The Minamata disaster has certainly been the most outstanding example recorded of the effects of industrial irresponsibility and will remain as a never to be forgotten lesson. In that respect, this review of the outbreak and of the neuropathology of the process, of which much though not by any means all was published in the Japanese literature of the period, is well justified. Several of these papers are printed here in the appendix to this survey of the clinical and pathological features of the illness. It must be admitted that while there are a great deal of data here, especially the detailed mercury analyses of nervous tissues, the book is more remarkable, perhaps, for what it leaves out.

The story of the outbreak of poisoning is briefly though incompletely told. There is the account of the shameful behaviour of the physician in charge of the hospital maintained by the factory in question, who realized that the illness of these patients could well be related to chemicals used in the factory, but was restrained by the management from publishing this opinion. Then there were the chemists who for a long while were ‘economical with the truth’ as to what was being put into the effluent from the factory and the reasons why the route of the factory effluent, that had earlier been run straight into the sea away from the town, was changed and run into the river that fed into the bay. The role of Dr Douglas McAlpine, who visited the area with Dr Araki, is also overlooked. Dr McAlpine was looking for unusual cases of Multiple Sclerosis, which was known to be uncommon in Japan at that time, and it was thought that the cases of this unknown encephalopathy in Minamata might be unusual forms of that disease. When he saw these Minamata patients, Dr McAlpine told me that he was forcibly reminded of the cases of methylmercury poisoning reported a few years earlier by Donald Hunter and colleagues, and so he sent Donald Hunter a telegram asking if he knew of any sources of mercury that might be entering the environment in the area. Donald Hunter immediately wired back to say that there was a vinyl-producing factory in Minamata using mercury as a catalyst in the preparation of acetaldehyde. Although Douglas McAlpine personally recounted this story to me, it was curious that in their Lancet paper (McAlpine
and Araki, *Lancet* ii, p. 629, 1958) this was not specifically mentioned, even though he was convinced, he assured me, of the clinical and pathological similarity of these Japanese patients to Hunter’s three cases.

Dr Takeuchi and his colleagues, who described the early cases of the condition, made over the ensuing years a remarkably thorough description of the neuropathological changes and these descriptions are reviewed in this book. However, despite the large number of cases he examined over several years, there were very few changes seen that had not been described by Dorothy Russell in her one case. Although he must have seen this topographical pattern of damage over and over again, Dr Takeuchi does not comment upon the remarkable restriction of the cortical and cerebellar damage to those regions with predominant numbers of small neurons, namely the visual cortex, the auditory cortex and cerebellar granular layer. Nor does he comment upon the remarkable sparing of large neurons, such as large neurons in the deeper cortical laminae, the Purkinje cells of the cerebellum and motor cells in the brainstem and spinal cord. Yet these striking differences must be mechanistically significant, since the mercury was more or less uniformly distributed throughout brain tissue. Only in peripheral nerves do we see large neurons being affected, namely the sensory ganglion cells, accounting for the almost exclusive sensory neuropathy that was such a common feature in almost all affected subjects. Knowing as we do that the spinal ganglia are not protected by a blood–nerve barrier and that mercury does not easily penetrate the blood–brain barrier, this localized damage should not be cause for surprise. It is for this reason that, in addition to organic mercury, dorsal root ganglion cells are also susceptible both to poisoning by both mercuric and to mercurous salts. Witness the now mercifully long defunct ‘Pink Disease’ of infants due to calomel in teething powders. When Dr Takeuchi thinks mechanistically of the changes in the brain, it is in terms of anoxia and vascular insufficiency rather than disturbances to metabolic function. Indeed, as seen in Russell’s case, depths of sulci are often selectively damaged suggesting focal vascular insufficiency, but reactionary oedema consequent upon the extensive neuron damage is more likely to be an additional mechanism.

What I find extraordinary, in addition, is the continued occurrence of new cases during the 15 years after the source of the problem had been identified. This can only mean, since the intoxication is a cumulative process, that locally caught fish and shellfish continued to find their way onto the Japanese tables. While cleansing of the bay would be an impossible task, the Prefectural Authorities should have placed an absolute ban on all fish and shellfish caught within a 50 km length of the coast, fully explaining the vital necessity for doing this, until the mercury levels had declined to safe levels. This does not seem to have been effectively done.

Whether this is a useful book or not depends much on whether a shortened history of this event is enough. The whole episode is still deeply embarrassing to the Japanese, and every now and then it must seem necessary to them, with more than 2000 people affected by this disaster and many of them cruelly crippled thereby, to go over the matter once again as a continuing process of admission of guilt that is difficult for them to express in any other manner.

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