cells with receptors for them, e.g. the glial cells of the brain, the mesangial cells of the retinal capillaries, the Kupfer cells of the liver, etc.(Fudenberg. Immune Dysregulation Syndromes in preparation)
The antigens are of 3 kinds, namely anthrax, squalene, and mercury. Unfortunately, only a very small minority of veterans with the Gulf War Illness have been tested for any of these antibodies. Whether the complexes are the cause or the result of the Immune Dysregulation present in these patients is conjectural, but the laboratory findings are present in almost all individuals with GWI (and also in a large percent of autistic children. And in most of the offspring of patients with the GWI sired after the onset of symptoms, are severely autistic or have other immunologic and/or neurologic problems (Fudenberg, in preparation). The placenta contains cells with Fc receptors (as doe's semen, footnote 3) which capture these complexes and pass them on to the glial cells in fetal brain.

In any event, individuals with classic forms of depression do not have laboratory findings of Immune Dysregulation; thus individuals positive with these tests have a documentable organic illness, these findings are not present in patients with ordinary depression, or with a Garden variety Post Traumatic Stress Syndrome (PTSS).

Hypothesis 2

The Mechanism whereby Mercury exerts its deleterious effects is unclear, but are due to l blocking of the ACH REC receptor; the symptoms resemble those of pyridostigmine toxicity; this compound increases production of ACH esterase (67) resulting in increased destruction of ACH. Mercury toxicity is also associated witn malfunction of the ACH receptor. It also causes apoptosis. (70-71). Organophosphates interfere with binding of ACH to its receptor (72-74).

I postulate that anthrax and/or squalene suppresses ACH production (Table1). Mercury blocks receptors v for acetylcholine and other neurotransmitters by immune complexes of mercury and its antibody; some support for this hypothesis is the finding that mercury potentates the adverse effects of organophosphates (74), and recent findings that organophosphates, like thimerosal, induces various neurologic diseases(67), and apoptosis (68). Furthermore, the combination of organophosphates and trace metals make the adverse effects of mercurials much worse, far greater than merely additive (74).

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TABLE I

COMPOUND	Mechanism
Pyridostigmine	increase in ACH esterase so ACH is destroyed more rapidly
Mercury	direct neurotoxic effect Mitochondrial dysfunction Blocking of ACH receptor
Squalene	decreased ACH synthesis? Many other mechanisms
Anthrax vaccine	No known action on ACH or ACHR; postuted- decrease ACH synthesis
Organophosphates	inhibit binding of ACH to the Ach receptor

K. D.

K. D., birthdate 5/5/64, a nurse, enjoyed good health until she joined the army in l986. She received an unknown number of vaccines shortly after joining, and developed dizziness, blurred vision, and memory and sleep difficulties. These lasted about 3 weeks, then disappeared.
In 1991, in preparation for going to the Gulf, she received additional immunizations, oral pyridostigmine, and organophosphate insecticides were placed in her pajamas. Her original symptoms returned in far greater severity. She also developed tubular vision, severe fatigability, difficulty reasoning, abdominal pain, constipation, and urinary frequency. Because of this, in late March of 1991 she was sent to Bethesda Naval Hospital instead of the Gulf. After 2 weeks she was discharged with diagnoses of Chronic Fatigue Syndrome, Functional Bowel Distress, and Hypoglycemia (fasting blood sugar 39). No immunologic tests were performed.
During the next 17 years her health gradually deteriorated and she developed new symptoms, including paresthesias, intermittent laryngeal paralysis and other neurologic symptoms including occasional difficulty in swallowing, suggesting atypical myasthenia gravis. She had 3 boys since 1991, all with severe immunologic illness, namely agammaglobulinemia, autism and regional ileitis, and severe allergies.

She and all 3 sons have antibodies to mercury. Red Cell mercury, which correlates well with total body mercury, was markedly increased in K.D. and in the one son in whom it was measured. She has other features of Immune dysregulation including abnormal Ig levels, impaired lymphocyte DNA synthesis in response to mitogens, and auto-antibodies to neural antigens. I attribute this to materials she received in the service. Her lab findings are those of mercury toxicity. Her neurologic symptoms are due to impairment of acetylcholine uptake by its receptors.

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