The Psychological and Neuropsychological Assessment of Chronic Organic Solvent Neurotoxicity: A Case Series

Jenni A. Ogden
Department of Psychology, University of Auckland,
Auckland, New Zealand.

Occupational solvent neurotoxicity (OSN) is defined, and suggestions for its assessment are described. The 1985 International Solvent Workshop hypothesised three severities of OSN; Type 1 (acute), Type 2 (chronic), and Type 3 (dementia). They further postulated that Type 2 OSN could be subclassified into Type 2A, characterised by sustained personality and mood changes, and Type 2B, characterised by chronic cognitive deficits. The psychological and neuropsychological assessment of a series of 13 cases of Type 2 OSN is described. The results of this series do not support the division into Type 2A and 2B OSN, as 11 of the men are equally debilitated by symptoms from both categories. Recovery of cognitive deficits is unlikely in this group, but other rehabilitation strategies that may be helpful are suggested.

Organic solvents are widely used in industry in paints, glues, adhesives, and degreasing/cleaning agents, and in the production of plastics, textiles, printing inks, polymers, dyes, agricultural products and pharmaceuticals. Long or very intense exposure to many of the solvents used in these industries can result in neurotoxicity. The chronic, and often slow and insidious effects of organic solvent neurotoxicity (OSN) include psychological and psychiatric symptoms and impairments in cognitive functioning.

On the basis of United States data (NIOSH, 1987) it has been calculated that approximately 100,000 New Zealanders work in environments that potentially expose them to organic solvents (Dryson & Ogden, 1992). In Scandinavian countries a considerable body of research has been carried out over many years in the area of occupational neurotoxicity, resulting in a general awareness of the problems it causes. New Zealand is only beginning to become aware of the very extensive symptoms that can affect exposed workers, and recently the Occupational and Health Service of the Department of Labour published a booklet outlining the diagnostic criteria for chronic OSN (Dryson & Ogden, 1992). As outlined in this booklet, a clinical interview that focuses on the psychological symptoms that can follow OSN, followed by a neuropsychological assessment, play a major part in diagnosing chronic neurotoxicity. In many cases cognitive impairments may be the only clear indicator of neurotoxicity.

Clinical psychologists, psychiatrists, neurologists and general medical practitioners may be approached by clients who have problems that are possibly related to long-term exposure to organic solvents. Given that the onset of symptoms of chronic neurotoxicity is usually gradual, and diagnosis is often confounded or compounded by other work and family-related problems, many sufferers may be unaware of the possible association of their symptoms with past or present solvent exposure. At this stage in the education of NZ health professionals, it is also not uncommon for the professionals themselves to be unaware of the consequences of OSN. It is therefore important for health professionals to gain some understanding of the syndrome so that they can conduct an informative clinical interview and assessment. In particular it is important to ask pertinent questions of clients and patients who present with one or more of the symptoms commonly associated with OSN and who have a history of working in an environment that increases the potential for solvent exposure.

The purpose of this paper is twofold: firstly to describe the causes and symptoms of OSN and its psychological and neuropsychological assessment, and secondly to illustrate by way of a series

Correspondence to: Dr. Jenni A. Ogden, Department of Psychology, University of Auckland, Private Bag 92019, Auckland, New Zealand. I would like to thank Dr. E. Dryson for referring the subjects to me, my post-graduate neuropsychology students for assistance with some of the assessments, and the subjects for their cooperation.

of cases the psychological and neuropsychological profiles of victims of OSN.

Causes of OSN

Before symptoms of OSN become apparent, 10 years or more of exposure to neurotoxic solvents at or above the appropriate NZ Workplace Exposure Standard (Department of Health, 1991) is usual. Not all solvents are neurotoxic and some are more dangerous than others. Table 1 lists the solvents that are clearly neurotoxic and that are commonly found in NZ workplaces, along with the industries in which they are typically used.

Accidental intake of solvents into the bloodstream is either via direct absorption through the skin, or via inhalation. Solvent abuse via inhalation is also common in young people. Johnson, Bachman and O'Malley (1979) estimated that 18.7% of high school seniors in the USA had tried solvent-based inhalants. Solvents accidentally or purposefully ingested (e.g., in suicide attempts) are readily absorbed from the gastrointestinal tract. The amount of solvent retained is dependent on various factors, including the blood and tissue solubility of the solvent, its toxicity, diurnal metabolic cycles of the individual, alcohol

Table 1: Neurotoxic solvents commonly used in NZ and their use in industries, occupations and the home

SOLVENT	USE IN INDUSTRY
Toluene (Methyl benzene) $CH_6H_6CH_3$	Cleaning and degreasing, and as a solvent for paint, varnish and rubber adhesive.
Styrene (Vinyl benzene) $C_6H_5CHCH_2$	Used in the manufacture of plastics, rubbers and in boat-building. In floor waxes and polishers, paints, adhesives, putty, metal cleaners, varnishes, tobacco smoke, automobile exhaust and in food stored in polystyrene containers.
Trichloroethylene (TCE, Triclene) $CHC1C1_2$	Cleaning, stripping and degreasing machinery, dry-cleaning, In house hold cleaners, lubricants, adhesives and typewriter correction fluids.
Tetrachloroethylene (PCE, Perchloroethylene) $CC1_2CC1_2$	Dry-cleaning, metal degreasing, fabric finishing. In lighter fluid and cleaning fluid.
Xylene $C_6H_4(CH_3)_2$	Used in the rubber and leather industries and in the preparation of histology slides. In paint solvents, glues, varnishes and printing inks.
Methyl chloride (Chloromethane) CH_3C1	In manufacture of synthetic rubber, plastic foams, antiknock fuel additives, methyl cellulose, tetramethyl lead. In insecticide propellants and refrigerants.
n-Hexane $CH_3(CH_2)_4CH_3$ and Methy butyl ketone (MBK)	Inexpensive solvents used as a component in white spirit, petrol, glues, lacquers, glue thinners. Used in the manufacture of perfumes, pharmaceuticals and cleaning agents.
Methyl ethyl ketone (MEK)	Used in printing industry.
Carbon disulphide CS_2	Limited use in NZ in manufacture of pharmaceuticals and in some laboratories.

use and possibly obesity (some solvents last longer in fat people than in thin people). The immediate exposure level of the solvent can be measured in urine, blood or exhaled air.

Today, safety standards in industry are constantly being updated as our knowledge and awareness of the effects of solvents and other industrial toxins increase. In the recent past, however, protective clothing was not always worn by workers at risk from solvent exposure, and even today workers may neglect to take the necessary precautions if they feel the clothing interferes with their ability to do the job effectively (e.g., gloves making it difficult to do fine work), or the clothing makes them too hot. Self-employed workers are particularly vulnerable, as they do not have peer pressure and support to encourage maintenance of safety standards. In addition it is important for them to get through as much work as possible and protective clothing may slow them down, and the cost of protective clothing may also act as a negative factor (e.g., the frequent need to replace rubber gloves).

Types of OSN

The 1985 International Solvent Workshop (Baker & Seppalainen, 1986) postulated, as a working hypothesis, three types of OSN as follows:

Type 1 OSN: This is the least severe presentation and is characterised by *subjective complaints of fatigue, irritability, depression, and episodes of anxiety.* No impairments are apparent on neuropsychological tests. This corresponds to the WHO classification of organic affective syndrome and it is reversible on removal from the solvent.

Clients with these symptoms as either their primary problem or as difficulties apparently secondary to a marital, work, or other stress-related problem may present to a clinical psychologist. Assessing the probability that these symptoms are caused by exposure to solvents can be problematical, given that combinations of these common symptoms can accompany infection and recovery from some viruses, or can occur as a result of work or family stress. If on interview it becomes apparent that the client is potentially exposed to neurotoxic solvents, he/she should take a break from that environment for at least two weeks (longer if possible), and during that time period both the client and a close relative should take careful notes of the times and durations of any of the symptoms complained about, as well as the context and client behaviours surrounding the onset of the symptoms. At the end of the exposure-free period he/she should return to the work environment and continue to note the occurrence of symptoms. If a symptom increase is closely correlated with a return to the solvent exposure, this gives some support for Type 1 OSN. Clearly it does not rule out non-solvent-related work stress as the cause of the symptoms, nor does it rule out social expectancy effects or malingering (Hawkins, 1990). If any of these are likely, then they should be thoroughly assessed and appropriate therapy/counselling given.

Type 2 OSN: This is a more severe and chronic disorder than Type 1, and neuropsychological and clinical assessments are required to demonstrate *chronic symptoms of neurotoxicity and cognitive impairments.* For research purposes, the Type 2 disorder can be divided into two subcategories, characterised primarily by emotional and cognitive problems respectively. The diagnostic features of the *Type 2A* disorder include *sustained personality or mood disturbances, fatigue, poor impulse control and poor motivation,* and *Type 2B* is diagnosed by deficits in intellectual functioning, including *impaired concentration, memory, learning, and psychomotor slowing.* The relationship between these two Type 2 disorders is unclear, but in practice symptoms of both subtypes are often present in the same individual. Mild symptoms may be apparent as early as 3 years with chronic industrial exposure, but a period of 10 years or more of exposure is usual before symptoms become debilitating.

Type 2 corresponds to the WHO classification of mild chronic toxic encephalopathy, and though the term "chronic" suggests long-term changes, in some cases the symptoms may become less severe as time since the last exposure to solvents lengthens, and recovery may be enhanced by appropriate counselling or rehabilitation. This level of OSN is the one most likely to be missed by a clinical psychologist, particularly if the client is no longer working in the solvent environment and presents with a problem that is not obviously associated with solvent neurotoxicity (e.g., a decreased tolerance to alcohol and violent and disinhibited behaviours). The post-concussional syndrome includes a similar constellation of symptoms (Gronwall & Wrightson, 1974) and therefore it is important to exclude closed head injury as the main or a contributing diagnosis.

Type 3 OSN: This is a dementia, and requires a *global and progressive deterioration in memory, other intellectual functions, and emotion.* It corresponds to the WHO classification of severe chronic toxic encephalopathy and is irreversible. This level of OSN is uncommon, and as safety standards in work places improve, and self-employed workers become more aware of the importance of safety measures when using solvents, this level of OSN will hopefully become very rare.

Psychological and Cognitive Assessment of OSN

Given the high number of New Zealanders involved in occupations that use neurotoxic solvents, it behoves the clinical psychologist to include general screening questions in the initial interview about the types of work and recreational activities clients have been involved in both currently and in the past. This is particularly important for clients who complain of memory and cognitive impairments, stress symptoms (e.g., fatigue, irritability, sleep disorders), intolerance to alcohol and a range of other psychological problems including marital difficulties, violent and aggressive behaviours, depression and anxiety.

Typically, people who suffer Type 2 OSN are men in their thirties or older, have a family and a mortgage, and are usually skilled or semi-skilled tradespeople (e.g., car and house painters, printers, dry-cleaners, workers in degreasing and extraction industries and in the manufacture of pharmaceuticals and agricultural sprays, leather workers, pest controllers and agricultural sprayers). It is not uncommon for clients with suspected OSN who are still working with solvents to express reluctance to give up their jobs, even when they at times experience the debilitating, acute affects of solvent neurotoxicity such as nausea, loss of appetite, vomiting and dermatitis (Arlien-Soberg, 1985). This is understandable in the present-day economy, given that it is unlikely that they could successfully retrain for and obtain a job in a new trade that does not involve solvents. Thus, the appearance of many potential victims of OSN at a health or counselling agency is often precipitated by a crisis in their marriage or work resulting from their changed personality, extreme fatigue, or significant memory problems, rather than by a concern about the acute or chronic effects of the solvents they earn their living by.

With clients who have suspected OSN, part of the initial interview should be conducted with a member of the family, close friend or workmate present to widen the information base, and in some cases add a more objective assessment of the client's subjective complaints. It is sometimes advantageous to carry out (with the client's permission) separate interviews with relatives and friends, or ask them to convey via letter, their views of the client's difficulties. The symptoms of fatigue, irritability, depression, anxiety, poor concentration and memory impairments that affect the client's daily routine should be carefully explored. Depression is a particularly common consequence of the syndrome. For example, OSN from the widely used industrial solvent, trichloroethylene (TCE) has been reported to frequently result in severe agitated depression, sometimes accompanied by violent behaviours towards self and others (White, Feldman, & Travers, 1990). The causes of depression, anxiety and irritability are often difficult or impossible to isolate, but in cases where OSN seems a likely diagnosis on a number of grounds these symptoms may be both directly and indirectly seen as a consequence of the OSN. Indirect causes for depression and anxiety could include a poor memory, lowered sexual drive, fatigue and low energy levels resulting in marital stress, and hypersensitivity to noise, constant headaches and other physical symptoms resulting in a lowered work capacity. Other "psychiatric" symptoms including hallucinations, confusion, inappropriate laughter, suicidal ideation and emotional lability can occur in various degrees of severity with chronic exposure to some solvents (e.g., toluene), although these symptoms are more likely in cases where the solvent exposure is current as well as chronic (e.g., in toluene abusers).

In addition, note should be taken of the physical symptoms of unwarranted headaches, dizziness, sleep disturbances, poor appetite, alcohol intolerance, heart palpitations, feelings of oppression in the chest, painful tingling in some parts of the body and excessive perspiring. Obviously a client who has these physical symptoms should have a medical check-up to rule out other causes.

Clinical psychologists with some training in neuropsychology can follow up a suggestive history or symptom presentation with some preliminary neuropsychological tests and if these further support the hypothesis of possible Type 2 OSN can then refer the client on to a specialist clinical neuropsychologist for a more extensive assessment. Other diagnostic procedures could include

a psychiatric assessment and a neurological examination, primarily to rule out other possible diagnoses, but also to assess neurological signs which although uncommon in mild to moderately severe chronic OSN, may be present in more severe cases (Juntunen, 1982). In particular, toluene and TCE can cause peripheral neuropathy and TCE can damage the trigeminal nerve. This can result in trigeminal anaesthesia, which can be permanent and spread to the other cranial nerves (Hartman, 1988).

Different neurotoxic solvents can result in different types of cognitive impairment, but there are a number of deficits that seem to accompany Type 2 OSN whichever neurotoxic solvent is causative. Many people who suffer OSN may, in any case, have been exposed to a mixture of solvents, making it difficult to taylor a test battery to the deficits thought to be caused by a specific solvent. The neuropsychological symptoms that are most commonly found in cases of OSN from any neurotoxic solvent are those associated with diffuse cerebral encephalopathy. In this respect, the picture painted by OSN is rather similar to that following a mild to moderate closed head injury in some people (i.e., the post-concussional syndrome). Thus, the tests the OSN sufferers are most likely to do poorly on are those that measure concentration, vigilance, psychomotor speed, reaction time, and memory for new material. Many sufferers also do poorly on complex tests of visuospatial perception and memory, and in more severe (but still Type 2) cases, impaired abstract thinking, organisation and planning abilities may also be apparent. These latter deficits are suggestive of frontal-lobe dysfunction.

Included in an assessment must be tests of functions that tend to remain stable following mild to moderate diffuse brain damage, and that can also be used, along with information about educational attainment, occupation and hobbies, to estimate premorbid intelligence. Such "holding" tests could include the NART (Nelson, 1976), the WAIS-R Vocabulary and Picture Completion subtests and Digit Span Forward. Clients who perform erratically or well below expected levels on these tests may be malingering, or attempting to exacerbate their problems so that their difficulties will be taken seriously and additional tests that are designed to assess these possibilities could be given at a later session.

Table 2 provides suggestions for a minimal battery of tests that will allow assessments of the areas of likely impairment along with tests that should not be impaired. Descriptions of these tests and normative data can be found in Lezak (1983) and Spreen and Strauss (1991). This battery includes tests that assess the same functions as the Core Battery recommended by the WHO/Nordic Council (1985), with additional tests of executive functions, memory, and complex visuospatial perception. There have been a number of published studies that support the use of similar test batteries (e.g., Cassitio, 1982; Hanninen, 1982).

It must be stressed that interpreting a test battery such as this in the context of other current and past psychological information is a specialist task, and should be carried out by a psychologist with appropriate training and experience in clinical neuropsychology. However, a clinical psychologist with good interviewing skills, a postgraduate training in neuropsychological assessment and some practical experience in administering and interpreting qualitative and quantitative neuropsychological data following closed head injury, could administer some of these tests as a screening measure before deciding whether to refer on for a more extensive assessment. As pointed out by Hawkins (1990), the various core batteries commonly used in the assessment of OSN may result in the overlooking of important impairments that are not covered by these batteries. As the WHO/Nordic Council (1985) note, the core battery should be supplemented as necessary and this is a task for a specialist in neuropsychology with knowledge of OSN.

A Case Series

Background of Subjects and Assessment Procedures

In order to see if any distinctive profiles of emotional and neuropsychological functioning emerged in Type 2 OSN, the results of the assessments of a consecutive series of 13 cases of acknowledged or probable Type 2 OSN referred to me by a Medical Specialist in Occupational Health were examined. Three of the subjects were printers, three were painters, six were involved in trades that used glues, dyes, cleaning and degreasing agents, fibreglass and paints (two boat builders, a screen printer, a leather worker, a rubber worker, an aircraft maintenance worker) and one was a pest controller.

All were men aged 25 to 64 years with confirmed histories of 10 to 40 years of occupational solvent exposure. All had been exposed to multiple solvents, but TCE, Methyl Ethyl Ketone

Table 2: A selection of neuropsychological tests useful for the assessment of Type 2 OSN. A minimal test battery would include one or more tests to assess each of the functions listed. Generally, tests in groups 1, 2 and 3 would not be impaired but impairments are possible in any of the other groups of tests. Descriptions, normative data and references for the tests can be found in Lezak (1983) and Spreen and Strauss (1991).

SUGGESTED TESTS	MAIN FUNCTIONS TESTED
1. New Adult Reading Test (NART), WAIS-R Vocabulary subtest.	Estimate of premorbid IQ.
2. WAIS-R Picture Completion.	Simple visuospatial perception.
3. WAIS-R Digit Span forward.	Immediate memory and attention.
4. WAIS-R Digit Span back, Cancellation test, Continuous Performance test.	Attention, concentration, mental tracking, vigilance, reaction time.
5. Finger Tapping, Pegboard test.	Fine motor coordination, motor speed.
6. WAIS-R Digit Symbol.	Psychomotor speed.
7. WAIS-R Block Design.	Complex visuospatial perception, abstraction and speed.
8. Rey-Osterreith Complex Figure, with 30–45 minute delayed recall.	Complex visuospatial perception, motor coordination, planning ahead, longer-term visuospatial memory.
9. Wechsler Memory Scale Logical Memory and Associate Learning, Rey Auditory Verbal Learning Test, Californian Verbal Learning Test, Verbal Selective Reminding.	Longer-term verbal memory (including long-term storage, retrieval and recognition memory).
10. WAIS-R Similarities, Oral Word Fluency, Trail-Making Test, Wisconsin Card Sorting Test, The copy trial of the Complex Figure.	Frontal-lobe functions including abstract thinking, organisation and planning, changing set, perseveration.

(MEK), toluene and styrene were common. In all cases the assessment took place following a solvent free period, usually of many months or years, but in some cases of 2 to 6 weeks only. All subjects had a long, stable work history and the eleven men below retiring age were under considerable pressure to continue at their trade in order to keep up with financial commitments. In three cases, other causes may have contributed to the mens' problems; a past (but not current) history of heavy alcohol intake in two cases and two minor head injuries 3 years previously in a third case.

Malingering and social expectancy effects were ruled out as best as possible in all cases on the basis of a combination of factors. These included a strong desire to continue working, a determination to persevere and do well on the neuropsychological tests, a test profile that was consistent with OSN and inconsistent with malingering or social expectancy effects, no or relatively minimal monetary compensation to be gained (i.e., from Accident Compensation Corporation) following an assessment of impairment, and clear evidence of months to years of distress and anxiety as a result of the symptoms, and increasing difficulties in working and in family relationships, confirmed by family members in many cases.

It was a common occurrence in the initial interviews for the client to break down in tears as he told his story. This was particularly poignant as it was clear that most of these men were not accustomed to crying and were often very embar-

rassed by their display of emotion in front of a stranger (and in some cases their wife who was present at the interview). True to the stereotype of the New Zealand "working class" male, most of the men tried valiantly to regain their "stiff upper lip" so they could get on with the neuropsychological tests. Whilst this sometimes worked, in some cases it was clear that some preliminary counselling was necessary before the necessary information could be gathered. When the client was particularly upset, the neuropsychological assessment was rescheduled for a later date. Some of the men, often with their partners, later returned to the Psychology Clinic for therapy sessions to help them cope with their problems.

Following the initial interview to assess history and psychological states, the men were given a neuropsychological assessment which included as a minimum the WAIS-R, the Wechsler Memory Scale, a more difficult verbal memory test (Oral Selective Reminding), the Rey Complex Figure copy and delayed call, and one or more tests of frontal-lobe abilities (Oral Word Fluency, Trail-Making test, Modified Wisconsin Card Sorting Test). Care was taken when interpreting test scores to take into account the premorbid abilities of the individual and to look at the pattern of scores (i.e., the differences between scores) rather than simply looking at absolute subtest scores, or overall IQ scores. For example, if an individual obtains age-scaled scores of 6, 7 and 8 across all the subtests of the WAIS-R, it is not valid to interpret a Digit Symbol score of 6 as impaired, even if many other individuals with OSN also score a 6 on Digit Symbol, but have scores of 10 and 11 on the other subtests. In this series, a score was judged to be impaired if it fell more than 1SD below the "expected" score for that individual. The WAIS-R Vocabulary, Information, and Picture Completion scores should not be affected by Type 2B OSN and were therefore used as "holding" scores with which to compare the other WAIS-R subtest scores. The scores of these holding subtests, along with the NART, were also used to assist in estimating each individual's premorbid IQ.

Acute Symptoms of OSN

Ten men reported that they had suffered acute physical symptoms of neurotoxity when working with solvents, including nausea, vomiting, diahorrhea, tingling in the fingers, dizziness, and dermatitis, all of which abated when they removed themselves from the solvents for a number of days or weeks. In cases when the men returned to the work environment, often after a holiday break, the symptoms returned in force and within hours or days of their return. For example, a leather worker complained of feeling severe anxiety when he smelled any spirit-based toxins such as leather dyes, petrol and even some felt-tipped pens. A screen printer described the acute effects of strong glue (toluene & xylene) as "dramatic". He would feel "quite zombied" by the end of the day and often experience "a weird feeling of not really being anywhere and a feeling of nausea and being incredibly tired when I got home".

An unusual occurrence in one printer who was exposed to MEK and other industrial solvents, was the onset of severe and sustained withdrawal symptoms on two occasions when he had a holiday from his work and some alleviation of these symptoms for a week when he returned to printing. The apparent withdrawal symptoms included severe leg tremors, lethargy, nausea, headaches, depression and memory loss, and were more severe and debilitating than the neurotoxic symptoms he suffered whilst in the solvent environment. At one point when he decided to leave printing permanently, he suffered severe withdrawal symptoms for 3 months such that he had difficulty leaving his bed and then improved with the help of a fitness program to the extent that 8 months after leaving his job he felt better than he had since his youth. He later returned to the trade, but in a situation where he was able to reduce his use of neurotoxic solvents significantly and was very careful to use protective clothing. Since then he has been able to survive "in a semi-mobile state". On assessment, following a 2 week holiday from his job, he impressed as a physically fit and highly intelligent person, but nevertheless still demonstrated significant and typical neuropsychological impairments of verbal and nonverbal memory, psychomotor slowing and visuospatial deficits. In addition he continues to experience excessive fatigue, headaches and occasional bouts of depression.

Symptoms of Type 2A OSN

A range of psychological and emotional symptoms have been postulated to characterise Type 2A OSN and all 13 men suffered chronically from some of these. All the men were frustrated by low energy levels and decreased motivation to get on with their work or recreational activities. A number commented on how energetic they had

been in the past. For example, one previously successful self-employed man said that in the last year of his working, he would often wake in the morning, look in his diary to find he had a job on that day and then return to bed and neglect to do the job or even telephone the client to cancel it. He commented that he knew he would lose valued clients this way, but simply did not have the energy to do anything about it. Excessive levels of fatigue was a related symptom experienced by 10 of the men. Many would have difficulty getting up in the morning, needed to sleep in the afternoon and went to bed early (e.g., 8pm) at night.

Eleven of the men said they were frequently irritable with little cause with family and friends and at work, unwarranted mood swings were reported by six men and pervasive and debilitating feelings of anxiety by three men. One man, a printer, had recently begun to experience symptoms of agoraphobia and another, a leather worker, was continually anxious that he had forgotten an appointment to the extent that during his neuropsychological assessment he had to telephone home and check with his wife. He said this was a frequent problem for him and much of the time he was unable to pinpoint what he was anxious about.

Nine of the men suffered bouts of depression; three were currently taking anti-depressive medication. Three others had suffered depression in the past, but did not do so now. A year following his neuropsychological assessment, one of the youngest men who had been a spray painter for 10 years was admitted as a psychiatric inpatient suffering from severe depression. Some months following his admission when it was considered that his depression had abated, a further neuropsychological assessment was strongly suggestive of dementia. He has continued to decline intellectually and emotionally since then, and would now fit the criteria for Type 3 OSN. While it is possible that his psychiatric and demented condition is independent of his exposure to solvents, the acute symptoms consistent with solvent neurotoxity he suffered prior to leaving his job, the constellation of emotional and neuropsychological symptoms he demonstrated 5 months after leaving his job and before becoming severely depressed, and his youth (in his late twenties), support the diagnosis of Type 3 OSN, at least as one major contributer to his dementia.

Two men admitted to ongoing uncontrollable violent behaviours towards their families, though they had not been violent when younger. Two additional men said that uncharacteristic episodes of violence and aggression had been major problems for them in recent years, but they were no longer violent since removing themselves from solvents and seeking therapy to help them with anger control. In three cases the men said their violent behaviours had been the primary reason for the breakup or near breakup of their marriages. As the supportive wife of the fourth (currently violent) man said, her husband (who had been a painter for 23 years) was normally a gentle, happy person, but there had been a change over the last few years when he was often angry and had sudden episodes of violence towards the children. As a result the family have been in family therapy and the father requires ongoing psychiatric help.

Symptoms of Type 2B OSN

All 13 men complained of debilitating problems of memory and concentration in their day-to-day life. Often it was their concern about these symptoms that brought them to the attention of a health professional. They may also have been suffering from a number of acute physical symptoms for which they did not seek medical help, often because they feared being forced to leave their jobs. They often blamed their symptoms of irritability and fatigue on general work and family stress. However, when the problems of memory became so pervasive that they and their families could no longer ignore them or put them down to a normal ageing process, their concerns about a possible neurological (e.g., a tumour, or dementia) or psychiatric condition (e.g., "going crazy"), forced them to seek help. For example, one man suffered severe physical symptoms for years, including vomiting, profuse sweating, dry wretching, diarrhoea, a tingling and burning sensation of the skin and scaly patches of skin, and was aware that these were related to his work with solvent-based insecticides. On one occasion he experienced marked hallucinations on a hot day in an enclosed space when lying directly under a surface on which he was applying a kerosene-based insecticide. In spite of this he did not seek medical advice or give up his work until he realised, on talking to his wife, that he had a total memory blank for a wedding of a close relative he had attended 5 years previously.

His daily problems with memory were similar to those reported by most of the other subjects. He had difficulty remembering the mixing in-

structions for his pesticide solutions, even though he had mixed them many times. He frequently could not remember what he was about to do, or what he went into a room to get. At times, he has taken a stove apart in order to apply an insecticide, and after putting it back together again has pulled it apart again having forgotten that he had already done it. Similarly, a printer said he often started one project and then moved onto another without either realising the change or finishing the first project. Forgetting appointments was a common problem and often a diary did not help as the men would forget where they had put it, or forget to keep it with them.

The inability to organise their lives and plan ahead was another common complaint. Though this is a consequence of a poor memory in some cases, it is also suggestive of some frontal-lobe disturbance in others. A leather worker said he had stopped working after 15 years because of an increasing difficulty organising the running of his business, due to memory problems and an inability to organise more than one thing at a time or to plan anything in advance.

Neuropsychological assessment provides some objective evidence for impairment of functions that are causing difficulty in daily life and that have been postulated to characterise Type 2B OSN. The majority of men, whatever their occupation and the mix of solvents they were exposed to demonstrated both verbal and nonverbal recent memory (new learning) deficits (12 men) and 11 men were impaired on Digit Symbol, indicating psychomotor slowing. Poor concentration and mental tracking ability was apparent in nine men who had poor Digit Span back scores. Eleven men had difficulties with the copy of the Rey Complex Figure (scores of 2SD or more below their age mean), possibly suggesting some impairment of visuospatial ability, although in some cases poor copies were likely to be associated with poor planning strategies, a problem usually associated with frontal-lobe dysfunction. Indeed, eight men had some indications of mild to moderate frontal-lobe impairment on more than one test (Word Fluency, Trail-Making test, Card Sorting, disorganised copy of the Rey Complex Figure).

In marked contrast to their poor performances on tests requiring concentration, memory, psychomotor speed, forward-planning abilities, and complex visuospatial abilities, all men performed normally (i.e., relative to estimates of their premorbid abilities) on tests of well-learned verbal material (WAIS-R Information, Vocabulary, & Comprehension), simple visuospatial perception (WAIS-R Picture Completion) and on tests of verbal and nonverbal visual reasoning and abstraction (WAIS-R Similarities, Picture Arrangement, Block Design & Object Assembly).

Three men had lower scores on most of the Performance tests relative to the Verbal tests, resulting in an 11 to 16 IQ point difference between the Verbal and Performance scales. This is consistent with psychomotor slowing, given that the Performance subtests are timed, and may also indicate problems with performing novel, visuospatial tasks, a common finding in cases of diffuse cerebral encephalopathy. The Arithmetic subtest was low for a number of subjects, but as this test relies on concentration, a good working memory and an adequate formal education in arithmetic, as well as the ability to calculate, it is not a useful measure of any specific function.

Discussion

The majority of men in this case series demonstrated definite cognitive impairments on many of the tests hypothesised to be associated with Type 2B OSN. On careful psychological assessment, most of the men also demonstrated some of the psychological symptoms hypothesised to characterise Type 2A OSN. Only one of the 13 cases, a printer and glassworker of 16 years who had been exposed to a mix of solvents, but particularly MEK and toluene, demonstrated a pattern that was clearly more aligned with the psychological and emotional pattern of Type 2A than the cognitive pattern of Type 2B. Even so, he showed some verbal and nonverbal memory deficits, though it is possible these were a result of his depressed mood rather than memory deficits per se. His relatively young age of 31 may have had some bearing on his presentation, if age interacts with the neurotoxic effects of solvents to cause greater cognitive impairment.

Similarly only one of the 13 men demonstrated a Type 2B pattern; that of greater cognitive impairment and fewer psychological and emotional impairments. This 44 year old man was a boat builder and fibreglasser, and had been exposed to styrene and toluene among other solvents over 20 years. Although he did not complain of depression and extreme fatigue, he did complain of many acute neurotoxic symptoms as well as ongoing irritability and intolerance to alcohol. Thus even he is not a clear case of Type 2B OSN. The

other 11 men were plagued equally by psychological and neuropsychological symptoms, and could therefore not be classified more specifically than as undifferentiated Type 2 OSN. This case series, therefore, does not support the diagnostic research criteria hypothesised by the 1985 International Solvent Workshop (Baker & Seppalainen, 1986).

It is often impossible to say with absolute certainty that cognitive and psychological symptoms are the result of neurotoxicity. One reason for this is the complex context the symptoms are often embedded in. For example, by the time the problems are assessed, the client's life may well be in disarray in both personal and work areas, making it difficult to tease apart the organic and psychological causes of depression, fatigue, poor concentration and memory problems in daily life. A second reason is the difficulty in demonstrating a clear correlation between deterioration of function and brain damage, as for example visualised by Computerised Tomographs (CT) or Magnetic Resonance Images (MRI) of the brain. While there are studies that show diffuse brain damage in individuals who demonstrate clear cognitive deficits as a result of toluene exposure (Cavanaugh, 1985; Fornazzari, Wilkinson, Kapur, & Carlen, 1983) and in painters who have been exposed to a mix of solvents (Arlien-Sobert, Bruhn, Gyldensted, & Melgaard, 1979; Bruhn, Arlien-Soberg, Gyldensted, & Christensen, 1981), many individuals who have equally severe cases of OSN do not show central nervous system (CNS) changes on CT or MRI. These inconsistencies in research and clinical findings may in part be explained by the different mechanisms used to damage the nervous system by different toxins, and perhaps by the same toxin in different individuals. In some cases CNS dysfunction may be caused by the destruction of neurons via the direct toxic effects of the solvent or its decomposition products, and in others by disruption of neurotransmitter mechanisms. Pre-existing conditions in individuals including systemic disease, other neurological conditions (e.g., alcohol-related damage, closed head injury) and various physical and psychiatric illnesses may make some individuals more vulnerable to neurotoxic effects than others. Gender may also influence the occurrence and/or symptoms of OSN. This case series does not include women and therefore is unable to comment on possible differences between men and women.

Given the range of toxins the men in this case series were exposed to, it is somewhat surprising that 11 of the 13 demonstrated such a similar psychological and neuropsychological profile. This profile is consistent with the lowered cortical arousal that often follows diffuse brain damage. Poor concentration and an inability to selectively process multiple bits of information simultaneously results from a poorly aroused cortex, and this in turn can lead to psychomotor slowing, difficulties in organising and planning ahead and impairments in learning new material. A lowered cortical arousal is synonymous with feelings of fatigue and low energy and it is not hard to see how a difficulty in processing multiple bits of information and sorting out what is important from background noise, can lead to irritability in a world in which we are constantly bombarded with trivia. Some specific toxins are likely to have specific and more focal effects on the brain and the memory deficits, visuospatial deficits and frontal-lobe deficits found in many of these men may, in addition, be a result of such damage. It is not possible to confirm this given the mix of solvents in all cases and the unavailability (and unreliability) of CT and MRI scans for this group.

The question of recovery and rehabilitation for sufferers of Type 2 OSN has not been satisfactorily addressed by the research literature to date. Arlien-Sobert, et al. (1979), in a longitudinal study of 26 painters exposed to solvent mixtures for an average of 28 years, found that two-thirds of these men had mild to moderate cerebal atrophy and that when reassessed neuropsychologically two solvent-free years following their first assessment, none of the subjects showed any improvement in their neuropsychological or neuroradiolgical status. Their conclusion was that a syndrome of chronic solvent exposure did exist and symptoms were likely to be irreversible once cerebral atrophy or intellectual changes were observed.

While this presents a gloomy picture, much can be done for individual clients. Obviously prevention through information, ongoing monitoring of solvent levels in the work place and correct use of protective clothing and safety procedures are the most important ways of decreasing this syndrome and it is rarely too late for individuals who already demonstrate some symptoms of OSN to take steps that will prevent further damage (preferably permanent removal from the solvent environment). Strategies can be taught to lessen the impact of cognitive impairments. The proper use of a diary to act as a memory aid is a simple

strategy, but very often fails unless the client is carefully instructed and monitored in its use until it becomes second nature. Problems with planning and organising can be alleviated with step by step instructions for various activities. As OSN can result in an intolerance to alcohol (also a solvent) and even small amounts of alcohol can further depress the under-aroused cortex, thus exacerbating many of the symptoms of OSN, it is important to monitor the intake of alcohol. If it is a problem, an appropriate programme aimed at reducing or terminating alcohol intake should be instigated.

Counselling and therapy can be of use in helping the victims with the many psychological problems that have built up around the solvent symptoms. Strategies to help fight depression and anxiety, to cope with fatigue and irritability, and to innoculate against stress can all be useful. Anger management is sometimes necessary in cases where, due to frontal-lobe dysfunction, impulsive and violent behaviours are a problem. Environmental adjustments can be made by the family to allow for the difficulties many victims have with noise levels and too many stimuli impinging at once. For example, important family discussions should take place in a quiet environment when the client is not tired, and family members as well as the client may need help to learn to read the signs that signal fatigue, irritability, depression, anger and aggressive and violent reactions. This will better enable them to work together to predict and prevent difficult situations.

Many clients will benefit from therapy to help them express and resolve the understandable anger, resentment and frustration that they feel as a result of becoming aware of the years of solvent toxicity they have been exposed to in their occupation, and grief for the loss of their health, jobs and often relationships. Marital and family therapy is very often essential, to help other family members as well as the primary victim.

Perhaps most important of all is the task of rebuilding the client's self-esteem. In many cases this will include exploring new job and recreational possibilities, ongoing assistance with job retraining and providing the client with strategies to cope with job difficulties and failures in the future, without losing his or her pride.

References

Arlien-Soberg, P. (1985). Chronic effects of organic solvents on the central nervous system and diagnostic criteria. In *Chronic effects of organic solvents on the central nervous system and diagnostic criteria* (pp. 197–218). Copenhagen: World Health Organisation.

Arlien-Soberg, P., Bruhn, P., Gyldensten, C., & Melgaard, B. (1979). Chronic painters' syndrome: Toxic encephalopathy in house painters. *Acta Neurologica Scandinavica, 60*, 149–156.

Baker, E. L., & Seppalainen, A. M. (1986). Session 3: Human aspects of solvent neurobehavioural effects. Report of the workshop of clinical and epidemiological topics. *Neurotoxicology, 7*, 43–56.

Bruhn, P., Arlien-Soberg, P., Gyldensted, C., & Christensen, E. L. (1981). Prognosis in chronic toxic encephalopathy. *Acta Neurological Scandinavica, 64*, 259–272.

Cassitio, M. G. (1982). Current behavioural techniques. In R. Gilioli, M. G. Cassitto, & V. Foa (Eds.), *Neurobehavioural Methods in Occupational Health* (pp. 27–38). Oxford: Pergamon Press.

Cavanaugh, J. B. (1985). Mechanisms of organic solvent toxicity: Morphological changes. In *Chronic effects of organic solvents on the central nervous system and diagnostic criteria* (pp. 110–135). Copenhagen: World Health Organisation.

Department of Health. (1991). *Workplace exposure standards for New Zealand.* Wellington.

Dryson, E., & Ogden, J. A. (1992). *Chronic organic solvent neurotoxicity: Diagnostic criteria.* Wellington: Occupational Safety and Health Service, Department of Labour.

Fornazzari, L., Wilkinson, D. A. Kapur, B. M., & Carlen, P. L. (1983). Cerebellar, cortical and functional impairment in toluene abusers. *Acta Neurologica Scandinavica, 67*, 319–329.

Gronwall, D., & Wrightson, P. (1974). Delayed recovery in intellectual function after minor head injury. *Lancet, 2*, 605–609.

Hanninen, H. (1982). Psychological test batteries: New trends and developments. In: R. Gilioli, M. G. Cassitto, & V. Foa (Eds.), *Neurobehavioural methods in occupational health* (pp. 123–129). Oxford: Pergamon Press.

Hartman, D. E. (1988). *Neuropsychological toxicology. Identification and assessment of human neurotoxic Syndromes* (pp. 108–159). New York, Pergamon Press.

Hawkins, K. A. (1990). Occupational neurotoxicology. Some neuropsychological issues and challenges. *Journal of Clinical and Experimental Neuropsychology, 12*, 664–680.

Johnson, L. D., Bachman, J. G., & O'Malley, P. M. (1979). *Drugs and the nation's high school students: Five year trends, 1979 highlights.* Rockville, Md. National Institute on Drug Abuse.

Juntunen, J. (1982). Neurological examination and assessment of the syndromes caused by exposure to neurotoxic agents. In R. Gilioli, M. G. Cassitto, & V. Foa (Eds.), *Neurobehavioural methods in occupational health* (pp. 3–10). Oxford: Pergamon Press.

Lezak, M. D. (1983). *Neuropsychological assessment, 2nd Edition.* New York, Oxford University Press.

Nelson, H. E. (1976). A modified card-sorting test sensitive to frontal lobe deficits. *Cortex, 12*, 313–324.

NIOSH (1987). *Current intelligence bulletin 48, organic solvent neurotoxicity.* Ohio: US Department of Health and Human Services.

Spreen, O., & Strauss, E. (1991). *A compendium of neuropsychological tests.* New York: Oxford University Press.

White, R. F., Feldman, R. G., & Travers, P. H. (1990). Neurobehavioral effects of toxicity due to metals, solvents, and insecticides. *Clinical Neuropharmacology, 13*, 392–412.

World Health Organisation, Nordic Council of Ministers. (1985). *Organic solvents and the central nervous system, EH5.* Copenhagen, Denmark: World Health Organisation.