

## Possible Foetotoxic Effects of Mercury Vapour: a Case Report

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A thirty-year-old dental surgeon who worked until the 35th week of pregnancy in a surgery in which mercury vapour concentrations in excess of the threshold limit value (TLV) had been detected, gave birth at 42 weeks to a small-for-dates baby with severe brain damage. The possibility that this baby's development might have been harmed by mercury is discussed and the literature relevant to teratogenicity of mercury reviewed. Attention is drawn to the need for further research into the effects on health and pregnancy outcome of mercury vapour in dental surgeries.

### Introduction

Since passage of the 1974 Health and Safety at Work Act, employers and employees have had a duty of care to ensure that there is no unreasonable risk to safety and health in the working environment. It is thus essential to review any potential hazards arising from misuse of dental surgery equipment and materials.

Mercury is a known teratogenic agent.<sup>1</sup> The foetotoxicity of alkyl mercury has been well documented, particularly following the Minamata epidemic.<sup>2</sup> A case of congenital brain damage after exposure to mercury vapour was reported by Carmona in 1982.<sup>3</sup> In spite of discussion in the profession regarding mercury hazards little mention has been made of the foetotoxic effects of mercury vapour. Some 80% of inhaled mercury vapour is retained.<sup>4</sup> Animal data show that such atomic mercury dissolved in blood rapidly crosses the placenta.<sup>5,6</sup> A recent Japanese study<sup>6</sup> has recorded high accumulations of mercury in the placenta of pregnant guinea pigs exposed to mercury vapour. The authors of that study postulate that elemental mercury rapidly crosses the placental barrier but that ionic mercury does not. Oxidation of elemental mercury in foetal blood and tissue inhibits movement across the placenta and other tissue barriers. Observations on *in utero* exposure to mercury indicate that the human foetus is more sensitive than the human adult.<sup>4,5,6</sup> Animal studies<sup>6</sup> show marked differences between mercury vapour metabolism in foetuses and their mothers. Much experimental work has been performed using methyl mercury. Routes are postulated for methylation of mercury in the body, but they have only been demonstrated *in vitro*.<sup>7</sup> Although the full effects of inorganic mercury and mercury vapour on the nervous system are unknown, it is known these substances can prevent normal foetal growth and development and cause later abnormal behaviour patterns in experimental animals.<sup>1</sup> Berlin *et al.*<sup>8</sup> have reported work on the uptake of mercury vapour in the brain of

mammals. In the light of research by a number of authors<sup>8-11</sup> it is hoped that the following case study will stimulate further consideration by the profession of the possibility of mercury vapour foetotoxicity.

In 1980, the Health and Safety Executive<sup>12</sup> set a threshold limit value (TLV) of 0.05 mg/m<sup>3</sup> for the concentration of mercury in a room. It refers to a time-weighted average concentration. Chronic mecurial poisoning can result from exposure over a period of time to mercury vapour levels above the TLV.

Because readings in the surgery observed in the case study were made with reference to the TLV this measure has been retained. However, it is now out of date. Instead, we should be referring to the time-weighted average (TWA) and short-term exposure limit exposed to mercury (STEL). The TWA is the time-weighted average concentration for a normal 8-hour working day or a 40-hour week to which a healthy worker may be repeatedly exposed, day after day, without adverse effect. The TWA for mercury vapour is also set at 0.05 mg/m<sup>3</sup>. The STEL is the maximum concentration at which workers can be exposed for a period of up to 10 minutes without ill effects. For mercury vapour it is 0.15 mg/m<sup>3</sup>. The general philosophy now appears to be that these are very much limits: all efforts should be made to control the concentration within the working environment to at most one-quarter of these values.

### A Case Study

Brown's study<sup>13</sup> with passive mercury monitoring discs of 704 dental surgeries throughout the United Kingdom demonstrated that over a period of one week some 10% had mercury vapour concentrations which exceeded the TLV of 0.05 mg/m<sup>3</sup>.

Working in one of these surgeries was a 30-year-old female dental surgeon in the first trimester of her first pregnancy. She had worked in this one surgery for four years. Brown reported a raised level of mercury vapour in her surgery and those of two colleagues at the same practice, despite the normal precautions regarding mercury hygiene apparently being taken. The Department of Community Dental Health offers a service to local general dental practitioners to help them check the concentration of mercury vapour in their surgeries. Advantage was taken of that service by the practice principal. The scuffed floor levels of vapour in this one surgery reached 26 times the currently accepted TLV (Table I). Other surgeries on the premises also presented readings greater than the TLV. No specific spillage site was identified. It was suggested that the problem may partly have been due to lack of

**Table I** Mercury vapour levels in the original dental practice at floor level

		Vapour levels in mg/m <sup>3</sup>		
		Surgery A	Surgery B	Surgery C
General	Static	0.02	0.04	0.01
	Scuffed	0.06	0.05	0.01
Dentist's chair side	Static	0.02	0.02	0.01
	Scuffed	1.05†	0.1*	0.07*
DSA's chair side	Static	0.04	0.03	0.01
	Scuffed	1.3‡	0.08*	0.05

\* = greater than TLV/TWA of 0.05 mg/m<sup>3</sup>

† = 21 × TLV

‡ = 26 × TLV

ventilation. The surgeries measured approximately 8' x 7' with low ceilings, and no ventilation other than the door. The dentist, who had been reassured by her medical advisers regarding possible foetotoxic effects, continued to work until the thirty-fifth week of pregnancy.

At the start of labour, 12 days post-term, old meconium was present, indicating possible hypoxia of the foetus. A foetal scalp electrode was therefore placed. A baby girl was born nine hours later by means of a forceps delivery. She weighed 2.53 kilograms (5lb 6oz). The baby was severely brain damaged and died nine days later. The child's mother was told at delivery that the placenta had been disproportionately small for the size of baby. Unfortunately, the placenta was not retained for examination.

The baby's blood mercury level at eight days was 16 µg/l blood (L. Stimmler, Personal Communication). Table II shows the normal and hazardous levels for blood and urine, but they refer to adults. This normal level is continually being re-defined. All analyses in this case study were performed by the National Poisons Unit. At the time of the initial analyses, they regarded normal as less than 20 µg/l. The current value is less than 5 µg/l.

During pregnancy, the mother's level of mercury in her urine dropped from 25 µg/l at nine weeks gestation to 15 µg/l at 35 weeks gestation, despite constant environmental exposure, the mother working the same number of hours per week in the one surgery. Difficulties involved in the analysis of the mercury content of biological material<sup>3</sup> suggest that no specific conclusions may justifiably be drawn from laboratory test results presented in this case. However, these results are of interest for two reasons. Firstly, although the maternal ionic mercury is itself unlikely to cross the placenta and harm the foetus, the maternal levels are presumably in part a reflection of the potentially foetotoxic mercury vapour concentrations in the working environment. Secondly, despite the fact that mercury concentration in urine may vary physiologically in an unpredictable way which so far has not been explained (M. Berlin, Personal Communication) the levels exhibit a general trend which is worthy of note.

### Discussion

There is no direct evidence that the baby's brain damage was caused by mercury vapour in addition to birth injury. However, Clarkson *et al.*'s work<sup>5</sup> on the bio-transformation of mercury in experimental animals shows that inhaled vapour is absorbed into the bloodstream. Despite the rapid oxidation in red blood cells, some elemental mercury remains dissolved in the blood long enough for it to be carried to the blood-brain barrier and to the placenta. Yoshida's work<sup>6</sup> confirms this fact. Lipid solubility and high diffusibility allow rapid transit across these barriers. It has been demonstrated in animal experiments that after brief (20 min) exposure to radioactive elemental mercury vapour, the radio active mercury easily penetrates the placental barrier. Tissue oxidation in brain and placenta serves as a trap to hold the mercury and leads to accumulation in brain and foetal tissues.<sup>4</sup>

**Table II** Normal and hazardous blood and urine levels\*

Specimen	Normal	Hazardous
Blood	Less than 5 µg/l	More than 50 µg/l
Urine	Less than 5 µg/l	More than 100 µg/l

\* Information supplied by National Poisons Unit

These findings support Koos and Longo's recommendation<sup>1</sup> that women of child bearing age should not be exposed to mercury vapour air concentration greater than 0.01 mg/m.<sup>3</sup>

Birth weight shows a class gradient.<sup>14</sup> 2.53 kilograms represents a relatively small baby for a fit and healthy, non-smoking mother, of registrar-general's social class I. A premature baby is regarded as one whose weight at birth is 2.50 kg or less.<sup>15</sup>

It is possible to speculate that the lower maternal tissue concentrations of mercury recorded at 35 weeks gestation might reflect a decreased total maternal mercury content resulting from mercury distribution into placental and foetal tissues, with the foetus acting as a sink in a similar way to that postulated by Koos and Longo.<sup>1</sup> Growth retardation, if it occurred, would result in a foetus less able to withstand the stress of labour. It is not known whether concentration of ionic mercury within the placenta can itself contribute to placental malfunction.

Nixon *et al.*<sup>16</sup> report that female dentists have a higher spontaneous abortion rate than doctors. Unfortunately, their pilot study had a disappointing response rate. The number of females on the dental register has since increased from 2,800 female dentists in 1976 to 4,995 in 1985. In a paper presented to an International Conference on Mercury Hazards in Dental Practice, one consultant obstetrician said: 'The third trimester of pregnancy is normally a time of obstetric tranquillity and the occurrence of late abortion and early premature labour in working dentists is disturbing and requires explanation.'<sup>16</sup> He pointed out that female dentists who work seem to have a higher than expected incidence of spontaneous abortion, a disturbing excess of late abortion and premature labour and a higher incidence of peri-natal mortality than would be expected of mothers in social class I.

Kuntz *et al.*<sup>18</sup> suggest that 'previous still birth exhibited significant positive correlation with blood mercury levels in both mother and infant'. However, Newman<sup>19</sup> feels that their data do not support this assertion. Kuntz *et al.* and Koos and Longo<sup>1</sup> both stress that epidemics of (organic) mercury poisoning in recent years have been associated with brain damage to infants in spite of little or no evidence of maternal toxicity. They believe that the foetus is especially sensitive to injury.

One point that emerges clearly from this discussion is the need for further research. The WHO Task Group on Environmental Health<sup>4</sup> makes the plea: 'Further epidemiological studies are needed on groups occupationally exposed to mercury vapour. Wherever possible collaborative studies should be carried out in which cohorts should be followed in time and different groups related to each other'. They also state that 'studies are needed on the dose received by the expectant mother and the effect on her infant including the development and growth of the child'.

The second point that emerges is the more immediate one regarding pre-conceptual assessment of any mercury vapour in a working environment.

MacDonald<sup>20</sup> states that mercury is a teratogenic agent and should be avoided by pregnant women. She suggests 'since damage to the foetus is most likely to occur during the first eight to ten weeks consideration should be given to attempts to eliminate all exposure prior to conception'. Perhaps this statement in the Journal of the California Dental Association might seem unnecessarily alarmist to British readers. However, it would seem prudent to assess vapour levels prior to conception, or at least as soon as a state of pregnancy is confirmed. Administrative dental officers when equipped to do so will respond sympathetically to a request to assess surgery mercury levels. However, such requests must obviously be initiated by the practice principal.

Bearing in mind the increasing numbers of females qualifying as dental surgeons, and the almost totally female paradental workforce – dental surgery assistants, hygienists and dental therapists, who are frequently of child bearing age – these indications of adverse

reproductive performance in relation to the complex area of mercury vapour toxicity are worthy of note. The discussion of mercury hazards within the profession should be broadened to cover not only alleged harm to patients and dentists but also foetotoxicity as a result of maternal exposure to mercury vapour.

It is not the authors' intention to draw any specific conclusions as a result of this single case study. The paper is written to bring to the profession's attention a possible case of foetotoxicity, and to review the evidence for the potential that mercury vapour exhibits for foetotoxicity, as already documented by other authors. For now, the TWA and STEL must be regarded as limits, but females working in dental surgeries are warned to take great care, especially when they are pregnant or trying to conceive. The profession is alerted to the clear need for further research to be carried out in relation to this problem.

### Postscript

Six months following the initial pregnancy, with blood and urine mercury levels within the then accepted normal limits, the woman in this case study conceived again. The surgery in which she was then working contained a mercury vapour absorbing fan as detailed by Brown.<sup>20</sup> In addition, the surgery mercury vapour level had been assessed by the Department of Community Dental Health as being safe. Nevertheless, the dentist's urine mercury levels rose from 9 µg/l prior to conception to 13 µg/l at 6 weeks of pregnancy to 21 µg/l at 8 weeks of pregnancy (C. Garrett, Personal Communication). These readings were standardized against urine creatinine. The pregnancy ended in a missed abortion at 12 weeks. The urine mercury level then fell to 8 µg/l. The significance of these events is difficult to assess. One possible explanation is that the increased biochemical demands of early pregnancy may cause old body stores of mercury, (e.g. in bone) to be mobilized.

Subsequently, a second early abortion occurred. A healthy baby has now been born.

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