A brain SPECT scan is produced for evaluating how the brain is functioning, not for elucidating how the brain is structured such as if an X-ray, CAT scan, or MRI were carried out. A brain SPECT scan is produced by an individual receiving an injection of the radioactive compound Tc-HMPAO, which travels through the blood vessels to the brain. Once in the brain this compound remains for a short while in effect giving off a message of how the brain is working.

When concentrating or relaxing, an individual is injected with the compound containing the radioactive technetium. This then travels throughout the body in the blood vessels. As a consequence, part of what a brain SPECT scan demonstrates is the blood flow throughout the brain. In addition to the technetium entering the blood vessels, it also passes into the brain cells and gets locked in chemically in two different ways: (i) the Tc-HMPAO binds to the antioxidant tripeptide called glutathione [2]; sufficient levels of glutathione need to be present in the brain cells for the Tc to be retained. (ii) the Tc-HMPAO binds to the mitochondria [3], which act as the power-houses of each cell in the body; sufficient mitochondrial function needs to be present in the brain cells for the Tc to be retained.

The brain SPECT scan thus provides an image of the level of blood flow to parts of the brain (too low, too high, or appropriate), the level of glutathione in parts of the brain (typically too low or appropriate), and the level of mitochondrial functioning in parts of the brain (too low, too high, or appropriate).

In considering the potential results at a cellular level, we can note the following: poor blood flow implies the delivery of decreased oxygen to the brain; low vitamin D leads to poor glutathione production [4]; the “energy-producing” enzyme cytochrome C oxidase in mitochondria may function poorly with insufficient iron levels [5]; and brain cells and their mitochondria will become activated in the presence of the excitatory neurotransmitter glutamate [6].

How would the knowledge from such a brain SPECT-based evaluation help to correct elevated glutamate levels, such as in individuals chronically subjected to low levels of organophosphates? Let us begin by recognizing some of the major difficulties with which these individuals struggle. They include symptoms such as memory problems, headaches, depression, and mood swings.

One of the theories regarding the toxicity of organophosphates is a consequent excessive rise in glutamate release [7]. This leads to activation of the brain cells by opening a transmembrane channel [1] and letting calcium flow in. This will energize the cells to work harder. The now activated brain cells use more energy and require their mitochondria to produce it. More blood will flow to these now-activated brain cells. Energized sufficiently, these active areas and the correspondingly increased blood flow can be seen on a technetium injection SPECT scan.

But if the cells are low in oxygen and iron, the production of this energy falters. These consequently underactive areas can be seen on a technetium injection SPECT scan.

After a brain injury, moderating levels of glutamate, and optimizing levels of mitochondrial functioning by optimizing oxygen and iron levels, may help to heal the brain. Using further the knowledge described above, is also reasonable to consider the possibility of preventing brain damage from organophosphate exposure. Optimizing vitamin D intake to enhance glutathione production and improve glutamate absorption before an organophosphate exposure, for example, may minimize the extent of impairment after an exposure. Optimizing vitamin B₆ intake to enhance the metabolism of glutamate may also minimize the extent of damage subsequent to organophos-
phate exposure [8]. Optimizing magnesium intake to block the action of glutamate before an organophosphate exposure may minimize the extent of future impairment [9]. And optimizing ferritin levels (the best way to measure total body iron status) before an organophosphate exposure may similarly minimize the extent of future impairment.

In summary, with a greater understanding of what seems to occur in the brain after organophosphate exposure, in part by the use of SPECT imaging, potential options for minimizing the effects of future exposure or addressing the damage of past exposure present themselves.

REFERENCES