Clinical Treatment Guidelines for Alcohol and Drug Clinicians
Co-occurring acquired brain injury / cognitive impairment and alcohol and other drug use disorders
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Published by Turning Point Alcohol and Drug Centre under funding provided by the Drug Treatment Services Unit, Department of Human Services, Victoria. The views expressed in this material are not necessarily those of the Victorian Government.

The correct citation for this publication is:

Cash, R. & Philactides, A. (2006). Clinical Treatment Guidelines for Alcohol and Drug Clinicians. No. 14: Co-occurring acquired brain injury / cognitive impairment and alcohol and other drug use disorders. Fitzroy, Victoria: Turning Point Alcohol and Drug Centre Inc.

ISBN: ??

Introduction

In late 2005 The Department Of Human Services Drug Policy Branch commissioned Turning Point Alcohol and Drug Centre to develop a set of clinical treatment guidelines for clinicians working with clients with comorbid acquired brain injury (ABI) and alcohol and drug use disorder.

The process of development for these guidelines has involved a widespread literature review, consultation with a key-informant group comprising brain injury and alcohol and drug specialists from a variety of backgrounds, and consultation with the state-wide group of alcohol and drug and ABI clinicians and consultants.

This clinical treatment guideline is made up of four parts: a literature review, a key informants issues paper, an assessment guide and a set of treatment resources.

These guidelines were developed by Dr Richard Cash, Turning Point Alcohol and Drug Centre, with assistance from Mandy Philactides, South East Alcohol and Drug Service.

Acknowledgements

We would like to thank the following organisations and people:

Martin Jackson, Latrobe University for his advice and expertise in Acquired Brain Injury

Silvia Alberti and Nicole Lee for their leadership and expertise in overseeing the ABI CTG project.

ABI Behaviour Consultancy, ARBIAS, Ballarat Community Health Centre, Caraniche, COATS, Eastern Drug & Alcohol Service, Headway, Turning Point Alcohol and Drug Centre for providing expertise for the Key Informant Interviews

Robin Fisher & Marion Simmonds, Department of Human Services for their support and guidance as part of the steering committee.

Anna Guthrie, Publications Unit, Turning Point Alcohol and Drug Centre for her editorial assistance

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ABI AOD Clinical Treatment Guidelines Part 1: Literature review

Introduction

This literature review forms the basis for development of a set of Clinical Treatment Guidelines for individuals with co-occurring alcohol and drug issues and acquired brain injury (ABI). This review focuses specifically on ABI in the context of alcohol and other drug use, although there are several limitations to the review, including:

- 1. The evidence is limited, with few controlled studies published. Most studies are case series or expert opinion papers.
- 2. Much of our understanding of ABI has been drawn from traumatic brain injury.
- Most of the available literature has focused on alcohol, with very little on ABI cooccurring with other drugs.

Both substance use and cognitive impairment in acquired brain injury are complex phenomena - making it difficult to isolate risk factors. Frequently, risk factors for acquiring an ABI as a result of head injury are similar to those for substance related brain injury, and the histories of individuals experiencing ABI may include both. Much of our understanding of cognitive dysfunction, recovery, and treatment efficacy after injury comes from the traumatic brain injury (TBI) literature. For these reasons, and due to the paucity of literature relating specifically to alcohol and other substance related ABI, it has been necessary to include literature relating to TBI in this review.

Much of the available literature has focused on alcohol, and this review will draw on this work, as well as using alcohol related brain injury as a vehicle for investigating the different theoretical models of cognitive dysfunction. Substance related brain injury is also investigated, with both commonly abused licit and illicit substances included. Whilst there are few recommendations as to specific programs of substance treatment available from the literature, general treatment recommendations are covered, and particular aspects of treatment are investigated in detail, including a detailed discussion of assessment of substance use in the context of cognitive dysfunction, and the relationship between substance use, cognitive dysfunction, and offending behaviour.

ABI: An overview

Acquired brain injury refers to a spectrum of disorders involving an injury to the brain which results in cognitive, physical, emotional, social and independent functioning (Ponsford, 1995; see also Box 1). The range of impairments arising as a result of an ABI may be permanent or temporary, and manifest across a range of levels of disability. ABI may result from a variety of causes, including traumatic head injury, infection, tumour, stroke, degenerative neurological disease, and alcohol and other substance use (referred to as substance related brain injury or SRBI).

Given that ABI refers to a wide range of possible presentations and clusters of deficits, it may be more helpful to consider cognitive impairments relating to substance use as a continuum. This understanding does not seek to minimise the complex physical, emotional and social expressions of ABI, nor minimise the difficulties experienced by individuals with ABI. Rather, a broader cognitive impairment model allows clinicians and researchers to better conceptualise the impact of substance use on brain functioning across a full range of client presentations. For example, viewing ABI and Cognitive Impairment (CI) as a continuum may assist with early intervention with clients, allow treatment to be delivered in accordance with principles of risk, need and responsivity.

Recognising sub-clinical manifestations of high-prevalence disorders such as anxiety and depression within alcohol and drug using populations and including mental health adjunct treatment to match client need has been shown to increase the efficacy of AOD interventions (REF). It is proposed that similar attention to emerging and established cognitive impairments within an AOD treatment context will similarly improve the efficacy of delivered interventions.

Box 1: Consequences of ABI

Neurological lipairment – motor, sensory and neurologic

Motor function impairment – coordination, balance, walking hand function, speech

Sensory loss – taste, touch, hearing, smell, vision

Sleep disturbance – insomnia, fatigue

Medical complications – spasticity, post-traumatic Epilepsy, hydrocephalus, heterotopic ossification

Sexual dysfunction and other alterations to sexual behaviour

Cognitive impairment

Memory impairment – difficulty with new learning, attention and concentration, reduced speed of information processing and reduced flexibility of thinking, impaired problem solving ability

Problems in problem solving, planning, organising, making decisions

Language problems – dysphasia, problems finding words, impaired reading and writing Impaired judgement and safety awareness

Personality and behavioural changes

Impaired social and coping skills, reduced self-esteem

Reduced emotional control – poor frustration tolerance and anger management

Anger and self-centeredness

Reduced insight, disinhibition, impulsivity

Psychiatric disorders – anxiety, depression, suicidal ideation, post traumatic stress disorder, psychosis

Apathy, amotivational syndrome

Lifestyle consequences

Social Isolation

Unemployment and financial hardship

Inadequate academic achievement

Lack of transportation and recreation

Difficulties in maintaining interpersonal relationships, marital break-up

Loss of pre-injury roles, loss of independence.

Prevalence of ABI

According to the 2003 Australian Institute of Health and Welfare Study, 2.2% of the Australian population were found to have an ABI (n=438,300), and of these, 157,500 were considered to have a severe or profound impairment (AIHW, 2005). The figures for alcohol related brain injury are harder to quantify, with 1993 AIHW statistics identifying 2,714 individuals with 'alcohol related ABI', although notes that the actual prevalence of this disorder is likely to be underestimated as the study relies on self-reported identification of ABI¹ (AIHW, 1999).

Substance use and ABI overview

The expression of acquired brain injury in individuals is often idiosyncratic, and appears to be mediated by a complex interaction of individual differences in substance use history, predisposing factors as well as pre-existing strengths and vulnerabilities, environmental and lifestyle factors. Needless to say, people experiencing ABI are a heterogeneous group, making classification and codification of diagnoses, assessments, treatment approaches and outcome measures difficult. Not surprisingly, this heterogeneity also impacts the ability to conduct research with clearly defined experimental groups, levels of dysfunction or standard outcome measures.

Whilst the actual mechanisms of structural and functional alterations to the brain in ABI are not fully understood, some reliable patterns of causative factors and expression of various deficits are becoming better understood. The application of neuropsychological assessments and increasingly sophisticated neuroimaging techniques have allowed the development of both structural and functional models of acquired brain injury. Increasingly, cognitive neuropsychological approaches are allowing comprehensive models of cognitive change following brain injury which allow for understandings of patterns of deficit based on the underlying cognitive processes mediating individual behaviours or functions. This review will detail relevant models of acquired brain injury

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¹Self-identification is problematic for several reasons including the general limitations of surveys using self-identification of behaviours or issues associated with stigma, as well as issues relating specifically to ABI including validity of self-report data, problem identification and insight deficits.

within the literature on alcohol in some detail, before highlighting the evidence of brain injury due to licit and illicit substance use.

Alcohol and cognitive impairment

Of all substances, alcohol features most heavily in the literature in association with acquired brain injury and the development of cognitive impairment. Alcohol is frequently implicated as a both a direct and indirect cause of traumatic brain injury (injury to brain resulting from a closed head injury such as a blow to the head from an assault, a fall or motor vehicle accident). There is considerable overlap between the TBI and ABI literature, and indeed overlap between TBI and ABI populations. Alcohol places people at higher risk of incurring a head injury and subsequent acquired brain injury, for instance being judgement-impaired whilst intoxicated and being involved in motor car accidents, falls, assaults etc (Corrigan, 1995, Corrigan, Rust & Lamb-Hart, 1995).

Alcohol's neurotoxicity (either as a direct or mediating factor in injury to brain structure or function), it's widespread availability and its abuse have combined with it's non-direct effects to elevate it to the most commonly recognised substance in the development of ABI. It should be noted that the relationship between risk factors, injury vectors, recovery and outcome is often complex, for instance alcohol use is thought to compromise both acute and long term recovery from TBI, particularly where the individual has had a premorbid alcohol problem. TBI may lead to increased use/abuse of alcohol for complex reasons explored later in this review, with the effects of increased alcohol increasing the risks of both direct and indirect ABI.

Alcohol related brain injury (ARBI) is known to manifest a range of disorders with varying severity of symptom profiles. These disorders range from mild and difficult to detect impairment to the distinct presentations of severe ARBI: Wernicke's encephalopathy and Korsakoff's Amnesia (Jacques & Stevenson, 2000). Whilst a comprehensive review of the development of understandings of ARBI is beyond the scope of this review, alcohol's role in mediating cognitive functioning is instructive and will be reviewed subsequently. The nature of the relationship between alcohol and ABI also provides an example of the difficulty in identifying causal mechanisms from the existing literature, as well as highlighting the dichotomy (perhaps false) between structural and functional models of brain dysfunction.

Spectrum of cognitive impairments with alcohol use

The relationship between alcohol use and the development of cognitive impairments has long been 'implicitly held' by both the community and the medical establishment, although the nature of this relationship (as measured empirically) is not always consistent. Within the literature some studies have highlighted an interested and possibly counter-intuitive relationship whereby light to moderate drinkers are found to have better cognitive functioning than non-drinkers (Elias et al, 1999; Mukamal et al, 2001, Zuccala et al, 2001; Kalmijin et al, 2002; Ruitenberg, 2002). This finding may represent the action of mediator variable(s) – for instance moderate drinkers may also maintain good mental, physical health and socio-economic position, both with are related to good cognitive performance (Rabbitt et al, 95; Holland & Rabbitt, 1991). Alternatively, low to moderate alcohol consumption may beneficially affect vascular function, itself associated with good cognitive functioning (Desmond et al, 1993; Launer et al, 1995; Sing-Manoux et al, 2003). Without doubt, the relationship between alcohol and CI changes direction as consumption rises, with heavy consumption reliably associated with poorer cognitive outcomes (Mukamal et al, 2001, Zuccala et al, 2001; Kalmijin et al, 2002; Ruitenberg, 2002; Britton, Singh-Manoux & Marmot, 20040, Evert & Oscar-Berman, 1995).

Evert & Oscar-Berman (1995) identify a continuum of cognitive change, with increasing cognitive problems developing progressively in correlation with the duration and degree of a persons use. This continuum is thought to include 'social drinkers' who exhibit no signs of CI, through to severe degradations to memory and other cognitive functions. Wernicke-Korsakoff syndrome is the most severe cognitive impairment arising as a consequence of alcohol abuse on this continuum. Although uncommon now, it is characterised by profound memory impairments (anterograde amnesia, or the inability to form new memories or learn new information), as well as deficits in abstraction and problem solving ability.

An implicit component of this model is the dose-dependent relationship between alcohol use and acquired brain injury, with an assumption that alcohol mediates graduated changes in brain structure, which in turn lead to the expression of cognitive impairment. This Continuum hypothesis, originally advanced by Ryan & Butters (1980) has had mixed support from studies that have directly tested it. Presumably, accurate measurement of alcohol use quantity, frequency and duration should correlate to impairments on tests of

cognition. Whilst this expected relationship has not always been consistently reported, strong associations do tend to be in the expected direction. Ryan & Butters (1980) found that performance on 'demanding' tests of memory and learning were found to lie on a progressive scale, which these authors divided into four categories:

- 1. Wernicke Korsakoff's clients exhibiting the worst performance
- 2. Chronic alcohol users without demonstrable symptoms of W-K, but who spontaneously report memory problems (without clinical signs of amnesia)
- 3. Alcohol users with no memory complaints.
- 4. Non alcohol users (controls)

Cermak et al (1974), found patterns of performance whereby alcoholics demonstrate similar performance on memory tests to control groups, and different to the performance of W-K clients. Delin & Lee (1992) reviewed the performance of 'social drinkers; - presumed to perform somewhere between abstainers and alcoholics on tests of cognitive impairment – and found that social drinkers exhibited no signs of the effects of alcohol on cognition.

Individual differences also appear to mediate the alcohol-cognitive impairment relationship. No single factor has been identified to reliably account for observed variability in the individual expression of cognitive impairments. Variables such as age, gender, diet (especially compromised thiamine levels), medical problems (eg. liver disease), emotional difficulties, and childhood behavioural or learning problems (such as conduct disorder, ADHD, antisocial personality and a family history of alcohol problems) have been advanced to account for this variability. The development of multifactorial models is limited as few studies uniformly report sufficient detail around the selection, measurement or relative contribution of factors (Parsons, 1993).

Theoretical models of cognitive impairment in alcohol related brain injury

Increasingly sophisticated imaging and histological investigations allowed closer scrutiny of brain structure as a method of investigating the underlying mechanisms of cognitive impairments. Lishman (1990) related shrinkage of the cerebral cortex and possibly the basal forebrain regions to the direct neurotoxic effects of alcohol, that is, alcohol in the

bloodstream causes direct changes in the structure of brain tissue. Lisman further noted that Thiamine deficiency, common in heavy abusers of alcohol, may result in injury to deeper structures in the brain such as the diencephalon. These two mechanisms led Lisman to hypothesise a dual sensitivity model – alcohol users who are susceptible to the neurotoxic effects of alcohol may develop permanent or transient CD associated with cortical shrinkage. Alcohol users susceptible to thiamine deficiency alone will develop a mild or transient Korsakoff state, with anterograde amnesia. Alcohol users with dual vulnerabilities will experience widespread injury to the cerebral cortex, as well as the deep brain structures, and as a result will exhibit severe anterograde amnesia as well as other lasting cognitive impairments.

Several theoretical models exist to account for these cognitive changes. The 'Structure-Function' model compares the performance of individuals with known injury to cortical regions with the performance of individuals exhibiting no demonstrable structural injury. Alternatively, the process approach (explored in section 2.7) examines the underlying nature of the observed cognitive functions or deficits, with little reference to brain structure, but rather, using models which attempt to describe underlying mental processes such as memory, attention, etc. (Evert & Oscar-Berman, 1995).

Using the structural approach with patients with known, localised areas of brain injury, (eg from stroke, tumours or focal lesions from penetrating head wounds), researchers have identified regions of the brain which, when injured, appear to be involved in mediating various aspects of cognition. This has led to certain regions of the brain being 'associated' with the patterns of deficit most reliably seen with alcohol abuse. Structural models of alcohol related brain injury have included, the "premature ageing model", the "right hemisphere" model, the "diffuse brain dysfunction" model and the "frontal lobe systems" model.

Britton, Singh-Manoux & Marmot, 2004 point out several key issues with studies evaluating these models. Many investigations are based on cohort studies comparing alcohol users with abstinent control groups, and this may introduce confounding factors as non-drinkers, particularly former problem alcohol users differ from drinkers in significant ways. Few studies are structured to appropriately match comparison groups. Similarly, few studies investigate the different expression of cognitive impairment within subgroups of alcohol users – which would allow more comprehensive testing of the potential role of

mediator variables such as socioeconomic status, general health, and emotional functioning.

The "Premature Ageing" model has identified similar patterns of cerebral atrophy in alcohol users as is found in the non-alcohol using aged population (Wilkinson & Carlen 1982). More recently Pfefferbaum et al (1992) used MRI imaging to identify increased cerebral atrophy in alcoholics compared to non-alcoholics where age had been controlled for. There appear to be two versions of the Premature Ageing model. One posits that alcohol accelerates the normal ageing of the brain, irrespective of the age at which the alcohol abuse begins. Alternatively, the Increased Vulnerability model states that vulnerability to the ageing effects of alcohol is magnified in people abusing alcohol over the age of 50. MRI evidence appears to support the assertion that older alcoholics exhibit more tissue loss than younger alcoholics, suggesting that they are more susceptible to the effects of alcohol.

The "Right Hemisphere model" hypothesises that the right hemisphere of the brain is more susceptible to the actions of alcohol². This model is based on the finding that alcoholics generally exhibit poorer relative performance and steeper decline in performance on tests of non-verbal ability than verbal ability (non-verbal tasks are typically mediated principally by the right cerebral hemisphere). For example, alcohol users show abnormally low scores on digit symbol, object assembly and block design subtests of IQ tests such as the WAIS-R which assess non-verbal information processing, thought to be largely mediated by the right hemisphere (Ellis, 1990).

Similar deficits have been identified where verbal and non-verbal materials are used in tasks which assess memory and attentional functioning. These deficits in long term alcohol users are similar to those found in investigations of clients with right hemisphere injury unrelated to alcohol use. The right-hemisphere model has also provided some support for the premature ageing hypothesis, with Ellis (1990) demonstrating that older alcoholics performed worse on the non-verbal subtests compared to younger alcoholics and older non-alcoholics.

² See Ellis & Oscar-Berman (1989) for a detailed review

There are several weaknesses with the right hemisphere model of alcohol related cognitive impairment. Ellis' (1990) results have not been replicated, and the majority of evidence does not support either version of the premature ageing hypothesis in relation to this model (Evert & Oscar_Berman, 1995). In addition, the right hemisphere model has been criticised for being descriptive rather than explanatory in that it does not account for the underlying mechanisms of damage or functional change – why is the right hemisphere of the brain particularly susceptible to the effects of alcohol? Lastly, the nature of tests used to investigate patterns of cognitive impairment may introduce artefact into models based on these tests. Sensitive tests of verbally-mediated cognitive functions have shown that alcoholics do exhibit deficits in verbal skills, meaning that the differences noted in 'hemispheric function' may be more parsimoniously explained by varying task demands rather than actual injury to one hemisphere.

The diffuse brain dysfunction model hypothesises that alcohol use leads to diffuse patterns of structural changes to the brain, with alcohol users exhibiting a wide range of cognitive impairments. Parsons et al. (1990) administered verbal and visuospatial tests comprising factors which test both left and right hemisphere functioning and found that alcoholics performed significantly worse than non alcoholic controls on all factors tested, suggesting that both hemispheres are affected by alcohol. Parsons further posits that particular cognitive impairments noted in alcohol users may arise from task difficulty alone, and/or the brains overall cognitive capacity as opposed to specific deficits arising from localised lesions in brain tissue.

Another single region model is the frontal lobe dysfunction model, which argues that alcohol use significantly disrupts those cognitive functions normally ascribed to the frontal lobes of the brain. Alcohol users show personality changes and cognitive impairments similar to individuals with frontal lobe injury unrelated to alcohol use, such as impaired impulse control, lack of insight, difficulty in adapting to change, poor performance on tests of planning, organising, problem solving and abstraction³. The most reliable finding in alcohol users which implicates dysfunction in the frontal lobe is abnormal perseverative responding – or the inability to recognise incorrect or unsuccessful strategies and alter them accordingly (Evert & Oscar-Berman, 1995).

³ For a review see Oscar-Berman & Hutner (1993).

In addition to these structural accounts of alcohol related changes to the functioning of the cortex, Sullivan identifies cerebellar atrophy (injury to cerebellum) as another reliable feature of alcohol related brain injury affecting more gross motor functions such as balance and coordination. The role of the cerebellum in the cognitive changes that occur in alcoholism is unknown but recent research has highlighted its probable involvement. (Sullivan et. al., 2000)

Process-oriented approaches to CI

Proposing an alternative to structural accounts of acquired brain injury following alcohol use, process oriented approaches seek to identify the underlying functions which are impaired in alcohol users without seeking to identify the brain structure involved. The component-process model (Parsons & Nixon, 1993, Nixon, 1993) is an example of a process-oriented approach to memory dysfunction. This model proposes two information stores comprising the episodic and knowledge information stores, with three component processes operating within each store – availability, access and efficiency.

Availability of information refers to the persistence of longevity of information over time, access refers to the ability to retrieve previously acquired information, whilst efficiency refers to the ability to use accurate and relevant information whilst ignoring or disregarding inaccurate or irrelevant material as appropriate. Under the Component-Process model, the nature of expressed deficits in an individual is dependent on the functioning of the component processes. For example, a deficit in the availability component may be characterised by impairments in accuracy of recall, measured by increased error rates over expected values.

Access impairments may be characterised by slowing of behaviour which can be measured via slower response times in impaired clients over expected values. Efficiency impairments may manifest in a person being less able to filter irrelevant material, and may be evidenced by over-inclusion and confabulation.

According to Evert & Oscar-Berman(1995), it is likely that efficiency processes within the knowledge information store are particularly susceptible to the effects of alcohol misuse. Nixon & Parsons (1991) investigated differences in abstraction abilities between alcoholics

and controls. Abstraction abilities are thought to represent knowledge store processes, and refers to the ability to comprehend organising principles, or the ability to draw specific conclusions from general information. Nixon & Parsons used the 'Plant Test" – a tool with more ecological validity (or 'real world applicability' than other measures of abstraction such as the Wisconsin Card Sorting Test (Berg, 1948; Grant & Berg, 1948). Subjects are shown healthy and unhealthy plants with descriptions of their maintenance, then are asked to rate the healthiness of an unseen plant given only a description of it's maintenance.

Nixon & Parsons predicted that availability deficits would be characterised by a subject's inability to correctly identify the relevant variable, that efficiency deficits would be characterised by the inclusion of irrelevant variables. Their results showed no statistically significant differences between alcohol users and non-using controls in correctly predicting the unseen plant's condition, however, the alcoholic subjects experienced significantly greater difficulty in identifying relevant predictor variables and screening irrelevant material. According to Nixon & Parsons (1991), this represents efficiency factors in the knowledge information store being compromised in their alcoholic experimental group.

To summarise this section on theoretical models of cognitive impairment following substance (alcohol) misuse, several structural models have been advanced to account for the demonstrable cognitive impairments affecting long term alcohol users. The hemispheric, frontal lobe dysfunction and premature ageing models have not been well supported in the literature (Evert & Oscar-Berman 1995). Whilst a combination of these structural understandings with functional models such as the Component Process model is likely to offer the most comprehensive picture of ARBI, it is possible that these models will remain descriptive in nature, and will not offer the level of sophistication in causal attribution necessary to make be clinically relevant in individual treatment.

Other illicit and licit substances and ABI

Whilst the actual mechanisms for alcohol related brain injury are complex and not fully understood, knowledge around the actual and potential harms arising from other substances are less well mapped. It is not the purpose of this review to address vectors for brain injury for the complete range of psychoactive substances, although a review of the evidence for commonly used and abused substances is instructive. Substance related

brain injury (SRBI) relates to persistent structural and functional alterations to the brain or parts of the brain as a result of substance use. Usually SRBI relates to illicit substances, although impairments are also associated with prescription medications such as benzodiazepines and are also subsumed under SRBI. Whilst there are acute effects of intoxication and withdrawal from various substances, which may share presenting features with ABI, it is the permanent or irreversible effects which are classed as SRBI. The following is a brief description of the cognitive and physiological effects of the main types of illicit substances or substances of abuse: cannabis, stimulants, opiates, inhalants, and benzodiazepines.

Cannabis

Considerable research has addressed the issue of cannabis related harms, most notably psychotic illness, given the reportedly high prevalence of use of this drug. According to the 2004 Household survey (AIHW, 2005), Cannabis is the most frequently used illicit substance in Australia – with lifetime use rates estimated at 33.6% of the population aged over 14, and with approximately 11.3% reporting recent use (last 12 months). Whilst cannabis' acute effects are known to compromise several cognitive functions including attention, memory, and executive function (Solowij, 1998), conclusive evidence for the deleterious long-term effects of cannabis on cognition is difficult to ascertain from the literature. Chronic cannabis users are likely to experience similar cognitive deficits within various subsystems of attention and memory related to their ongoing use, (Solowij, 1998, Block & Ghoneim, 1993; Solowij, et al., 2002)

Most literature investigating long term effects has ruled out the presence of gross impairment (Pope, Gruber & Yurgelun-Todd, 1995; Solowij, 1998), such as is found in long term alcohol users, although impairments to specific areas of cognition remain controversial (Pope, 2002). The reason for this lack of conclusive evidence is in part due to the methodological limitations on retrospective studies of substance users, (similar to the concerns previously described about alcohol research) and as the less global, subtler nature of the deficits related to cannabis.

The lack of a 'baseline' measurement of cognitive ability before cannabis users commenced use limits the causal inferences that can be drawn from retrospective designs. Further potential confounds to establishing relationships between cannabis use and cognitive impairments relate to the selection of experimental and control groups samples.

In 'matched control designs' it is typical to attempt to match the experimental (i.e. a treatment seeking group) with a control group (usually non-clinical) on as many variables as possible – age, socioeconomic status, education. However, the reality of experimental design and sampling means that often it is very difficult or unfeasible to satisfactorily control for all potential confounding variables (Pope, 2002). Pope (2002) points out that studies often exclude or comorbid substance problems or psychotic disorders without excluding high prevalence disorders such as anxiety and depression, or clients receiving large doses of medications such as benzodiazepines – each potentially contributing to poor performance on tests of cognition. Exclusion of all co-morbidity within cannabis user sampling, however, risks selecting a artificially high-functioning group, and may serve to obscure genuine deficits in the using population as a whole.

Psychostimulants

Few studies have addressed the question of cognitive impairment following psychostimulant use. Cocaine use has been associated with memory impairment, impaired visuospatial skills, concentration and slowed speed of information processing (Trx ref 2002). High doses of methamphetamine can cause long term neurochemical and structural changes to the brain with loss of tissue in the pre-frontal cortex, impaired signs of brain activation in functional brain imaging and cognitive impairments in areas such as memory and executive functioning (Rogers et al., 1999).

Similarly, MDMA has been identified as a neurotoxic substance in both animal and human experiments (Ricaurte et al., 1992; McCann, et al., 1998). However, linking the functional deficits associated with changes in brain metabolism or structure to the psychostimulant use is again difficult due to methodological limitations of studies, including the heterogeneity of clinical samples of psychostimulant users (Gouzoulis-Mayfrank et al, 2000). As with other single factor designs, it is very difficult to reliably isolate psychostimulant use and its potential effects from the wide range of other risk factors for developing cognitive impairment.

Opiates

Traditionally, opiates as a class of substance have been held to be largely free of damaging effects to cognitive functioning, as they are not directly neurotoxic substances. However, the risk of overdose and respiratory arrest leading to anoxic brain injury (no oxygen reaching the brain) is significant for opiate users. Similarly, 'respiratory

depression' during periods of acute intoxication can lead to hypoxic brain injury (lower than optimal levels of oxygen reaching the brain). Overdose and respiratory depression or failure is particularly likely if opiates have been mixed with other central nervous system depressants, or the strength of the opiate is unknown prior to use – both common situations for many opiate users. The pattern of cognitive impairments following prolonged hypoxia includes slowed information processing, attentional deficits, impaired complex reasoning skills, concrete and inflexible cognitive styles, and disturbances to memory function (Darke, Sims, McDonald & Wickles, 2000).

Despite the common difficulty in weighing the relative contributions of traumatic brain injury as well as comorbid alcohol use to the development of acquired brain injury, opiate-related overdose and hypoxia are significant vectors for the development of a substance related cognitive impairment or acquired brain injury.

In a recent study investigating the potential cognitive effects of methadone maintenance programs, Darke et al (2000) demonstrated a group of methadone maintenance clients showed poorer performances across a range of neuropsychological tests of information processing, attention, short term and delayed visual memory, short term and delayed verbal memory and problem solving. However, Darke points out that the group of methadone maintenance clients tested had significantly higher rates of alcohol dependence, heroin overdose and head injury than the non-opiate dependent controls, who were matched for age, gender and education. In this study, the likelihood of cognitive impairment was better predicted by alcohol dependence history as well as exposure to overdose events.

Inhalants / solvents

Organic solvents inhaled or sniffed constitute a class of substances with significant risks for development of cognitive impairments and potentially, acquired brain injury. The neurotoxicity of organic solvents has been demonstrated via neuroimaging, neurophysiological and neuropsychological assessment (Baker, 1994; White, 1992; White & Proctor 1997). White and Proctor (1997) summarise the findings of structural changes in the tissue of the frontal lobe and cerebellum, white matter lesions, as well as neuropsychological changes including alterations to attentional capacity, executive functioning, visuospatial skills, short term memory and the regulation of mood.

Apart from the direct neurotoxic effects, there are also secondary risk factors for developing ABI via associated with of solvent use. Depression of the breathing, vasovagal inhibition (slowing of the heart rate and fall in blood pressure), can lead to loss of consciousness, hypoxia, and potentially brain injury. Inhalant intoxication is also associated with accidental injury, such as falling, inhalation of vomit, asphyxia from plastic bag, and traumatic brain injury from risky behaviours undertaken whilst intoxicated.

Benzodiazepines

The family of depressant drugs known as benzodiazepines are one of the most commonly prescribed medications for anxiety, insomnia, panic disorder, as well as some psychotic states, depression, social phobia, OCD, substance withdrawal, and as a side effect medication for other antidepressants and antipsychotics. Additionally, benzodiazepines are a commonly 'abused' licit substance, with their sedative and hypnotic effects valued either as a drug of choice, as a substitute for other CNS depressants, or as part of a polysubstance using pattern.

Benzodiazepine dependence or abuse is often clinically difficult to manage due to widespread availability of the drug, and perceptions that as the substance is 'prescribed' it is innocuous or safe. Benzodiazepines are frequently implicated in causing or contributing to CNS overdose. Withdrawal from benzodiazepines can also be clinically complex, given rebound effects of withdrawal and likely heightening of the problematic symptoms which led to the initiation of benzodiazepines use. For this reason individuals using benzodiazepines often find it difficult to cease use, and physiological dependence is often cited as the main disadvantage of their use (Spiegel, 1999). Between 0.5 and 5.8% of the population are thought to use benzodiazepines on a long term (greater than one year) basis.

In a recent meta-analysis, Barker et al (2004) investigated the effects on cognition of long term benzodiazepine use, citing both increasing evidence of detrimental cognitive effects as well as a degree of controversy about this relationship. Barker and colleagues outline frequently reported effects of long term benzodiazepine use in the following areas of cognition:

- Memory
- Concentration and Attention

- Visuospatial abilities
- General intellectual functioning
- Motor speed and fine motor control
- Reaction time
- Arousal
- Psychomotor speed
- Conceptual tracking abilities
- Speed of information processing

Similarly to the literature addressing alcohol mediated changes in cognitive functioning, Barker et al (2004) highlight significant heterogeneity in the literature, limiting the generalisability and comparability of studies. These limitations include the divergence in samples with respect to reason for using benzodiazepines, psychiatric diagnosis, length of use (and definitions of 'long term use'), range of other substance used, ranges of doses, types of benzodiazepines studied, and the types of instruments used to measure for cognitive impairments.

Barker et al (2004) indicate that many studies tend to be retrospective and cross sectional in design, limiting the degree of causal inference which can be made, and lastly, many studies fail to sufficiently differentiate between the acute and chronic effects of benzodiazepine use i.e. 'intoxication effects' are not sufficiently demarcated from lasting, permanent effects.

Despite these limitations in the extant literature, the Barker et al meta-analysis included studies with sufficiently robust design, allowing the calculation of effect size. Consistently, long term benzodiazepine users were found to be more impaired than control groups across all cognitive indices measured, with all effect sizes significant, ranging from –1.3 to –0.4.

Summary – Substance related brain injury

The literature addressing substance related brain injury does not offer the same level of certainty regarding neurotoxicity as exists for alcohol. Whilst the substantive evidence for drug related cognitive impairment may be less conclusive, the 'clinical' picture will

necessarily be affected not only by the users direct drug using behaviour, but by the range of associated risk factors and injury vectors associated with a regular substance using lifestyle. Of particular note are the anoxic brain injury vectors associated with CNS depressant abuse, particularly in poly-substance using individuals, and the role of disinhibition and risk taking in facilitating injury or assault. 'Clinical wisdom' often points to daily use of a substance (to intoxication) for 10 years or more as being associated with probable cognitive impairment, although in this very vague formulation the relative contribution of neurotoxicity, associated risk, and the individual's level of rehearsal and application of complex cognitive skills is largely unknown.

Recovery and abstinence

The effects of heavy, prolonged use of alcohol and certain other substances may be associated with changes to brain structure or function, but the role of abstinence in facilitating recovery in function is less well understood. At cessation of continued heavy alcohol use, users show poor performance on cognitive tests, related to the 'general malaise' of acute withdrawal. Improvements are generally seen following this period, continuing for weeks, months or years, with some users never recovering full cognitive function (Goldman, 1995).

The rate of improvement and the ultimate level of functioning reached vary with the type of cognitive processing involved, and the age of the individual (Goldman, 1995). Other factors thought to mediate cognitive recovery relate to the general health and environmental factors experienced by the individual. These factors include nutrition, exposure to social and cognitive stimulation. This recovery may not manifest continuously or steadily, meaning sensitive testing over two or more occasions is necessary to plot improvements. Wherever multiple testing of cognitive abilities is conducted, practice effects need to be minimised in order to ensure that the testing is measuring true cognitive recovery, rather than the individuals experience with the test being used.

Practice effects can be controlled for clinically by using 'alternate forms' of tests (where more than one set of equivalent materials or stimuli is available), and experimentally by using a matched group design with staggered measurement intervals for the two groups (Goldman, 1995). Multiple assessments of cognitive ability via neuropsychological

assessment may be difficult to facilitate. The use of selected subtests from neuropsychological batteries may allow assessments to target problematic areas of cognition, alternatively self-reported cognitive difficulties scales or observation / collateral reports, may facilitate ongoing monitoring. Accurate tracking of changes in an individuals cognitive functioning, be it improvement or decline is however, crucial for planning effective and appropriate treatment and support services, as well as assessing the effectiveness of treatment or rehabilitation programs.

Clinical complexity – Acquired brain injury, traumatic brain injury and substance use

The nexus of substance abuse and cognitive impairment due to brain injury is complex and multifactorial. Added to the direct effects of alcohol and other substance use on the brain already discussed, there are the indirect effects of substance use (such as poor general health, compromised self-care, psychosocial instability), as well as associated risks for developing functional deficits through traumatic brain injury. The association between substance use and traumatic brain injury is well documented in the literature. In a recent review, Taylor, Kreutzer, Demm & Meade (2003) outline the complexity of this association. The literature identifies substance use, particularly alcohol use, as a major risk factor for sustaining traumatic brain injury, but also identifies post-injury substance use as a major feature of the management of rehabilitation and recovery⁴.

According to Corrigan (1995), 2/3 of individuals incurring a traumatic brain injury have had a history of pre-injury substance abuse, and 1/3 to ½ are actually intoxicated at the time of their injury (which can compromise immediate treatment and complicate recovery). A broader sample of the literature indicates levels of pre-injury alcohol abuse range between 44% and 79%, whilst between 21 and 37% report histories of illicit substance abuse prior to their injury. Kreutzer et al (1991) stated that 20% TBI clients reported abstinence from illicit drugs, 12 claimed light or infrequent use, 30% moderate use and 38% heavy use, with cannabis the most commonly endorsed illicit substance used.

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⁴ See Taylor, Kreutzer, Demm & Meade (2003) for a review.

Drubach et al (1993) used DSM-IV criteria to assess the substance use status of TBI clients and found that 33% met criteria for alcohol abuse prior to their injury, 8% for substance abuse, and 29% for combined substance and alcohol abuse diagnoses. Taylor et al (2003) note that the literature consistently indicates that histories of alcohol abuse, lack of post high-school education, being male, unmarried, and unemployed are significant risk factors for incurring a traumatic brain injury. Kretzer et al. (1996) note that individuals intoxicated at the time of their injury are more likely to have pre and post injury substance abuse problems, although increasing levels of injury can decrease alcohol use. Between 25% and 75% of admissions for TBI are likely to be intoxicated at time of injury (Corrigan, 1995; Corrigan et al, 2001; Kreutzer et al 1991).

Following TBI the evidence for problematic substance use is less definitive. Several studies have identified lower levels of substance abuse following injury (Kreutzer et al 1990a; Kreutzer et al 1991b; Kreutzer et al, 1995, Corrigan et al 1995b). However, there are some factors which appear to mediate this relationship, relating to the severity of the injury and the length of follow-up of the study. Kreutzer et al, (1996b) linked increasing impairment with decreasing alcohol use. Kreutzer et al, (1996a), with a sample of 73 younger TBI clients showed that abstinence rates for alcohol use were lower at a 28 month follow up than they had been at 8 month follow-up.

Similarly, Corrigan et al (1998) showed that whilst their sample of 95 TBI clients were uniformly abstinent at 2 years post injury, one quarter had developed alcohol or illicit substance abuse problems when they were followed up at five year post-injury. There are a myriad of factors contributing to predict likely patterns of substance use post-injury, psychological, social, economic and also pragmatic. Taylor et al (2003) summarised risk factors for developing alcohol or drug problems and signs for identifying these problems early, these factors are presented in Box 2.

Box 2: Risk factors for substance abuse following traumatic brain injury

- Pre-injury history of alcohol or substance abuse
- Intoxicated status at time of injury
- History of legal problems related to substance use
- Substance abuse problems present in family or significant peer network
- Denial or lack of knowledge of dangers or substance abuse
- Age less than 25
- Physically healthy with income and transport available

Subtle signs of substance abuse in traumatic brain injury

- Irregular vocational or educational history frequent changes of employment, absenteeism, tardiness
- Frequent participation in social activities involving alcohol and drug use
- Denial or minimisation of alcohol or substance issues
- Self-Identifying a need to moderate or control use
- Express guilt about alcohol or substance use
- Becomes emotive when probed about alcohol or substance use
- Early morning alcohol or substance use
- Preoccupation with alcohol or substance related topics, steers conversation towards use.

(From Taylor et al, 2003)

Indicators of alcohol or substance use status following traumatic brain injury are likely to be affected as much by the nature of the injury as by the nature of the environment or rehabilitation program offered to TBI clients. The perhaps counter-intuitive finding that those with fewer impairments demonstrate more alcohol and substance use issues may be an artefact of the fact that those with more severe injuries are often placed in more structured, supervised environments, with longer periods of follow-up support in the community. Whilst the less-impaired individual may have fewer severe cognitive impairments, they may also be less supported, have more insight into the nature of their loss, suffer less understanding and support from peers or family, and be more able to access alcohol and substances as a way of managing their post-injury experience.

Thus a comprehensive history of head trauma is a vital assessment domain in planning alcohol and drug interventions, just as assessment of substance use is recognised as a vital assessment domain in planning recovery from traumatic brain injury. Accurate assessment of risk factors for involvement in head trauma and substance use, such as impulsivity, need to factored into a range of decisions regarding appropriate clinical management, including appropriateness of different types of treatment context, accommodation, and social support programs.

Assessment and diagnosis overview

Assessment of substance abuse is frequently an uncertain prospect, even with no demonstrable cognitive impairment interfering with the process of gathering data. In populations not exhibiting cognitive impairments, there is abundant evidence that self-reporting across a wide range of behaviours is strongly affected by the fallibility and general inaccuracy of memory, particularly episodic memory⁵. (Anglin et al., 1993). Fundamental constraints of the mechanisms of attention, memory storage and retrieval limit the general validity of recollections of past behaviour. The shortcomings of memory function are potentiated in substance abusing populations, quite apart from the well established pattern of memory impairment in acquired brain injury populations.

Memory is affected by substance use directly via the acute physiological effects of intoxication and withdrawal, and chronic substance abuse can also mediate changes in memory function which persist beyond the period of intoxication (Hammersley, 1994). Where cognitive functioning has been shown to be compromised by either acute or chronic substance use effects it has been demonstrated that self-reports of behaviour will generally be less valid and reliable (Sinha & Easton, 1999). With many self-report instruments demanding a period of recall greater than one month, the difficulties of recalling behaviour which is not considered salient at the time are magnified.

⁵ Our recall of events, and our ability to place events correctly in time

Obtaining valid assessments

The necessity of gaining accurate date concerning substance use behaviour has led researchers and clinicians to search for more 'objective' means of assessment. Objective sources of data concerning an individual's substance use include blood or urine based drug screens, breathalyser tests, laboratory tests, collateral informant reports and official records such as hospital admissions or arrest records. Biological assays (toxicology) perhaps offer the most objective means of assessing substance intake. Analysis of urine samples - urinalysis or urine drug screens (UDS) are most often used for detection of substance use, although blood, saliva, perspiration and hair are also used to detect substance use (Smith & Liu, 1986).

In alcohol treatment, several abnormal laboratory results are taken as indicators of heavy drinking, and are frequently used to monitor alcohol use, and verify abstinence and relapse. Tests such as mean corpuscular volume (MCV) and gamma glutamyl transpeptidase (GGT) have been demonstrated to be related to recent alcohol use (Kricka & Clarke, 1979). Although Cushman, Jackobson, Barboriak and Anderson (1984) point out that the lack of sensitivity of these measures has limited their utility in either treatment or research as a criterion of recent alcohol use. For example, an elevation in GGT may be due to medication or non-alcoholic liver disease, whilst MCV may be determined by nutritional factors and cigarette smoking (Monteiro & Masur, 1986).

Collateral reports and official records are relatively poor 'objective' indicators compared to toxicology, and are particularly limited by incompleteness of collateral /official knowledge of the subject's pattern of use, however, much more sophisticated and detailed information on substance use may be collected via collateral interviews. Here the clinician or researcher attempts to gather information regarding the subject's history of use from the report of significant others such as spouses or regular sexual partners, other family members, employers and health and medical professionals.

In a review of the literature relating to alcohol users, O'Farrell and Maisto (1987) reported that agreement between self-reported alcohol use and collateral-reported use tended to be in the range of 90% for alcohol related events, but less perfect for actual amounts consumed, with a median correlation in the range of 0.50. Collateral informant reports are sometimes assumed to have 'objective' validity as the collateral informant is seen to have

a capacity to directly observe the subjects use behaviour. However, collateral interviews do not constitute 'objective' measurement in the same way biological assays do.

Collateral informant data in fact suffers from the same general limitations of direct self-report, principally related to limitations of human memory and various biases in how personally sensitive information is communicated. The assumption of objectivity has also been challenged on the basis of independence (Platt, 1980). For collateral informants reports to be objective, they need to be completely independent of the subject's stated history of use. Ehrman and Robbins (1994) note that collateral informants reports often suffer from contamination by the subject's own self-reported behaviour. Where the collateral cannot directly observe all instances of the subjects substance use they themselves rely on the retrospective report of the user.

In many cases collateral informants may simply have much less exposure to the substance use behaviour than the subject themselves, meaning their data are less complete (Del Boca & Noll, 2000). Additionally, different collateral informants have varying types of contact and amounts of observational opportunity with the substance user, and this variation in the informant's knowledge can introduce another source of error, lowering the validity of this technique (Fisher & Harford, 1983).

Despite these limitations, judicious use of toxicology assessment and collateral informants to detect and measure substance use has special applicability when working with clients with ABI. Disparities in collateral validation are more frequently seen where the assessment requires attributions regarding behaviour. In one such study, Watson, et al(1984) found agreement between self-report and collateral sources on frequencies and amounts of consumption, but disagreements as to the degree of control over the behaviour. Where used judiciously for data gathering, collateral informants are an invaluable assessment source, particularly given that including significant others in assessment is likely to better engage them to support and remain involved in treatment programs.

Assessment and diagnostic tools

Formal diagnoses in alcohol and drug treatment tend to include the DSM-IV classifications of dependence, and abuse for various substances (see Box 3). Most alcohol and drug clinicians do not employ structured assessments or interviews in order to make these diagnoses systematically, rather diagnostic criteria are usually retrospectively identified from generic or unstructured assessments of substance use. An example of a generic assessment is the Victorian Alcohol and other Drug Assessment module used in most funded agencies. This tool represents a comprehensive assessment tool for investigating substance use and related issues, including medical, psychiatric, and cognitive issues.

This assessment is however, rarely comprehensively conducted, particularly it's cognitive assessment component, the Cognitive Status Examination. The CSE itself is of limited utility in planning interventions or treatment selection as it is intended as a screening instrument, however it is a valuable indicator of some level of dysfunction, and can usefully indicate where referral to a neuropsychological assessment is appropriate. The Tool uses a digit-symbol task, and a 'cognitive difficulties scale'. The digit-symbol task, similar to those found in IQ tests and neuropsychological batteries is a timed task which asks client's to match unfamiliar symbols with letters, and measures working memory and processing speed. The cognitive difficulties scale is a self-report task in which client's rate the severity of a range of cognitive and motor tasks.

Turning Point Alcohol and Drug Centre has produced an additional "Trigger Sheet" tool to be used in alcohol and other drug assessments in order to highlight probably causal and presenting factors for ABI in a brief, easy to score instrument. This instrument has not undergone any formal evaluation, however it is not intended as a diagnostic tool, rather as a signpost indicating where further comprehensive assessment is indicated. Perhaps the most appealing addition to the existing compliment of alcohol and drug assessment domains, (as recommended in the Queensland Government's ABI and Alcohol and Drug Manual) is the readiness to change measure. Traditionally clients self-present to AOD agencies and a basic level of motivation to change is expected.

In populations experiencing comorbidity, the proportion of clients being referred by an agency rather than self-identifying an issue is higher. With agency-based referrals the 'real' motivation to change behaviour may rest with the agency which has made the

referral rather than the client. Such referrals are very common in situations where some degree of coercion is present in the referral, most notably mandatory substance treatment dictated by legal sanction i.e. child protection cases and non-custodial sentencing options for drug-related crime.

Whilst the majority of AOD counsellors and assessors will make implicit judgements and assessments of a client's readiness to change, and value methods of understanding the relationship between motivation and outcome such as the Prochaska & DiClemente (Prochaska & DiClemente, 1992) stages of change model, few would routinely use a structured approach to measuring motivation, and fewer will monitor motivation in an ongoing way.

Box 3: DSM IV criteria for substance-related diagnoses (APA, 2000)

DSM IV dependence

A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12-month period:

- tolerance, as defined by either of the following:
 - a need for markedly increased amounts of the substance to achieve intoxication or desired effect
 - markedly diminished effect with continued use of the same amount of substance
- withdrawal, as manifested by either of the following:
 - the characteristic withdrawal syndrome for the substance
 - the same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms
- the substance is often taken in larger amounts or over a longer period than was intended
- there is a persistent desire or unsuccessful efforts to cut down or control substance
- a great deal of time is spent in activities to obtain the substance, use the substance, or recover from its effects
- important social, occupational or recreational activities are given up or reduced because of substance use
- the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (e.g., continued drinking despite recognition that an ulcer was made worse by alcohol consumption)

DSM-IV substance abuse

A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by one (or more) of the following, occurring within a 12-month period:

recurrent substance use resulting in a failure to fulfil major role obligations at work,
 school, home (e.g. repeated absences or poor work performance related to

- substance use; substance-related absences, suspensions, or expulsions from school; neglect of children or household)
- recurrent substance use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by substance use)
- recurrent substance-related legal problems (e.g., arrests for substance-related disorderly conduct)
- continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (e.g., arguments with spouse about consequences of intoxication, physical fights)
- The symptoms have never met the criteria for Substance Dependence for this class of substances.

Multi-method assessment

Taylor, Kreutzer, Demm & Meade (2003) recommend that ongoing, multi-method assessment be the preferred methodology of investigating substance use with TBI clients, including biological assays of use, structured interviews, collateral interviews, and record reviews (File Audit). Before discussing the specialist instruments available for measurement of alcohol and substance abuse it is appropriate to outline the above techniques in general. Skinner (1984) outlines a scheme for considering assessment methods in substance use: a) prospective methods, b) retrospective methods and c) objective indicators.

Prospective methods generally consist of differing types of client (or research subject) self-monitoring and recording of use, such as the use of drug use diaries. This method is commonly considered to be the most accurate alternative to direct observation of use, however it has limitations, particularly with clients experiencing cognitive impairments. Prospective assessment requires the client to have the necessary skills, motivation and resources to complete the assessment task on a regular basis. Retrospective methods entail having the client recall and report previous substance use, usually over a designated time period, and are obviously heavily dependent on the accuracy of the client's memory functioning, again, an area commonly compromised with acquired brain injury.

Retrospective instruments are the most commonly used methodology in substance use treatment and research, and include comprehensive measures such as the Addiction Severity Index (ASI, McLellan, Luborsky, Woody & O'Brien, 1980), the Time Line Follow Back Interview (Sobell & Sobell, 1985), and the Opiate Treatment Index (Darke, Hall, Wodak, Heather & Ward, 1992). Simpler instruments such as the Michigan Alcohol Screening test (MAST, Selzer 1971), the CAGE (Ewing, 1984), and the AUDIT (Bush et al 1998) are considerably less involved instruments and suffer from the same basic limitations of self-report measures of substance use and offer considerably less detail to the assessor regarding details of use.

Recommendations from the Queensland ABI Drug and Alcohol Manual (REF) cover assessment issues such as raising the issue of substances, assessing patterns of use, severity, drug related problems, situational use, and the client's readiness to change. Crucial aspects of substance use assessment are covered in Box 4.

Box 4: Assessing patterns of substance use

- Types of substances used currently
- Types of substances used in the past
- Quantity measurements (may need to be multimodal assessment, especially for illicit drugs where quantities/strengths may be variable or hard to gauge. Multi modal assessment includes amounts spent, numbers of injections, weight, etc.
- Frequency measures how often using
- Time and place of substance use
- Social context of substance use
- Past treatment for substance use patterns including where the treatment was obtained, and how successful it was.
- Periods of abstinence how long, circumstances
- Patterns of substitution
- Cues and triggers for relapse
- Client's understanding of the role of their substance use and it's effects positive and negative.
- Future plans or goals for substance use
- Costs including social, emotional, psychological, cognitive and general medical consequences

(Adapted from QLD ABI Drug and Alcohol Manual, with additions REF)

Models of ABI sensitive AOD treatment

The existing literature has very few reported evaluations of specialist treatment packages for individuals with comorbid ABI and substance abuse issues. Taylor et al (2003) have reviewed available treatment options for TBI clients, although the level of objective program evaluation does not allow any definitive conclusions regarding efficacy. In a non-evaluated program, Langley et al (1990) describe a 'comprehensive' alcohol abuse treatment package for clients with comorbid TBI. This program included initial assessment with the use of screening measures and a criterion based interview. Delivery of treatment involved client and family education, promotion of lifestyle change, interventions to alter

specific beliefs about alcohol, motivational enhancement for sustained alcohol behaviour change, as well as provision of support for family members.

Corrigan et al (1995b) presented a community based model of treatment based in a 'network' of traumatic brain injury services and making use of interdisciplinary teams with expertise in vocational rehabilitation, specialists in TBI and specialists in alcohol and drug treatment. The program involved a 'comprehensive assessment of substance use history, a neuropsychological assessment, followed by integrated service planning and monitoring. Program components were delivered with outreach components, and included vocational development with prospective employers and families, support and advocacy in accessing services, social and emotional support. Corrigan and colleagues reported 37 client vocational outcomes for the program indicating more employment at 6 months than for clients not involved in the vocational support program. Bogner et al (1997) reviewed this TBI network program and found significantly improved vocational status, and increased rates of abstinence.

Delmonico, et al. (1998) reported on a group psychotherapy model for comorbid ABI-substance using clients based on harm minimisation principles. Delivered to inpatients and outpatients, these open groups were facilitated, and had goals of identifying substance use problems, maladaptive coping styles, triggers to use, recognising the consequences of drug use, and developing effective relapse prevention and coping strategies. Group members were provided with written materials and encouraged to make notes during groups. These authors report indicators of treatment success with this group including reduced clinic presentations, fewer emergency calls for substance related health problems, and increased stability of housing, although no data was reported for 'objective' indicators of changed substance-use behaviour.

Taylor et al (2003) report 'critical features' of successful substance abuse treatment with clients with comorbid TBI. These factors, whilst relating specifically to clients who have incurred an ABI through traumatic injury and who receive treatment within a TBI rehabilitation framework nevertheless may have wider application to individuals with cognitive impairment, particularly given the lack of specific treatment recommendations for clients with a comorbid ABI and alcohol or drug disorder. These factors are listed in box 5 as a set of expectations/recommendations for clinicians working with this group.

Box 6: Recommendations for clinicians working with ABI-AOD clients

- Clinicians must identify substance abuse risk factors, subtle signs of current abuse with clients, individual's patterns of use and treatment needs, the available resources and challenges to the client's recovery.
- Clinicians must monitor substance abuse over time with routine assessments, particularly for clients with indicators of high risk.
- Clinicians must assist clients in recognising their substance abuse problems and encourage them to take responsibility for this behaviour.
- Clinicians must involve family members and significant friends or supports in assessment and treatment.
- Clinicians must educate clients and significant others about the dangers associated
 with substance use, the differences between substance related problems and
 issues related to the consequences of brain injury. Whilst the clinician should
 provide education about available treatment options, education and prevention are
 emphasised over treatment.
- Clinicians must be pro-active
- Clinicians must assist clients to seek treatment and recognise the positive aspects
 of choosing abstinence, and avoid negative treatment of clients who make
 maladaptive choices in relation to their substance use.
- Clinicians should help clients discuss the effect of substance use on their feelings, self-esteem and relationships
- Clinicians should use repetition, and present information in a variety of modalities, including role play, visual aids, peer modelling, videotaping, and coaching in order to promote effective learning.
- Clinicians should measure goals regularly, and offer the client praise for attaining or working successfully towards their goals.
- Clinicians should recognise and prepare for the relapses which are likely for their clients.
- Clinicians should help clients and families make contact with community resources (i.e. NA, AA)
- Clinicians should encourage participation in alcohol and drug-free social and recreation activities.
- Clinicians should carefully consider the pros and cons of referring for outpatient and

inpatient substance abuse treatment programs.

 Clinicians should provide information to community agencies and treatment programs about the effects of ABI and the need for accommodations to be made for clients.

Adapted from Taylor et al., (2003)

Taylor et al (2003) also highlight seven commonly encountered issues in delivering alcohol and drug treatment to clients with a comorbid brain injury. These issues represent both potential confounds or barriers to treatment, but also suggest where adjunct support and sensitive treatment selection may improve efficacy, and are presented in Box 6.

Box 7: Issues in delivery of AOD-ABI sensitive treatment

- Few vocational opportunities exist for clients following traumatic brain injury.
 Alcohol and drug use further reduce the likelihood of a client securing employment, reduces available income, increases the risk of re-injury, and exacerbates the effects of injury related cognitive impairments. Alcohol in particular is identified as affecting health, relationships and full participation in activities.
- Alcohol and substance use can significantly undermine rehabilitation from brain injury, and regardless of the rehabilitation setting (inpatient, outpatient), substance use should be monitored for several years post-injury.
- The effects of substance abuse may be difficult to distinguish from the effects of the brain injury. Neuropsychological tests can be used to differentiate these two factors, given the ability to measure the abstinent client. The effects of substances are likely to be more significant where clients are older, with longer histories of substance use, histories of substance-related health problems (such as liver disease), a history of overdose.
- Cognitive effects of brain injury, particularly attention concentration, learning and
 memory affect a client's ability to learn and recall all information presented in
 treatment. Frontal lobe impairments in particular affect the client's ability to be selfaware, to be actively anticipating the consequences of their behaviour, to inhibit
 behaviour, to plan solutions and implement strategies.
- Information should be presented in complimentary modalities to account for 4.
 Teaching or coaching should progress slowly, with the use of paraphrasing and repetition encouraged. Clients may need to be encouraged to attend groups where their particular dual disability needs are understood, adequate structure is provided whilst still encouraging participation, and using the application of behavioural principles of treatment.
- Resistance to change can be significant with ABI AOD clients, commonly from a combination of psychological 'denial' processes, as well as awareness deficits

caused by their injury. Managing resistance may involve including family and significant others in assessment, treatment, education and prevention efforts. Assessment results should be discussed with clients and others they have involved in their treatment. Referrals should be made actively – assisting the client to engage successfully with appropriate treatment. Resistance may also be countered by assisting the client to recognise how substance use is affecting their goal attainment, what the potential benefits of treatment are and what are the barriers to treatment. Encouragement of clients rather than criticism and threats are recommended, in keeping with principles of motivational interviewing.

• Families have significant roles in treatment. For example, they may be close observers of client behaviour, and be able to provide more detailed information on patterns of substance use, as well as general functioning. Families can exert strong influences on behaviour, reinforcing positive behaviours, facilitating involvement in drug and alcohol free activities. Families, can also undermine successful treatment by distancing themselves from the client, encouraging substance use, downplaying risks or dangers of use, or the efficacy of treatment.

Adapted from Taylor et al., (2003)

Within the Victorian AOD treatment context, the general treatment issues highlighted above should be considered within the parameters of the existing service delivery model; treatment modalities available for clients with a dual ABI diagnosis, and treatment modality that best suits any given individual's needs. There are significant differences between the services available to clients incurring a brain injury through 'trauma' and those incurring through substance use which Taylor's summary does not address.

Structured rehabilitation environments are much better able to manage complex behaviour such as alcohol or drug use. Clients referred to alcohol and drug services are often no longer involved with rehabilitation, are isolated, may not have any services involved, may never have completed or undertaken cognitive rehabilitation, may be intransigent and have more complex and significant substance use histories. Whilst this literature review does not seek to codify the range of existing treatment options available in Victoria nor make specific comment on appropriateness for a heterogeneous client group, pragmatic issues

dictate that the majority of clients with cognitive impairments can engage in residential or home based withdrawal service, pharmacotherapy programs, diversion programs, outpatient counselling, outreach counselling or support, residential rehabilitation (medium to long term), or a self-help program. Furthermore, it is likely that the within the available treatment options, selection of an appropriate level of intervention, selection of an abstinence based versus controlled use based intervention, and the potential role of family and community supports will be crucial decision in planning effective ABI treatment.

Similarly, selection of appropriate staff for delivering ABI sensitive treatment involves careful consideration of worker characteristics, knowledge areas, competencies, training history, supervision resources in working with complex client groups. Within the Victorian treatment network, specialist roles have been developed which focus on both alcohol and drug treatment and acquired brain injury. It is usually advantageous to make use of neuropsychological reports to inform this process of treatment planning, and service or clinician selection. Ongoing liaison with the assessing neuropsychologist, other treatment providers or significant others familiar with the case is considered crucial to monitor the effectiveness of treatment strategies. Referral to neuropsychological assessment and the interpretation of reports specifically for alcohol and drug treatment will be considered in later sections of this report.

Incorporating cognitive retraining / rehabilitation into AOD treatment

In recent years the identification of neuropsychological contributions to substance misuse conditions such as 'frontal lobe dysfunction like' symptoms has encouraged treatment responses including cognitive rehabilitation. As mentioned previously, following acute withdrawal, alcohol users typically recover much (but not necessarily all) cognitive functioning, although the improvement may occur over weeks, months or even years. Goldman (1995) presents a summary of the literature investigating facilitated recovery of cognitive functioning in alcohol users who have become abstinent.

The rate of improvement of cognitive functions depends on the age of the individual and the type of cognitive skill under examination. Younger drinkers (under 40) typically demonstrate substantial recovery of all cognitive functions, with only the most demanding tests indicating residual deficits. Older alcohol users tend to improve more slowly, and

tests of visuospatial and problem solving tasks typically taking longer to show improvement (Brandt et al, 1983). The effect of an individuals drinking history is less clear, long term users have not been demonstrated to have more lasting impairments than those with shorter alcohol use histories. Goldman (1995) indicates that the mechanism for this lack of consistency is not clear, however it may be related to the greater resilience or adaptability of the brains of individuals with shorter drinking careers.

Experience-Dependent Recovery refers to the concept that cognitive recovery may not be an 'automatic' process reflecting an intrinsic neurophysiological healing, but rather is a process which may be influenced and enhanced by environmental factors. Under experience dependent recovery, it is hypothesised that cognitive recovery may be induced or facilitated via practice and rehearsal of exercises designed to tap various cognitive skills, such as memory, attention and decision making skills.

Goldman (1990) reported that recovery can be accelerated if subjects are asked to use cognitive skills at a level equal or slightly beyond their current functioning. Again, measurement of change, and the exclusion of practice effects is difficult, although alternate form retesting and the use of related cognitive tests can illustrate the enhancement of cognitive skills (Forsberg & Goldman, 1985). Whether rehearsal of discrete cognitive exercises generalises to other cognitive skills not directly rehearsed is unknown, as is the effect of cognitive retraining on other treatment goals such as alcohol or drug treatment (Goldman 1995), however, the cognitive demands of alcohol and drug treatment are considerable (integrating new learning, generation of novel situational behaviours, response selection and inhibition, etc).

Goldman (1995) argues that attention given to strengthening the cognitive basis for change and adaptation facilitates successful treatment, particularly treatment such as Cognitive Behavioural Therapy, which has intrinsically high cognitive demands. Roehrich and Goldman (1993) used repeated rehearsal of neuropsychological tools with good ecological validity (real world relevance) to facilitate the improvement of cognition of individuals shortly after leaving detox, and demonstrated improved recall, which generalised to include relapse prevention material.

Bellack (1992) has expressed concerns that there is little existing evidence that cognitive rehabilitation can be viewed as amenable to rehearsal-repetition type improvement –

particularly given concerns about the 'real world transferability' of skills developed using neuropsychological exercises with little ecological validity – such as card sorting exercises. Goldberg & Bougakov (2000) however, indicate cognitive rehabilitation is at least conceptually, a useful addition to existing treatment programs – at the very least it facilitates measurement of cognitive skill and presentation of cognitively appropriate material to clients.

In a limited pilot study, Davalos et al (2002) assessed dysexecutive syndrome in a group of five schizophrenic patients, then effected a cognitive rehabilitation intervention using a tool which encourages clients to develop established routines, develop well-rehearsed strategies and acquire compensatory strategies through training and homework (Brainwave-R program, Malia et al 1997). Using a Jacobson and Truax (1991) 'clinical significance' approach, Davolos and colleagues identified improvements made in cognitive function, with subjects moving from 'abnormal' ranges of functioning towards 'normal' functioning. Whilst this is a small, uncontrolled study did not specifically address ABI, it is illustrative of the beneficial effects on day-to-day functioning of cognitive rehabilitation carried out within a dual diagnosis population.

Quite apart from the enhancement of internal cognitive skills, it is important to recognise the enormous functional impact of assisting clients to adapt to their impairments, adopt alternative strategies, and use external compensatory aids such as calendar, diaries, alarms, personal digital assistants. Most studies reviewing cognitive recovery identify these compensatory strategies as most effective (Ponsford, 2004).

Relationship between substance use, brain injury and offending behaviour

Various forms of acquired brain injury have long associations with increased impulsivity and offending behaviour (Pick, 1989). Several clinical studies have outlined increased incidence of violent offending within groups of brain injured individuals, particularly where their injury involves frontal lobe functions (Vogenthaler, 1987; Grafman et al, 1996; Nedopil, 2000). Brennan et al (2000) likewise found elevated rates of offending, particularly violent offending within a community sample of individuals with acquired brain injury.

The relationship between substance use and increased rates of offending behaviour, and the overlap between presentations of substance dependence or misuse and cognitive impairment or acquired brain injury makes it difficult in many cases to clearly identify the relative weight of causal factors (Mullen, 2001). Similarly, limitations in the methodology of studies investigating these relationships mean that only associative, rather than causal conclusions may be drawn.

Mullen (2001) notes that the lack of multivariate research limits the ability of researchers to make conclusions as to the relative contributions of substance use and acquired brain injury to offending behaviour. Multivariate studies would preferably include a wide range of other potential factors bearing on offending behaviour, such as age, socioeconomic status, prior criminal history, relationship and employment status. By relying on bivariate studies (measuring, for instance, only substance use and one area of comorbidity), and by confining sampling to select client groups, much of the literature fails to offer explanatory power beyond associative generalisations.

Impulsivity review

The clinical presentation of impulsivity is common to many criminal offenders, clients experiencing acquired brain injury, as well as those with substance abuse issues. Impulsivity has been variously defined as behaviour without adequate thought, and the tendency to act with less forethought than do most individuals with equal ability and knowledge (Moeller et al, 2001). Patton et al (1995) have divided the concept of impulsivity into three factors – acting on the spur of the moment (motor activation), not focusing on the task at hand (attention), and not planning and thinking carefully (lack of planning).

Both biological and psychological models of impulsivity have been advanced to account for clinical presentations of impulsivity, as well as to provide a basis for research. Either model type appears to draw explanatory power from the method of measurement of impulsivity, be it evoked potentials on EEG tests, or performance on laboratory behavioural tests of impulsivity. Moeller et al combine these factors to describe impulsivity as

1. Decreased sensitivity to negative consequences of behaviour,

- 2. Rapid unplanned reactions to stimuli before complete processing of information, and
- 3. Lack of regard for long term consequences.

Moeller et al note that there are social aspects to impulsivity as well, including aspects of learned behaviour stemming from reinforcement of goal directed behaviour within the family.

Impulsivity is usefully distinguished from compulsive behaviour and from impaired judgement, particularly where the impulsivity is defined as rapid unplanned action occurring before conscious cognition about consequences. Management or 'treatment' of impulsivity includes various modalities including insight oriented psychotherapy, cognitive behavioural therapy, and pharmacotherapy. Whilst the small case-series reporting of insight oriented therapies do not allow systematic evaluation of this treatment modality, similarly the cognitive behavioural literature (whilst supporting this treatment type, and the amenability of particular impulsive behaviours to treatment) often does not report on the direct alteration of impulsivity itself.

Pharmacological treatments for impulsivity often focus on the management of aggressive behaviours, via mood stabilisers such as lithium, antidepressants such as SSRI's, anticonvulsants, antipsychotics, and psychostimulants. Evaluation of these medications with respect to the 'core' aspects of impulsivity is difficult as the majority of studies used aggression as the outcome measure, which may have other contributory processes other than impulsivity itself.

Contingency management (CM) offers another behavioural approach to intervening around impulsivity. Under CM, predetermined positive and negative consequences are set up in order to reinforce or extinguish a particular behaviour. CM has become popular in the management of substance use disorders, and commonly in fixing abstinence outcomes for clients likely to relapse. Whilst measures of impulsivity are not typically included as outcome measures in CM studies investigating substance use change, Higgins et al (1986; 2000) have demonstrated efficacy for reducing substance use behaviours.

Moeller et al note that often the benefits of CM are poorly generalised and maintained, with behaviours often re-occurring once the reinforcement and extinguishment contingencies

are removed. For clients with offending histories, particularly mandated clients whose involvement with treatment and support services is likely to be sustained, stable CM principles, applied over time, offer a tool with significant utility for managing impulsive substance use and related behaviours.

Introduction

As part of the process of developing clinical treatment guidelines, a number of key agencies and individuals in both the ABI and AOD sectors were approached to provide expert advice and consultation. A Key Informant Questionnaire was formulated to tap experiences of service delivery and explore potential modifications to the service delivery framework that could potentially improve outcomes for this client group.

Responses from the participating key informants have been summarised according to a simplified set of key themes emerging from the original questionnaire. Certain questions have been collapsed, as they elicited no unique information in their separate forms. Where responses received multiple endorsements this has been noted in the summary of key themes. This approach is thought to capture the breadth of key informant's responses whilst condensing the material into a more readable format. The collation process was undertaken by two project investigators to ensure minimal loss of information from the original interview data, and the collated responses were compared to the original interviews to ensure that the key themes were accurate and representative.

For each section, the original questions presented to key informants have been included, and responses summarised under the simplified key themes.

Assessment of existing AOD service provision for ABI clients

For this section covering existing service provision the following caveat was read to respondents:

This questionnaire does not represent an evaluation of service provision; it is intended to allow respondent's expertise in the area of ABI and AOD to be included in the CTG process. Your responses will be kept confidential and no specific comments or identifying information will be included in the final report.

This section assessed the current framework of AOD ABI services available to Key informants or their service, covered any issues with service provision, access, referral and level of utilisation. The following questions were included:

- 1. What types of AOD or ABI services would you or your service access in supporting clients with substance use and brain injury dual diagnosis? i.e. withdrawal, counselling, pharmacotherapy referrals?
- 2. What are the access / referral protocols in place?
- 3. What issues are involved in referral / handover / shared care?
- 4. To what extent do consultations with AOD providers form part of your assessment / referral / management process?
- 5. What aspects of the existing framework of AOD service delivery is useful to you or your service in managing complex AOD-ABI presentations. What aspects of your management of these issues would be helpful for other services to make use of or adopt / adapt?

This section includes summarised responses to Question 5: What aspects of the existing framework of AOD service delivery is useful to you or your service in managing complex AOD-ABI presentations. What aspects of your management of these issues would be helpful for other services to make use of or adopt / adapt?

Key Theme 1: What services are useful in responding to the AOD treatment needs of ABI clients?

Responses highlight the wide variety of useful services within the AOD treatment framework. All alcohol and drug treatment options were nominated as useful and appropriate for ABI clients depending on the client, their situation and needs. Certain modifiers were thought to increase the effectiveness of AOD treatment: where services included outreach components, high levels of cross sector expertise, and the ability to provide secondary. Ongoing support, especially the provision of services for longer periods

of time was also nominated as particularly helpful with contact periods of up to 12 months seen as vital for supporting significant behaviour change.

Also identified as helpful adjunct services were community health centres and GPs involved in managing aspects of primary health, and mental health dual diagnosis teams, where either regular appointments or regular visits to clients provided regular monitoring and assertive follow-up.

Outreach forms of treatment were highly endorsed as particularly useful with this client group, even for counselling interventions, where outreach is not traditionally used. The benefits included more comprehensive assessment of the client and their environment, more powerful engagement, circumvention of a client's potential mobility and motivational issues, which may compromise attendance at outpatient appointments. ABI specific case management was also nominated as being effective in supporting AOD treatment.

AOD ABI clinicians were nominated as particular useful consultation and referral resources with endorsement from ABI and AOD sectors, due to their cross-sector expertise, experience with brain injury services and resources, ability to outreach and provide secondary consultation, and length of engagement with ABI clients.

Forensic referrals for AOD treatment for ABI clients primarily made use of CCCCs interventions, although withdrawal and rehabilitation options were also exercised less frequently.

Key Theme 2: Process issues: What works well in treatment delivery? What aspects of service delivery might predict better outcomes?

Several respondents nominated 'integrated care' between services as predictive of treatment success – the ability to work across multiple sectors and within sectors seamlessly. Integrated care involved information and expertise sharing, common treatment plans across services, and coordinated timing of services. The needs of ABI clients were particularly well served where service transitions were managed well, for example: a short stay in a detoxification service timed with the entry point to other residential services,

allowing no unstructured time where a client may relapse). Several residential services were preferred for their rapid response to ABI referrals.

Regarding provision of residential rehabilitation, respondents nominated a focus on individual symptom management rather than 'standardised program for everyone' as most useful, as ABI clients may have difficulty with standard rehabilitation models. In the traditional therapeutic community model barriers may exist to entering and participating in services for ABI clients: motivation needs to be maintained and appropriately communicated (i.e. remembering to phone a triage line at a certain time of day); individual needs are subsumed in group priorities, meaning ABI clients may not receive sufficient individual support to comply with behavioural guidelines, or participate meaningfully in some aspects of treatment like psychotherapeutic group work. Behavioural issues may be attributed to personality or motivation rather than disability. Despite this, respondents identified that the structured inpatient environment of some detoxification and rehabilitation services were useful where there was a focus on activities rather than therapies. Longer periods in residential detox (greater than one week) were nominated as being more useful than short stays.

Several respondents nominated a preference for making use of services with an explicit focus on prevention of further alcohol or substance related ABI, including provision of harm-minimisation techniques, policies to limit access to substances, access to alcohol and substance-free residential environments.

Respondents noted that where treatment providers used multidisciplinary teams this strengthens treatment. The AOD sector was viewed as having relatively broad staffing profiles, as well as good access and linkages to other professionals or services that their ABI clients may require.

Also nominated as helpful was the ability for services to provide information, guidance and support to families to assist with supporting clients, monitoring and implementing behavioural strategies, and managing stressors relating to carer roles.

Key Theme 3: What aspects of the existing service sector do not meet the needs of ABI clients? What aspects of service delivery are detrimental to the success of AOD interventions?

The following service or clinician characteristics and factors were nominated as being potentially detrimental to delivery of successful AOD interventions for clients with ABI. Services or clinicians may be reluctant to engage with ABI clients as they are seen as too 'difficult' or requiring a 'specialist' service (which may not exist). These barriers may limit the treatment options available to clients and prevent the AOD sector developing experience with ABI clients. Lack of familiarity with ABI and cognitive impairment may also potentially limit the ability for AOD workers to play an 'early intervention' role with clients who are at risk of developing ABI.

Respondents nominated that lack of expertise (equating to specific training in both the ABI and AOD fields) can be a significant 'gate keeping' factor for entry to the AOD sector, across most service types.

Whilst it was identified that the ABI AOD Clinician-Consultants are skilled enough to work with a range of ABI presentations, generalist AOD clinicians may be able to provide high quality interventions if they have adequate understanding of ABI, or via a process of consultation with the ABI-AOD clinicians.

Barriers to effective treatment may also involve service limitations:

- The service may have Limited outreach, meaning it cannot engage or work within the client's own environment,
- The program may lack flexibility of service delivery eg strict rules around entry criteria (phone calls every day/week at a particular time).
- The service may not be flexible in terms of seeking or accepting the guidance and advice given by consultants. There may be systemic reasons why services are unable or unwilling to alter their service delivery on the recommendation of external experts such as ABI agencies and ABI-AOD clinicians (for example, wanting to maintain a uniform program for all clients).

These barriers may exist for relatively 'minor' aspects of delivery, such unwillingness for an agency to vary their communication strategies with ABI clients.

There exists some confusion as to how long clients need to be substance free before being suitable for neuropsychological assessment.

Respondents nominated difficulties where services are 'funded' to deliver specialist interventions to ABI clients and do not for various reasons (including re-allocation of resources, difficulty with staffing specialist positions).

Respondents highlighted that treatment delivery suffered where shared care principles are not enacted – usually via lack of case planning meetings, poor communication between treating agencies or workers, or poor consistency with implementation of behaviour modification plans).

Some forms of residential services with strict behavioural requirements for entry, continuation and progress may inadvertently put barriers in place for some ABI clients. Where cognitive impairment leads to difficulties for clients in behavioural terms (such as initiating and inhibiting responses) clients may be disadvantaged as the treating team misapprehends their behaviour.

Examples nominated included clients failing to maintain attendance for preparation groups (or waiting list maintenance) due to memory problems where the lack of consistency was misinterpreted as lack of motivation. Wandering behaviour or difficulties inhibiting routine behaviours can be misinterpreted as attitudinal problems (such as unwillingness to adhere to the rules of the service). Failure to participate with group therapy environments due to slowed responses, communication problems or difficulties with understanding complex interpersonal factors can be misinterpreted as undermining, rather than representing the inappropriateness of the treatment type. Even where an adequate skill level and understanding around the complexity of ABI is present, lack of routine assessment of cognitive function can mean clients with cognitive difficulties are poorly recognised, representing a barrier to effective treatment.

Poor discharge planning was identified as a limiter of treatment efficacy – particularly in the move from residential to outpatient based treatment. Poor communication, lack of awareness of treatment relationships, and lack of provision of discharge summaries to

community case-managers and counsellors can mean that important diagnostic and other information is not transferred to the ongoing treatment team.

Lack of assertive follow-up in appointment-based services can limit the efficacy of treatment. ABI clients can often miss appointments, or disengage with services as a result of their ABI or related problems. Respondents nominated that without assertive follow-up, AOD agencies can misapprehend disorganisation for poor motivation: where clients do not consistently articulate their service needs an agency may assume they do not have any treatment needs.

Lastly, philosophical difference between services which work from a directive framework, and those working from a client centred approach may cause conflict where the 'goals' of the organisations are incompatible, and no attempt has been made to identify or reconcile these conflicts. Typically ABI agencies are more likely to work from a directive framework and AOD agencies are likely to favour a client centred framework.

Assessment of AOD issues with ABI clients

This section addressed opinions and experiences with performing assessments with dual ABI AOD clients. Respondents were asked to nominate assessment domains with relevance to planning interventions with this client group, and recommendations as to what assessment tools are most useful. The following questions were presented to key informants:

- 1. Who should ideally conduct AOD assessments for ABI clients, what should their background be like in terms of discipline, experience etc.?
- 2. Which tools should be used?
- 3. What areas of a client's history and functioning are relevant to an AOD assessment?
- 4. What formal measures should be used to assess AOD problems with ABI clients?
- 5. When should reassessment occur?
- 6. How should AOD assessments be recorded and communicated to clients / collaterals / other treatment providers?

- 7. How should families and significant others be included in the assessment procedure?
- 8. What feedback mechanisms to families / collaterals / significant others should be used?
- 9. What should the process be for using the results of assessments to plan treatment, develop or modify services?

Key Theme 1: Who should conduct ABI AOD assessments, and what should be assessed?

Please note that this section relates to specific assessments of alcohol and other drug problems in the context of ABI, and does not relate to the separate process of neuropsychological assessment for exploring ABI in detail.

Respondents uniformly nominated that ABI AOD assessments should be conducted by senior, experienced clinicians with knowledge of both Alcohol and Drug, as well as Acquired Brain Injury. The ABI AOD clinician-consultant positions received many endorsements as representing workers with this cross-sector knowledge. Strong ABI and AOD education and training and experience were highlighted as crucial to building a working understanding of the client, their needs, and choosing the most effective treatment option available.

Respondents nominated that a clinician who has established good rapport with the client may be best placed to conduct or collaborate with a specialist assessor in some instances (for example, where the client is guarded about communicating with new workers) in order to have access to the most reliable information. Generalist AOD clinicians were viewed as requiring ongoing training in order to better recognise and assess ABI or cognitive impairment, and provide appropriate interventions. Respondents also highlighted that workers with an interest/passion in assisting clients with ABI were often more effective.

Key Theme 2: What should be assessed in an ABI AOD assessment?

Respondents with AOD assessment experience nominated the DHS Specialist AOD Assessment tool as allowing comprehensive assessment of AOD use, and indicated that additional domains of assessment would be relevant to understanding a client's ABI or cognitive impairments. Few specific tools or scales were nominated other than the existing Cognitive Status Examination (part of the DHS Specialist Assessment) or similar non Neuropsychologist-administered brief cognitive screens (such as the Heidelberg Cognitive Screen).

Respondents nominated a range of factors to be included in assessment such as AOD treatment history, relapse risk assessment, ABI treatment history, client-identified cognitive problems, general psychosocial functioning, forensic history, inventory of behavioural changes or behavioural concerns, full medical history (including medication regimes), and psychiatric history. The use of collateral informants to build a more comprehensive picture of an individual with ABI was strongly endorsed.

Several respondents highlighted the importance of determining whether there were any substance use issues prior to the individual incurring the ABI, because pre-existing substance use problems were thought to be related to greater difficulties in meeting controlled use or abstinence goals.

Several respondents highlighted that where assessment occurs in the client's own environment this process has the potential to produce a richer information base, particularly where a clinician seeks to recommend behavioural strategies, problem solve for the client, or where the client has difficulty fully apprehending or communicating environmental factors related to substance use.

The issue of AOD clinicians making use of cognitive screening was raised by several respondents, with endorsement for limited screening to detect potential cognitive difficulties, but strong recommendations that once a threshold level of concern has been raised, that referral for neuropsychological assessment be sought.

Assessment of AOD program outcomes with ABI clients

This section addressed respondent's beliefs on what represents 'reasonable goals' in successful AOD ABI services or interventions, in terms of meeting the needs of clients, significant others, services and the community as a whole. Respondents were reminded that the questions were not intended to form an evaluation of any particular service, agency, or philosophical approach to treatment, but rather to inform the selection of appropriate goal areas and outcome measures for the CTG project. The following questions were presented to key informants:

- 1. What are appropriate goals for an AOD program with ABI clients?
- 2. What are appropriate individual goals for a client with a dual ABI-AOD diagnosis?
- 3. How should the goals of such a service be chosen?
- 4. What role for client and community needs analysis?
- 5. What role for research, policy or other information sources in program development?
- 6. How should treatment efficacy be tracked?
- 7. How should an individual's progress be measured?
- 8. What kind of follow-up is appropriate?
- 9. How should overall programs be evaluated?
- 10. Should cost-benefit models be used in evaluating efficacy?
- 11. How should program utilisation be assessed?
- 12. How should client satisfaction be measured should significant other satisfaction also be assessed?
- 13. How should families, significant others, collaterals be included in choosing pertinent goals, assessing outcomes?

Key Theme 3: What are appropriate treatment goals for ABI clients?

Respondents nominated that ABI clients benefit from access to structured AOD programs which offer similar interventions to those provided to non-ABI clients. The range of appropriate goal frameworks nominated included harm minimisation, alcohol or substance

use reduction programs, and abstinence programs. Respondents typically identified a principle goal of maintaining a focus on reduction in AOD related harms and management of risks, with focus also paid to supporting clients to make improvements to their lifestyle, with this process guided by the client's choices, goals and motivations. Respondents highlighted that effecting significant, longstanding changes with ABI clients often requires longer involvement with treatment, and longer periods of post-treatment follow-up or support.

Key Theme 4: How should treatment goals for ABI clients be measured?

Measurement of success was seen as vital; with both self-report and collateral reports seen as important measures of success. Several respondents noted the importance of both formal measurement of outcomes or results (such as lower scores on depression inventory) as well as measuring functional outcomes – such as positive behaviour change carefully and systematically. Functionality is seen as more important than arbitrary conditions such as absolute abstinence, although measurement of functional states (such as 'levels of involvement in non-alcohol or drug related activity) require more creative assessment solutions.

Key Theme 3: How directive should clinicians be in working with ABI clients?

Multiple respondents raised the tension around adopting paternalistic/maternalistic or directive approaches in assisting ABI clients in choosing goals, related strongly to the nature and severity of the client's ABI. For some clients, insight or awareness deficits may necessitate a level of worker or family led decision making which can often be 'at odds' which the client-centred, motivational approach most often used in AOD interventions. The question was elegantly expressed in this dilemma: Which is more important out of autonomy and welfare? ABI agencies and workers typically reported being more comfortable adopting a more directive approach than AOD agencies and workers.

Respondents identified that difficulties can arise where a particular service, worker or family member favours goals which are in conflict with the client's stated preferences (i.e. abstinence versus continued substance use). In these situations, third party consultants

and advocacy agencies are often called upon to mediate. Respondents from these organisations recommended that meeting the needs of all parties often meant working from a 'client centred' position, but not necessarily a 'content free' position. This may mean inclusion of family and community issues and concerns in selecting appropriate goals and strategies for clients.

With respect to clients experiencing some degree of insight or problem-solving deficit, more directive strategies in goal selection were recommended, ("holding the client's best interests in mind") whilst remaining within a motivational framework. Respondents nominated that in many cases, clinicians will quickly recognise where goals are totally inappropriate but are advised to motivate the client towards more appropriate goals rather than fail to provide a service. Respondents recognised that issues of trust, low self-efficacy and negative expectancies for success can limit the client's goal choice and working with small achievable goals can assist in moving the client to a point where more significant behaviour change can be contemplated and enacted.

Key Theme 4: How to reconcile differing service/client goals?

Respondents nominated motivational interviewing approaches as most useful for reconciling the divergent goals of different stakeholders. Respondents identified a need for educating clients, clinicians, services and families who may have unrealistically high expectations of change, or alternatively very low expectations of the potential for useful intervention. The importance of maintaining motivation and efficacy through choice of appropriate targets for change was highlighted again. These issues are particularly pertinent in the delivery of forensic treatment, where external stakeholders have the ability to place particular conditions on clients and services to fulfil particular goals (these external parameters also apply to Child Protection referrals and other instances of 'mandatory' treatment).

Bearing in mind the recommendations subsumed under key theme 3.1, respondents also recommended bargaining and trial and error to explore what goals are achievable, and stressed honesty and frankness about the limitations of services, and treatments and clients.

Modifications to the existing AOD treatment system for ABI clients

This section asked respondents to nominate what modifications to the existing framework of AOD ABI service provision would be beneficial to clients with a dual ABI-AOD diagnosis? The key theme subsumes the original question posed to key informants.

Key Theme 1: What modifications would you recommend to improve the capacity of the services system to respond to ABI clients?

Respondents consistently nominated increased capacity across the ABI-AOD sectors as crucial, with specific recommendations that more ABI AOD clinicians / consultant positions be made available, as well as ABI-AOD case managers. Respondents indicated that the demonstrated cross-sector experience and knowledge, secondary consultation and outreach capacity made these positions particularly effective.

Specific ABI AOD training delivered uniformly to generalist ABI, generalist AOD and generalist forensic workers was also frequently nominated as a strategy to improve the service system's capacity to meet the needs of ABI clients. This training would include detailed cross-sector knowledge for ABI, AOD and forensic sector staff, and would have explicit processes for encouraging the use of secondary consultation and external supervision / mentoring models. Several respondents nominated that a system of posting clinicians to a relevant service in the other sector would improve clinicians' knowledge, improve referral linkages, and strengthen shared-care arrangements, as well as familiarising clinicians with models of multidisciplinary input into treatment planning.

Respondents highlighted that greater flexibility in alcohol and other drug treatment service delivery would improve access and utility for ABI clients. Specifically, AOD services which can increase their outreach capacity were thought to be able to maximise successful engagement, comprehensive assessment, also allowing treatment to be provided to clients with access and mobility issues.

Multiple respondents called for residential 'rehabilitation' models to be made available to ABI clients with a lower level of expectations for entry to the service, more of a focus on structured activities and skill building, and less of a traditional psychotherapeutic approach,

and a focus on individualised support programs which make use of neuropsychological assessments to plan interventions around client's cognitive impairments. Involvement of family in treatment was also rated as an effective modification.

Respondents highlighted the issue of linking the provision of residential services to other aspects of the AOD treatment network in a timely fashion (such as timed pre-rehabilitation detox, and speedy access to post-rehabilitation support counselling or case management). Quite apart from rehabilitation, multiple respondents nominated non therapeutic community, medium-stay residential care, recognising that respite and geographical isolation may have benefits for carers and significant others as well as the client. Some respondents indicated that a mandatory aspect to this service would be useful for very high-risk clients.

Other recommendations included a dedicated ABI AOD helpline (possibly involving training identified staff at Directline), providing family support components, allowing services sufficient discretionary funding to broker additional services where existing provision falls short (including treatment, access to residential facilities, attendant care, carer respite, and recreational activity programs). Respondents indicated that a 'no wrong door policy' – AOD or ABI agencies facilitating access to multiple treatments and sectors would assist clients navigating the treatment system. Also nominated was a preference for ABI –AOD clients to have access to a singular case manager with wide-ranging mandate to assist clients across multiple treatment and support domains rather than segmented responsibility model.

Other relevant approaches to managing AOD issues with ABI clients

This section drew upon respondent's knowledge of alternative treatment models from Australia and internationally specifically targeting ABI, or relevant treatment models which whilst not currently available, would be of benefit in providing services to ABI AOD clients.

Key Theme 2: Are you aware of other programs or approaches that are not available to you but would improve the capacity of the service system to respond to the needs of clients with a dual ABI-AOD diagnosis?

Few respondents could identify novel approaches to managing ABI-AOD issues which were not previously mentioned under the 'modifications to existing treatment systems' questions, however some themes in existing services could be applied to the ABI-AOD field. The allocation of greater amounts of time and resources to conducting comprehensive assessments – similar to the model of forensic assessments currently in place - allows for inclusive data gathering. This would explicitly identify assessment of ABI as a complex process requiring additional time, and reduce pressures on clinicians to formulate treatment responses for clients without a full understanding of the alcohol and drug use issues, ABI issues, and general biopsychosocial situation.

The self-support "bear in mind" model was also nominated as a helpful approach – involving face to face and electronic communication modes, as we the Headway model of 'carer support', and the Mental Health sectors support model for carers.

In relation to forensic models of treatment provision with propensity to be useful in responding to ABI clients, the Ward & Brown 'Good life model' was nominated, being an adaptation of existing relapse prevention 'risk-need-responsivity' treatments to a more holistic approach to treating offenders. The good life model was thought to be appropriate primarily for it's focus on 'meeting client needs for positive experiences' as well as managing risks of relapse to substance use or offending behaviour.

ABI AOD Clinical Treatment Guidelines Part 3: Alcohol and drug / acquired brain injury assessment module

Introduction

This alcohol and drug / acquired brain injury assessment module will assist you to provide treatment and support services for clients experiencing concurrent substance use and acquired brain injury issues. The tool is designed to facilitate a comprehensive approach to assessing problematic substance use and related high-risk behaviours, including offending behaviour with clients experiencing an ABI. The module is designed to assist clinicians decide on the level of treatment need of the client, and recommend a treatment pathway appropriate to the level of risk, need, and the client's likely responsiveness to interventions.

As recovery and deterioration are dynamic or changing states and can affect a client's presentation and needs, it is recommended that clinicians working with this client group adopt an 'iterative' approach to assessment – that is, assessment as an ongoing process which constantly reviews goals, motivation, strengths and weaknesses in order to best frame a service response. This module recommends that clinicians work towards classifying clients into three levels of treatment need, mirroring both the dual diagnosis three tier system, and allied to a nominal 'mild-moderate-severe' framework of judging severity of ABI.

This assessment tool does not constitute a formal diagnostic instrument for detection or assessment of an ABI condition. In fact it is not possible at the AOD assessment point to make definitive determinations about a client's cognitive status. Rather, neuropsychological assessment is always recommended for clients experiencing cognitive problems, as only a neuropsychological assessment can offer the required detailed assessment necessary for accurate formulation of a client's strengths and weaknesses in planning treatment and support services.

This assessment, however, should allow the generalist clinician to quickly screen and identify likely ABI issues with AOD clients, then assess in details the relevant areas critical to treatment delivery. In particular, a comprehensive assessment will facilitate consultation

with specialist ABI-AOD clinicians or consultants, encouraging skill and capacity development across the AOD sector.

Levels of ABI treatment needs

Level one - Low risk-need group

Level one describes a group of clients who are currently presenting with problematic, low-to medium risk alcohol and drug behaviour (not meeting DSM-IV diagnoses for dependent or hazardous use), and low level (mild) ABI. These clients are presenting as motivated for treatment and have sufficient resources to attend outpatient appointments, carry out behavioural change in their own environment, and to self-identify goals and needs, potential escalations in risk behaviour, and emerging problems. These client's may not present with confirmed ABI diagnoses, and may be unaware of the impact of previous behaviours / incidents on current cognitive functioning. Emerging ABI symptomatology may have accounted for difficulties in accessing or following through with AOD interventions in the past.

AOD: The client has a level of ongoing substance use which is amenable to maintenance of control or reduction / cessation, or is at risk of relapse to substance use and requires support. The client may be well motivated to work on substance use issues, or very well supported to carry out planned interventions around substance use. An example would be a low level of alcohol use with adequate internal resources to modify use (motivation, healthy beliefs about substance use, positive goals to maintain cognitive functioning), as well as good external resources and supports to maintain low levels of use.

ABI: The client has an emerging or identified ABI with a 'mild' overall estimate of impairment. The client's impairments may be limited in nature (i.e. has memory problems although other cognitive abilities are unaffected), or well adjusted for (i.e. client has multiple cognitive difficulties but has accommodated to these deficits well, or is very well supported to manage the deficits). The client may be unaware of the relationship between substance use or risk behaviours and potential cognitive impairments but is not resistant to or unable to explore these relationships further. The client may need support around initiating assessment of their cognitive impairments via neuropsychological assessment, as well as relevant follow-up, including cognitive rehabilitation and ongoing monitoring.

Management options for low risk-need group

This client group may best receive management of their needs in either the primary health care system or AOD system without requiring intervention by specialist ABI agencies, and in particular, would not require intensive case management or rehabilitation services. Most services can be delivered in an outpatient, appointment based system.

Provision of counselling, pharmacotherapy, AOD case management could be delivered in conjunction with primary health provider such as a GP to meet the client's needs or goals. Some modification of standard delivery of information and therapeutic services may be required to match the clients needs. In general, these clients can manage their self-care in the community without requiring specialist supports, but may from time to time benefit from community support referrals (such as education or vocational support).

Level two - Medium risk-need group

Level two describes a group of clients presenting with dependent or hazardous AOD use (as defined by DSM) and mild to moderate ABI (as per neuropsychological assessment). These clients may self-present or be referred due to the concerning nature of their substance use, because their ABI has recently been identified or diagnosed, or due to increasing problems with meeting significant obligations or legal conditions. There may be awareness or insight into the cognitive impairments faced and the difficulties in changing substance use behaviour, or the client may be unaware of changes in their cognition due to substance use effects.

Cognitive impairments may mean the client has gained minimal benefit from alcohol and drug interventions or other treatment and support services in the past. Because of the impact of their ABI, the client will require their treatment to be modified in order to match their individual needs. Other targeted supports may be required to support the client in making changes in their substance use behaviour.

AOD: The client has a moderate to severe level of ongoing substance use which is amenable to harm minimisation, control or reduction / cessation. The client may be somewhat motivated to work on substance use issues, or well supported to carry out planned interventions around substance use. An example would be a significant level of alcohol use (Daily use over the WHO safe limits) with plans to decrease or cease use and

relevant supports to assist with goal attainment. The client has some internal resources (motivation, positive beliefs about substance-free lifestyle), and some external supports.

ABI: The client has an identified ABI with a mild to moderate overall estimate of impairment as judged by neuropsychological assessment. Client's impairments may be limited in nature (i.e. has memory problems although other cognitive abilities are unaffected), or well adjusted for (i.e. client has multiple cognitive difficulties but has accommodated to these deficits well, or is very well supported to manage the deficits). Client may be unaware of ABI symptoms and require assessment and follow-up to track changes in cognitive functioning over time if deterioration is likely. Client has the necessary awareness to link risk behaviour with changes in cognition and other health indications

Management options for medium risk-need group

This client group may best find support and treatment services offered primarily via the AOD sector and primary health care sector, with support and consultation from ABI specialist services where required.

For example, AOD treatment would focus on areas such as provision of harm minimisation information, engagement into modified counselling or case management services, induction and maintenance into pharmacotherapy programs, referral to withdrawal services or other residential rehabilitation services. These interventions may be best supported with close consultation with the AOD-ABI clinician/consultant, or with consultation with an ABI case management service, community mental health service, etc.

These clients may require intermittent intensive ABI case management or support services around deterioration in circumstances, health or psychosocial functioning – i.e. where legal issues arise, where accommodation or health situation deteriorates, where substance use escalates to risky or hazardous levels. Without significant substance use, these clients would ordinarily be able to manage most of their own care in the community without specialist supports.

These clients may require a degree of service coordination and planning of interventions around the ABI needs. Shared care or potential for shared care may require obtaining consent to share assessment results and consult with other agencies in order to design

treatment plans. Referrals and consultations need to be related to managing risk and meeting the client's goals and needs, ensuring suitable resources and supports, enhancing motivation, as well as supporting significant others involved in the client's care.

Roles, expectations and requirements of other service providers of key stakeholders need to be made explicit (to client, service providers and significant others). Planning for medium to long term needs (especially deterioration and increased need for services such as outreach case management, residential support facilities such as SRS) may require mapping the potential roles to be performed by various agencies, planning for likely setbacks, relapses, etc.

The treatment resource section provides information on adjusting counselling and general AOD service provision strategies to account for mild to moderate cognitive impairments, including:

- Modifying communication and intervention strategies with ABI clients to enhance understanding and comprehension.
- Making use of Neuropsychological reports to better recognise a client's cognitive impairments in treatment provision and adjust treatment strategy accordingly.
- Strategies for matching counselling style to an ABI client's needs
- Enhancing memory, concentration and attention in traditional counselling environments

Level three – High risk-need group

Level three represents clients presenting with serious issues of AOD dependency, polysubstance use and or hazardous substance use (as defined by DSM-IV), with concurrent moderate to severe symptoms of ABI. These clients may self-present due to acute crisis or be referred due to the at-risk nature of their substance use or self care. It is unlikely this client group will present with significant insight into the nature of their ABI, or the relationship between their cognitive impairments, substance use or related psychosocial problems. Their ABI may be longstanding (particularly due to long term alcohol use), or have been recently been identified or diagnosed, (particularly due to acute trauma such as head injury or overdose) or due to increasing problems with self-care, primary health, meeting significant obligations or legal conditions.

There may be limited awareness or insight into the cognitive impairments, or the client may be unaware of changes in their cognition due to substance use effects. Cognitive impairments may mean the client has gained minimal benefit, or had difficulties engaging with alcohol and drug interventions or other services in the past. Because of the severity of their ABI, the client will require their treatment to be modified in order to match their individual needs. It is highly likely a range of other supports may be required to support the client.

AOD: The client has a severe level of ongoing substance dependence or hazardous use with significant risk of further or ongoing ABI (i.e. falls when intoxicated, or SRBI), or other related risks to health or welfare (Overdose, BBV, safety). The client may have low intrinsic motivation to consider modifying or ceasing substance use, or not be well supported to carry out interventions around substance use (e.g. living in an unsupported environment with other at-risk users). The client may have a dangerous pattern of drug and alcohol related risk behaviours (such as impulsive offending behaviour, risk-taking behaviour).

Due to the severity of the ABI the client will be less able to distinguish high-risk behaviours, make reasonable decisions about risk avoidance and may not be able to spontaneously instigate alternative behaviours and strategies designed to control their substance use. An example would be a client with a significant hazardous level of alcohol use or polysubstance use, with corresponding poor general self-care, health and psychosocial supports. The client has few internal resources for positive change (motivation, positive beliefs about substance-free lifestyle, cognitive resources), and few or poorly organised external supports. Typically the client will have difficulty engaging with AOD services due to poorly articulated treatment needs, substance affected presentations, missing appointments or behavioural dysregulation (acting out).

ABI: The client has an identified ABI with a moderate to severe overall estimate of impairment as judged by neuropsychological assessment. The client's impairments may be profound, yet limited to one domain (i.e. has severe memory problems, or a severe language disorder), or generalised (i.e. client has multiple severe cognitive difficulties across areas such as memory, attention and concentration, frontal lobe deficits or executive dysfunction, behavioural dysregulation).

The client may be unaware of ABI and its ramifications, and require residential or home based support some or all of the time. Client will more than likely be involved with a specialist ABI service, although may require support from other agencies with coordinating assessment and follow-up assessments to track changes in cognitive functioning over time if deterioration is likely. The client will likely lack the necessary awareness to link risk behaviour with the need to implement changes behaviour and will require prompting and structure rather than insight-oriented interventions.

The client may also lack the ability to make changes in their behaviours without significant reinforcement, cueing, and support. Note this client group may include those with very recent ABI diagnoses, who may improve over time with rehabilitation and natural processes of recovery, in which case, their care needs and goals will necessarily change over time.

Management options for high risk-need group

For this client group, the most appropriate care may be facilitated via disability or specialised ABI agencies. Where clients are not appropriate for appointment-based support services, the ABI network maintains the bulk of service delivery (i.e. intensive or outreach case management, accommodation support, behavioural interventions, rehabilitation management). Alcohol and drug interventions may be delivered by either AOD or ABI agencies, with intensive support and consultation from the AOD sector. For example, a client's primary service delivery may be via a case management service with ABI specialisation, where AOD interventions may be primarily behaviour modification or harm minimisation rather than psychotherapeutic in nature.

Interventions are planned across multiple agencies with consultation with the AOD sector, and delivered in a coordinated manner, with activities such as case conferences forming the main intervention point for AOD and ABI workers. These clients may require an assessment and consultation with the AOD-ABI clinician consultant rather than be referred for assessments with a generalist AOD counsellor. AOD workers may integrate into a coordinated treatment system and incorporate elements of AOD case management such as pharmacotherapy compliance monitoring, outreach support, service coordination and supporting non-AOD agencies and staff to respond to the client's needs. For this client group, AOD clinicians need to pay particular attention to link clients into the available ABI and disability support / rehabilitation services and agencies.

The importance of making use of secondary consultation with AOD-ABI Consultant and relevant ABI and disability sector agencies should also be stressed. Clients with particularly complex needs will benefit from shared care delivery models. Effective behaviour modification requires a coordinated approach to reinforcing and shaping behaviour. Excellent communication of treatment plans, including specific strategies, shared data collection, and feedback on successes and setbacks between service providers are vital to delivering consistent behavioural interventions for clients with severe cognitive impairments.

Regardless of which agency takes the lead in case-management, case-planning meetings allow clear communication or roles and responses. AOD ABI consultants may be called upon to participate in assessment assist with treatment planning – both in terms of immediate shared care arrangements, and planning for potential relapse or deterioration in the client's situation which may lead to increased or changed treatment needs. Ongoing treatment or monitoring arrangement should be inclusive of risk indicators for any signs of deterioration including behavioural checklists, clear referral pathways for emergencies and agreed service responsibilities.

Categorising clients into levels of treatment needs

It should be noted that this assessment module is not designed as an actuarial tool, nor is it a diagnostic tool. The levels of need outlined above are designed to allow clinicians to judge overall levels of risk, make decisions about treatment suitability, and to design and communicate treatment plans where multiple agencies are involved in a client's care.

To make the most appropriate decision about which level of treatment need both ABI factors and AOD factors need to be judged together. As mentioned above, some ABI presentations involve minimal global interference, and are comparatively easily accommodated for, with minimal support. Some clients with low-level ABI conditions will experience much more significant problems than can be accounted for by their cognitive status alone, due to the effects of their substance use.

Some clients may have significant AOD problems which are quite independent of their cognitive impairments. Although the pattern of deficit may vary, all of these clients would more than likely receive appropriate support in the AOD sector. For client's whose level of ABI treatment need will not be met within the AOD sector however, consultation or referral to a disability or ABI sector agency will likely improve the result of AOD interventions, as well as opening the door to potential interventions specifically tailored to cognitive recovery, improving independent functioning, or ensuring appropriate support structures.

Where clinicians are having difficulty planning treatment delivery, whether it be around making overall judgements about the best referral and linkage options or planning individual interventions, it is recommended that they contact their regional AOD ABI clinician/consultant. These specialist roles are designed to bridge the two areas of expertise, and to support workers from both sectors in meeting the treatment and support needs of this client group. Consultation with the ABI-AOD clinician can be a one-off secondary consultation, a clinical contact (assessment or treatment provision), or to assist with service development at any level.

ABI AOD assessment module

This assessment guide has been designed to allow you to assess your client's alcohol and drug use and potential acquired brain injury status and to make decisions about the level of treatment and support required.

The assessment guide comprises several components covering known risk factors for acquired brain injury, and will allow you to make a determination as to the best strategy for delivering AOD treatment to clients with ABI.

Please note this is NOT a diagnostic guide, however it will assist with planning the most appropriate treatment plans for your clients, and assist you in gathering resources and expertise to assist with your service provision.

The assessment will cover the following areas:

Initial screening for ABI potential (Trigger Sheet)

Substance use

Lifetime and current substance use

AOD treatment history

AOD-ABI risk behaviours

Motivational assessment

Impact of ABI and substance use on cognitive functioning

Client perceptions and Heidelberg Cognitive Status Exam

Medical history (including licit substance use)

AOD relapse and risk assessment

Psychosocial history

Psychiatric history (Including Suicide / Self Harm assessment)

Forensic / legal assessment

Working with diverse and NES populations

Expected outputs

This assessment tool will allow you to place your client into one of three risk/need categories, which roughly correspond to the severity of the client's AOD and ABI status. A

comprehensive assessment will allow formulation of treatment plans, particularly where consultation is sought from the ABI-AOD clinician consultant.

When to use the ABI-AOD specialist assessment module

The short "Screening Tool" is a useful way of quickly assessing a client's potential for ABI diagnoses before conducting a more thorough assessment of ABI related presenting issues using this module.

Acquired brain injury screening tool

Alcohol: Most studies identify a 10+ year history of regular alcohol use (8+ standard
drinks per day for men and 5+ standard drinks per day for women) as being sufficient for
incurring alcohol related brain damage, particularly if drinking occurs concurrently with
periods of homelessness or inadequate nutrition. If physical signs of alcohol related brain
damage are present (peripheral neuropathy, etc) consider alcohol related brain injury very
likely. Alcohol related brain injury is likely to interfere with awareness and insight and
clients may be totally unaware of any changes in their cognition, memory, affect, etc.
Specify
Heroin / opiates: As heroin is not neurotoxic, investigate possibility of anoxic brain
damage via assessing the client's history of overdose or loss of consciousness (LOC) for
any significant period of time (minutes to hours). Prolonged periods of 'shallow' overdose -
respiratory suppression during periods of heavy intoxication can cause cumulative anoxic
brain damage. Pay attention polysubstance use with other CNS depressants.
<u>Specify</u>
Benzodiazepines: Long term benzodiazepine use (5+ years) has been associated with
neuropsychological deficits, and it is possible for impairments to be found with therapeutic
dose ranges (5-15mg Diazepam equivalent).
<u>Specify</u>

Other substance use: Has the client used any other high-risk substance types
(Inhalants, polysubstance use), or used any other substance at a daily, to intoxication level
for a significant period of time?
<u>Specify</u>
Medical: Past history of loss of consciousness (LOC) from falls, assaults, accidents
(particularly motorbike or motorcar), Current or past history of blackouts/headaches,
bleeds, space-occupying lesions (tumours).
<u>Specify</u>
Psychiatric: History of suicide attempts with hypoxic damage / loss of consciousness.
(Probe for number, chronicity and recency)
Specify
Other indicators: Impulsivity, marked personality change not accounted for by other
condition, loss of function following adverse event, history of higher previous functioning
not otherwise accounted for, collateral or informant information relating to client
functioning.
Specify

Previous ABI Assessment / Treatment History

□ Current Treatment	☐ Past Treatment	☐ Past Assessmen	t
☐ ARBIAS	□ Mell	oourne City Mission	☐ Occupational Therapy
☐ General Prac	ctitioner	S	□ Neuropsychologist
☐ Royal Talbot	☐ Hea	adway	☐ Other

Substance use assessment

It is recommended that clinicians adopt an assessment approach that matches their engagement style, the context of the assessment and the client's level of engagement and motivation. A tabular format of recording alcohol and drug use information similar to that found in the DHS Assessment Module provides a convenient summary of information once collected. Below is a sample AOD use summary which presents many important substance use variables in a convenient table.

Figure 1: Sample AOD history

	Inhalants	Cannabis	Alcohol	Amphetamines	Heroin	Nicotine
First used	14	16	14	18	19	13
Age regular	16	17	14	30	30	14
use						
Route of	Inhales	Smokes	Drinks	Intravenous	Intravenous	Smokes
administration		bongs	mainly			
			beer			
Average daily	1 large	\$20 gram	Drinks	\$50-200 1-2	\$50-200 1-	1 pack
use	can per	per day	until	times per	2 times per	day - 25
	day		intoxicated	fortnight	fortnight	cigarettes
						12-16mg
Days used in	7 / 7	7/7	4/7	2/7	2/7	7/7
past week						
Days used in	25 / 28	28 / 28	20 / 28	5 / 28	6 / 28	28 / 28
past month						
When last	Day	Day of	2 days	5 days before	4 days	1 hour
used	before	interview	before	interview	before	before
	interview		interview		interview	interview
How long	3 years	5 + years	5 + years	5 + years	5 + years	25 years
using like this						

Additional Information regarding substance use history

For all substances, useful assessment information includes the variables contained above in the table, but more detailed information can be sought for each factor. For illicit substances it is often vital to record quantity-frequency measures in multiple units: for example you may wish to record the number of substance administrations throughout a typical day (i.e. bongs smoked, injections, numbers of glasses of wine), however it is also considered prudent to measure amounts used by some other yardstick – such as amount by weight, by amount of money spent, by number of times the bottle shop is visited. As well as the details contained in the table regarding lifetime use patterns –(when first tried, when used regularly), additional detail about the nature of the substance use is vital to considering possible interventions points.

The following areas of assessment may be important in forming a comprehensive picture of the function of a client's substance use:

- The degree of substance involvement (how much of a day is consumed by seeking, procuring, funding, using, managing consequences).
- Degree of involvement in activities which support the substance use, particularly
 where these activities have some degree of intrinsic value to the client such as
 socialisation, identity development, areas of self-efficacy.
- Periods of abstinence from substance use: including the timing, duration, reason for cessation.
- Patterns of substitution reasons for substitution, possible patterns of polysubstance use.
- Licit substances used in manner not indicated or prescribed, including capacity for "doctor shopping" behaviour.
- Is the client using to intoxication? To what degree?
- Binge patterns also need to be assessed as well as dependent patterns.
- Concurrent malnutrition is important as well as general health, self-care, and accommodation issues.

AOD treatment history

In addition to information about an individuals pattern of substance use, planning a successful intervention may depend on a knowledge of what has been tried in the past, and in particular the reasons for any successes or setbacks in previous AOD treatment experiences.

Consider assessing the following aspects of previous involvement in treatment: what level of intervention, in what context, who initiated, what was the outcome; what is the client's attitudes, beliefs or expectancies about potential treatment options. For each treatment type available, there may be some pertinent factors which are specific to that treatment type:

Withdrawal history – Date, setting (outpatient, home based withdrawal, inpatient), which substance, any complications with withdrawal (i.e. seizures, behavioural dysregulation, absconding), medications used, outcome.

Counselling history – Individual, group counselling, self-help groups (e.g. AA) – type of intervention, length of intervention, outcome of counselling.

Other therapeutic interventions – Residential rehabilitation (e.g. therapeutic community), counselling or drug information courses conducted whilst incarcerated.

Pharmacotherapy interventions – Pharmacotherapy maintenance history – has the client used methadone, buprenorphine, suboxone, naltrexone, acamprosate, etc.? Length of program, dose range, effective dose, prescriber (MO), setting (e.g. community pharmacotherapy, specialist clinic), outcome.

Other interventions - Client nominated strategies or behaviours to reduce substance use or related problems – for example geographical isolation, substitution, financial controls – self imposed or externally imposed).

Periods of abstinence or controlled use - What factors assisted the client in meeting substance use goals at this time? What allied supports or services (e.g. case management, generalist counselling, supported accommodation services) were used?

Inventory of ABI-AOD associated risk-taking behaviour

Probe for risky activities directly related to substance use (route of administration, injecting safety, sharing equipment with other users, high risk patterns of substance use such as polysubstance use, using alone, using in public places).

Probe for activities to support substance use (offending behaviour, dealing, theft, fraud, prostitution). Do offending behaviours constitute skills sets which may be generalised to maximise self-efficacy?

Probe for activities secondary to substance use (spending time in high risk environments, neglecting other areas of self-care in order to focus on substance use).

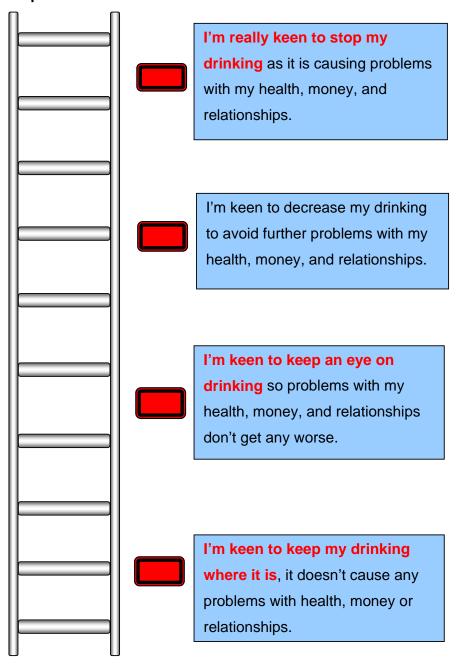
Motivational assessment

Motivational assessment involves asking the client for a rating of intention or motivation to meet a particular goal in relation to substance use, a particular behaviour change related to substance use, or to reduce their offending behaviour. Motivational assessment is one component of motivational interviewing, a set of techniques and skills designed to enhance client motivation towards a particular goal. Motivational interviewing techniques tend to be most useful when applied consistently across all stages of interventions, although they are usually most salient during motivational assessment and goal selection stages. A good understanding of the principles of Prochaska and DiClemente's (1982) Stages of Change Model is crucial for effective and responsive motivational interviewing.

Motivational assessment may begin with a motivational enhancement using an exercise such as the decisional balance. Successful motivational enhancement or identification may lead to the clinician eliciting or supporting a client to formulate behaviour change goals. For clients with marginal motivation, a generalised harm minimisation goal, or separate goals for each substance or behaviour under consideration may be required. Depending

on the client's resources and responses, you may wish to distinguish between immediate and longer-term goals. A diagram-based 'motivational ladder', may make it easier for clients to rate their levels of motivation, and provide a guide for which goal to address first in treatment. Below is an example of a motivational ladder with therapist-provided goal statements which may be relevant to a particular client experiencing ambivalence about their alcohol use.

Figure 2: Sample motivational assessment tool



A measure of motivation such as the above example can be linked to a self efficacy measure for each goal or related sets of goals to further explore capacity for change. Asking how confident or capable the client is that they can reach a given goal may identify resource and skill gaps which alcohol and drug treatment can address – such as drink or drug refusal skills, monitoring skills, relaxation techniques, and decision making techniques.

Assessing the impact of cognitive deficits and the role of substance use

A brief assessment of cognitive function is included within the DHS Specialist Alcohol and Drug Assessment module (The Heidelberg Cognitive Screen). Whilst clinicians can readily use this assessment in order to screen for the potential for ABI and the usefulness of a referral to a Neuropsychologist, it may not capture the full impact of the cognitive impairments on the clients functioning.

Insert Heidelberg Cognitive Screen with Administration and Scoring Instructions here.

Assessing the impact of cognitive deficits involves asking the client to describe the functional impacts on their general day-to-day functioning. It may be difficult to distinguish between the functional effects of ongoing substance use and the consequences of ABI, although asking for the client's own beliefs about these factors will be useful in both overall treatment planning, as well as choosing likely harm minimisation and motivational approaches. Such an assessment may allow a clinician to assess whether the client is fully aware of the consequences of their condition since client's with disorders of insight client's may not have full appreciation of the effects of their acquired brain injury. A workers appreciation of likely consequences of brain injury, as well as collateral information sources such as significant others, family members, other workers may be required to assess these effects more broadly.

Depending on the material spontaneously nominated by a client and the context of the assessment, clinicians may wish to assess the impact of deficits using the following prompts. Note it is the client's theories concerning the nature of their deficits, and the function of substance use which are of interest.

Effects on cognition - Has the client noticed any affects of their ABI or drug use on their thinking skills – such as paying attention, remembering, making decisions, task completion?

Effects on emotional functioning - In what way does their ABI or substance use affect their emotional functioning? Do they view their ABI or their substance use as having detrimental or beneficial effects? Are they aware of any long term effects of their use on their ability to regulate or manage feelings?

Effects on living skills - What effects does the client see their substance use having on their general day-to-day functioning including ability carry out tasks such as self care activities, finances, leisure, and social obligations? Are there particular behaviours which are problematic for clients (self-care activities, managing bills, cleaning, mobility, etc.)?

Effects on relationships - Can the client nominate any effects of their ABI or substance use on their capacity to initiate, maintain, and develop healthy relationships? Would a significant other have a different view of these possible effects?

Effects on vocations / employment - Is the client aware of any impacts of their ABI or substance use on their ability to secure employment or purposeful activity in the community?

Effects on offending behaviour - Does the client see any relationships between their ABI and substance use and their offending behaviour? Does the client recognise offending behaviour as problematic, does their ABI or substance use affect the way in which their offences are committed?

Effects on health - Is the client aware of the effects on their general health of their substance use or ABI? Where substance use has particular effects (such as liver damage) are they aware of the potential harms? What monitoring is in place (if any)? What is the general level of knowledge of substance related harms, particularly in relation to ongoing cognitive impairment?

Medical history and indicators of potential brain injury

Assessing medical history indicators for ABI may be helpful in building a picture of a client's potential for injury in an exploratory assessment, it may help in building a strong referral for neuropsychological evaluation, and it may also assist with engagement by allowing the treating AOD clinician to demonstrate interest and commitment to accurate understanding of the client's cognitive difficulties. Clinicians are recommended to be 'inclusive' in exploring potential areas of brain injury – asking clients specifically about various injury mechanisms to ensure all possible sources of brain injury have been assessed.

Areas of assessment

Anoxic brain damage - Explore potential incidents of anoxic brain damage, whereby oxygen supply to the brain has been limited or ceased. Note there are many potential risks for anoxic brain damage including near-drowning, complications in childbirth, suicide attempts, heart attacks, accidents, etc.

Closed head injuries - Blows to the head not causing skull fractures / penetrating head wounds. This form of head injury includes concussions, motor car accidents, falls, assaults, sporting injuries. Probe for period of loss of consciousness. Probe for period of post traumatic amnesia (PTA).

Open head injuries - Also referred to as penetrating head injuries (involving a skull fracture and rupture of the meninges (protective layers of tissue surrounding the brain). Probe for period of loss of consciousness. Probe for period of post traumatic amnesia (PTA).

Other BI Indicators from medical history

Stroke – Can the client specify where the stroke was? Are they aware of any neurological or neuropsychological sequelae of the stroke? What impairments followed the CVA if any? Are there any lasting effects? What rehabilitation was indicated? Did the client comply with rehabilitation program or any ongoing treatment?

Degenerative neurological disease – Type, level of dysfunction, prognosis, schedule of neurological and neuropsychological follow-up – e.g. When is the next assessment or scan? Is the client complying with a medication regime or treatment regime?

Methods of assessing ABI from injury severity - Glasgow Coma Scale

The GCS introduced by Teasdale and Jennett in 1974⁶ provides the best initial measure of severity of head injury. The score is the sum of the scale's three measures of eye opening, and best motor and verbal responses. This ranges from a score of 3 for a patient with no motor or verbal response or eye opening to painful stimuli, to 15 for a patient who is orientated, follows commands, and has spontaneous eye opening. Patients who do not follow commands, speak or open their eyes, with a score of 8 or less, are by definition in coma. TBI is defined as *mild* by an admission GCS score of either 13 or 14–15, *moderate* by a score of 9–12 or 13, and *severe* by a score of 3–8. The score on admission, and its prognostic usefulness, are obviously easily confounded by factors other than TBI, particularly substance misuse, but its sequential recording after admission plays a crucial role in recording early progress and in management.

Methods of assessing ABI from injury severity – Duration of post traumatic amnesia

Another standard classification of head trauma severity based on the duration of post-traumatic amnesia (PTA), (Jennett & Teasdale, 1981), and is reproduced in Figure 3. This classification system is an expanded version of the system developed by Russell & Smith (1961). Once the patient is out of PTA (once memory functioning has returned to normal or the immediate effects of the injury on gross memory function have resolved), they should be classified according to this system. Age and any prior history of concussion or head injury will complicate this picture although it is held to be reliable for most instances of traumatic brain injury. PTA is calculated from the time of the accident, it therefore includes the period of coma. Many clients will require assistance with following up the length of PTA by contacting and requesting relevant documentation such as hospital discharge records.

Figure 3: Classification of post-traumatic amnesia (PTA)

Duration of PTA	Severity of Injury
Less than 5 minutes	Very mild
5 - 60 minutes	Mild
1 - 24 hours	Moderate
1 - 7 days	Severe
1 - 4 weeks	Very severe
Greater than 4 weeks	Extremely severe

Licit substance use history

Many clients can have complicated medication histories, sometimes reflecting their involvement with separated treatment providers (such as a generalist GP, psychiatrist, and Addiction Medicine specialist who are not aware of each other's role). In addition, the role of medication in alleviating various symptoms of ABI may be poorly understood, meaning medications are often incorrectly prescribed, or prescribed for longer periods than would be usually considered appropriate. In particular, medications which may be helpful for short term management of conditions like anxiety, sleeping problems and pain may continue to be prescribed to the point where the client develops a secondary dependency to the substances beyond their purported therapeutic effects.

Long-term prescription of benzodiazepine class drugs is a frequent occurrence, and as these drugs have significant potential for abuse, their use should be monitored and reviewed regularly. Doctor shopping behaviour for benzodiazepines and opiates in particular may be retained as an intact 'skill' in clients who have a history of abusing prescription medications, who have a 'medicalised' attribution about their substance use 'problem', or who can no longer fund illicit substance use. Distinguishing between 'therapeutic' and 'intoxicating' uses of medications can be problematic, although clues such as running out of scripts early, being involved with multiple prescribers or pharmacies, secrecy around relationships with prescribers, staunchly avoiding discussions

of medication effects and reasons for prescription, and hoarding medication are potential signposts to licit substance problems.

An accurate history of medications currently being prescribed can be used to investigate the potential for abuse of licit substances. Taking this history whilst focussing on other medical issues may also serve to diminish client perceptions that medicalised substance abuse will be 'targeted' and lead to more accurate self-report behaviour. Clinicians may require secondary consultations with medical officers or psychiatrists to identify legitimate and questionable medications being used, as well as check with families / significant others / service providers to assess compliance.

Clinicians may also wish to explore past history of prescribed medication, reason prescribed, effectiveness, reason for cessation, particularly where issues of non-compliance or abuse are present.

Figure 4: Medication history

Medication	Dose	Taking as	Duration of	Reason for	Prescriber
		prescribed?	treatment	prescription	
		Method of			
		administration			

Alcohol and drug use relapse / Risk assessment

Cognitive indicators

- Nature of beliefs regarding substance use and substance control/abstinence.
- Nature of expectancies regarding substance use and substance control / abstinence.
- Overt planning for substance use or planning for high risk activities.

Behavioural indicators

- Increasing high risk behaviours, testing self-control behaviours, low level self-harm behaviours, diminishing self-care behaviours, fewer positive behaviours.
- Acquiring substances or means to use substances, initiating contact with other users, becoming withdrawn or secretive with workers and significant others.
- Non-attendance at appointments.

Motivational indicators

- Reduced risk management behaviours.
- Reduced motivation to engage in alternative activities or to implement strategies designed to manage substance use or offending behaviour.
- Reduced motivation to engage with counsellor or case manager.

Psychosocial history

Taking an effective psychosocial history allows the AOD clinician to understand the broader context of an individual's substance use and related concerns, building a comprehensive picture of the client and their social environment. Psychosocial histories allow a clinician to capture material which is potentially the most engaging and illustrative of a client's strengths and weaknesses. Given that levels of functioning within the psychosocial sphere (such as connectedness, relationship functioning, access to support) can be so crucial to supporting positive outcomes for ABI clients it is a particularly relevant area of assessment with this client group.

It is frequently via the psychosocial context that areas such as pre-morbid levels of functioning, personality style, relationship functioning, attitudes and expectations of self and others, and supports are elicited. Typically a comprehensive psychosocial history

might also contain a history of the client's early learning environment, particularly if counselling interventions are planned and a clinician wants to assess belief structures, core themes in personality organisation and potential cognitive distortions. Areas of relevance in psychosocial histories vary considerably with the context, however the following are some domains which may be helpful to review with the ABI-AOD client.

- Family relationships, connections and linkages (useful to use a genogram here), including patterns of contact / support / conflict. It is also useful to consider these patterns both before and after the ABI was incurred.
- Current 'significant other' relationships partners, friends, supports.
- Patterns in relationships including expectations of others and self (can be
 particularly useful for mapping potential issues in treatment relationships, for
 example, unrealistic expectations or patterns of dependency).
- Interests, skills, strengths, hobbies, etc.
- Occupational and educational history.

Psychiatric history

As mentioned in the literature review for these guidelines, clients presenting with Acquired Brain Injury frequently experience increased rates of psychiatric disorder, including anxiety spectrum disorders, depression and psychosis. A comprehensive psychiatric history may not be appropriate to all assessment contexts, however as a minimum, clinicians are recommended to probe clients about potential current symptom levels, current and past treatment history, and if indicated, carry out relevant risk assessment and follow through with referral and or support where indicated.

Clinicians may wish to use the following suicide / self-harm risk checklist and structure an appropriate contingency plan for their clients. The role of increased impulsivity, emotional dysregulation and impoverishment of response choices should be taken into consideration when conducting risk assessments with ABI clients, and in selecting support strategies and interventions. Where clients are unable to spontaneously recognise an appropriate response to a worsening in psychiatric symptoms they may need additional resources to cue the appropriate response.

Suicide/Self harm risk checklist

This form is designed to help you determine the level of risk of suicide and self-harm for your client. It is intended as a guide only. Health professionals must exercise individual judgement in individual cases. Discuss your assessment or management plan with your supervisor or a senior health professional if you are in doubt.

Based on a detailed clinical assessment, judgements are made as to what category of risk best matches the client's presentation. The most salient risk factors occur at the start of the form, with factors less closely associated with for suicide and self harm are presented last.

Risk factors	Low risk	Moderate risk	High risk	
Previous attempts	Consider potential lethality and recency of attempts: Recent multiple attempts by			
	moderately lethal means or any previous attempts of high potential lethality represent high			
	risk. More recent and letha	al attempts by family or friends rep	resent higher risk.	
Self				
	Previous parasuicidal /	Previous suicide/self harm	Previous high lethality	
	self harm behaviour of	behaviour of moderate lethality	suicide behaviour, high	
	low lethality or frequency.	or high frequency.	frequency attempts.	
Family members or close friends				

=			
Current suicidal ideation	Consider how the suicidal	ideation has been communicated	. Non-disclosure may not
	indicate low risk. Commur	nication of plans and intentions ar	e indicative of high risk.
	Consider non-direct or non	verbal expressions of suicidal id	eation such as depressive
	body language, 'good byes	s', drawing up of wills, unexplaine	d termination of treatment
	relationships etc.		
	Consider also homicidal id	eation or murder / suicide ideation	n.
Intent			
	No specific intention to	Intention to suicide in near	An immediate intention to
	act or no strict time frame	future but not immediate.	act or highly specific
	is communicated.		time/date is communicated.
Plan			
	Communicates a vague	Communicates a viable plan,	Communicates a detailed
	plan, few details elicited.	but with limited detail.	plan: when, were and how
			with high probability of
			completion.
Means			
	Means not available or	Means available but not	Means obtained and
	easily accessible.	obtained.	immediately available.

Lethality			
	Minor self-harm	Planned overdose, motor	Firearms, hanging, jumping,
	behaviour such as binge	vehicle accident, serious	carbon monoxide.
	substance use,	cutting.	
	superficial cutting.	Intervention by other possible	Intervention unlikely (social
	High probability of	(e.g. telephone call or	isolation, remote location).
	intervention by other.	neighbour).	
Mental health factors			
History of or current depression			
	Lowered mood which can	Lowered mood without	Diagnosed major depressive
	resolve situationally.	resolution with additional	episode (DSM-IV)
		symptoms of depression.	
Mental health disorder or			
symptoms	Some clinically significant	Pronounced clinically	Multiple or pronounced
	symptoms of mental	significant signs of mental	symptoms of mental illness
	illness (e.g. overvalued	illness with some concurrent	with little or no management
	ideas of worthlessness or	management.	e.g. untreated psychotic
	persecution) Or		illness with hallucinations

significant mental illness well managed.

and delusions present.

Protective factors	Protective factors include social supports, ability or inclination to make use of support where available, family involvement, work or school relationships, stability of lifestyle,				
		Many: Client is highly	Some: Client is somewhat	Few: Client is highly	
	interconnected and	isolated or supported, has	isolated, has few supports		
	supported and has many	some internal resources and	and coping mechanisms		
	internal resources and	some coping strategies.	and/or few internal		
	flexible coping strategies.		resources.		
Coping skills and resources					
Family, friendships and other					
support networks					
Stable lifestyle					
Communication skills / ability to					
make use of supports					

Self harm risk assessment

Level of risk

No or minimal risk

Low risk: Some thoughts but minimal risk factors, no previous attempts, no specific plan, intention or means, evidence of minor self harm, protective factors (e.g. available supports).

Moderate risk: Thoughts, some risk factors, plan has some specific detail, means are available, intention to act in near future but not immediately, some protective factors (e.g. inconsistent supports).

High risk: Thoughts, previous attempts, risk factors, clear and detailed plan, immediate intent to act, means are available (and lethal), social isolation.

Action

Monitor as required.

Monitor closely and agree on a verbal or written contingency plan* with client, provide support numbers and obtain commitment to follow contingency plan should feelings escalate.

Offer or refer for further assessment/contact with metal health or other appropriate service. Agree on a written contingency plan with client clearly outlining relevant supports to be contacted if feelings escalate. Request permission to inform emergency monitoring team (CATT) and/or family. Consultation with supervisor as necessary.

Immediate referral to hospital mental health services or emergency mental health team, or call ambulance/police if necessary, break confidentiality, management plan, obtain

support from supervisor if required.

- * Note contingency plan should include the following to me most useful.
 - A number of behavioural alternatives to suicide/self harm formulated by clinician and client.
 - An agreement that these alternatives will be followed before instituting suicidal or self-harm plans.
 - A statement concerning the time period over which the contract applies.
 - A formal statement of treatment goals for resolving the suicidal ideation and the responsibilities of each signatory.
 - (Although this document seeks to provide alternatives to self-harm and is not a 'suicide contract' per se it may also include a formal statement where the client undertakes not to attempt suicide or self harm).

Legal and forensic history

Assessment of legal issues and forensic behaviour are in many instances dependent on the context of assessment (particularly where mandatory treatment has been sanctioned). Entering into discussions of criminality and offending behaviour is frequently seen as potentially disengaging in treatment, particularly where the limitations of confidentiality become complicated by the existence of 'third parties' in the treatment relationship (such as the courts).

Clients may feel compromised in engaging with a treatment provider where they have concerns about issues such as confidentiality and mandatory reporting.

In general, an honest approach towards clarifying reporting relationships and the limitations of confidentiality is seen to be the preferred route for building sincere therapeutic relationships, however, particular attention may need to be paid to how this information is conveyed when working with clients with comorbid ABI and AOD issues.

Information related to offending behaviour and legal issues may be better elicited under other assessment domains in order to more firmly embed the treatment of offending behaviour into the treatment of substance use issues. Eliciting relevant offending behaviour information during assessment of substance use (for example, behaviours carried out to support substance use), or the motivational assessment (beliefs about changing offending behaviour) may offer the clinician more clues as to potential interventions than by approaching offending behaviour as a separate assessment section.

This type of assessment may allow clients to view their offending behaviour as part of a broader set of choices and responses – and may both help build motivation to address substance use behaviour, as well as suggesting behavioural interventions not immediately connected with offending (such as improving general self-care skills).

Working with clients from diverse and non-English speaking backgrounds

Provision of services to non-English-speaking background individuals and those from diverse cultural backgrounds can present some unique challenges to clinicians managing

complex care needs. Language and cultural barriers can complicate successful engagement, assessment and treatment provision, particularly where access to interpreter services or culturally sensitive or congruent staff is limited.

Some cultural groups have particular patterns of substance use which may involve elevated levels of risk for the development of cognitive deficits or acquired brain injury, either because of that group's knowledge or perception of health consequences of substance use, or associated risk factors such as rates of victimisation, involvement in particular types of substance use such as inhalant use.

A comprehensive approach to management of cultural and linguistic diversity in the context of co-existing alcohol and drug and acquired brain injury issues is beyond the scope of these guidelines. Clinicians are recommended to seek referral to, or consultation with culturally relevant support groups and organisations in order to meet the needs of their clients in a sensitive and effective manner.

ABI AOD Clinical Treatment Guidelines Part 4: ABI-AOD treatment resources

Introduction

The following resources have been prepared to assist you with understanding your client's individual pattern of cognitive impairments and in planning some appropriate alcohol and drug interventions strategies.

This resource section is divided into five sections:

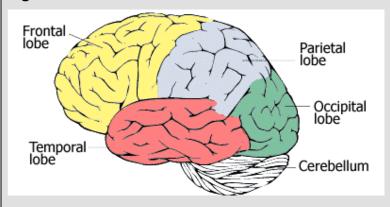
- 1. Acquired brain injury: What damage does to different areas of the brain.
- 2. Common problem areas associated with ABI
- 3. Tailoring treatment: Strategies useful for particular deficits
- 4. Integrating forensic treatment into AOD service provision
- 5. Case examples: Two fictional client vignettes

Acquired brain injury: What damage does to different areas of the brain

Neuropsychological reports and brain imaging scans often cite damage to one or more areas of the brain following traumatic brain injury, stroke, lesions and tumors. The following boxes summarise commonly occurring consequences of focal brain damage.

Bear in mind individual expression of deficits is often idiosyncratic and lesions in the same areas of the brain may have different effects for different people.

Figure 5: Different areas of the brain



Frontal lobe damage

The frontal lobes of the brain are responsible for a range of complex, integrative cognitive functions, often referred to as 'executive functions', including decision-making and aspects of self-awareness. Damage to the frontal lobes of the brain is thought to be particularly concerning due to the effects on thinking, emotions and behaviour. The frontal lobe is also involved in the initiation and monitoring of voluntary movement, so physical disabilities can also result from frontal lobe damage. The following issues are frequently noted with damage to the frontal lobes of the brain:

- Planning and organising
- Problem solving
- Learning from mistakes
- Short term memory
- Flexibility in thinking and behaviour
- Seeing consequences of behaviour

- Putting plans into action
- Making new decisions
- Flexibility
- · Loss of initiative
- Rigidity and preservation
- Impulsiveness and disinhibition
- Initiating and monitoring voluntary behaviour

Limbic System

The limbic system is deep in the brain and controls emotions, learning and memory It traverses 4 anatomical areas

Is the "pleasure" or "reward" pathway of the brain

Temporal lobe damage

- Difficulty remembering new things
- Difficulty with language (aphasias) (dominant hemisphere)
- Disorders of sensation and sensory integration (dominant hemisphere)
- Behaviour changes including episodic hyperirritability, anger, aggressive outbursts and sudden onset of dysphoric mood states.

Parietal lobe damage

- Can't tell left from right.
- Difficulty reading and writing.
- Difficulty with initiating and monitoring voluntary movement.
- Spatial disorientation.
- Ignoring opposite side of body.
- Difficulty putting things together.

Occipital lobe damage

- Inability to recognise objects and faces even when able to see them
- Loss of parts of visual field

Cerebellar damage

Poor coordination.

- Jerky movements.
- Impaired balance.

Brain stem damage

Loss of consciousness.

Common problem areas associated with ABI

'Clinical problems' arising from ABI can be grouped into the following general problem areas. Not all clients with an acquired brain injury will experience all of these issues, although many clients will experience multiple issues depending on the level of impairment, their pre-morbid capacity, the success of their rehabilitation, the level of support they experience and the services available. Problem areas include:

- Cognitive impairment
- Communication problems
- · Behavioural problems
- Psychiatric problems
- Other issues

Cognitive impairment

Cognitive impairment includes any degradation in 'thinking skills', including changes to attending, storing and recalling information, alterations to the efficiency, speed and capacity of individuals to manipulate information and make decisions, as well as alterations to the cognitive aspects of managing mood and behaviour.

Common cognitive impairments

- Difficulty concentrating, quick to fatigue note this is not physical fatigue, but refers to mental tiredness, inability to concentrate, and integrate new information.
- Difficulty sequencing tasks and organisation the client may understand the correct steps in a given task (such as cleaning a car), but may not carry out the tasks in the correct order, or may not prepare adequately, or ensure they have the necessary resources before carrying out a task.
- Difficulty putting plans into action
- Difficulties learning new information or tasks
- Difficulty solving problems, particularly new problems.
- Difficulties coping with or adapting to new situations
- Rigid, inflexible thinking (concrete thinking)
- Difficulty understanding complex communication
- Difficulty in expressive language--saying what is meant.
- Slowness in responding
- Short term memory problems
- Perseveration--repeating the same mistakes
- Perceptual problems vision, hearing, touch, smell impairment
- Confusion

Probing for cognitive impairment: What to ask your client

- Have you noticed any changes in your thinking?
- Have you noticed that things are harder to do than they used to be?
- What kinds of things are taking longer to do than they used to?
- Has anyone else commented on how well you get things done?
- Have these changes had any other effects (substance use, legal, relationships, occupational, educational, mood, hobbies/interests etc)?

Communication problems

Communication problems associated with ABI may arise as a result of damage to the speech production or comprehension areas of the brain (aphasias), as well as damage to

the parts of the brain which control the muscles involved in speech. Speech and communications problems can involve partial or complete interruption to many or one mode of communication, although the best known are Broca's and Wernicke's Aphasia. In most people Broca's aphasia relates to damage to a specific area in the lower part of the left frontal lobe of the brain. It is one of the main areas in the cerebral cortex responsible for language because it controls the motor (muscle movement) aspects of speech.

Clients with a Broca aphasia can usually understand what words mean, but have trouble performing the motor or output aspects of speech. Broca's Aphasia is also referred to as 'expressive' or 'motor' aphasia. Depending on the severity of the lesion to Broca's area, the symptoms can range from the mildest type (cortical dysarthria) with intact comprehension and the ability to communicate through writing to a complete loss of the ability to speak out loud.

Wernicke's aphasia consists of loss of comprehension of spoken language, loss of ability to read (silently) and write, and distortion of articulate speech. The affected clients may speak fluently with a natural language rhythm, but the result has neither understandable meaning nor syntax. Despite the loss of comprehension, the word memory is preserved and words are often chosen correctly. Alexia, agraphia, acalculia, and paraphasia are frequently associated. Some patients are euphoric and/or paranoid. Wernicke's aphasia is due to lesions in the temporal lobe.

Behavioural problems

Behavioural issues arising as a result of Acquired Brain Injury subsume a wide range of problems, including interruptions to 'normal' behaviours such as an inability to perform a particular behaviour at the appropriate time, to an inability to inhibit a behaviour at an inappropriate time. Behaviour change can also involve the occurrence of behaviours not previously demonstrated prior to the ABI, including threatening and challenging behaviours and high risk activities, sexual disinhibition, and emotional dysregulation.

Often clients with behavioural problems will not self-present for treatment, as they may lack the ability to perceive or control their problematic behaviours. Families, carers and other workers involved in the client's care may be much more significantly affected by a client's behaviour and may initiate. Behavioural interventions are designed to reinforce positive behaviours and 'extinguish' negative ones. Behaviour modification usually

involves a detailed functional analysis of the behaviour, involving accurate recording of incidents and an analysis of the likely need the behaviour is serving for the client (if inappropriately). Quite often the bulk of behaviour modification interventions occur 'around' the client, with carers, families and services adjusting the environment and their responses to behaviour in order to minimise adverse outcomes, and if possible, shape the client's behaviours in a more appropriate direction. Examples of behaviour modification include assisting clients with initiating self-care activities like showering, and managing sexually inappropriate behaviour.

Common behavioural problems

- Increased disinhibition
- Lack of initiative (inertia)
- · Low levels of motivation
- Recklessness, lack of ability to self monitor behaviour
- Self-centeredness
- Sexual dysfunction
- Reluctance to shower
- Wandering behaviour
- Confusion
- Withdrawal
- Aggressive outbursts
- Unwillingness to eat
- Forgetting to go to the toilet
- Oppositional / defiant behaviour: e.g. saying "no" all the time
- Uninhibited grieving

Exploring behavioural concerns

As mentioned above, clients may not self-present for behavioural concerns, and undertaking interventions to manage behaviour needs to be carefully managed, with accurate definition and location of the problem (who's problem), accurate data gathering about the problem (when, where, how often, in response to what etc), and a realistic approach to management (operate on the environment or intervene with the individual).

Often environmental manipulation can be more effective than attempting to change a clients reaction to that environment – particularly for low-incidence behaviours. Clinicians will need to be realistic in communicating the likely outcomes of behaviour modification, and consultation with ABI consultants or similar experienced consulting agencies or clinicians is recommended.

Psychiatric problems

Acquired brain injury is associated with increased rates of psychiatric illness, particularly anxiety and depressive spectrum illnesses. People experiencing significant loss, undergoing difficult changes in role, relationships and levels of functioning may have difficulty coping. Treatment for psychiatric illnesses is vital for ensuring the best functional outcome for the client and in ensuring that alcohol and drug interventions have maximum potential benefit.

Common psychiatric outcomes of ABI

- Mood changes (dysregulation due to frontal lobe damage especially significant)
- Anxiety disorders generalised anxiety disorder, phobias, OCD.
- Depression, suicidal or self-harming behaviour
- PTSD (particularly with traumatic brain injury)
- Overactivity / hypomania / bipolar affective disorder
- Euphoria / lack of insight

Exploring psychiatric concerns with ABI clients

Bear in mind that where ABI begins to interfere with processes of self-awareness and insight, a client's ability to draw inferences about their own mental state and contributing stressors may be diminished. Whilst clients may be able to give accurate information about subjective symptoms, collateral information may need to be sought in order to explore issues such as chronicity, onset, and relationship with stressors or precipitants.

Family members and other treating clinicians may also have more accurate recall about details such as past and current medications, existing treatment relationships or interventions.

Quite apart from changes in cognition and mood, individuals experiencing acquired brain injury often experience changes in personality functioning. These changes may be mediated by a range of other altered functions – such as diminished cognitive capabilities, or more labile mood. Personality changes may manifest in a range of altered reactions, beliefs, expectations and behaviours. Individuals with significant frontal lobe damage often

exhibit changes to personality functioning. Families, carers and significant others are usually more aware of the changes in a clients personality than the individual themselves, and the effects of these changes can be more devastating to maintenance of relationships and supports.

Common personality changes following ABI

- May become irritated more quickly
- May have emotional outbursts
- May not always think about others
- May talk more, jumping from one topic to another, making it hard to understand the point
- Might be impulsive
- · May have less emotional control
- May not notice they have changed
- May have social problems
- May be more suspicious

Probing for personality changes with ABI clients

- Have you noticed your reactions to things changing?
- Are your emotions more intense (or more changeable)?
- How are you getting along with other people lately?
- Have your views about the world changed?

Other problems experienced by clients with ABI

Adjusting to an acquired brain injury has ramification beyond an individuals cognitive and emotional capacity. The combination of functional deficits, changes in self-concept, and the ways in which society perceives brain injury may lead to broader problems and barriers to recovery or treatment endeavours. The following is a list of commonly reported issues faced following ABI.

- Social isolation and lack of appropriate opportunity for socialisation.
- Loss of role-identity (occupational, family, relationship).
- Loss of self-esteem and self-efficacy.
- Diminished capacity to advocate for self / locate and access services.
- Financial hardships diminished resources as well as diminished capacity to manage finances, increased financial vulnerability.

The unseen injury – Minor head injury

Where clients have experienced very minor head injury, clinicians still have an opportunity to pass on information regarding optimum recovery management and improve long term outcomes for clients. Up to 95% of all head injuries are minor with no outward sign of injury. In many cases of minor head injury the head itself may not be struck. Often the injury occurs as a result of sudden violent motion or acceleration / deceleration e.g. whiplash, being shaken. Minor head injuries are common in sports including football, and boxing. In cases of minor injury where there is no focal damage or lesion MRI scans can often identify damaged nerve fibers which have been stretched, torn or bruised. Most people receive no follow up after visiting casualty despite the requirement for careful recovery from minor head injury.

Minor head injury may still have noticeable symptoms such as headaches, excessive tiredness, poor concentration and memory, emotional problems, reduced tolerance of alcohol and drugs. Whilst these symptoms will gradually disappear over weeks or months it is vital that the brain be allowed to recover without undue stress. Mental activity should be reduced to routine tasks that are undemanding. Major decisions, changes of routine should be delayed and return to work should be gradual. Often it is helpful to educate others (including families, employers, coaches) about subtle hidden problems of minor head injury.

Neuropsychological testing

Referral to neuropsychological testing is the most commonly made referral for assessing or re-assessing cognitive impairments or acquired brain injury. Most aspects of AOD treatment delivery will be more effectively implemented if a clients cognitive strengths and weaknesses are taken into consideration via a neuropsychological report. Whilst it is possible for sophisticated imaging techniques such as MRI, PET and SPECT to show

detailed representations of brain structure and function, these types of investigations are generally not readily accessible or interpretable by the majority of clinicians.

Neuropsychological evaluations tend to be based much more around real-world functions, and most reports should include at the very least a practical translation of the 'real world' implications of test results into plain English. Of particular utility are reports where suggested strategies for engaging with and working with clients with respect to their particular pattern of cognitive strengths and weaknesses are included.

When is neuropsychological assessment appropriate?

- When ABI risk factors are identified on A&D assessment (i.e. long history of alcohol
 use, incidents of head injury with loss of consciousness, etc).
- When the client or significant other reports any sudden or unaccounted for changes in cognitive functioning.
- When there is a medical condition which may have impact on brain functioning.

How does neuropsychological assessment help the AOD clinician?

- Making treatment plans cognitively appropriate to the client.
- Assessing suitability for AOD interventions (i.e. capacity to engage in different modes of counseling, pharmacotherapy regimes, residential services).
- Determining appropriate levels of intervention (i.e. harm reduction / relapse prevention / insight oriented therapies).
- Structuring appropriate treatment goals, appropriate pace of interventions, managing expectations of recovery speed.
- Identifying range of potentially helpful treatment interventions (e.g. Identifying mental health concerns secondary to ABI, sleep hygiene).
- To facilitate referral to relevant support programs, (e.g. supported accommodation, primary health care, occupational therapy, speech therapy, vocational support, structured activity programs etc).
- Determining the limitations of informed consent to engage in treatment under the legal definition of consent, a client with a full-scale IQ of 70 or less may not be able to provide consent to engage in treatment, and consent of a legal guardian or administrator may need to be sought.

Contra-indications to neuropsychological testing

- Active and unstable psychiatric condition such as major depressive episode, manic episode, psychotic episode.
- Acute medical issues which may compromise performance.
- Acute intoxication and withdrawal states.
- Low client motivation, which is not amenable to enhancement.
- Unclear or chaotic pattern of substance use (I.e. cannot establish with certainty intoxication or withdrawal status).
- Recent Neuropsychological testing has been conducted but further deterioration or improvement in condition is likely and required re-assessment.

Important steps before a neuropsychological referral

- Have you conducted an ABI screening / assessment and identified possible causes, symptoms and presenting issues?
- Have you formulated a clear referral question?
- Why are you referring now?
- Does the client have a good understanding of why the assessment is being done?
- Do wait times make testing relevant to your involvement or would the results be part of a client's follow-up with another agency?
- Do you have an accurate history have you determined whether a recent neuropsychological assessment has been conducted?
- How will the results be interpreted / used, who will be giving the client feedback?
- Who will be communicating results to family / significant others / other service providers involved with the client?

Tailoring treatment: Strategies useful for particular deficits

The following section contains a summary of frequent clinical complaints found when working with clients with ABI or cognitive impairments. The presenting features of the symptom or problem are summarised, and strategies for overcoming or adjusting treatment provision to accommodate the problem are also suggested.

Deficit area: Memory problems

Presenting features / symptoms

Client forgets names, appointments, messages, phone calls, information which has been read, where things have been put, gets lost in familiar streets, forgets what they are doing or speaking about in sessions.

- As prospective memory problems involve forgetting to do something or remembering it at a future time, clients can be highly amenable to the use of compensatory aids. It can be helpful to distinguish between problems with verbal / auditory memory and visual memory – some clients can remember what they hear but not what they read.
- Assist the client in developing "compensators" for their particular memory deficit.
 Bulletin boards, calendars, designated places for important items, lists of where things are kept, whiteboards, pictorial calendars are all helpful household compensators.
- Encourage the client to carry a multi-section notebook or diary and record things
 that need to be done, how to get from one place to another, important dates, names
 places, etc. These can all be recorded in one place which means "check the book'
 may be the only skill that needs to be learnt.
- Electronic diaries and Personal Digital Assistants are particularly powerful for
 prospective memory problems (remembering to do things at some time in the
 future) as they can cue action via alarms and reminders. However, acquiring the
 skill and confidence to use electronic tools may be a barrier for some clients who
 have no familiarity with these devices.
- Help formulate reminders for clients to write and refer to their notebook / diary, and model using the notebook or diary during sessions.
- Help clients make their notebook / diary relevant for planning ahead by anticipating

- and recording important details, goals, strategies, due dates, appointments, etc with them in sessions.
- Repeat instructions and ensure that agreed goals, actions or simply a record of topics covered in each session are recorded in the client's notebook / diary. (Again, modeling recording important information may help to destignatise and encourage the behaviour).
- Encourage mnemonics and check they assist recall it may be possible for word association, acronyms etc to be useful in remembering names, orders of carrying out tasks, lists of things, etc.
- Identify and manage memory blockers such as patterns of substance use, depression or anxiety symptoms, preoccupations, and poor attention processes (failure to attend to important information).

Deficit area: Poor concentration

Presenting features / symptoms

Client loses concentration and becomes easily distracted. Complains of being bored, no longer enjoys activities requiring concentration, has a short concentration span – jumping from one thing to another and has difficulty concentrating on more than one task at a time.

- Help provide an environment free from distractions this may include visual and auditory stimulation, social distractions, etc.
- Break down activities and sessions into short bursts with regular breaks.
- Focus on realistic goal setting, assist the client to break up difficult goals into manageable 'chunks'.
- Focus on physical activities requiring less concentration to counter boredom or lack of interest e.g. walking / exercise, gardening, housework, practical hobbies.
- Consider role of anxiety / depression in contributing to attention and motivation.

Deficit area: Slowed responses

Presenting features / symptoms

Client is slow to answer questions or perform tasks and has difficulty keeping up in conversation. Client recognises the correct strategy to use in a situation but acts too late, meaning his/her ability to respond quickly in an emergency may be lost.

- Allow your client more time to respond and complete tasks. Be patient.
- Manage your own presentation of material and 'thinking speed' so as not to get ahead of your client in a discussion.
- Coach clients in strategies which allow them more time to decide what to do this
 may include rehearsing behaviours which can be used in multiple situations (such
 as going for a walk, using the toilet, phoning a supportive friend or worker).
- Avoid suggesting behaviours in which the client may get into situations of high risk (e.g. assess local areas and travel routes for relapse risk, safety considerations).
- You may need to organise assessment of your client's suitability to drive.
- Client may need support with managing home safety (e.g. assistance with smoke detectors, installing a simple 'fire plan' on back of front door).

Deficit area: Planning and problem solving deficits

Presenting features / symptoms

Client has difficulty solving problems, planning and organising new activities. They may present as listless or unmotivated due to difficulty with initiating activity or solving behavioural dilemma. They may also be initiating habitual behaviours (such as substance use or offending) due to lack of alternatives in meeting some need.

Suggested strategies

 Brainstorm all alternative solutions with the client and use motivational interviewing to focus in on the simplest workable option, assisting the client to plan concrete steps.

OR

- Give client several concrete solutions from which to choose, with the benefits and drawbacks of these solutions made clear and unambiguous.
- Structure complex tasks in a step-by-step fashion.
- Record steps simply (pictures or diagrams may be helpful e.g. how to get to rehab
 via detox with behavioural expectations, time spent, etc.).
- Structure a blank decision making / planning pro-forma and put into diary or notebook along with other examples that are working. The client may choose to use this tool to work though a new problem or situation.
- Follow up to verify that the chosen strategy was helpful / successful.

Deficit area: Lack of Initiative

Presenting features / symptoms

Client may sit around all day watching TV, not get out of bed or initiate self-care activities. He/she may need prompting to engage with activities, attend appointments, etc. and may no longer maintain relationships, seek friends or initiate conversation.

- Assist the client, family or service to provide structure to the client's day both to prompt behaviours and to avoid long periods of inactivity.
- Don't rely on the client to take initiative with respect to treatment (they may need a reminder call the day before the appointment as well as a few hours before).
- Assist the client with formulating a written / diagrammatic / pictorial daily schedule
 which includes positive or enjoyable activities as well as self-care behaviours (e.g.
 structure chores around favourite TV program).
- Encourage the use of an alarm, watch or timer to initiate activities.
- Educate client, families and workers that lack of initiative is not due to laziness, but a result of the head injury.

Deficit Area: Lack of cognitive flexibility

Presenting features / symptoms

Client has difficulty with changes to routine, can't always change their train of thought, tends to repeat themselves and has difficulty seeing other's point of view.

- Avoid sudden changes in routine such as changes to appointments, clinicians, or service providers.
- Ignore it when a client repeats themselves or perseverates. Use a distraction technique such as a quick empathic summary and assertive topic change.
- Recognise where you are perseverating as a clinician particularly where you may have chosen an ineffective communication strategy for your client.
- Accept that you may need to explain repeatedly why decisions that affect the client have been made (such as taking annual leave, service limitations, etc.).
- Give as much warning and explanation as possible around change to minimise agitation – if staff are changing, handover periods may assist with transitions.

Deficit area: Impulsivity

Presenting features / symptoms

Client has lost filtering system or control to stop them from "jumping in" (often associated with patterns of frontal lobe damage). He/she makes potentially dangerous mistakes, and keeps making the same mistakes despite feedback that the response is not working. He/she may be incapable of understanding traditional 'relapse prevention plan' frameworks if there is difficulty with inhibiting the habitual behaviour and recognising when to implement a new strategy. The client's impulsivity in social situations can lead to embarrassment and marginalisation. Impulsivity in services can lead to difficulties managing behaviour particuarly when he/she keeps refusing service.

- Be prepared to look at environmental manipulation as a strategy over direct intervention with the client's behaviour. Limiting stressors in an environment (such as scheduling appointments during quiet times in a busy clinic) may be more effective than coaching the client to inhibit reactions and responses to that environment.
- Be prepared to 'take on the role' of foreseeing and preparing for potential areas of
 impulsive behaviour for your client recognising high risk situations and likely
 relapse triggers for the client and formulating responses on their behalf. This may
 involve lengthy coaching / rehearsal of appropriate behaviour prior to a situation.
- Avoid strategies or solutions which may place the client in dangerous situations e.g. entering high risk areas to access services without supports, attending stressful appointments on their own.
- Coach the client to slow down and think before acting. Recognising a situation as high risk can be difficult, and clinicians may need to help clients recognise cues other than traditional craving cognitions (such as physical sensations or behaviours).
- Where clients can use internal or environmental cues to recognise high risk situations, they may benefit from external decision making aids to initiate the new behaviour (such as Stop-Think-Act coaching cards: behavioural option cards with various appropriate strategies carried in the wallet or diary).
- Reinforcement, praise and giving short-term rewards for brief periods of self-control
 can be effective (reinforcement needs to be done soon after positive behaviour to

be effective).

- Clinicians need to be aware of realistic goals in managing behaviour change.
- Psychoeducation may be introduced to alleviate a client's (or family's) anxiety about why they are responding in inappropriate ways at times.

Deficit area: Irritability / anger management

Presenting features / symptoms

Client has lower frustration tolerance and may become agitated if kept waiting or environment is noisy or chaotic (e.g. children making noise). He/she may also become irritable and lose temper easily, particularly when fatigued. Anger may be completely out of proportion to the situation. Client may become unreasonably suspicious and paranoid.

- If possible, remove the client from the situation that provoked the anger.
- Remove the immediate stressor, change the topic, or the environment.
- Identify precipitating factors and try to manage them in future situations.
- When the client has calmed down, explore or suggest better ways of dealing with the situation next time. Clinicians may need to demonstrate and rehearse the behavioural alternatives with explicit links made to the client's own goals.
- Consider a structured behavioural anger management program. Insight based anger programs may not be effective if the primary anger management issue is neuropsychological rather than attitudinally based.

Deficit area: Excessive talking

Presenting features / symptoms

Client is not able to inhibit verbal output – talks too long, or talks over people, has lost knowledge of 'conversational rules' like give and take. Listening skills are poor and the client perseverates when listener has lost interest. He/she may ramble and jump from topic to topic.

- Don't confront the problem too aggressively
- Be empathic but assertive in changing the subject
- Let them know they have already said that by demonstrating understanding rather than labeling the repetition.
- Respond consistently manage your own frustration!
- Distraction or assertive changing of subject may be more beneficial than trying to build insight / understanding about the nature of the communication problem
- Psychoeducation may be indicated to educate clients and significant others about the reasons why communication skills or behaviours have changed.

Deficit area: Socially inappropriate behaviour

Presenting features / symptoms

Client has difficulty judging how to behave in social situations and difficulty changing from one set of social expectations to another. He/she may approach strangers and behave in an inappropriate manner, be over-familiar with strangers, clinicians, therapists and make inappropriate sexual advances.

- See also the strategies for Impulsivity and Irritability / anger management.
- Coach before social events as to appropriate behaviour and rehearse appropriate examples (e.g. context appropriate drink refusal skills).
- Don't reinforce inappropriate behaviour by catastrophising, or trying to cover up the behaviour or its consequences (this may require behavioural coaching of carers / family members).
- Articulate service and personal boundaries clearly and often.
- Try not to 'react' emotionally to inappropriate sexual comments make clear statements and investigate potential reinforcers of the behaviour.
- Consider appropriate staffing arrangements for sexually disinhibited clients.
- Clients may need support finding appropriate outlets for their sexual needs.

Deficit area: Social isolation and dependency

Presenting features / symptoms

Client lacks self-efficacy and is highly self-centered. A loss of role and identity may mean the client is overly reliant on workers or supports. He/she may demonstrate difficulty coping with social situations and lacks initiative. May also have a paradoxical attachment to a 'sick' role where this is instrumental in meeting practical, social or psychological needs.

- Be clear about roles, boundaries and limitations. Reinforce and encourage significant others to clarify their own limitations.
- Try not to let yourself become your client's only "friend", involve other services and reiterate the nature of your relationship with the client.
- Provide opportunities for your client to socialise outside treatment provision contexts.
- Add new people one at a time and try to present a uniform set of values and objectives to minimise potential for splitting.
- Try not to be over protective / too concerned and recognise where your own behaviours or values are sponsoring dependency. Consider the role of codependency and make use of supervision or other reflective forums to understand your helping role.
- Encourage clients to engage with peers via brain injury support groups.
- Consider worker rotation and service rotation for difficult clients or clients with potential for dependence.
- Regardless of issues of ABI, clinicians with highly dependent clients are advised to
 explore pre-morbid predilection to dependency (i.e. personality based), and
 dependency as a result of ABI.

Deficit area: Emotional liability

Presenting features / symptoms

Client has difficulty controlling emotions - may cry too much/too often, laugh inappropriately and experience rapid mood changes. The underlying emotion may not be that strong even though the client's response seems dramatic.

- Ignore the behaviour (unless its antisocial).
- Model calm behaviour and tolerance of emotions.
- Time contacts with the client around periods of emotional resilience (e.g. morning times, or where the client has psychosocial support available).
- Praise appropriately when emotions are kept under control, reinforce times and events for which the client manages their emotions successfully.
- Consider emotional control techniques (de-catastrophisation, breathing and relaxation techniques, exercise, managing environmental stressors, exploring where emotional outbursts are positively reinforced).

Deficit area: Depression / anxiety

Presenting features / symptoms

This is a very common emotional consequence of ABI and usually occurs some time after the injury. Client typically has a lowered mood, appetite and sleep changes, lack of interest in activities, altered self-esteem, negative expectancies and evaluations of self and the world. He/she can represent a paradoxical sign of progress and be more aware of deficits and issues of loss.

- Seek help to deal with depression as with any other client including referral to GP
 / psychiatrist / psychologist / mental health agency.
- Assess for suicidal ideation / means / level of impulsivity / past and recent history of suicidal and parasuicidal behaviour using included tool.
- Encourage diversionary activities and strategies to avoid depressive thoughts.
- Plan and monitor small, easily achieved goals.
- Daily exercise program, meal plans, consider also sleep hygiene (regularising sleep/wake cycles can have a beneficial effect on mood functioning and emotional resilience).
- · Structure activities and activity scheduling.
- Counselling for issues of grief, loss and identity change.

Deficit Area: Self-centeredness

Presenting features / symptoms

Client fails to see other people's point of view (particularly if 'theory of mind' is impaired) and become overly preoccupied with own problems, unaware of the consequences of behaviour on others. He/she may have unrealistic expectations of their counsellor / case manager – they may see the counsellor as existing only to meet *their* needs. They may also become jealous or paranoid if they perceive that their needs are not being met.

Suggested strategies

- Don't let your client come to expect all their demands will be met. Give frequent and clear personal and service limitations to your client, and reiterate to significant others to be clear with their limitations.
- Don't expect clients to respect your rights (or feelings).
- Be consistent with limits and inform other agencies or stakeholders of your limits (to prevent possible splitting).
- Provide support to carers, other workers and families.

Managing cognitive impairments in counselling

Counselling is a commonly sought intervention for assisting ABI clients with low and medium levels of impairment. The following box provides a summary of strategies that may be helpful in tailoring a counselling intervention to better meet the needs of a client with cognitive impairment.

Suggested practical strategies

- Attendance may be enhanced by a reminder system such as calling the client the day before or immediately before the session to confirm their attendance. Keeping times consistent helps.
- Counselling sessions may need to be shorter than usual to account for cognitive fatigue. Consider providing two short sessions in a week rather than one long session – this may allow increased repetition and exposure to new material.
 Breaking up a longer session with a short rest may also be effective.
- Encourage the use of a log book, diary, notebook or folder for keeping a record of counselling sessions, in order to provide more consistency between sessions, and to encourage the client to continue their work 'out of session'. It may include client summaries of sessions: material discussed, goals and homework set, behaviours and strategies to implement for each session. If your client has problems with organisation, it may be helpful for both you and the client to retain a copy of what you did in the session (photocopying the summary at the end of the session).
- Be aware of working memory and attentional limitations use learning aids such as whiteboards in order to decrease the cognitive load (particularly in decisional balance exercises).
- Avoid abstract thinking tasks and use of complex analogy.
- Use simple, appropriate language and present ideas in concrete ways.
- Diagrams are helpful as alternative communication strategies particularly behaviour chains which allow clients to see the progression of events, decisions and outcomes in a clear and unambiguous way (great for looking at relapse cycles).
- A picture may be a more effective way of communicating a complex idea such as a high-risk situation or seemingly irrelevant decision.

Therapeutic approach

For the majority of clients with cognitive impairment or ABI, structured behavioural
or cognitive behavioural approaches work best, with the degree of 'cognitive work'
related to the severity of the deficits, client's ability and motivation to engage
actively in counselling, capacity to engage in homework and goodness of fit with the
counsellor.

- Avoid insight oriented psychotherapy approaches such as gestalt, narrative, and
 psychodynamic therapies, unless the cognitive deficits are minimal or limited to
 'simple' domains (such as uncomplicated memory impairment), and client and
 clinician can demonstrate clear benefits by way of behaviour change and objective
 improvement in psychosocial functioning. Clients with a pre-existing history of
 success with one mode of therapy may not achieve the same successes following
 an ABI.
- Focusing on behavioural strategies to build skills and extend areas of competence
 is generally seen as more effective in drug and alcohol interventions than focussing
 on emotional processing, although recognition of areas of emotional impact and
 assisting with emotional adjustment to ABI is important.
- Clinicians may have to be more directive in style, due to clients difficulties with self-monitoring, time management, attention and concentration. This stylistic change can subsume 'how' a session is organised and guided by the clinician, but does not necessarily mean the clinician is more directive about actual treatment goals.

Providing structure

- Giving clients a plan or structure for the intervention/session may help them to make better use of the available time and lead to empowerment.
- Useful strategies to communicate session format include using a diagram of a clock with shaded sections relating to the different components of a session for example the total length of the session can be set, with sections indicating what will be discussed such as catching up on the client's week, reviewing homework or following up on activity schedules, discussion of new strategies, setting homework or scheduling for the upcoming week or rehearsal of a behaviour, and a wrap-up segment. Such a format demonstrates and models breaking complicated goals up into smaller achievable steps, and encourages regular recording and review of goals and strategies.

Successful referral, shared care and consultation

- There is no single 'best fit' approach to providing AOD treatment for all individuals with cognitive deficits or ABI. Most strategies and techniques employed with 'general' AOD clients are valid and have efficacy with ABI-AOD clients especially the key interventions of providing harm minimisation information, undertaking motivational interviewing and relapse prevention counselling, provision of pharmacotherapy programs, withdrawal programs, and residential rehabilitation.
- Different individuals respond to different treatment approaches and this can change with time, changing patterns of substance use, changing psychosocial circumstances, and motivation.
- Matching individuals to optimal treatments increases treatment effectiveness and efficiency, and clinicians are encouraged to seek guidance and assistance with treatment matching, via referral, shared care and consultancy processes.

Referral options

- ABI/AOD clinician for assistance with assessment, secondary consultation, clientspecific supervision, or handover.
- Neuropsychologist for specialist assessment and recommendations.
- Specialist ABI agency for intensive interventions, case management, brokerage of specialist services, financial or other administration.
- Other AOD services (i.e. residential services)
- Support groups / structured accommodation / community support agencies / recreation and activity groups (adjunct treatment and support services).

Improving referral outcomes

- Target agencies or clinicians with knowledge and interest in working with ABI (or who are amenable to receiving input on working with ABI clients).
- Target services that are accessible and / or include provision of outreach services as part of their assessment or intervention process.
- Target agencies providing assertive, holistic case management.

- For clients with challenging behaviours, consider developing plans for rotation of workers or services with requisite resources and experience.
- Frequent liaison between AOD and ABI services valuable (e.g. shared case plan meetings, joint sessions).
- Look for residential withdrawal services that do not involve lengthy waiting periods (motivation transitory).
- Structured, 24 hour supported accommodation is valuable in the post-withdrawal period, until the new abstinence or controlled use 'habits' are established.

Improving shared care outcomes

- Where possible, agencies or clinicians should seek to share assessment results, interventions and treatment programs.
- Clarification of the roles of all workers involved and commitment to high quality communication in both directions.
- Periodic but regular face-to-face contact between the relevant clinicians. Clinical forums with a 'case review focus' help cement holistic and consistent delivery of services.
- Shared care, and in particular, service transition is facilitated by flexibility in arrangements, taking into consideration both client 'crisis' as well as windows of motivation and opportunities for intervention.
- Ongoing education and training for AOD and ABI clinicians strengthens both sectors' ability to respond.
- Evaluation and review of shared treatment services, resources and interventions allows for ongoing improvement in service provision across sectors.

Improving outcomes of secondary consultation

- Effective secondary consultation involves workers liaising with specialist clinicians
 or consultants for advice, assistance and advocacy where the explicit purpose is to
 equip the referring clinician with the skills, knowledge, strategies or guidance to
 allow best practice delivery of treatment or support interventions.
- Secondary consultation may be more effective where the consultant has some contact with client (usually via an assessment, or sitting in on one or two regular

appointments).

- Where the referring clinician already has a comprehensive assessment, discussions and meetings with the consultant may be best utilised for assistance with treatment planning, assessment of outcomes.
- Effective secondary consultation may comprise 'one-off advice' or ongoing clinical supervision around a particular client.
- It is particularly successful if clinicians seeking consultation have access to assessment information, that issues of cognitive deficit and alcohol or drug use have been explored and the consultation questions are clear.

Integrating forensic treatment into AOD service provision

Responding to forensic issues can be challenging for AOD clinicians who do not have specific experience in offender treatment. Many clinicians and clients are able to formulate responses to offending behaviour as part of substance abuse treatment by exploring the relationship between substance use and offending behaviour and in particular, using relevant goals, and successful strategies from substance use treatment to influence offending behaviour.

For example:

Henry is a 40 year old man who has been referred for counselling for his alcohol use. He was recently assessed as having a mild alcohol related brain injury, and has a pending court case due to his tendency to become violent when intoxicated in pubs and clubs. Henry has stated he doesn't wish to become 'legless in pubs' because he is frightened of more brain damage, and would prefer to 'just get along with people instead of getting agro and fighting them".

The main substance use goal explored by the client and clinician in this example may be to reduce binge drinking in licensed venues, prompted by the shared understanding that this constitutes the highest risk alcohol use behaviour for the client. This alcohol change goal may bear directly on offending behaviour where the link between intoxication, disinhibition and reduced social skills are considered.

In keeping with the client's intrinsic goal to minimise further alcohol related harms, reduce conflict, and incarceration or legal issues, as well as to have a fulfilling social life, the clinician and client could work towards either reduction of alcohol use or avoidance of high risk venues. Going beyond goals which are primarily avoidant or negative is facilitated when clinicians consider also the role of meeting pro-social needs appropriately. In the above example, attention may be paid to the client's social needs which require an alternative outlet if visits to licensed venues are not manageable. Often, 'positive' goals are left out of traditional offender treatment.

Traditional risk-need treatment

Three general principles underpin the traditional 'risk-need' approach to offender treatment (Andrews & Bonta, 1998):

- 1. The risk principle is concerned with matching the level of risk and the actual amount or dose of treatment received.
- The need principle recognises that treatment programs should target primarily criminogenic needs, or the dynamic risk factors associated with recidivism.
 (Dynamic risk factors refer to non-stable characteristics thought to be associated with offending behaviour – as opposed to static risk factors like personality)
- 3. The responsivity principle relates to the ability of a program to actually make sense to the participants for whom it was designed is the content appropriate?

Under the risk-need model the treatment approach is to help offenders understand their offence pattern and cope with situational and psychological factors which place them at risk of offending or relapse. Under this model, the best way to reduce or eliminate recidivism is to identify and reduce or eliminate risk factors for the individual. These risks may be clinical needs or problems (such as alcohol or substance dependence), and should be explicitly targeted in order to reduce offending.

Whilst this is a empirically supported model of treatment, there are areas of weakness – centred around the issue of offender responsivity and the difficulty of motivating offenders using this approach where the goals are largely 'negative' – i.e. identifying problem (negative) behaviours and working out strategies to avoid or decrease those behaviours.

Good Lives model of offender treatment (GLM)

Ward and Mann's Good Lives model offers an alternative approach to offender treatment which deviates from typical relapse prevention models in that it includes consideration of the needs for positive life change as well as the typical avoidant and negative treatment goals.

According to Ward and Mann (2004) an adequate model of rehabilitation for offender treatment should include the following components:

- Conceptual resources to create a bridge between aetiology and treatment
- Specific treatment targets
- Provision of a rationale and theoretical basis for the importance of forming positive attitudes towards offenders and thereby clarify the role of the therapeutic alliance
- A capacity to deal with individual agency and identity
- Be strength based
- Explain the relationship between risk and goods (i.e. balance the need to manage and control relapse risk whilst at the same time meeting the basic human needs of the offender for positive experiences)
- Provide concrete suggestions for the assessment and treatment of offenders

Assessing personal goals and priorities

Ward and Mann recommend a clinical interview approach for assessing priorities and goals as there are no psychometric measure available for this type of individual offender assessment. An example of such an approach is given below:

"Research has shown that there are a number of important activities and experiences which people need if they are to experience good, fulfilling lives. I would like to discuss some of these things with you and find out which you feel you have achieved and those which you may want to work towards. We can talk about how your treatment may help you to focus on those areas you would like to improve. We will need to explore your strengths, and think about how to build on the positives."

Questions for exploring personal goals should explore not only the goal or priority, but also the individual's strategies for realising the goal.

- What does this activity / goal mean to you?
- How is this important? Has this changed in importance over time?
- How have you tried to achieve this in the past? Which strategies have worked the best? Which have worked least well?
- What do you think has prevented you from achieving this goal so far?
- Where would you like to be in respect to this goal in one year, five years, ten years?

Additional questions for the *clinician* to consider when assisting the client in choosing and following up treatment goals may include:

- Is there a restricted scope of goals i.e. is the client focussing on some goals to the detriment of others?
- Are some goals pursued with inappropriate means strategies which are counter productive?
- Is there inherent conflict amongst goals?
- Does the client have the requisite capacity and resources to meet their goals? Are the goals realistic?

Collaborative assessment

Where motivations between the assessor and the client are divergent (i.e. the assessor wants to minimise the risk of relapse to harmful substance use or offending behaviour, and the client is focussed on minimising their self-reported use), collaborative assessment is vital to ensure good communication, accurate recording of data, and strong therapeutic alliance. Mann and Shingler (2001) have reported recommendations for re-conceptualising 'risk' assessment as 'need' assessment in order to increase the collaborative aspect of assessing dangerous or risky behaviours. These authors recommend:

- Genuine commitment from the clinician to work in a transparent and respectful fashion, emphasising that the client's best interests should be served by the assessment process.
- Assessments presented for collaborative investigation should include not only issues of relapse risk but also positive life goals, strengths and achievements.

Approach goals

Work with AOD clients has demonstrated that individuals working toward 'approach goals' are less likely to relapse than clients working towards avoidant goals (Cox, Klinger & Blount, 1991). Approach goals allow the client and clinician to frame change goals in terms of positive outcomes or improvements in quality of life, rather than negative outcomes to be avoided. Including approach goals is likely to have beneficial effects on engagement and therapeutic relationships as well.

For clients with cognitive deficits or ABI, the salience of functional, emotional and psychosocial issues is likely to bias an assessor or treating clinician towards a 'problem'

identification' framework, particularly where legal or forensic issues offer additional salience to a client's problems. Using the GLM to understand issues of assessment, rapport building and risk-need management should assist clinicians to take a broader, holistic view to meeting client's needs, resulting in strengthened engagement, greater retention in treatment, more positive treatment outcomes, and reduced relapse and reoffence rates.

Case examples: Vignettes of clients with acquired brain injury and alcohol and drug misuse

(Note these vignettes are for educational purposes only, any similarities to real individuals is coincidental).

Vignette 1: Medium level of risk / need

Barry is referred to alcohol and drug treatment by his sister, who requests information about and treatment for inhalant use following increased concerns about Barry's frequent intoxication and deteriorating self-care. Barry is brought to his initial appointment by his sister, and presented as a 34-year-old with both an identified ABI from several serious motor vehicle accidents, premorbid borderline intelligence, and longstanding history of polysubstance use. Barry's psychosocial situation has been deteriorating as his use of substances, particularly chrome-based paint, has escalated in the preceding six months.

Interview and assessment

Barry's psychosocial history is elicited over three appointments. Barry fatigues quickly in remembering details of his life, particularly past substance use, and session length is limited to 30 minutes, meaning that a comprehensive assessment, as well as preparation of an individual treatment plan takes longer than usual to achieve. The need for a 'really good picture' of substance use patterns is explained carefully to Barry with comprehension checks so that motivation to provide accurate information is sustained. Collateral information is also sought from Barry's sister (with Barry's permission), who is able to provide details about historical substance use as well as Barry's early learning environment, general functioning and the events surrounding his acquired brain injury.

Social / Family history

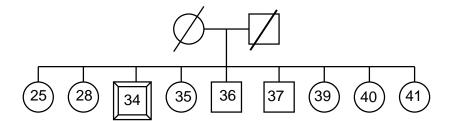
Barry is the third youngest of 9 siblings, who he describes as 'doing much better than me" – all engaged in full time employment, with no significant histories of alcohol or other drug concerns. Similarly there is no history of psychiatric issues within the family. Barry has significant contact with several of his sisters but generally limited contact with the family as a unit (he has failed to attend several large family gatherings due to feelings of shame). Barry's sisters frequently provide financial assistance and support around issues of accommodation / advocacy with the department of housing, shopping and social outings. Barry's sisters indicate to the counsellor that they are unable/unwilling to continue to

provide the same level of involvement and assistance as they are feeling ineffective and frustrated with Barry's deterioration, in particular with his ongoing substance use.

Barry's social contacts are relatively limited. He spends time with other substance users, predominantly those in his local area who share similar patterns of substance use (daily alcohol and cannabis use and intermittent heroin and amphetamine use). It appears that Barry's compromised intellectual functioning and poor assertiveness skills means he is often manipulated by others to fund substance use or to provide the space for use to occur. Barry expresses considerable ambivalence in early sessions about these relationships, stating he likes the contact and company and the generally non-judgemental nature of other users, but he often feels "ripped off" by them. Barry purchases beer or cannabis more often than not, and feels unable to enforce boundaries with his 'friends' – particularly when he has a number of people in his flat and he wants to go to bed. The friends won't leave and continue to use his alcohol and cannabis.

Barry has in the past had significant social relationships with non-substance-users, in particular several friends from high school whom Barry would visit, or go on fishing trips with. He would use small amounts of alcohol with these friends. Barry indicates that he feels these friends have 'moved on' and wouldn't want anything to do with him now that they have children. Further questioning reveals Barry is also demotivated to contact these friends as he is ashamed of his escalating chrome paint use.

Figure 6: Barry's family history



Medical history

Barry's sisters are able to provide some details regarding Barry's medical history, although medical records, discharge summaries and neuropsychological assessment reports were not available. Barry was a very low birth weight baby, and was slow to reach developmental milestones (i.e. walking, talking, reading).

At age 15 Barry was involved in a motor vehicle accident as a passenger. This accident involved an open head injury (fractured skull), and Barry spent 10 days in a coma with a significant period of post-traumatic amnesia. Barry's sisters are able to describe diffuse cerebral damage with no focal lesions and no gross loss of functioning (i.e. Barry did not lose specific functions such as speech). Problems arising from this accident included (according to Barry) – difficulties with balance, slowness in completing tasks, lack of general motivation, impairment to memory functioning and epilepsy.

Barry's sisters include reports of behavioural dysregulation (increased physical aggression and impulsivity), which is consistent with frontal lobe damage and executive functioning deficits. Barry's recollection of this period is that he got into several fights. He also traces his paranoia about being assaulted to this time.

At age 18 Barry was involved in another motor vehicle accident, this time as a pedestrian being struck by a moving car at an intersection. The details of this accident are difficult to elicit from either family members or from Barry himself. He recalls that this accident involved a blow to the head, although it did not involve a skull fracture, and his period of lost consciousness was of the order of 2-4 hours, with some limited memory loss for the period of the accident itself. Barry admits that he was intoxicated at the time of this accident (alcohol). It is possible that both this intoxication and impaired decision-making and impulsivity acquired from the previous accident may have contributed to unsafe behaviour on Barry's part.

Barry at present is Hepatitis C positive and has stomach ulcers which "flare up", causing pain at times of increased stress.

Alcohol and drug use history

Barry's substance use commenced at age 14, when he began using alcohol on a regular (weekly) basis after leaving school. His use of alcohol at this time would be considered 'binge' use – drinking 10 or more standard drinks per session. He commenced smoking cigarettes at age 16 when he started an apprenticeship working in a mower repair shop.

Barry's use of cannabis began at age 18 during a period of unemployment and social withdrawal following his second motor vehicle accident. Barry describes spending much of

his time in his room at home smoking throughout the day and evening. He also reports increasing paranoia and delusional thinking (beliefs that people who looked at him in public were ridiculing him and were going to assault him). Barry relates this withdrawal from friends and family and increased use of drugs to highly traumatic experiences of workplace bullying which occurred during his apprenticeship, and to his difficulty coping with the effects of his acquired brain injury.

At the time of assessment, Barry is using chrome paint, cannabis and nicotine daily, with frequent use of alcohol, and intermittent use of amphetamines and heroin. His substance use history is presented in the following table (from AOD assessment module).

Figure 7: Barry's alcohol and drug history

	Inhalants	Cannabis	Alcohol	Amphetamines	Heroin	Nicotine
First used	14	16	14	18	19	13
Age regular	16	17	14	30	30	14
use						
Route of	Inhales	Smokes	Drinks	Intravenous	Intravenous	Smokes
administration		bongs	mainly			
			beer			
Average daily	1 large	\$20 gram	Drinks	\$50-200 1-2	\$50-200 1-	1 pack
use	can per	per day	until	times per	2 times per	day – 25
	day		intoxicated	fortnight	fortnight	cigarettes
						12-16mg
Days used in	7/7	7/7	4/7	2/7	2/7	7 / 7
past week						
Days used in	25 / 28	28 / 28	20 / 28	5 / 28	6 / 28	28 / 28
past month						
When last	Day	Day of	2 days	5 days before	4 days	1 hour
used	before	interview	before	interview	before	before
	interview		interview		interview	interview
How long	3 years	5 + years	5 + years	5 + years	5 + years	25 years
using like this						

Diagnostics

Barry is assessed as meeting the criteria for dependence for inhalant and cannabis use. Criteria met are **marked in bold** to follow the diagnostic process.

DSM-IV diagnostic criteria

Inhalant abuse: A destructive pattern of inhalant use, leading to significant social, occupational, or medical impairment.

Must have three (or more) of the following, occurring when inhalant use is at its worst:

- Inhalant tolerance: Either need for markedly increased amounts of inhalant to achieve intoxication, or markedly diminished effect with continued use of the same amount of inhalant.
- Greater use of inhalant than intended: Inhalant is often taken in larger amounts or over a longer period than is intended.
- Unsuccessful efforts to cut down:
 Persistent desire or unsuccessful attempts to cut down or control inhalant use. A great deal of time is spent using inhalant or recovering from hangovers.
- Reduction in social, occupational or recreational activities: Important activities are given up or reduced because of inhalant use.
- Continued inhalant use despite knowing it causes significant problems: Use continues despite

DSM-IV diagnostic criteria

Cannabis abuse: A destructive pattern of cannabis use, leading to significant social, occupational, or medical impairment.

Must have three (or more) of the following, occurring when cannabis use is at its worst:

- Cannabis tolerance: Either need for markedly increased amounts of cannabis to achieve intoxication, or markedly diminished effect with continued use of the same amount of cannabis.
- Greater use of cannabis than intended: Cannabis is often taken in larger amounts or over a longer period than is intended.
- Unsuccessful efforts to cut down:
 Persistent desire or unsuccessful attempts to cut down or control cannabis use. A great deal of time is spent in using cannabis or recovering from hangovers.
- Reduction in social, occupational or recreational activities: Important activities are given up or reduced because of cannabis use.
- Continued using cannabis despite knowing it causes significant problems: Use continues despite

- knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or worsened by inhalant.
- Associated features: Learning
 problems, dysarthria or involuntary
 movement, hypoactivity /
 hyperactivity, psychosis, euphoric
 mood, depressed mood, somatic or
 sexual dysfunction, addiction,
 sexually deviant behaviour, dramatic,
 erratic or antisocial personality.
- knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or worsened by inhalant.
- Associated features: Learning problems, psychosis, euphoric mood, depressed mood, somatic or sexual dysfunction, addiction, odd, eccentric or suspicious personality.

Barry is also assessed as meeting criteria for heroin and amphetamine abuse. See the diagnostic criteria below for substance abuse.

Criteria for substance abuse

- A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by one (or more) of the following, occurring within a 12-month period:
 - a) Recurrent substance use resulting in a failure to fulfil major role obligations at work, school, or home (e.g. repeated absences or poor work performance related to substance use, substance-related absences, suspensions, or expulsions from school, neglect of children or household).
 - b) Recurrent substance use in situations in which it is physically hazardous (e.g. driving an automobile or operating a machine when impaired by substance use).
 - c) Recurrent substance-related legal problems (e.g. arrests for substance-related disorderly conduct).
 - d) Continued substance use despite having persistent or recurrent social

or interpersonal problems caused or exacerbated by the effects of the substance (e.g. arguments with spouse about consequences of intoxication, physical fights).

2. The symptoms have never met the criteria for substance dependence for this class of substance.

Alcohol and drug treatment history

Barry reports nil previous alcohol and drug treatment prior to his involvement with this service. Barry reports seeing a psychiatrist at age 16 or 17 for what he describes as a 'complex'. Barry understands that this treatment helped him with his anxiety about ridicule and assault, and helped him counter punitive thoughts.

Barry has made previous attempts to limit/cease substance use, and has managed several periods of abstinence, ranging from 1-4 months, from all illicit drugs, although at that time Barry reports his use of alcohol increases significantly (15+ standard drinks per day with no alcohol free days in a usual week).

Engagement in treatment and outcomes

Barry is assessed as being best suited to follow-up in the medium risk/need group. Whist his cognitive status and substance use involves complex problems, his high levels of motivation for treatment and capacity to tolerate one-on-one counselling increases the responsivity of treatment interventions.

Barry is able to formulate several goals for the current episode of alcohol and drug counselling, namely abstinence from inhalant use, heroin use and amphetamine use, and controlled use of alcohol and cannabis. His specific goal with relation to alcohol use is to drink in moderation (3-4 standard drinks per day with 3 alcohol free days).

Following assessment, Barry is provided with outpatient counselling and after an initial period of 'wary' engagement with the service, Barry is able to make good use of counselling to moderate his substance use. It is necessary to concentrate much attention

on motivational enhancement to maintain Barry's attendance and adherence to treatment goals.

Barry is able to reach his goals and maintain change over approximately 6 months of counselling contact. Barry's treatment also involves some aspects of cognitive behavioural therapy as he begins to report lowered mood and difficulties managing aggressive moods and self-punitive cognitions. With increased self-efficacy and trust, Barry is able to work towards several approach goals – including more drug-free outlets for social interaction and re-engaging with his family.

Vignette 2: High level of risk / need

Rachel, a 40-year-old, is referred to the ABI-AOD clinician by her case manager from an AOD service for re-assessment, following recent deterioration in her physical condition. Rachel has had several unsuccessful attempts at detoxification and has been consuming methylated spirits and cask wine daily in the context of isolation and possible suicidality.

The assessment is requested to provide a comprehensive picture of Rachel's alcohol and drug use, her mental state, the relationship between her substance use and suicidality, and to make recommendations concerning appropriate alcohol and drug interventions. An ongoing secondary consultation role is brokered between the ABI-AOD consultant and the AOD case manager.

Interview and assessment

A long assessment process involves interviews carried out at a number of locations: at Rachel's residential detox service, at a supported accommodation unit, and at the alcohol and drug agency. Follow-up discussion with the AOD case manager is required as eliciting a full history from Rachel is not possible within the assessment sessions due to her cognitive deficits, intoxication and fatigue effects. Multiple assessments in different contexts are thought to give a more accurate picture of the range of Rachel's functioning.

These assessment interviews include Rachel's AOD case manager, and workers from her supported accommodation and detox agencies. Consideration is also given to discharge summaries from detox services and the results of neuropsychological assessments.

On interview, Rachel is difficult to engage due to some paranoid thoughts concerning intrusion into her affairs by 'the government' (related to previous assessments for guardianship orders). She also complains about 'having done too many assessments' whilst in detox.

The effects of intoxication and withdrawal also impacted on the assessment, notably Rachel's agitated mood, psychomotor agitation (pacing, difficulty in remaining in the assessment room), aggressive verbal outbursts, fatigue and cognitive disorganisation, such as confusion, confabulation (making up aspects of history unintentionally), and derailment (losing track of what is being discussed).

Rachel is initially seen at the detox agency, where she has spent the preceding 9 days withdrawing from alcohol. Whilst Rachel reports feeling 'scattered and confused' this location provides an opportunity to make an assessment while Rachel is not intoxicated and represents her highest functioning during assessment. Rachel is able to provide a consistent alcohol and drug history, treatment history and an assessment of motivation is also conducted (using a decisional balance exercise).

Subsequent assessments track a decline in functioning as alcohol use recommences and escalates. Where Rachel is intoxicated, the interviewer does not continue a formal assessment, other than observations as to the level of intoxication (via use of portable blood alcohol meter), patterns of use and the effects of alcohol use on motivation to change. These situations also provide opportunities to liaise with other workers involved in her care (supported accommodation staff) and verify Rachel's self-reported levels of consumption and associated activities (frequency of alcohol purchase, types of alcohol found in Rachel's room, etc).

Social and family history

A detailed picture of Rachel's family history, early learning environment and level of premorbid functioning is compromised by several factors during assessment. Rachel has limited recall of the events of her childhood and is not willing to allow the assessor to contact family members for collateral interviews. It is judged that it may be distressing to discuss family history due to indications of physical and/or sexual abuse in previous assessment reports. It is decided that the need to limit the risk of re-traumatisation and maintain a positive engagement outweighs the need for first-hand assessment of this area.

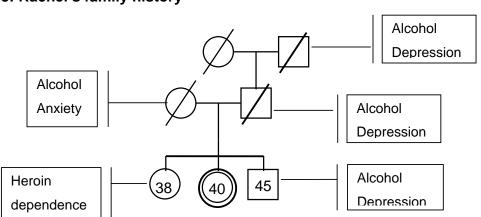


Figure 8: Rachel's family history

Rachel's family history is notable for the incidence of alcohol and drug and mental health issues. Rachel was made a ward of the state at age 12 following the death of her mother (alcohol-related, few details available), and lost contact with her father. Her paternal grandfather suffered from depression and was a heavy alcohol user, and died from alcohol use (unknown pathology, possibly liver failure). Rachel's parents also both used alcohol heavily, and both eventually died as a result of their use (possibly liver failure). Rachel has limited contact with her older brother, who is also an alcohol user and who suffers from depression, but has not seen her younger sister for several years due to ongoing conflict.

Educational / employment history

Rachel attended school until early Year 9 (age 14), when she was expelled for truancy and fights with teachers and peers (which Rachel relates to increasing alcohol and drug use). Rachel reports she had enjoyed schoolwork and had done comparatively well, finishing in the top quarter of her class in Year 8. Following school she found part-time employment until age 18 as a factory hand, but increasing substance use made maintaining work difficult. Rachel has received unemployment benefits and disability pensions since this time, although at times she has involved herself in voluntary work.

Medical history

Rachel's case manager is able to provide a neuropsychological report dating from 18 months prior to the index assessment. As this assessment was conducted over several weeks where Rachel had been using alcohol moderately for several months, it represents a high water mark in terms of her cognitive functioning and level of insight. This report highlights several potential vectors for acquired brain injury. Several incidents involving closed head injuries are identified. In 1990 Rachel was struck by a moving car crossing an intersection whilst intoxicated, although this accident did not involve any significant period of loss of consciousness, and medical treatment was not sought. Rachel also reported receiving several blows to the head during assaults in the period from 1988 to the present, although similarly these injuries did not involve loss of consciousness.

It appears likely that alcohol related brain damage is the most probable cause for Rachel's ABI. The assessment identifies moderate and significant impairment of memory and attention, and moderate and significant impairment of executive functioning. The assessment notes mild impairment to new learning skills. Strengths identified during the

assessment include 'reasonably intact' domains such as regulation of behaviour, flexibility of thinking and problem solving skills. More significantly, the assessment identifies a clear relationship between levels of recent alcohol use, anxiety and performance on the neuropsychological tests – meaning that whilst memory, attentional and executive functions are impaired, improvements are possible with abstinence and lowered levels of stress / anxiety.

Alcohol and drug use history

Rachel describes a pattern of heavy alcohol use over a period of 19 years which classifies her consumption as 'at risk' given the amounts of use and generally poor self-care (poor nutrition, lack of attention to acute and chronic health complaints, pattern of risky relationships, alcohol precipitating polysubstance use and sensation seeking behaviour such as driving whilst intoxicated). It is likely that the combination of impaired decision-making due to her ABI and acute intoxication effects contribute to this pattern of use.

Rachel's alcohol use has involved the use of spirits, beer and wine, although at the time of assessment, her preferred drink was cask wine and/or methylated spirits. Rachel's pattern of consumption involves constant alcohol use throughout the day. Rachel describes taking some pains to conceal her use when out of the house, including decanting wine into 'raspberry lemonade' bottles to allow consumption on public transport, and in doctor's offices. Rachel funds her consumption via her pension, and through 'making friends' with people in pubs in order for them to buy her alcohol. Rachel reports this behaviour has frequently placed her at risk of physical and sexual assault.

Rachel reports using cannabis intermittently with friends, rarely purchasing it herself. Similarly Rachel's use of amphetamines and heroin appears to be context-dependent, associated with contact with a group of polysubstance users who Rachel has intermittent (fortnightly) contact with. Rachel's use of benzodiazepines was initially considered to involve regular prescribing of small amounts for treatment of anxiety and sleep disturbance, although discussions with staff at supported accommodation services indicated that bottles of Valium and Serepax have been discovered in Rachel's room from a variety of prescribers – indicating doctor shopping. Rachel is unwilling to discuss her benzodiazepine use initially. Although with some psychoeducation as to the role of benzodiazepine class drugs on exacerbating cognitive deficits, and gentle probing around

the relationship between binge use and critical incidents (such as assaults, evictions and blackouts), Rachel offers more information and commits to trying to limit use.

Rachel's substance use history is presented in the following table (from AOD assessment module).

Figure 9: Rachel's alcohol and drug history

	Alcohol	Cannabis	Benzo-	Stimulants	Heroin	Nicotine
			diazepines			
First used	12	14	12	18	18	14
Age regular	12	14	14	20	20	14
use						
Route of	Drinks	Smokes	Ingests,	Intravenous	Intravenous	Smokes
administration		bongs	occassionally			
			injects			
Average daily	2-4 litres	Less than	2-5 x 5mg	\$60 1 time	\$60 1-2	1 pack
use	of cask wine or	1 gram	diazepam	per month	times per	day – 25
	0.5-1 litre	per	daily, binges		fortnight	cigarettes
	of methylated	fortnight	up to			12mg
	spirits		20 x 5mg			
			diazepam			
Days used in	0 / 7 (in	0/7	4/7	0/7	2/7	7/7
past week	detox)					
Days used in	19 / 28	10 / 28	20 / 28	1 / 28	6 / 28	28 / 28
past month						
When last	9 days	10 days	2 days	2 weeks	4 days	Prior to
used	before	before	before	before	before	and
	interview	interview	interview	interview	interview	during
						interview
How long	18 years	5 + years	5-10 years	10 years	10 years	25 years
using like this						

Alcohol and drug treatment history

Rachel describes significant involvement with alcohol and drug treatment prior to the index treatment. Rachel has undergone upwards of 30 inpatient detox programs over a period of 15 years. She relates that most of these detoxification attempts have been instigated by 'social workers poking around in my business', and have been largely unsuccessful, although on two occasions she maintained abstinence from alcohol for periods of 6-8 months. Rachel reports seeing 'a counsellor' for her alcohol use on these occasions, although she is generally critical of the efficacy of counselling.

Rachel describes numerous 'home based detox' episodes, although it appears that this refers to attempts to stop alcohol use with benzodiazepines without much structure (i.e. no daily dispensing of medication, no ongoing contact with treating doctor following prescription).

Rachel is currently well engaged with her case manager who has considerable experience as an alcohol and drug worker, but who requires support to tailor her interventions to Rachel's needs.

Motivational assessment

Rachel's motivation to address her substance use varies markedly depending on her alcohol and drug use status. When abstinent or drinking at a moderate level, Rachel demonstrates limited insight into the hazardous effects of alcohol and benzodiazepine abuse and the risks associated with spasmodic intravenous drug use. The decisional balance exercise completed during the initial assessment interview highlights an understanding of the chronic health and psychosocial difficulties associated with ongoing alcohol use, and the disinhibitive effects of binge benzodiazepine use.

Rachel is able to identify numerous additional negatives to add to this balance during subsequent alcohol-free sessions with her case manager. Rachel identifies the positives of her use as relating to alleviating chronic feelings of low-self esteem, loneliness and hopelessness. Alcohol and substance use also provides a means of engaging with non-judgemental peers, although Rachel's impaired decision-making often means she feels 'ripped off' in situations where she uses or drinks with others.

Engagement in treatment and outcomes

Rachel is believed to fall into the higher risk-need group (level 3), and the majority of treatment interventions are planned to be delivered outside of a traditional appointment-based service environment. Supportive counselling and relapse prevention pharmacotherapy are identified as interventions with potential efficacy in increasing/sustaining Rachel's motivation to limit her alcohol intake to safe limits, as well as provide additional support around possible further deterioration of mental state, and monitor requirements for inpatient admissions.

Rachel's accommodation in a supported facility allows the ABI/AOD clinician and her AOD case manager to work with residential staff to monitor levels of alcohol and other drug use, mental state and general health regularly. The AOD case manager is able to continue supportive counselling and facilitate the pharmacotherapy with occasional support from the ABI AOD consultant.

Pharmacotherapy interventions

Two alcohol pharmacotherapy options are considered appropriate for Rachel - Naltrexone and Acamprosate. Both of these medications have proven efficacy in assisting people maintain abstinence or control alcohol intake, by decreasing the intensity of cravings to drink and also diminishing the pleasurable effects of alcohol use. The supported residential facility makes ensuring compliance with these medications easier than if Rachel had been living in a more unstructured environment.

Doctor shopping behavioural intervention

Rachel's usual GP is approached and is willing to continue to prescribe benzodiazepines to Rachel with a treatment plan that includes a dose increase initially (to allow for the total 'doctor shopped' amounts, then a slow reduction over around 6 months. All doses of benzodiazepines will be dispensed daily from a local pharmacy (along with the chosen pharmacotherapy for alcohol use). A release of information is sought from Rachel to apply to the Health Insurance Commission for information relating to benzodiazepine prescription. The health insurance commission process does not guarantee that Rachel will not visit another prescriber to obtain benzodiazepines, but it does allow these visits to be retrospectively identified. This process is explained clearly to Rachel, with clearly identified consequences associated with continued doctor shopping including cessation of prescribing.

Supportive treatment framework

Rachel's excellent level of engagement with her AOD case manager is of considerable benefit in ensuring reasonable compliance with the agreed treatment plan. However, boundary management strategies are also necessary, particularly around home visits when Rachel is intoxicated, or when spurious or unnecessary visits are requested. It is identified that interactions with Rachel when she is at all intoxicated are counter productive for any 'work' around alcohol or drug use, and also increases the levels of shame and guilt in subsequent engagements.

Counselling strategies deemed to be most effective are highly structured, containing few basic elements per session, and involve short durations. Of particular need is repetition and reinforcement of the decisional balance exercise and integration of feedback around improving functioning into reasons to abstain or limit alcohol and drug use. Where relapse prevention strategies are used, they are kept conceptually simple, and involve a structured and simple analysis of high risk situations and moods and alternative behavioural responses to these situations. Pictorial records of these high-risk situations are made by Rachel with support from her case manager.

Counselling focusses on avoidance strategies for particularly high risk situations and relationships, some assertiveness skills training (such as role-playing drink and drug refusal skills), and general empathic and supportive work around improving general relationship skills (approach goal). Careful attention is paid during counselling to maintaining a practical, behaviour-focused and motivational approach to interactions with Rachel.

References

American Psychiatric Association 2000. (DSM-IV-TR) Diagnostic and statistical manual of mental disorders, 4th edition, text revision. Washington, DC: American Psychiatric Press, Inc.

Anglin, M. D., Hser, Y. & Chou, C. (1993). Reliability and Validity of Retrospective Behavioural Self-Report by Narcotic Addicts. *Evaluation Review*, *17(1)*, 91-108.

Australian Institute of Health and Welfare (1999). The Definition, incidence and prevalence of acquired brain injury in Australia. Australian Institute of Health and Welfare, Canberra.

Australian Institute of Health and Welfare (2005) Australia's Welfare 2005. Australian Institute of Health and Welfare, Canberra.

Australian Institute of Health and Welfare. (2005). 2004 National Drug Strategy Household Survey: First Results. AIHW Cat No. PHE 57. Canberra AIHW (Drug Statistics Series No. 13).

Baker, E.L. (1994) A review of recent research on health effects of human occupational exposure to organic solvents. J Occup Med; 36: 1079-92.

Barker, M.J., Jackson, M., Greenwood, K.M., Crowe, S. (2003). Cognitive Effects of Benzodiazepine Use: A Review. Australian Psychologist, Vol 38; pp 202-213.

Bellack, A.S. (1992). Cognitive rehabilitation for schizophrenia: Is it possible, Is it necessary? Schizophrenia Bulletin, 18: 43-50.

Berg, E.A. (1948). A simple objective test for measuring flexibility in thinking. Journal of General Psychology, 39: 15-22.

Block, R.I., & Ghoneim, M.M. (1993). Effects of chronic marijuana use on human cognition. Psychopharmacology; 110: 219-228.

Bogner, J.A., Corrigan, J.D., Spafford, D.E. & Lamb-Hart, G.L. (1997). Integrating

substance abuse treatment and vocational rehabilitation after traumatic brain injury. Journal of Head Trauma Rehabilitation, 12(5); 57-71.

Brandt, L., Butters, N., Ryan, C. & Bayog, R. (1983) Cognitive loss and recovery in long-term alcohol abusers. Archives of General Psychiatry, 40(4): 435-442.

Brennan, P.A., Mednick, S.A. & Hodgins, S. (2000). Major mental disorders and criminal violence in a Danish birth cohort. Archives of General Psychiatry, 57(5): 494-500.

Bush, K., Kivlahan, D.R., McDonell, M.B., Fihn, S.D., Bradley, K.A. (1998). The AUDIT Alcohol Consumption Questions (AUDIT-C) An Effective Brief Screening Test for Problem Drinking. *Arch Intern Med.* 1998;158:1789-1795.

Cermak, L.S., Butters, N., & Moreines, J. (1974). Some analyses of the verbal encoding deficit of alcoholic Korsakoff patients. Brain and Language 1: 141-150

Corrigan, J. D. (1995). Substance abuse as a mediating factor in outcome from traumatic brain injury. Archives of Physical Medicine and Rehabilitation, 76 (4), 302-309.

Corrigan, J. D., Rust, E., & Lamb-Hart, G. L. (1995). The nature and extent of substance abuse problems among persons with traumatic brain injuries. Journal of Head Trauma Rehabilitation, 10 (3), 29-45

Corrigan, J.D. (1995). Substance abuse as a mediating factor in outcome from traumatic brain injury. Archives of Physical Medicine and Rehabilitation, 76(4); 302-309.

Corrigan, J.D., Bogner, J.A., Mysiw, W.J., Clinchot, DE., & Fugate, L. (2001). Life satisfaction after traumatic brain injury. Journal of Head Trauma Rehabilitation 16(6). 543-555.

Corrigan, J.D., Rust, E., & Lamb-Hart, G.L. (1995). The nature and extent of substance abuse problems in persons with traumatic brain injury. Journal of Head Trauma Rehabilitation; 10(3): 29-46.

Corrigan, J.D., Smith-Knapp, K. & Granger, C.V. (1998) Outcomes in the first 5 years after

traumatic brain injury. Archives of Physical Medicine & Rehabilitation, 79(3): 298-305.

Cushman, P., Jackobson, G., Barboriak, J.J. and Anderson, A.J. (1984). Biochemical markers for alcoholism: Sensitivity problems. *Alcsm clin. Exp. Res.*, *8*, 253-257.

Darke, S., Hall, W., Wodak, A., Heather, N & Ward, J. (1992). Development and validation of a multi-dimensional instrument for assessing outcome of treatment among opiate users; the Opiate Treatment Index. *British Journal of Addiction*, *87*, 733-742.

Darke, S., Sims, J., McDonald, S., & Wickes, W. (2000). Cognitive impairment among methadone patients. Addiction; May 2000, 95 (5), 687-695.

Davalos, D.B., Green, M. & Rial, D. (2002). Enhancement of executive function skills: An additional tier in the treatment of schizophrenia. Community Mental Health Journal, Vol 38(5). 403-412.

Del Boca, F. K., & Noll, J.A. (2000). Truth or consequences: The validity of self-report data in health services research on addictions. *Addiction*, *95*(*suppl.* 3), s347-s360.

Delin, C.R. & Lee, T.H. (1992) Drinking and the brain: Current evidence. Alcohol and Alcoholism 27(2): 117-126.

Delmonico, R.L., Hanley-Peterson, P. & Engelander, J. (1998). Group psychotherapy for persons with traumatic brain injury: Management of frustration and substance abuse. Journal of Head Trauma Rehabilitation. 13(6), 10-22.

Desmond, D.W., Tatemichi, T.K., Paik, M., et al. (1993). Risk factors for cerebrovascular disease as correlates of cognitive function in a stroke-free cohort. Arch Neurol; 50: 162-166.

Drubach, D.A., Kelly, M.P., Winslow, M.M. & Flynn, J.P. (1993). Substance abuse as a factor in the causality, severity and recurrence rate of traumatic brain injury. Maryland Medical Journal, 42(10); 989-993.

Ehrman, R. N., & Robbins, S.J. (1994). Reliability and Validity of 6-month timeline reports

of cocaine and heroin use in a methadone population. *Journal of Consulting and Clinical Psychology*, 62(4), 843-850.

Elias, P.K., Elias, M.F., D'Agnostino, R.B., et al. (1999) Alcohol Consumption and Cognitive Performance in the Framingham Heart Study. Am J Epidemiol; 150: 580-589.

Ellis, R.J. (1990). Dichotic asymmetries in aging and alcoholic subjects. Alcoholism: Clinical and Experimental Research; 14(6): 863-871.

Evert, D.L., Oscar-Berman, M. (1995) Alcohol Related Cognitive Impairments. Alcohol Health and Research World, Vol 19, No. 2. 89-96.

Ewing, J.A. (1984). Detecting Alcoholism. The CAGE questionnaire. JAMA, Vol 252(14).

Fisher, J., & Harford, T. (1983). Contextual correlates of the duration of drinking: Confirmation of ethnographic findings with a self-report instrument. *Addictive Behaviors, 8*, 193-200.

Forsberg, L.K. & Goldman, M.S. (1985) Experience-dependent recovery of visuospatial functioning in older alcoholic persons. Journal of Abnormal Psychology. 94(4): 519-529.

Goldberg, E. & Bougakov, D. (2000). Novel approaches to the diagnosis and treatment of frontal lobe dysfunction. In Christensen, A. & Uzzell, B et al. (Eds). International handbook of Neuropsychological Rehabilitation. Critical Issues in Neuropsychology. Pp 93-112). New York: Kluwer Academic.

Goldman, M.S. (1990). Experience-dependent neuropsychological recovery and the treatment of chronic alcoholism. Neuropsychology Review 1: 75-101.

Goldman, M.S. 1995. Recovery of Cognitive Function in Alcoholics: The Relationship to Treatment. Alcohol Health and Research World, Vol. 19(2).

Goldman, M.S. 1995. Recovery of Cognitive Function in Alcoholics: The Relationship to Treatment. Alcohol Health and Research World, Vol. 19(2).

Gouzoulis-Mayfrank, E., Daumann, J., Tuchtenhagen, F., Pelz, S., Becker, S., Kunert, H., Fimm, B., Sass" H. (2000) Impaired cognitive performance in drug free users of recreational ecstasy (MDMA) *J Neurol Neurosurg Psychiatry* 2000; 68: 719-725.

Grafman, J. Schwab, K., Warden, D. Pridgen, A., Brown, H.,R. & Salazar, A.M. (1996). Frontal Lobe injuries and aggression. Neurology, 46: 1231-1238.

Grant, D.A. & Berg, E.A. (1948). A behavioural analysis of degree of reinforcement and ease of shifting to new responses in a Weigl-type card sorting problem. Journal of Experimental Psychology, 34: 404-411.

Hammersley, R. (1994). A digest of memory phenomena for addiction research. *Addiction*, 89(3), 283-293.

Higgins, S.T., Stitzer, M.L., Bigelow, G.E., Liebson, I.A (1986). Contingent methadone delivery: effects on illicit opiate use. Drug and Alcohol Dependence 17: 311-322.

Higgins, S.T., Wong, C.J., Badger, G.J. Ogden, D.E., Dantrona, R.L. (2000) Contingent reinforcement increases cocaine abstinence during outpatient treatment and 1 year follow-up. Journal of Consulting and Clinical Psychology. 68: 64-72.

Holland, C.A., & Rabbitt, P. (1991). The course and causes of cognitive challenge with advancing age. Rev Clinic Gerontol; 1: 81-96.

J. Prochaska and C. DiClemente. *Stages of Change in the Modification of Problem Behavior*. In M. Hersen, R. Eisler, and P. Miller, editors, Progress in Behavior Modification, volume 28. Sycamore, IL: Sycamore Publishing Company, 1992.

Jackobson, N.S. & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. Journal of Consulting and Clinical Psychology, 59(1), 12-19.

Jacques, A. and Stevenson, G. (2000) Korsakoff's syndrome and other chronic alcohol related brain damage. Dementia Services Development Centre: Stirling.

Kalmaijn, S., van Boxtel, M.P.J., Verschuren, M.W.M., et al. (2002). Cigarette smoking and alcohol consumption in relation to cognitive performance in middle age. Am J Epidemiol; 156: 936-944.

Kreutzer, J.S., Doherty, K.R., Harris, J.A. & Zasler, N.D. (1990a) Alcohol use among persons with traumatic brain injury. Journal of Head Trauma Rehabilitation, 5(3); 9-20.

Kreutzer, J.S., Marwitz, J.H. & Witol, A.D. (1995). Interrelationships between crime, substance abuse and aggressive behaviours among persons with traumatic brain injury. Brain Injury. 9(8); 757-768.

Kreutzer, J.S., Wehman, P.H., Harris, J.A., Burns, C.T. & Young, H.F. (1991). Substance abuse and crime patterns among person with traumatic brain injury referred for supported employment. Brain Injury, 5(2); 177-187.

Kreutzer, J.S., Witol, A.D. & Marwitz, J.H. (1996a). Alcohol and drug use among young persons with traumatic brain injury. Journal of Learning Disabilities 29(6): 643-651.

Kreutzer, J.S., Witol, A.D., Sander, A.M., Cifu, D.X., Marwitz, J.H. & Delmonico, R. (1996b). A prospective longitudinal multicentre analysis of alcohol use patterns among persons with traumatic brain injury. Journal of Head Trauma Rehabilitation, 11(5); 58-69.

Kricka, L. J., & Clarke, P.M.S. (1979). *Biochemistry of alcohol and alcoholism*. New York: Halsted.

Langley, M.J., Lindsay, W.P., Lam, C.S. & Priddy, D.A. (1990). A Comprehensive alcohol abuse treatment program for persons with traumatic brain injury. Brain Injury, 4(1); 77-86.

Launer, L.J., Masaki, K., Petrovitch, H., et al. (1995). The association between midlife blood pressure levels and late-life cognitive function. J Am Geriatr Soc; 51: 1445-1450.

Lishman, W.A. (1990). Alcohol and the brain. British Journal of Psychiatry 156: 635-644.

Malia, K.B. Berwick, K.C., Raymond, M.J. & Bennet, T.L. (1997). Brainwave-R: Cognitive strategies and techniques for brain injury recovery: Executive functions (Client workbook

and therapist workbook). PRO-ED: Austin, Tx.

McCann DU, Szabo Z, Scheffel U, et al. Positron emission tomographic evidence of toxic effect of MDMA (ecstasy) on brain serotonin neurons in human beings. *Lancet* 1998;352:1433-1437

McLellan, A. T., Luborsky, L., Woody, G.E., & O'Brien, C.P. (1980). An improved diagnostic evaluation instrument for substance abuse patients: the Addiction Severity Index. *Journal of Nervous and Mental Disorders*, *168*, 26-33.

Moeller, G.G., Barrat, E.S., Dougherty, D.M., Schmitz, J.M & Swann, A.C. (2001). Psychiatric Aspects of Impulsivity. Am Journal Psychiatry, 158(11): 1783-1793.

Monteiro, M. G., & Masur, J. (1986). Monitoring alcoholism treatment: The appropriateness of choice between GGT and MCV evaluation after a short time of abstinence. *Alcohol, 3,* 223-226.

Mukamal, K.J., Longstreth, W.T., Mittleman, M.A., et al. (2001). Alcohol consumption and subclinical findings on magnetic resonance imaging of the brain in older adults. The Cardiovascular Health Study. Stroke: 32: 1939-1946.

Mullen, P.E. (2001). Mental Health and Criminal Justice. A review of the relationship between mental disorders and offending behaviours and on the management of mentally abnormal offenders in the health and criminal justice services. Criminology Research Council Australia.

National Health and Medical Research Council. A guide to the development, implementation and evaluation of clinical practice guidelines. Canberra: NHMRC, 1999. Available at: http://www.nhmrc.gov.au/publications/synopses/cp30syn.htm (accessed Jan 2006).

Nedopil, N. (2000). Offenders with Brain Damage. In: Hodgins S. & Muller-Isberner, R. (Eds). Violence, Crime and Mentally Disordered Offenders. Wiley Chichester, 39-62.

Nixon, S.J. & Parsons, O.A. (1991). Alcohol-related efficiency deficits using an ecologically valid test. Alcoholism: Clinical and Experimental Research 15(4): 601-606.

Nixon, S.J. (1993). Application of theoretical models to the study of alcohol-induced brain damage. In Hunt, W.A. and Nixon, S.J. eds. Alcohol Induced Brain Damage. National Institute on Alcohol Abuse and Alcoholism Research Monograph No. 22. NIH Publication no. 93-3549. Bethesda, MD: The Institute.213-228

O'Farrell, T. J., & Maisto, S.A. (1987). The utility of self-report and biological measures of alcohol consumption in alcoholism treatment outcome studies. *Advances in Behaviour Research and Therapy*, *9*, 91-125.

Oscar-Berman, M., & Hutner, N. (1993). Frontal lobe changes after chronic alcohol ingestion. In Hunt, W.A. & Nixon, S.J. eds. Alcohol – Induced Brain Damage. National Institute on Alcohol Abuse and Alcoholism Research Monograph No. 22. NIH Publication no. 93-3549. Bethesda, MD: The Institute. Pp 121-156.

Parons, O.A., & Nixon, S.J. (1993). Neurobehavioural sequelae of alcoholism. Neurologic Clinics; 11(1): 205-218.

Parsons, O.A. (1993) Impaired neuropsychological cognitive functioning in sober alcoholics. In Hunt, W.A. and Nixon, S.J. (eds) Alcohol Induced Brain Damage. National Institute on Alcohol Abuse and Alcoholism. Research Monograph No. 22. NIH Publication No. 93-3549. Bethesda, MD: The Institute. Pp 173-194.

Parsons, O.A., Sinha, R., & Williams, H.L. (1990). Relationships between neuropsychological test performance and event-related potentials in alcoholic and non-alcoholic samples. Alcoholism: Clinical and Experimental Research; 14(5): 746-755.

Patton, J.H., Standford, M.S. & Barratt, E.S. (1995). Factor structure of the Barratt impulsiveness scale. Journal of Clinical Psychology; 51: 768-774.

Pfefferbaum, A., Lim, K.O., Zipursky, R.B., Mathalon, D.H., Rosenbloom, M.J., Lane, B., Ha, C.N. & Sullivan, E.V. (1992). Brain grey and white matter volume loss accelerates with aging in chronic alcoholics: A quantitative MRI study. Alcoholism: Clinical and

Experimental Research; 16(6): 1078-1089.

Pick, D. (1989) Faces of Degeneration. Cambridge University Press. Cambridge.

Platt. (1980). On establishing the validity of 'objective' data: Can we rely on cross-interview agreement. *Psychological Medicine*, *10*, 573-581.

Ponsford, J. Ed. (2004) Cognitive and Behavioural Rehabilitation, from Neurobiology to Clinical Practice; Guilford Press, new York.

Pope, H.G. (2002). Cannabis, cognition and residual confounding. JAMA; 287: 1172-1174.

Pope, H.G., Gruber, A.J., Yurgelun-Todd, D. (1995). The residual neuropsychological effects of cannabis; the current state of the research. Drug Alcohol Dependence; 38: 25-34.

Rabbitt, P., Donlan, C. Watson, P et al. (1995). Unique and interactive effects of depression, age, socioeconomic advantage and gender on cognitive performance of normal healthy older people. Psychol Aging; 10: 307-313.

Ricaurte GA, Forno LS, Wilson MA, *et al.* MDMA selectively damages central serotonergic neurons in the primate. *JAMA* 1988:260:51-55

Roehrich, L. & Goldman, M.S. Experience-dependent neuropsychological recovery and the treatment of alcoholism. Journal of Consulting and Clinical Psychology 61(5): 812-821.

Rogers, R.D., Everitt, B.J., Baldacchino, A., Blackshaw, A.J. Swainson, R., Wynne, K., Maker, N.B., Hunter, J., Carthy, T., Booker, E., London, M., Deakin, J.F.>, Sahakian, B.J. & Robbins, T.W. (1999). Dissociable deficits in the decision making cognition of chronic amphetamine abusers, opiate abuses, patients with focal damage to prefrontal cortex, and tryptophan-depleted normal volunteers: evidence for monoaminergic mechanisms. Neuropsychopharmacology 20: 322-339.

Ruitenberg, A., van Swieten, J.C., Witteman, J.C.M et al. (2002). Alcohol consumption and risk of dementia; the Rotterdam Study. Lancet; 359: 281-286.

Ryan, C., & Butters, N. (1980). Learning and memory impairments in young and old alcoholics: Evidence for the pre-mature aging hypothesis. *Alcoholism, 4*, 288-293

SELZER, M.L. (1971) The Michigan Alcoholism Screening Test: The Quest for a New Diagnostic Instrument American Journal of Psychiatry 127:1653-1658.

Singh-Manoux, A., Britton, A., Marmot, M. (2003) Vascular disease and cognitive function. J Am Geriatr Soc; 51: 1445-1450.

Sinha, R., & Easton, C. (1999). Substance use and criminality. *Journal of the American Academy of Psychiatry and the Law, 27(4),* 513-526.

Skinner, H. A. (1984). Assessing alcohol use by patients in treatment. In R. G. Smart, Cappell, H.D., Glaser, F.B., Israel, Y., Kalant, H., Schmidt, W & Sellers, E.M. (Ed.), Research Advances in Alcohol and Drug Problems. New York: Plenum Press.

Smith, F., & Liu, R. (1986). Detection of phenobarbitol in bloodstains, semen, seminal stains, perspiration and hair. *Journal of Forensic Sciences*, *26*, 582-586.

Sobell, L. C., & Sobell, M.B. (1985). Alcohol Timeline Followback. In J. P. Allen, & M. Columbus, (Ed.), *Assessing alcohol problems: A guide for clinicians and researchers. NIAAA Treatment Handbook Series 4* (pp. 241-252). Bethesda: NIAAA.

Solowij, N. (1998). Cannabis and cognitive functioning. Cambridge, England: Cambridge University Press.

Solowij, N., Stephens, R.S., Roffman, R.A., Babor, T., Kadden, R., Miller, M., Christiansen, K., McRee, B., Vendetti, J. (2002). Cognitive functioning of long-term heavy cannabis users seeking treatment. JAMA; 287(9): 1123-1131.

Spiegel DA. (1999). Psychological strategies for discontinuing benzodiazepine treatment. Journal of Clinical Psychopharmacology 19(6 Supplement): 17S-22S.

Sullivan, E.V., Deshmukh, A., Desmond, J.E. et al. (2000) Cerebellar volume decline in

normal aging, alcoholism, and Korsakoff's syndrome: Relation to ataxia. Neuropsychology; 14: 341-352.

Taylor, L.A., Kreutzer, J.S., Demm, S.R. & Meade, M.A. (2003). Traumatic brain injury and substance abuse: A review and analysis of the literature. Neuropsychological Rehabilitation 13, (1/2) 165-188.

Volgenathaler, D.R. (1987). An overview of head injury: Its consequences and rehabilitation. Brain Injury 1: 113-127.

Ward, T., & Brown, M. (2004). The Good Lives Model and conceptual issues in offender rehabilitation, Psychology, Crime & Law, Vol. 10(3), 243-257.

Watson, C. G., Tilleskjor, C., Hoodecheck-Schow, E.A. Pucel, J & Jacobs, L. (1984). Do Alcoholics give valid self-reports? *Journal of studies on Alcohol, 45,* 169-175.

White RF, Feldman RG, Proctor SP. Neurobehavioral effects of toxic exposures. In: White RF, ed. Clinical syndromes in adult neuropsychology: the practitioner's handbook. Amsterdam: Elsevier Science Publishers, 1992: 1-51.

White, R.F; & Proctor, S.P. (1997) Solvents and neurotoxicity. The Lancet, Vol 349(9060), pp 1239-1243.

Wilkinson, D.A. & Carlen, P.L. (1982) Morphological abnormalities in the brains of alcoholics: Relationship to age, psychological test scores and patient type. In Wood, W.G.and Elias, M.F. eds. Alcoholism and Aging: Advances in Research. Boca Raton, FL, CRC Press, pp 61-77.