

Scientific Aspects of Welding Rod Cases

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I. Welding Products and How They Are Used

Welds are formed from the weld puddle created through contact of flame, work piece (metal) and welding rod, if used. Welding joins metal materials by heating them. The materials change from a solid to a semi-solid/ liquid state. Once the metal cools, it returns to a solid state.

There are two general categories of welding equipment. One is referred to as arc equipment. Arc equipment relies on electricity as its heat source. Gas equipment describes the second type of welding equipment. The gas equipment uses gas flame as the heat source for welding. Both gas welding equipment and arc welding equipment may involve the use of various types of welding rods and welding wires. For instance, depending on the materials used in the welding process, different properties are necessary in the weld puddle to achieve a solid and reliable weld. This involves the molecular changes in the alloys used to form the welding puddle. Manganese is one of several elements used in welding wires and welding rods. The alloys involved in the welding process will determine different levels and percentages of various elements used by manufacturers of welding products to formulate welding wires and welding rods.

A condition called de-oxidation is needed in the weld puddle to minimize carbon (C) formation and porosity. The soundness of the weld metal is characterized by lack of porosity, good fusion and freedom from cracking. Carbon can become trapped as the weld cools forming porous (porosity).

To avoid carbon formation and porosity, welding wires and rods contain elements that when combined with oxygen and carbon, form a harmless slag and diminish the formation of carbon and the porous conditions in the weld area. These elements called deoxidizers are manganese (Mn), silicon (Si), titanium (Ti), aluminum (Al), and zirconium (Zr).

A typical gas equipment welding set-up consists of an oxygen cylinder with oxygen regulator, an acetylene or other gas fuel cylinder with regulator, hoses leading from the regulators on the oxygen cylinder and gas cylinder to a torch and welding head attachment, welding rods, and a can or container of a material

referred to as flux (defined as a fuseable material used in welding or oxygen-cutting to dissolve and facilitate removal of oxides and other undesirable substances). Arc welding generally involves an electric power source for heat and the use of a torch component to focus the heat.

It is the use of manganese in the welding process that has spurred the current litigation against the manufacturers and suppliers of welding equipment and components, the trade associations for these companies and commercial entities that use welding in the course of its business operations.

II. Manganese Exposure From Welding Products

A. Mechanism of Exposure

Exposure to manganese during the welding process may occur when welding rods or wire are melted with the welding torch. The welding rods and wire give off fumes of sulfur, lead and manganese that allegedly cause irreparable brain damage. Manganese is a known toxin, and overexposure can cause a condition called Manganism, a disorder that closely resembles Parkinson's disease.

Medical experts dispute whether or not the amount of manganese absorbed through the lungs and into the brain can be quantified and then linked to Parkinson's disease. The related medical issue concerns the amount of fumes generated during welding that exceeds a known threshold of exposure to manganese that damages the brain.

B. The Scientific Debate

Parkinson's disease is described as a progressive disorder of the central nervous system caused by the degeneration of dopaminergic neurons in the substantia nigra of the midbrain. According to an article published in the American Family Physician, April 15, 1999, Parkinson's disease has been reported to affect approximately 1% of Americans over 50 years of age, but unrecognized early symptoms of the disease may be present in as many as 10% over the age of 60. The article goes on to describe the early onset of Parkinson's disease affecting persons in their 20s and that epidemiological studies have found that Parkinson's disease is more prevalent in men than in women by a ratio of 3 to 2. The author of the American Family Physician article, Rosabel Young, M.D., M.S. (King-Drew Medical Center, Los Angeles, California) states that pesticides and other toxins have been suspected but none of has been proved to be a definite causative factor for Parkinson's disease.

Encyclopedia references to Parkinson's disease or Parkinsonism describe a degenerative brain disorder first noted by English surgeon James Parkinson in 1817. When there is no known cause, the disease usually appears after age 40 and is referred to as Parkinson's disease. The term "Parkinsonism" usually refers to similar symptoms resulting from head injury, encephalitis, syphilis, carbon monoxide poisoning, cerebral arterial sclerosis or the use of a synthetic narcotic described as MPTP (N-methyl-4-phenyl-1, 2, 3, 6 – tetrahydropyridine).

In the legal context, the debate centers on whether or not reliable scientific testimony links symptoms generally associated with Parkinson's-type disease with exposure to welding fumes.

III. The Litigation History

Larry Elam may go down in history as the man who started the welding rod litigation. In a news article published in the Plain Dealer on November 29, 2003, it was reported that Elam was awarded a million dollars in damages in a suit brought against BOC Group, Hobart Brothers, and Lincoln Electric. According to the Plain Dealer article, Elam was the first plaintiff to win a case in ten such trials.

Pending before the District Court in Cleveland, Ohio are more than 100 lawsuits from across the country. These cases are consolidated for hearing before U.S. District Judge Kate O'Malley.

In California, cases are consolidated in the Oakland Courthouse for Alameda County. The Honorable Ronald M. Sabraw is the coordinating judge. Law firms involved in asbestos litigation, tobacco litigation and other mass toxic tort claims are in the early stages of litigation in the Alameda County action referred to as the *Welding Products Cases*.

The Master Complaint under submission for the Alameda Court's approval gives an overview of the types of target defendants and legal theories asserted in welding fumes exposure cases. The *Welding Products Cases* groups defendants into four general categories: (1) manufacturers and sellers of welding products; (2) manufacturers of welding machines and welding consumables; (3) trade organizations; and (4) premises/contractor defendants.

Examples of defendants by category are stated below:

Manufacturers and Sellers of Welding Products	Manufacturers of Welding Machines and Welding Consumables	Trade Organizations	Premises/ Contractor Defendants
Lincoln Electric Company	Westinghouse Electric Corporation	American Welding Society	Kinder Morgan Energy Partners, LP
Union Carbide Corporatoin	Miller Electric Manufacturing Company	National Electrical Manufacturer's Association	Proctor & Gamble Paper Products Company
Thermadyne Holding Corporation	Victor Equipment Company	Feroalloys Association	Texaco Downstream Properties Inc.
Deloro Stellit Company			Chevron USA Inc.
			Union Oil Company
			Crown Beverage Packing Inc.
			Shell Oil Company
			Shell Chemical LP
			Atlantic Richfield Company
			C&H Sugar Company

According to the Master Complaint "all of the defendants" were members of trade organizations.

The legal theories set out in the *Welding Products Cases* Master Complaint include: negligence; negligent misrepresentation; negligence-sale of product; negligence-voluntary undertaking (trade organizations); breach of express warranty; breach of implied warranty; strict liability-failure to warn; strict liability-design defect; strict products liability; fraud/deceit by suppression/concealment; conspiracy and concert of action; violation of Business & Profession Codes; premises owner/contractor liability; and loss of consortium.

The lynchpin to all pending and anticipated litigation is the judicial determination of admissible evidence establishing medical causation linking welding fumes to Parkinson's disease/Parkinsonism. In the consolidated actions before Judge O'Malley (federal District Court – Ohio) are numerous evidentiary motions brought by plaintiffs and defendants. Judge O'Malley must determine

who may give expert testimony and what qualifies as scientific studies that may be relied upon in forming expert opinions under the Federal Rules of Evidence.

IV. Parkinson's Disease and Manganese

The scientific debate now pending before civil courts involved in welding fumes litigation centers on the connection between exposure to welding fumes and Parkinson's disease or Parkinsonism. In the *Elam* trial mentioned above, evidence was presented that Elam was diagnosed with Parkinson's disease at age 57. Expert testimony offered by Elam's counsel showed that Parkinson's disease stemmed from years of using welding rods. Elam's attorney relied on a study published in the journal Neurology in 2001 that suggested welding acts as an accelerant to cause Parkinson's disease and that there is a link between exposure to manganese fumes and the early onset of Parkinson symptoms. The testimony in favour of plaintiff was provided by Brad A. Racette, M.D., a researcher with the Washington University School of Medicine in St. Louis, Missouri, who conducted the 2001 study.

V. Science and Causation

A. Overview

The *Elam* decision illustrates how a physician's study may be used as evidence to show a causal connection between the toxin, manganese and a clinically recognized medical condition, Parkinson's disease. According to Dr. Racette's study, patients who worked as welders tended to develop symptoms about 15 years earlier than other patients.

Legal counsel for defendant Lincoln Electric, John Beisner, relied on evidence concerning studies of workers in manganese mine operations that are exposed to manganese at greater concentrations than welders, and there are only rare circumstances of neurological disorders suffered by those workers.

The dispute concerning scientific evidence and causation also centers on the ability to accurately diagnose a disease linked to manganese.

To defend these claims, medical testimony is being offered on behalf of the welding manufacturers to show that only a very small number of welders are suffering the toxic effects of manganese exposure. Included in the motions pending before Judge O'Malley is the evidentiary dispute over whether or not diagnostic testing with positron emission tomography (PET) and single-photon emission computed tomography (SPECT) distinguishes Parkinson's disease from Manganism. This legal and scientific debate involves complex neurological theories about how Parkinson's disease affects brain functioning and whether

that disease has characteristics that are distinguishable from neurological disorders linked to manganese. In very simplistic terms, diagnostic findings using SPECT and PET can track increase and decrease levels of dopamine neurons in the brain. Medical opinions by neurologists have been offered to show that PET and SPECT testing of certain parts of the brain will distinguish manganese induced Parkinsonism from Parkinson's disease without influence from manganese exposure.

Complicating the debate is the ability of a given physician to make an accurate diagnosis of Parkinson's disease and relate that diagnosis to a patient's history. The hallmark physical signs of Parkinson's disease (tremor, rigidity and bradykinesia [slowed movement and non-volitional movements]) may be linked to patient histories that have nothing to do with Manganism. According to the April 15, 1999 article in the American Family Physician authored by Rosabel Young, M.D., certain prescription drugs, such as Haldol, Compazine, Reglan and Serpasil can produce a "constellation" of symptoms resembling Parkinson's disease.

B. Clinical Diagnosis of Parkinson's Disease

On file in the Welding Rod Products Liability litigation pending in the United States District Court, Northern District of Ohio, Eastern Division (Case No. 1:03-CV-17000; MDL Docket No. 1535; Judge Kathleen O'Malley) is a declaration of David Eidelberg, M.D. Eidelberg is a neurologist at Northshore University Hospital in Manhasset, New York. He is also the Director of Neuroscience for the Institute of Medical Research at Northshore-Long Island Jewish Research Institute. In the Welding Rod Products Liability litigation case, plaintiffs' counsel take exception to the opinion by Eidelberg regarding the use of PET and SPECT diagnostic testing for ruling out manganese exposure in patients who claim the onset of Parkinson's Disease results from exposure to welding fumes. According to Eidelberg, Parkinsonisms, including Parkinson's Disease (PD) are a heterogeneous group of neurodegenerative movement disorders that have overlapping clinical features. Eidelberg's declaration correctly states Parkinsonisms result from abnormalities in the functioning of the brain network responsible for controlling motor function and that the brain network involved is formed by neurocells (neurons) in the basal ganglia, a structure of the brain that includes the striatum (consisting of the putamen and caudate nucleus), the substantia nigra pars compacta, the subthalamic nucleus and the globus pallidus.

In his explanation of the brain function, Eidelberg notes the substantia nigra pars compacta contains the cell bodies of specialized neurons that produce the neurotransmitter dopamine and these dopamine neurons extend or project

into the striatum. At the end of these neurons are terminals that release dopamine into the spaces between the neurons (synapses in the striatum).

In textbook fashion, Eidelberg explains the dopamine levels in the striatum play a critical role in the normal basal ganglia function by modulating and stabilizing its activity. The various Parkinsonisms are characterized by neuronal loss or degeneration in specific regions within this network. Symptoms of Parkinsonisms “can result from damages or degeneration at any time the neural structures comprise the basal ganglia network.” Eidelberg offers an example; the significant loss of dopamine neurons in the substantia nigra pars compacta such as occurs in PD, resulting in a decrease of dopamine levels in the striatum. Eidelberg’s opinion centers on this decrease in striatal dopamine levels that impairs basal ganglia function and results in movement disorders.

Eidelberg believes that there is sufficient scientific validation for relying upon PET imaging to diagnose and assess Parkinsonisms, including PD in living patients. A diagnosis of PD can be made with a high degree of sensitivity with PET or SPECT imaging according to Eidelberg. Characteristic patterns of neurochemical loss within the basal ganglia that are specific for Parkinson’s Disease as opposed to other forms of Parkinsonism are revealed in PET and SPECT imaging studies according to Eidelberg. The specific clinical observation identified by Eidelberg is the reduced uptake of positron-emitting dopaminergic tracers in the posterior putamen. It is this characteristic and reliable feature of Parkinson’s Disease that Eidelberg believes is identified through PET and SPECT diagnostic testing.

Eidelberg explains that PET diagnostic testing provides reliable data on the viability of dopamine neurons in the substantia nigra pars compacta and their projections into the striatum. This is accomplished by quantifying the rate at which F-floradopa (Fdopa), a radio labelled chemical precursor or tracer of dopamine, is taken up by the nigral dopamine terminals in the putamen and is inverted by a specific enzyme (dopa decarboxylase) into dopamine. Fdopa uptake on PET, according to Eidelberg, reflects the rate of this enzymatic process that serves as an index of the integrity of the dopamine neurons in the substantia nigra pars compacta. When significant numbers of dopamine neurons in the substantia nigra pars compacta have been lost, as occurs in PD, the amount of measurable enzymatic activity in the terminals of the dopamine neurons will decline and PET imaging will reveal a decreased signal or reduced uptake of Fdopa. Eidelberg relies on various peer review studies of Fdopa PET imaging and the use of that diagnostic tool that identified a difference between Parkinson’s Disease and Parkinsonism and studies regarding SPECT imaging and the early diagnosis of Parkinson’s Disease.

Central to Eidelberg's opinion is a reduction in the posterior putamenal Fdopa uptake on PET imaging that is specific and consistent with PD and correlates with SPECT imaging.

C. Parkinsonism Secondary To Elevated Manganese Exposure

According to Eidelberg, in contrast to characteristic PET imaging findings in PD, there are Parkinsonian syndromes that clinically resemble PD where Fdopa uptake is intact on PET imaging. He notes that degeneration of neurons in the striatum can produce advanced clinical Parkinsonism with normal Fdopa uptake that it is revealed in the PET testing.

Eidelberg believes this finding is due to the fact that disease processes involving the striatum cause Parkinsonism through damage to structures downstream from the Fdopa uptake. He concludes the nigrostriatal dopaminergic inputs to the system are not involved and this contrasts with what occurs with PD.

Parkinsonism secondary to elevated manganese exposure involves pathological damage that is primarily localized to the globus pallidus and striatum and these basal ganglia structures are downstream from the substantia nigra pars compacta and its striatal projections and these findings, according to Eidelberg, shows a disorder where symptoms of Parkinsonism occur without evidence of cell death in the substantia nigra pars compacta. Eidelberg relies on consistent peer review published studies and reports that reveal intact striatal uptake of Fdopa in patients with Parkinsonism secondary to elevated manganese exposure.

D. Welders With Parkinson's Disease

Plaintiffs contend that welders with Parkinson's Disease contracted that condition as a result of welding fumes. As indicated by the declaration of Dr. Eidelberg described above, diagnostic testing using PET and SPECT will distinguish between those PD patients who have experienced manganese exposure from those who suffer from Parkinson's Disease that is not traced to manganese exposure from welding fumes.

Plaintiffs rely on the studies by Dr. Mark Guttman published in "Parkinson's Disease Diagnosis and Clinical Management," Volume 259 (2002), stating that there is no evidence that PET scans are a reliable tool in determining a differential diagnosis (Parkinsonism versus Parkinson's Disease). Plaintiffs also cite to an article entitled "Ideopathic Parkinsonism With Superimposed Manganese Exposure: Utility Of Positron Emission Tomography" 28 Neurotoxicology, pages 249 – 251 (1999).

It is not clear as of the submission of this paper whether the Federal Court in the Ohio case will allow defense expert opinions based upon PET and SPECT scans. If the defendants prevail, they will have a useful weapon in refuting the plaintiffs' claims that Parkinson's Disease presents an increased risk to welders due to manganese exposure.

VI. Managing the Risk

Litigation involving welders and their exposures to welding fumes poses a wide array of challenges to target defendants, their insurers and legal counsel. The approach by the plaintiffs driving this litigation uses a combination of the pleading and evidentiary techniques developed during the course of asbestos and tobacco litigation. Like asbestos litigation, there is a wide range of possible levels of exposures and sources for exposures that are linked to asbestos-related diseases. The same is true with welding fumes.

Like the tobacco litigation, plaintiffs are focusing on the alleged concealment of information about the risks associated with welding fumes. Counsel for the plaintiffs allege in the lawsuits that since 1837 manganese has been medically recognized as toxic to the central nervous system. Allegations by plaintiff's counsel reference medical articles from 1932 in a Metropolitan Life Insurance booklet regarding the risks associated with manganese and the involvement of various trade associations, including NEMA, that accumulated, discussed and took positions on the risks associated with welding fumes, manganese and neurological disorders that mislead welders about the dangers linked to welding fumes.

These claims must be evaluated in light of the on-going screening process that is used to assemble potential litigants. An attorney-based organization called the Welder Health Fund has been established to provide disease screening for welders.

Appropriate plans must be pursued in accumulating information for defending these claims. Thorough knowledge concerning the particular defendant and its role in the use or distribution of welding products is necessary to determine the opportunity for exposure. Further, as with any other occupational exposure case, detailed medical and occupational histories are necessary to determine any links between the defendants' products or activities and the claims regarding exposure to welding fumes. Additionally, careful consideration must be given to the means and methods for the diagnosis of disease due to manganese exposure.

Unfortunately, these cases involve many different venues, law firms, consolidated civil procedures and massive resources from both the plaintiffs and

the defendants. The ability of any one defendant to obtain immediate resolution of any given claim is severely impaired by the current status of litigation. The expertise of legal counsel, the talents of the insurance company litigation specialists and the dedication of resources by defendants are essential to managing and achieving favourable disposition of these claims. If the defendants are to prevail against these claims, they must know more about welding products and the science and medical evidence linking them to Parkinsonism than the trial attorneys and medical experts pursuing these cases.