Professor Maths H Berlin M.D.
Box 2081
S-182 02 DANDERYD
Sweden

Statement to be presented in court.

Having been asked to evaluate the contribution of occupational exposure at the municipal dental clinic of Boe in Telemark, Norway to the present state of illness shown by Tordis Klausen, I would like to make the following statement.

My evaluation is based on the information in the material delivered to me by Advokatfirma Robert Robertsen. The material includes copies of excerpts from hospital records, statements by clinical specialists, results from clinical examinations, laboratory analyses and clinical tests, the verdict by the district court, Nedre Telemark herredsrett, concerning this matter, and my own examination of Tordis Klausen.

Occupational exposure.

It is evident that chloroform and copper amalgam were used regularly during the seventies at the dental clinic, that no special point exhausts or ventilated hoods were present and that the ventilation mainly was upheld by natural ventilation. It is well funded in literature and occupational hygiene experience that under such conditions severe poisoning from both chloroform and mercury vapor exposure can occur. Mercury concentrations may easily exceed 1 mg/m^3, in the breathing zone of a person handling copper amalgam without special precautions.

The area of the mercury to air surfaces, the temperature and the degree of agitation of the mercury source determine the mercury concentration in air. Liquid mercury tends to cover itself with an oxide membrane, which prevents evaporation but stirring or dividing the source will facilitate evaporation, so also increased temperature. Spill on hands and clothes can considerably increase the exposure to a person. The ventilation or the rate of air change in the room influences the duration and extent of exposure. The temperature gradient between outdoor and indoor and wind pressure outdoor are the major factors determining the rate of air change in a room without mechanical ventilation.

From what is said above and considering the nature of the work in a dental clinic it is evident that one should expect a wide variation in mercury concentration and exposure of the personnel. The work phase, the way of handling, the season of the year and the workload are factors determining this variation.

Urine samples were collected from a female dental assistant working in the same clinic as Tordis Klausen at two occasions. The samples were analyzed for mercury. The mercury concentration in morning urine was 59 µg/l at both occasions indicating an exposure level exceeding or close to the hygienic threshold limit 50µg/m^3 air. Considering that the mercury concentration was similar at the two occasions, and assuming that the sampling was not done during a hot summer period it seems likely that these samples represent the most dominant exposure level. How much, how often and how long the exposure level exceeded this level at unfavorable temperature conditions and at contaminated working conditions can not be estimated from available information. The lack of further urine samples during the seventies is remarkable considering the concentrations found. Only a reconstruction of the working conditions can provide information about what concentrations may have been reached under the most unfavorable circumstances.

The uncertainty interval for an estimate of the mercury exposure level for Tordis Klausen is wide and includes lethal exposure – lethal cases of mercury poisoning have been described caused by handling amalgam in dental surgeries – on one end, and mild functional disturbances in a small and sensitive fraction of the population on the other end. The majority of dental assistants handling copper amalgam at this time in Norway did not show any obvious signs of mercury poisoning. Most probably however, the mercury exposure exceeded the hygienic threshold limit during long periods and it can not be excluded that the exposure reached neurotoxic levels. Only the existence of functional insufficiencies and organic injuries of the central nervous system can indicate how serious the exposure has been.

The exposure to chloroform is not elucidated in the available material. Chloroform is very volatile. There is no reason to assume that any large quantities of chloroform have been used. The rate of air change should have been the most determining factor for the extent of exposure. The first adverse effect from exposure to chloroform is a narcotic effect, which increases during the day, since chloroform accumulates in fatty tissue serving as a continuous source for exposure of CNS. Prolonged exposure to chloroform causes severe liver damage.

Relation between occupational exposure and signs and symptoms.

Tordis Klausen´s central nervous symptoms started in the seventies. She had then been working for several years as dental assistant in Boe. Professor Sverre Landgård describes her symptoms in detail in his occupational medicine statement and so also Docent Bo Nilsson in his statement. Tordis Klausen had periods of fatigue after work hours and pronounced sensitivity to alcohol. Alcohol induced acute fatigue. These symptoms are consistent with what can be expected from toxic exposure to a narcotic solvent like chloroform. She also described an episode with nausea and dark colored urine possibly indicating a liver damage. No laboratory tests confirming this are available. Laboratory tests of liver function performed in September 1992 showed normal values.

The local cytotoxic effects and interference with the immune system by mercury vapor can explain her protracted symptoms from the upper respiratory tract. During the seventies and the eighties She experienced several unspecific mental symptoms of neurasthenic type with increased irritability, tendency to depression and anxiety, all symptoms common in mercury poisoning. Her symptoms caused her doctor to treat her with a neuroleptic drug, fluanxol 0.5 mg daily. This treatment induced extrapyramidal side effects, which are unusual at such a low dose. This may however, be explained by the effect of mercury on the basal ganglia. Mercury accumulates in the basal ganglia on exposure to mercury vapor.

A neuro-ophthalmologic examination objectively showed that the convergence and accommodation control of her eyes was disturbed in a way indicating central nervous damage. Magnetic resonance imaging (MRI) examination of the brain reveals brain atrophy and signs of organic damage in basal ganglia. Both signs are consistent with what can be expected in chronic mercury poisoning based on animal experimental data and clinical reports in the literature. In the verdict by Nedre Telemarkens herredsrett it is stated that the brain images of the MRI examination are within the normal variation in the population and referring to the statements by the neurology specialists Kjuus and Aaserud. I have not been able to find any support for this statement in any of theirs written statements. I therefore accept the interpretation by the examining radiologist, that the images illustrate pathological changes in the brain.

It is important to consider that women retain the mercury at exposure to mercury vapor almost twice as effectively as men do, when evaluating the relation between exposure level and signs and symptoms. This gender difference already appears in the material published by Kussmaul 1861[1] and was recently pointed out by Berglund[2]. The same gender difference has been reported from animal

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1 Kussmaul, A. Untersuchungen über den constitutionellen Mercurialismus. Würtzburg,1861

2 Berglund, F. Kvinnor dubbelt så känsliga som män för kvicksilverånga.Hygiea, 1998:Hefte 2.

xperiments and is also evident in the results of the study of Norwegian dental personnel performed by Yrkesmedisinsk institutt in Oslo. The report of this study is one of the documents made available to me. The threshold limit value for mercury vapor and occupational medicine experience are to a large extent based on studies of male chloralkali industry workers. Thus, it can be expected that serious signs of poisoning appear in women at significantly lower exposure levels than in men. The neurologists Kjuss and Aaserud did not consider this fact in theirs statements.

Alternative causes to Tordis Klausens central nervous signs and symptoms.

It is difficult to find an alternative cause to neurotoxic damage, which covers the central nervous symptoms, the signs of organic brain damage such as the increased sensitivity to neuroleptics, the disturbance of visual functions and the neuromorphologic injury. There is no information about any other significant exposure to a neurotoxic agent. A neuro-degenerative disease of genetic etiology might be a possibility. However, the examining neurologists have made no such suggestion. Such a disease would be rare.

One psychiatrist suggested that Tordis Klausen suffers from of an anxiety disorder, a panic attack syndrome, which is discussed in the verdict of the district court. The reasoning in the verdict is not compatible with medical science. This psychiatric diagnosis of anxiety disorder is based on the existence of a defined symptom complex. The etiology to these symptoms can vary. Genetic disposition is probably a contributing factor, and internal and external milieu factors may induce the symptoms. Mercury vapor is one external milieu factor, which commonly induces anxiety at toxic exposure. Mercury accumulates in the basal ganglia of the brain and also in locus coeruleus. It also interferes with the release of catecholamines in the brain. Disposition to panic attacks can enhance the effect of mercury vapor exposure. In this case there is no indication before her employment as a dental assistant, that she has such a disposition. An anxiety disorder can not explain her organic brain damage. Most of the evidence points to her occupational exposure to mercury vapor as the cause of her brain damage.

Summary and evaluation.

This case is about a 56 years old women, who 10-12 years was occupationally exposed to chloroform and mercury vapor. She worked full time 1969-1993 as a dental assistant at a dental clinic. Copper amalgam was daily used up to 1981 at the clinic. The work environment at the clinic was hazardous,with no functioning mechanical ventilation or point exhausts over hazardous workplaces. To the best of her recollection she was not aware of any risks with

he above-mentioned chemicals and therefore did not take any special precautions. Her anamnesis indicates that she experienced periods of fatigue after work hours and decreased tolerance to alcohol intake, suggesting adverse effect of her chloroform exposure. Urine samples from a fellow dental assistant at the same clinic collected at two occasions in 1972 showed mercury concentrations indicating exposure levels around or above the hygienic threshold limit. In violation of good occupational hygiene practice no investigation of the work environment or follow up of the urine sampling was performed. No information about present risks was given to the employees concerned. One consequence of this is that there is no detailed data about the real exposure level. No evidence contradicts that exposure periodically can have reached neurotoxic levels.

After some years as dental assistant she experienced symptoms from the upper respiratory tract. These symptoms disappeared when she was away from the clinic. Also psychoasthenic symptoms of unspecific type appeared such as tendency to depression, irritability and anxiety. She was treated with a low dose of a neuroleptic drug, which induced extrapyramidal side effects, suggesting other toxic interference with brain function or brain damage. An investigation of her failing visual function showed disturbances in the regulation of convergence and accommodation suggesting brain disorder and a MRI examination of the brain showed brain atrophy and signs of injury in basal ganglia.

Chronic mercury poisoning can cause these clinical signs and the symptoms from the central nervous system. No other single cause can explain all clinical observations. I therefore conclude that chronic mercury poisoning is the most likely cause of Tordis Klausens brain injury and resulting functional insufficiency. I estimate the probability for this cause to be more than 75%. The way this case been handled may have hampered her rehabilitation and therefore also contributed to her functional disability.

Danderyd, November 5, 1998

Maths Berlin, MD
Professor Emeritus of Environmental Medicine, University of Lund.

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Maths Berlin

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Replik

der Autoren des „Kieler Amalgam-Gutachtens 1997"

zu der „Stellungnahme zum ‚Kieler Amalgam-Gutachten'"
der Autoren Prof. Dr. S. Halbach et al.,
im Jahre 1999 veröffentlicht als Buch unter dem Titel
„Amalgam im Spiegel kritischer Auseinandersetzungen",
Deutscher Ärzte-Verlag, Köln 1999

Otmar Wassermann, Martin Weitz, Carsten Alsen-Hinrichs

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Friberg, den die Autoren Halbach et al. zu Recht als international anerkannten Quecksilber-Toxikologen bezeichnen (S. 23 im Buch), ist Mitherausgeber des „Handbook on the toxicology of metals". In diesem Kompendium wird in Band II, 2. Aufl., Elsevier Amsterdam 1986, im Kapitel über Quecksilber (S. 387 - 445) der Mikromerkurialismus ohne jede Einschränkung als medizinisches Faktum mitgeteilt. Autor ist der ebenfalls als Quecksilber-Experte international renommierte M. Berlin.

Friberg ist zudem **Mitautor** der Veröffentlichung von Friberg, L., Eneström, S.: Toxicology of inorganic mercury, in: Dayan, A. D., et al. (Hrsg.): Immunotoxicity of metals and immunotoxicology, Plenum Press, New York 1990, S. 163 - 173. Auch Friberg bestätigt vorbehaltlos den quecksilberbedingten Symptomenkomplex des Mikromerkurialismus mit den Worten:

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Effects on the central nervous system

After long-term exposure to metallic mercury vapour, the critical organ in the vast majority of people is the central nervous system. At low exposure levels, nonspecific asthenic and vegetative symptoms (often called micromercurialism) are seen, but at higher exposure levels tremor and/or severe behavioural and personality changes dominate. Prolonged exposure to mercury levels in air of 100 µg/m³, corresponding to a urinary excretion of mercury at 100 µg/g creatinine, confers a high probability of developing the classical signs of mercurial poisoning (tremor and erethism) and proteinuria. At lower levels of exposure, the effects are less frequent and less severe. Subtle effects have been reported in a few studies after long-term exposure to concentrations as low as 25-35 µg/m³ (or µg/g creatinine). There is no evidence that a no-effect threshold level exists. Recently, the World Health Organization (1990) published a review of available information on the subject in an Environmental Health Criteria Document on inorganic mercury.

Friberg entzieht zu Recht den Autoren Halbach et al. jede Grundlage für ihren untauglichen Versuch, die wissenschaftliche Aussagekraft des „Kieler Amalgam-Gutachtens 1997" durch eine Berufung auf Fribergs Feststellungen zum Mikromerkurialismus in Zweifel zu ziehen. Dieser Versuch Halbachs et al. hat die Tendenz, gesundheitliche, toxisch bedingte Auswirkungen bereits niedriger Quecksilberdampf-Expositionen, wie sie z. B. aus Amalgamfüllungen resultieren können, in Abrede zu stellen. Die Behauptung der Autoren Halbach et al, der Mikromerkurialismus werde in der Fachliteratur nicht mehr erwähnt, ist eine massive Irreführung des Lesers, der vom Symptomenkomplex des Mikromerkurialismus betroffenen Patienten sowie u. a. der Ärzte und Juristen, die über die gebotene medizinische bzw. anderweitige Hilfestellung für die Betroffenen zu befinden haben.