MANAGING COGNITIVE IMPAIRMENT IN AOD TREATMENT

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MANAGING COGNITIVE IMPAIRMENT IN AOD TREATMENT

PRACTICE GUIDELINES FOR HEALTHCARE PROFESSIONALS

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INTRODUCTION

Alcohol and other drugs (AOD) influence brain function and subsequently our thinking, emotions and behaviour. Loss of control and continued substance use despite negative consequences are hallmark characteristics of addiction. People seeking treatment for AOD use disorders often experience impaired cognitive functioning, particularly poor attention, working memory, inhibition, planning, organisation and decision-making. Cognitive impairment (CI) can adversely influence treatment engagement, adherence, completion and outcomes.

Clients are often concerned about the harmful effects of AOD use on brain health, and thus an understanding of the nature, severity and duration of AOD-related CI can inform care-plans. Specifically, knowing an individual's cognitive strengths and difficulties can help shape the delivery of psychosocial interventions, the need for referral to neuropsychological services, the application of compensatory strategies, and the potential benefit of cognitive rehabilitation. However, whilst considering an individual's level of cognitive functioning can be helpful clinically, it is important to avoid using diagnostic labels such as "Acquired Brain Injury" or ABI unless a formal diagnosis has been conveyed, as these labels could be detrimental to self-efficacy and motivation for behaviour change.

These guidelines provide practical strategies for the management of CI informed by the latest scientific and grey literature concerning best practice, emerging approaches and clinical expertise. The development of these guidelines involved an extensive review of the literature, consultation with clinical neuropsychologists and other AOD clinicians. It is important to emphasise that these guidelines should be read in conjunction with workplace policies, and should never replace the application of sound clinical judgment.

WHAT IS COGNITIVE FUNCTIONING?

Cognition refers to the brains ability to collect, assimilate, organise, store and manipulate information. As such, cognitive functioning is an umbrella term that encompasses a range of integrated skills and domains that allow us to process and respond to information within our environment.

These domains range from basic functioning (e.g., processing speed, attention) through to highly complex goal-oriented skills (e.g., executive functioning such as planning or decision making). An example of the pyramid model of cognitive functioning is shown below in **Figure 1**, which highlights the key domains and their relative level of complexity (with more complex functions represented towards the top of the pyramid).

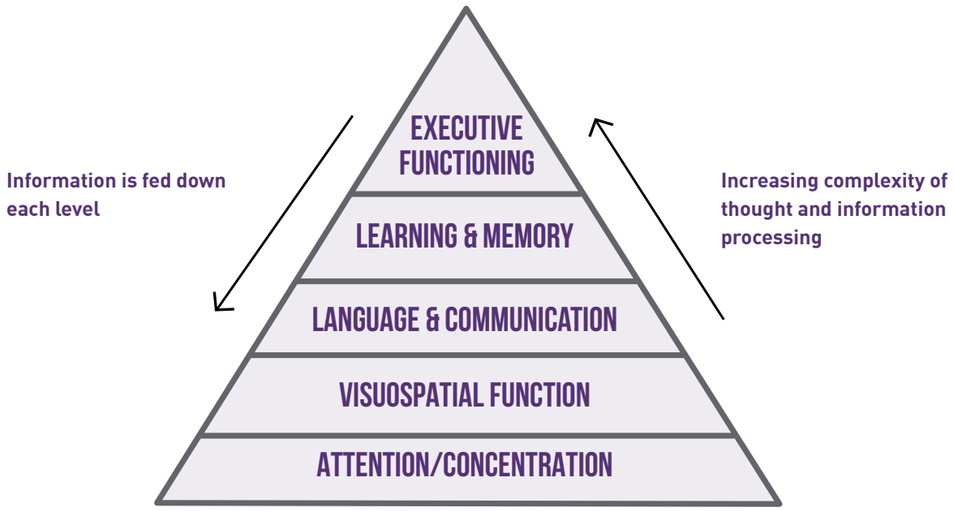


Figure 1. Hierarchy of Cognitive Functioning

INFORMATION PROCESSING SPEED

Processing or 'thinking' speed refers to how quickly the brain can process sensory information or perform a mental task.

ATTENTION

Attention refers to one's ability to process incoming information from the body's sensory systems. This domain is made up of various skills, including, focussed or selective attention (i.e. concentration), sustained attention or vigilance - and at the higher end of complexity, divided attention (performing multiple tasks at the same time). Generally these skills underpin higher level cognitive functions and consequently poor attention can impact on a variety of other cognitive domains.

VISUOSPATIAL FUNCTION

Visuoperceptual and visuospatial functioning describes abilities that allow the brain to make sense of sensory information (e.g., object or face recognition) and interpret this information in a spatial context (e.g. judging depth, distance, the spatial orientation of objects).

LANGUAGE AND COMMUNICATION

Language skills comprise two broad categories:

a) **Expressive language skills:** Communicating via speech, writing or gesture;
b) **Receptive language:** The ability to understand and comprehend language.

Language skills also include object naming, word finding, fluency, grammar and syntax.

MEMORY AND LEARNING

Memory and learning refers to our ability to hold and retain information over time and is reliant on specific structures within the temporal lobes of the brain. The stages of memory formation are as follows:

1) **Encoding:** This is the process by which information is acquired;
2) **Consolidation:** The process of storing information in long term memory;
3) **Retrieval:** The process of accessing or retrieving information from long term memory stores.

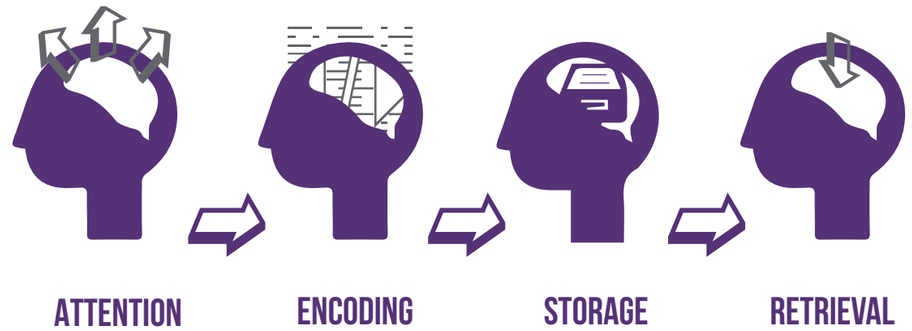


Figure 2. Stages of Memory Formation

MEMORY SYSTEMS

Memory functions can be classified into several distinct processes, each of which serves a particular role in our ability to remember information. This model is illustrated below, however, for most clinicians it may be more helpful to consider the following (more practical) distinctions:

- **Verbal & Visual Memory:** The left cerebral hemisphere (left hand side of the brain) typically process verbal information and the right cerebral hemisphere visual or spatial information. We are sometimes better at learning or remembering visual information (diagrams, pictures or faces) than verbal information (e.g. spoken or written information or names) or vice versa.

- **Recent versus Remote Memory:** This refers to the temporal sequence of memories. In the case of memory impairment, recollection of more recent events or memories is likely to be impacted (e.g. due to damage preventing adequate encoding and storage of information) rather than to well preserved, remote (long-term) memories that were processed prior to any damage occurring.
- **Prospective Memory:** Involves remembering to perform an action or recall an intention at some point in the future (i.e., remembering to pay a bill in two days' time).
- **Priming:** Exposure to a stimulus can influence one's response to a later stimulus.

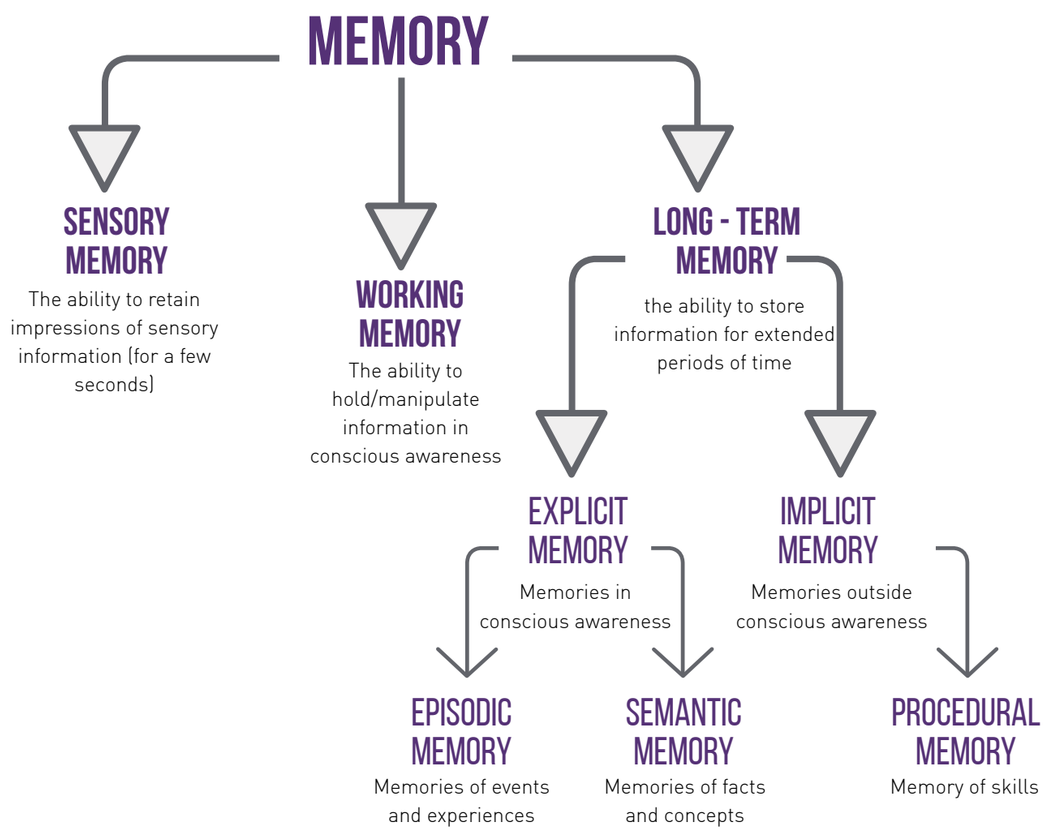


Figure 3. **Overview of Memory Types**

EXECUTIVE FUNCTIONING

Executive functioning is an umbrella term that refers to a set of effortful processes involved in goal-directed beahiours, involving inhibition, mental flexability and working memory. Theses processes enable us to make adaptive responses to novel, complex or ambiguous situations. Executive functions are predominantly governed by the frontal lobes of the brain. Executive functioning is sometimes split in to "hot" and "cold" neurocognitive functions (EF) in adults with a substance use disorder. Hot EFs are those involving emotional awareness, affective responses and social perception (e.g., social cognition), whilst Cold EFs refers to the more mechanistic higher-order cognitive operations (e.g., working memory) and rational decision-making.

Table 1. **Executive Functioning Skills**

EXECUTIVE FUNCTION	HEALTHY FUNCTIONING
Planning	The ability to organise, prioritise and plan a task
Decision-making	The ability to solve problems & weigh up alternatives
Inhibition	The ability to restrain or inhibit an action
Concept formation	The ability to form an understanding of an idea
Abstract thinking	The ability to "think outside the box" or reason with and consider principles and ideas removed from the objects themselves
Mental flexibility	The ability to hold multiple pieces of information in one's mind or switch between sets of information

WORKING MEMORY

Working Memory is a specific cognitive domain which refers to the ability to hold information in mind and manipulate it in order to perform a specific task (e.g., mental arithmetic, dialling a phone number, or following a set of directions). Some researchers suggest that working memory is a foundational executive function which underpins most cognitive functions [1].

SOCIAL & EMOTIONAL COGNITION

Social cognition refers to the capacity to detect emotions and intentions in others. The two subsets of social cognition are:

- **Theory of Mind:** Relates to the capacity to attribute beliefs, desires and intentions to both oneself and to others. This helps us understand why someone acts in a certain way and allows us to predict how someone might act.
- **Emotion Recognition:** Relates to the ability to infer an emotional state in someone else.

GENERAL INTELLECTUAL FUNCTIONING (IQ)

General intellectual functioning or 'crystallised intelligence' can be conceptualised as a global or unitary estimate of an individual's cognitive functioning across a range of domains. Commonly employed measures of intelligence yield an intelligence quotient (IQ), a total score derived from a collection of tests assessing multiple domains that are standardised based on an individuals' chronological age. IQ scores can be helpful in contributing to diagnostic formulations (e.g., for intellectual disability) or evaluating an individual's ability, however, in other circumstances they may not appropriately reflect an individual's cognitive functioning or level of CI.

WHAT IS COGNITIVE IMPAIRMENT?

Cognitive impairment (CI) refers to suboptimal cognitive functioning involving decline in one or more cognitive domains as a result of damage or disruption to neural structures or networks. When considering whether an individual is cognitively impaired, one must first consider what is normal for an individual and relative to other individuals their age.

Overall IQ (intelligence quotient) scores are a good example of this. In a 'normal' healthy population, 50% of individuals would be expected to score between 90 (lower IQ) and 110 (higher IQ) on an IQ test, and very few would be expected to score 130 (very high IQ) or more. If someone scored 85 but had previously scored 110, this could be considered an impairment, however, if that same person had always scored 85, then this could be considered normal (i.e., no impairment). It is important to remember that some skills are highly influenced by an individual's age and level of education. A final consideration is that we all have our own strengths and weaknesses, and some people are naturally better with words and verbal information while others might be better with processing visual or pictorial information.

BEHAVIOURAL INDICATORS OF COGNITIVE IMPAIRMENT

When individuals initially report cognitive difficulties it can be challenging to tease apart which cognitive domains may be impacted. Impairment in one cognitive domain can impact other cognitive domains. For instance, impaired attention can lead to memory problems because the individual is not attending to information sufficiently for it to be encoded properly. Nevertheless, some general patterns and common complaints are highlighted in **Table 2.** It may be helpful to refer back to this table when reading these guidelines as it offers context into the types of everyday behaviours clients may present with. Where CI is the result of serious brain damage there may also be additional impairments in sensory, motor, behaviour or emotional domains. Other common behaviours reflecting executive functioning deficits in particular, include increased frustration/irritability, socially inapproproiatre behaviour, we well as changes in emotional responsiveness/ expression (e.,g being flat or elevated) or reduced emotional regulation (e.g., volatile reactions).

Table 2. **Behaviours Indicative of CI**

Cognitive Domain	Example behaviour or complaint
Attention & Concentration	• Client makes repeat requests to clarify or explain information • Client lacks focus, is easily distracted, or misplaces items
Information Processing Speed	• The client is slow to respond to questions • Client is easily overwhelmed or appears to miss information
Working Memory	• Client loses track of what someone else is saying in conversations, or forgets what they were going to say • Can't follow plots (books or film) • Client has difficulty keeping multiple pieces of information in mind (e.g. makes requests to repeat a phone number that needs to be dialled) • Trouble working through problems in one's head (e.g. calculating change owed)
Visuospatial Perception	• Client bumps into stationary objects, neglects or ignores distinct areas in their environment • Client gets easily lost in otherwise familiar environments. • Client misidentifies objects • Navigational difficulties
Language	• Non-fluent (clunky) or effortful speech • Word finding difficulties (frequent pauses in conversation, or may describe the word they cannot recall) • Difficulty comprehending others speech
Memory	• Client forgets appointments, previous conversations, names of people or places, details, events, objects • Vague recall of events • Difficulties learning new information such as a new skill • Poor 'orientation' (time, place, date)
Executive Function	• Inability to multitask (e.g., can't focus on more than one thing at a time) • Problems communicating details of a story in a sequential/ organised way • Impulsive behaviour • Lack of insight • Concrete rigid/thinking (can't generalise learned info/ understand metaphors) • Difficulty monitoring one's behaviour • Difficulty adapting to changing situations or environments • Difficulty planning and organising behaviour • Changes in thinking patterns and sometimes personality • Difficulty problem-solving
Social Cognition	• Difficulty discerning emotional cues, misinterptreting others' thoughts and intentions • Hostile defensive, argumentative behaviour

CAUSES OF COGNITIVE IMPAIRMENT

HOW DO SUBSTANCES AFFECT COGNITIVE FUNCTIONING?

Psychoactive substances dysregulate several neurotransmitter systems that underpin cognitive functioning. For example, long term stimulant use (i.e., methamphetamines) causes both short-term and long-term neuroadaptations in dopamine, noradrenaline and serotonin systems responsible for (among other things) attention, learning, mood, and memory formation. Alcohol primarily disrupts the GABA and glutamate system, cannabis disrupts the endocannabinoid system, and opioids (i.e., heroin) effect the endogenous opioid system (Mu-receptor agonists). Each of these substances ultimately effect dopamine, altering the fronto-striatal system which governs executive functioning, decision-making and emotion regulation. It may be helpful to explain to clients that substance use can cause neurotransmitters to become depleted, as the brain learns that it no longer needs to produce as much dopamine, serotonin or other neurotransmitters mimicked by the drug. After a binge-episode, it can take a few days for these neurotransmitters to be restored to their normal levels, during which time the client may experience negative symptoms such as fatigue and low mood. With chronic long-term use, the time required for neurotransmitters to replenish increases, which can reinforce the cyclic nature of substance use.

WHAT CAUSES COGNITIVE IMPAIRMENT?

There are many factors that can cause or contribute to CI. This is most notable in the AOD sector, where individuals can present with multiple comorbid diagnoses, injury, and various environmental and developmental stressors. It is important to note that not all causes of CI result in permanent change and many are transient or modifiable. As such, it is important to obtain a comprehensive history from clients to identify areas for immediate multidisciplinary intervention and treatment, and refer on to specialist assessment if required. The following section details both persistent and transient causes of CI.

Table 3. Persistent and Transient Causes of CI

PERSISTENT CAUSES	TRANSIENT CAUSES
Acquired Brain Injury*	**Psychological conditions**
Traumatic Brain Injury*	**Acute substance use/intoxication**
Neurodegenerative conditions	**Sleep deprivation & insomnia**
Chronic medical conditions	**Acute medical conditions**
Developmental disorders (i.e., ADHD, Autism, intellectual disability)	**Stress caused by homelessness, social isolation, abuse, socioeconomic disadvantage**

*The persistence of impairments associated with ABI and TBI will vary based on severity of injury and individual differences.

Sarah's Story: Transient CI

Sarah's story illustrates the value of residential rehabilitation, nutrition and improved mental health on cognitive functioning.

Sarah is a 65-year-old female who was referred to neuropsychological assessment after being admitted to a long-term residential AOD rehabilitation program. This was the second time she had been admitted and clinicians noticed she had declined cognitively and were concerned about her having an acquired brain injury or dementia.

Sarah was suffering from grief after losing several family members and had been drinking alcohol every day for over 20 years as a coping mechanism. Prior to being admitted for rehabilitation, Sarah had not been looking after herself, felt depressed, had poor nutrition, could not clean her house, and avoided socialising.

During detox, Sarah was treated with thiamine, and during rehabilitation regularly saw a psychiatrist who prescribed anti-depressants and helped Sarah to discuss her grief and trauma. By the time she was seen for an assessment she had been abstinent for two months and was regularly participating in social and recreational programs at the rehabilitation centre. She was eating and sleeping well and getting exercise by swimming in the pool. Sarah was feeling more positive about life. Her neuropsychological assessment showed only mild weaknesses in some higher level executive skills such as planning and dividing attention, while all her other skills fell in the normal range for someone her age and as such there was no evidence for dementia or an acquired brain injury.

During the feedback session Sarah was relieved to hear this news as it had been weighing on her mind. She was advised to continue engaging in all the activities she had been doing which were making a big difference in helping her feel productive and positive, and her support workers were planning how to ensure a smooth transition for her return to the community.

ACQUIRED BRAIN INJURY (ABI)

ABI refers to damage that has occurred to the brain after birth resulting in the deterioration of cognitive, physical, sensory, emotional or independent functioning. Impairment can arise from traumatic injuries (referred to as Traumatic Brain Injury [TBI]) or non-traumatic injuries such as stroke, hypoxia or anoxia, infection (i.e., meningitis or encephalitis), poisoning, brain tumours, prolonged AOD misuse or drug overdose. ABI is differentiated from developmental disability, psychiatric illness, or degenerative neurological diseases (i.e., Alzheimer's disease, Multiple Sclerosis, and Motor Neurone Disease), however, these disorders can and do co-occur. Around 1 in 45 (or 432,700) Australians have an ABI [2], however the prevalence is often higher in certain populations (i.e., prison and indigenous populations and those with substance dependence or severe psychiatric illness).

TRAUMATIC BRAIN INJURY (TBI)

TBI is defined as an alteration in brain functioning as a result of external force (i.e., when the head has been subjected to a severe blunt force trauma or a penetrating injury from a projectile or sharp object). These injuries can result in the brain shifting inside the skull, causing damage to areas that make contact with the skull (contusions), rupture of blood vessels, or neuronal damage leading to a range of sensory, motor, cognitive, emotional and behavioural changes [3]. There is often a loss of conciousness for several minutes, neurological investigation and hospitalisation. The impact of these changes can range from being very mild through to exceptionally disabling affecting a client's day-to-day functioning (depending on the brain regions affected). It is important to note that not all head injuries result in a TBI. Studies have shown that approximately 80% of individuals admitted to hospital with TBI are classed as having a mild TBI, and that the majority of symptoms are generally expected to resolve within several months [3, 4].

In the AOD sector, clients frequently report incidents of head injury [5]. For instance, in a recent review of clients presenting to a specialist neuropsychological assessment service, 41% of clients reported having sustained a head injury [6]. However, of these incidents, TBI was considered to be a major contributing factor in only 7% of these cases. Consequently, clinicians should avoid potentially misattributing their clients CI to relatively benign instances of head injury – particularly when that injury is not followed by an extended period of loss of consciousness (>30 minutes).

TBI versus Concussion

Compare the two cases: one demonstrated significant cognitive impairment while the other performed normally on assessment.

Brett was assaulted while on a night out with friends. He struck his head and briefly lost consciousness but left the scene before the ambulance arrived. He has a history of depression and anxiety in addition to polysubstance use. He feels his memory is worse since the assault.

Larry was assaulted while on a night out with friends. He struck his head, was knocked unconsciousness and taken to hospital by ambulance. Medical notes indicate he was intubated and ventilated on arrival to hospital and remained in a coma for several days. Brain imaging revealed he had experienced bleeding on the brain in addition to a skull fracture. He has no recollection of the event and had to be told what happened. His first recollection was waking up in hospital but he was unsure how long after this was. He reported having a fuzzy recall of events in the days following the assault. After waking up he was unable to remember new information for 3 days. He was transferred to a rehabilitation hospital where he stayed as an inpatient for two weeks. In the weeks following the incident he noted feeling irritable, frustrated, slowed down and was having difficulty recalling information.

Brett's case is an example of a mild or concussive injury where a full recovery would likely be expected and his reported complaints are likely secondary to substance use or mental health difficulties. Brett would be referred for psychological assessment and support. Larry's case is an example of a client who sustained a severe traumatic brain injury and showed significant cognitive impairments on assessment. Larry would receive continued multidisciplinary rehabilitation support with input from neuropsychology, psychology, occupational therapy and social workers.

TBI & AOD

Those with pre-existing TBI, post-injury and pre-existing CI are more likely to subsequently develop a substance use disorder (SUD), particularly when injury is sustained during childhood through to young adulthood [7, 8]. Among those individuals who sustain a TBI, it is well established that alcohol consumption can undermine rehabilitative outcomes, increase seizure rates (including alcohol withdrawal seizures), psychiatric difficulties, and the likelihood of further TBI [9, 10]. Pagulayan, Temkin, Machamer, & Dikmen (2016) found that approximately half of their patients with TBI drank in the moderate (1–2 drinks between three and six times per week, or 3–4 drinks on 1–4 occasions per week) to heavy (consuming alcohol seven or more times a week) range prior to their head injury [11]. Six months after sustaining a TBI, 30% had returned to drinking, with 20% of these drinking heavily. The authors highlighted that early intervention in the first 6 months post-injury is crucial in terms of managing alcohol use post TBI.

STROKE

Strokes are classified as either ischaemic (a blockage or clot in an artery causing an obstruction in blood flow) or as haemorrhagic (bleeding in the brain). Stroke's that are haemorrhagic are often the result of either an arteriovenous malformation (AVM) (an abnormal tangle of blood vessels disrupting blood supply to the brain), or an aneurysm (an abnormal bulge in a blood vessel wall bursts) [12].

The Australian Stroke Foundation recommends the **'F.A.S.T'** test to help recognise signs of stroke [13].

1. Face: Check their face. Has their mouth drooped?
2. Arms: Can they lift both arms?
3. Speech: Is their speech slurred? Do they understand you?
4. Time: Is critical. If you see any of these signs call 000 straight away.

A number of drugs can increase the risk of stroke: e.g., methamphetamine, MDMA, cocaine, inhalants by increasing blood pressure (hypertension), and the composition of blood vessels (i.e., weakening of vessel walls) [14]. Alcohol is also a risk factor for stroke, particularly haemorrhagic stroke [15].

The degree of damage caused to the brain by a stroke depends on the area affected and how rapidly treatment has been obtained. Ischaemic strokes usually cause damage to certain confined parts of the brain, causing very specific sensory, motor, cognitive, emotional or behavioural impairments. Haemorrhagic strokes can have a more generalised and expansive effect, especially if very severe. In the event that a client reports having experienced a stroke, it is recommended that clinicians explore what rehabilitation and assessments were undertaken during and after hospital admission, as these may explain any difficulties observed. Often a client may have received neuropsychological assessment during a hospital admission, which may include useful recommendations and strategies that can be implemented in the treatment of their AOD problem.

HYPOXIC BRAIN INJURY

Hypoxic brain injuries are caused by extended oxygen deprivation. During respiratory arrest breathing is supressed, but the heart keeps pumping, and oxygen continues to be supplied to the brain. However, during cardiac arrest, the heart stops beating, and there is an elevated risk of brain injury (called an anoxic brain injury), especially when respiratory arrest precedes cardiac arrest (i.e., suffocation) [16]. Any of these incidents should be documented, and clinicians are recommended to explore the length of any required hospital stay and interventions received (e.g., Did the individual require ventilation and/or intensive care? Or were they admitted only briefly for observation?). Significant hypoxic brain injuries from drug overdose typically require hospitalisation and rehabilitation. These events can result in 'global' (generalised) CI and difficulties with daily living, which may persist long-term and require long-term support [17].

NEURODEGENERATIVE AND OTHER NEUROLOGICAL CONDITIONS

In older clients (>60/65 years) progressive cognitive decline can be indicative of a neurodegenerative condition such as Alzheimer's disease, other forms of Dementia, Parkinson's disease, or Huntington's disease. If there are concerns about significant decline in cognitive or functional ability, or an undiagnosed neurodegenerative condition, this should be documented and a GP referral sought in the first instance.

Common measures or screens of CI will not reliably detect dementia. A person with a good education may score very well on these scales and still have dementia, while a person who is poorly educated may score below the cut-off and not have dementia. Many other issues affect the score, including: vision and hearing problems, psychiatric illness, anxiety, depression, being unwell on the day, cultural and linguistic diversity, neurological problems, manual dexterity for questions requiring use of a paper and pencil, and practice effects. Observing a deterioration over 6–12 months is a more reliable indicator of dementia than a single assessment. An interview with an informant who reports deterioration is also helpful.

Specialist referrals can be made to a memory service, such as the 'Cognitive Dementia and Memory Service' (CDAMS) or a geriatrician. It is important to note that many CDAMS do not see people who are currently dependent on substances. More information on CDAMS and referral can be found at:
www.easternhealth.org.au/services/item/196-cognitive-dementia-and-memory-service-cdams.

Importantly, it is best-practice to provide any referral service with a clear history of what you have observed over time, with any changes. If you have information from a family member or close friend who has known the person for a long period of time, this information should be relayed. Clinical practice guidelines for the management of dementia can be found at:
www.dementia.org.au/resources/clinical-practice-guidelines.

BLOOD BORNE VIRUSES

Blood borne viruses can cause CI, particularly in the advanced stages of disease progression. Human Immunodeficiency Virus (HIV) in particular can be associated with significant cognitive dysfunction, and this has led to the development of a classification system for various 'HIV Associated Neurocognitive Disorders' (HAND) [18]. Hepatitis C is also a well-documented cause of CI, particularly affecting attention, concentration and psychomotor speed [19]. The prevalence of Hepatitis C is extremely high among people who inject drugs and those who have been incarcerated [20]. Liver cirrhosis and liver failure as well as complications associated with untreated Hepatitis C can result in acute changes to cognitive functioning (hepatic encephalopathy), resulting in symptoms such as confusion and disorientation through to coma. Fortunately, Hepatitis C can now be treated through a 12-week course of medicines known as 'Direct Acting Antivirals' (DAA's), which are subsidised under the Australian Governments Pharmaceutical Benefits Scheme (PBS).

COGNITIVE IMPAIRMENT MEANING & SIGNIFICANCE

THE BRAIN DISEASE MODEL OF ADDICTION

The Brain Disease Model of Addiction considers addiction to be a 'brain disease' where chronic substance use 'hijacks' the brain's reward systems, making it difficult for people to stop using them. Advocates of the model argue that neuroimaging technologies will help diagnose addiction problems, whilst pharmacological, neurocognitive and brain stimulation approaches will help treat them. Some argue that the model reduces moral judgment, stigma and discrimination and that its use with clients can increase insight into their condition and improve access to treatment. Its critics argue that it places too much emphasis on medical solutions to social problems and it can have a negative impact on clients by making people feel helpless about their recovery and reducing their self-efficacy.

WHAT IS A 'CLINICALLY MEANINGFUL' IMPAIRMENT?

Cognitive impairment is the noticable and measurable decline in a persons thinking skills, such that they experience difficulties remembring, learning new things, concentrating or making everyday decisions - and can range from very mild to severe. Many studies conclude that individuals with AOD use disorders have CI based on there being a 'statistically significant' ($p < .05$) difference in the average neuropsychological test performance relative to 'healthy controls'. However, in reality the actual scores may indicate only marginal differences (e.g., recalling only 1 more letter, or scoring 1 more point on a test than controls) and is not what clinicians would consider to be 'clinically meaningful'. Moreover participants may still be functioning within a 'normal' or unimpaired range [21]. In general CI is used to describe performance that is more than 1.3 standard deviations from population norms, or substantially below an individuals expected level of performance [22].

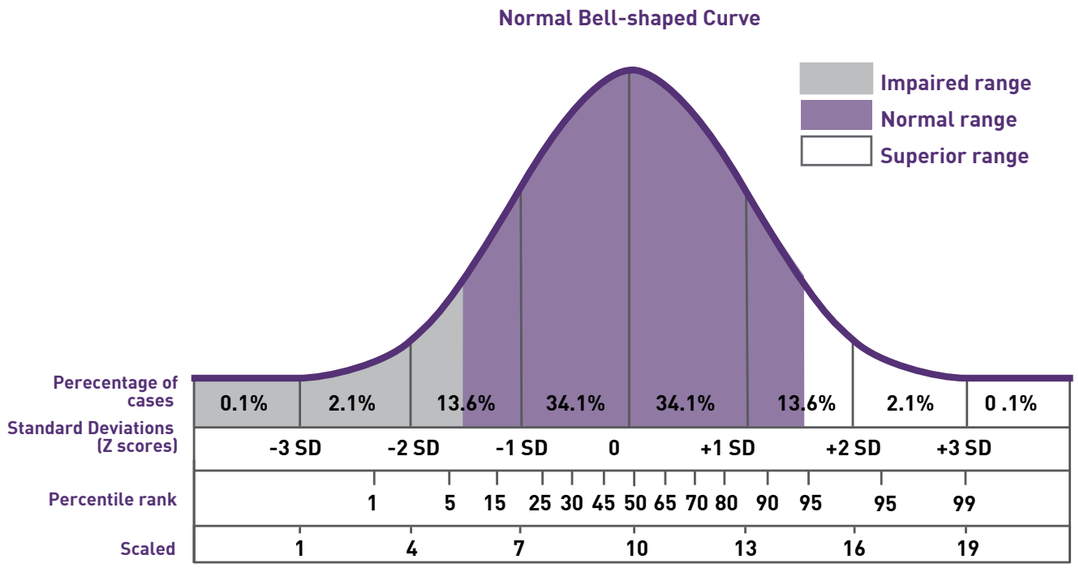


Figure 4. Overview of Neuropsychological Test Performance Metrics

It is expected that on any aptitude-like test 34% of the population will achieve a score within 1 SD below the mean, that 13.6% will have a score 1-2 SD below the mean and around <2% will have a score of 2 or more SD below the mean **(See Figure 4).** A score of 1 SD below the mean represents a score in the 15th percentile (i.e., 85% of the population would score higher), and a score of 2 SD below the mean represents a score in the 2nd percentile (i.e., around 98% of the population would score higher). Whilst there are numerous ways to quantify an individual's performance on neuropsychological tests (e.g., Z-score, T-score, percentile, scaled score), typically, clinically meaningful CI refers to a performance around 1.3 or more standard deviations below population norms (or a scaled score of <6). These scores correspond to the Borderline and Extremely Low categorisations of impairment on the seminal and widely used IQ test known as the Wechsler Adult Intelligence Scale (1955) [23].

Consequently, when interpreting the scientific literature on CI in the AOD sector, it is important to keep in mind that the findings of studies provide merely a guide, illustrating possible impairments that may be associated with prolonged and severe AOD use and that comprehensive neuropsychological assessment is required to confirm CI in individuals.

INTERPRETING AOD LITERATURE ON CI

Whilst there are hundreds of studies demonstrating CI in people experiencing AOD use disorders, often this is limited to one or two tests within a comprehensive battery. However it is important to keep in mind the limitations of this body of evdience which includes:

- Inconsistent approaches to reporting test performance.
- Limitations in methodology (i.e., small sample sizes, cross-sectional designs where causation cannot be inferred, lack of appropriate controls [matched on age, education, etc.], heterogeneity in use onset, duration of use, quantity and frequency of use, dependence vs. recreational use);
- Inability to compare between studies (i.e., different domains assessed, different tests/measures of functioning with a domain, different performance parameters with the same test);
- Publication bias (i.e., negative results showing no differences or unimpaired functioning are less likely to be published);
- Reporting statistical rather than clinically meaningful differences
- Polysubstance use is the norm (i.e., it is difficult to measure the effect of a single drug on cognition when other substances are typically also used);
- AOD use is unlikely to be the only causative factor (i.e., ageing, other psychosocial/environmental factors, TBI could play a role). CI may have pre-disposed them to AOD use disorders rather than be a consequence of AOD use disorders.
- Expert consensus has identified additional cognitive changes not typically assessed by traditional neuropsychological measures (i.e., negative affect and distorted reward processing).

OVERALL PREVALENCE OF CI IN PEOPLE WITH CURRENT AOD PROBLEMS

It is estimated that 50-80% of individuals with alcohol use disorder (AUD) [24-27], and up to to 75% of individuals with poly-substance use disorder experience CI [28-36]. The most commonly observed impairments include altered processing speed, selective and sustained attention, executive functions (working memory, inhibition, set-shifting) and poorer decision-making [36-40]. However, it can sometimes be unclear if CI predates or exists as consequence of AOD problems. Research has found that the cognitive deficits associated with long-term substance use disorders (particularly in executive functioning) may have moderate longevity, and that despite significant abstinence related cognitive recovery, impairments may continue to undermine treatment (increasing relapse and reducing retention) in the initial recovery stage (up to 6-months post discontinuation of use) [27, 41, 42]. A recent systematic review of the literature concluded that processing speed, attention and reasoning task performance were the only consistent predictors of treatment retention, whereas decision-making performance was the primary predictor of relapse to substance use [43]. The extent of CI depends on multiple factors such as the duration and severity of AOD problems, age of onset, frequency and quantity and extent to which normal psychosocial development was interupted.

Recent research has also indicated that the key cognitive domains associated with recovery and relapse may be substance-specific. Higher-order executive function skills, including problem solving, planning and decision making, predict alcohol relapse, whilst attention and generalised executive functioning is associated with opioid relapse, whereas working memory and response inhibition predict relapse to cannabis and stimulant use [38, 44]. This makes higher-order executive functioning viable treatment targets and training programs and interventions that seek to ameliorate specific deficits may show promise in improving treatment outcomes.

The following section examines cognition functioning in relation to specific substances. This literature should be interpreted with caution given the high rates of polysubstance use among treatment seekers (particularly among treatment seekers with a primary drug of concern other than alcohol).

COGNITIVE IMPAIRMENT

ALCOHOL RELATED BRAIN INJURY

Alcohol related brain injury (ARBI) is an umbrella term used to describe CI resulting from excessive alcohol consumption. Other terms in the literature include 'alcohol related dementia' (ARD) and 'alcohol related brain damage' (ARBD). A recent 2018 study examining the global burden of disease concluded that alcohol is a leading risk factor for disease and is the primary cause of approximately 10% of deaths of people between the ages of 15-49 [43]. Recent data from the Australian Institute of Health and Welfare (AIHW) showed that alcohol was the most common reason that people entered publically-funded AOD treatment in 2019-2020, representing 34% of all treatment episodes [46]. Until recently there was a common misperception that low levels of alcohol consumption have a protective effect on health [47]. It is now understood that even low levels of alcohol consumption can increase physical health risks, and any of the potentially protective cardiac effects of drinking are offset by the increased risk of cancers associated with alcohol consumption. However, generally, only regular, moderate to heavy alcohol use is associated with CI.

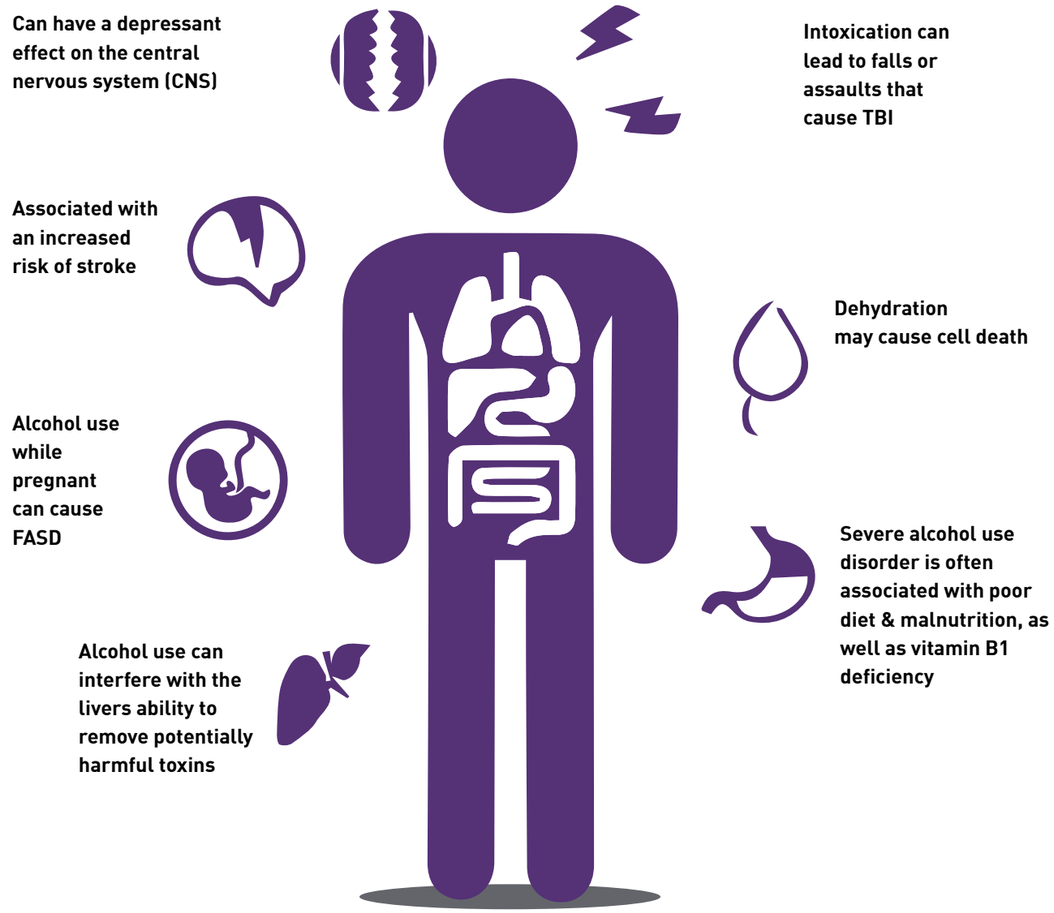


Figure 5. Ways in which alcohol can affect cognitive functioning

HARMS ASSOCIATED WITH CHRONIC ALCOHOL USE

Traumatic Brain Injury	Alcohol intoxication is a leading indirect cause of TBI - increasing the likelihood of motor-vehicle accidents, assaults and falls due to poor coordination and loss of consciousness [48].
Peripheral Neuropathy	Peripheral neuropathy is caused by damage to peripheral nerves (the portion of the nervous system that is outside the brain and spinal cord) which can lead to a sensory disturbance causing numbness, tingling sensations, burning and sometimes pain in the hands, feet and legs, which may progress to loss of limb reflexes and eventual muscle atrophy. This is thought to be related to both the neurotoxic effects of alcohol and poor nutrition.
Cerebellar Atrophy	Cerebellar atrophy is the result of cell loss in the cerebellum (the part of the brain responsible for coordinated movement). It can lead to balance and coordination difficulties, particularly affecting the lower limbs leading to a wide-based gait known as 'ataxia', which further increases the risk of falls.
Hepatic Encephalopathy	Hepatic Encephalopathy (HE) is a neurological condition caused by acute or chronic liver disease (alcoholic cirrhosis) due to high levels of ammonia in the blood [49]. Common symptoms associated with this condition include confusion, disorientation and poor coordination. Some research suggests that mild HE can occur in up to 80% of cirrhotic patients, with overt HE in 45%. Hepatic encephalopathy can be acute (short-term) or chronic (long-term), and is usually treatable [49].

HEALTH COMPLICATIONS RESULTING FROM INTOXICATION

Alcohol is a CNS depressant, and at elevated Blood Alcohol Concentrations (BACs) the risk of aspiration (liquids, saliva or vomit is breathed into the lungs), haemodynamic shock (where blood cannot reach major organs) and compromised cardiac functioning increases, potentially causing death.

SEIZURES

Seizures are a symptom of an underlying abnormal electrical discharges in the brain, and can occur when an individual is intoxicated, overdoses, or is in withdrawal. In heavy dependent drinkers, seizures can occur 6-48 hours after drinking cessation (i.e., during withdrawal) as blood alcohol levels decrease [50]. Among those who use alcohol heavily for a long time, there is also an elevated risk of brain haemorrhage. Finally, withdrawal can cause changes in heart rhythm (which can cause seizures or even death [51]. The risks of any of these adverse events are substantially mitigated by appropriate withdrawal treatment and use if medications such as benzodiazipines.

Some AOD clients may report a history of epilepsy and may be taking or have taken anti-epilepsy medications in the past. Among clients who have sustained some form of brain injury, AOD use can increase seizure frequency, and these clients may require special care [52, 53].

Clients with a history of epilepsy can be supported by helping them understand potential seizure triggers (i.e., sleep deprivation, AOD use, and non-compliance with anti-epilepsy medications. If the client has not been reviewed recently by a neurologist or has experienced an increase in seizure frequency, they should be encouraged to see their neurologist. Good seizure control can reduce the potential long-term cognitive impact of seizures [51].

FETAL ALCOHOL SPECTRUM DISORDERS (FASD)

Alcohol is a teratogen, and impacts the development of the fetus. The impact of alcohol on the developing brain (in utero) depends on maternal consumption levels and fetal developmental stage. As there is no lower limit of alcohol use that can be guaranteed to be completely safe, pregnant clients should be encouraged to stop drinking altogether whilst pregnant or breast feeding, and if medically necessary, receive monitored withdrawal treatment in an inpatient unit.

Children exposed to alcohol in utero are at risk of developing Fetal Alcohol Spectrum Disorders (FASD), characterised by cognitive (i.e., learning), behavioural, developmental, health, and physical (particularly facial) abnormalities. Binge drinking (consuming 4 or more standard drinks on a single drinking occasion) while pregnant can be particularly damaging [54]. Underdiagnoses and misdiagnosis of FASD is common in this group, leading to its characterisation as an 'invisible disability'. Neuropsychological assessment is one tool used to identify if a person may have a FASD, however little is known about how FASD progresses into adulthood [55].

COGNITIVE IMPACT OF ALCOHOL USE

Alcohol use can have both **direct** (e.g., neurotoxic effect on the central nervous system) and **indirect** effects (e.g., increasing the risk of falls) on the brain, and at high levels over a long period of time, is the substance most consistently associated with long term CI [56]. Sustained and heavy alcohol use impacts on the structure and function of the brain, contributing to marked atrophy in the frontal lobes, limbic system (particularly the hippocampus), cerebellum, peripheral nerves as well as reduced neural connectivity and white matter volume [27, 39, 57]. A recent study suggested that even moderate drinking reduces hippocampal volume (the brain area responsible for memory formation) in a dose–response manner [58]. Alcohol can also effect cognitive functioning through altered metabolism, blood flow, poor diet, and dehydration causing neuronal cell death.

ACUTE INTOXICATION

Alcohol hampers cognitive functioning during intoxication [56]. Whilst the toxic alcohol dose varies from person to person as tolerance builds up over time, use of alcohol resulting in a BAC of greater .05 can adversely disrupt cognition in infrequent drinkers. Cerebellar symptoms (e.g., dizziness) tend to occur at higher levels of intoxication (BAC >.08%). Blackouts are common following episodes of binge drinking, and have been associated with increased risk of TBI. Common physical and cognitive effects of alcohol at different BACs are shown below in **Table 4**.

Table 4. Intoxication effects and risk of harm with increasing blood alcohol concentrations.

BAC	EFFECTS
up to .05g%	Feeling of wellbeing, talkative, more relaxed, increased confidence.
.05 - .08 g%	At risk of impaired judgment and reduced inhibitions.
.08 - .15 g%	Moderate risk, slurred speech, impaired balance and coordination, unstable emotions, possibly nausea and vomiting.
.15 - .30 g%	High risk, potential for compromised breathing, unable to walk without assistance, loss of bladder control and possible loss of consciousness.
.30 g% and above	Coma or death.

WITHDRAWAL

In cases of severe alcohol use disorder (AUD) and physiological dependence, alcohol withdrawal symptoms can occur within 6 hours of the last alcoholic drink. Alcohol withdrawal can cause a range of physical and cognitive symptoms including confusion, sweating, diarrhoea, anxiety, loss of appetite, nausea, vomiting, hallucinations, delusions, and insomnia. Repeated withdrawal episodes have been associated with increased CI (most likely attributed to repeated withdrawal seizures) [59]. To minimise the risk of seizures, alcohol withdrawal is usually managed with benzodiazepines. Patients typically require sedation and hydration and are generally best managed in an inpatient setting.

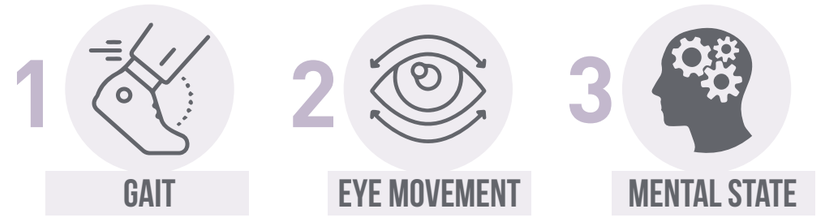
For more information on withdrawal management see Manning, V., Arunogiri, S., Frei, M., Lubman, D., Ridley, K., Mroz, K., & Campbell, S. (2018). Alcohol and Other Drug Withdrawal: Practice Guidelines, 3rd ed. Richmond, Victoria: Turning Point.

THIAMINE DEFICIENCY

Sustained, heavy alcohol use can lead to underlying pathologies arising from nutritional thiamine B1 deficiency. In fact, it has been suggested that the primary mechanism by which cognition is impaired in people with alcohol use disorder is thiamine deficiency [60]. Alcohol impairs the body's ability to process thiamine, and individuals who drink at heavy levels are often undernourished [61].

WERNICKE-KORSAKOFF SYNDROME (WKS)

Wernicke's encephalopathy is an acute condition affecting (1) gait, (2) eye movement and (3) mental state (severe confusion, attentional issues) secondary to nutritional deficiency, and can occur in a range of disorders where nutrient intake is poor (i.e., cancer, gastric surgery, anorexia nervosa) [62]. Not everyone presents with all three sets of symptoms (the "classic triad"), and confusion is the most common presenting problem. This can lead to under-diagnosis of the condition which is undesirable because if left untreated or inadequately managed, it can progress to Wernicke-Korsakoffs syndrome (WKS) [63]. Korsakoffs syndrome is characterised by severe and permanent memory impairment and confabulation (fabricated or misinterpreted memories and disordered speech) [64]. It is therefore crucial that people who drink alcohol heavily are treated with thiamine as early as possible to prevent progression to WKS, and prevent irreversible disability [62].



RESIDUAL AND LONG-TERM EFFECTS OF ALCOHOL

The most common symptoms of alcohol-related CI include problems with executive functioning, visuospatial, attention, processing speed and short-term memory, while general intellectual ability (IQ), episodic and procedural memory and verball skills remain largely intact [27,65]. These cognitive deficits have the potential to undermine AOD treatment engagement and outcomes [39, 66]. A description of how these impairments might maninfest is provided in **Table 5**.

Table 5. Alcohol-related CI

COGNITIVE DOMAIN	EVIDENCE OF IMPAIRMENT?	WHAT DOES IT LOOK LIKE IN REAL LIFE?
Executive Functioning	Marked deficits in decision-making, working memory, inhibition, cognitive flexibility, goal-directed behaviour [27, 39, 66]	Disinhibited or inappropriate behaviour Impulsive Poor decision making Problems doing more than one thing at a time Lack of insight of CI
Memory	Evidence of deficits in verbal, prospective, episodic memory [65]. Difficulties learning new information [27] with this deficit most pronounced in people with WKS [62]	Difficulty recalling recently-learned (new information) without prompts Poor chronological time tagging, and unable to give specific dates Individuals with WKS will have anterograde memory deficits (i.e., problems learning new information) but memories of the past, especially the distant past are largely intact Individuals with WKS can confabulate (fill in the gaps in their memory)
Attention	Deficits in selective and sustained attention [27]	Difficulty paying attention to a task over an extended period of time and difficulty ignoring distractors
Processing Speed	Reduced performance on simple reaction time tasks [27, 65, 66]	They may be slower to take in information, or respond, or complete tasks
Visuo-spatial functioning	Marked deficits in visuospatial skills [27, 67], processing, memory, visual learning and visuospatial organization	Changes in handwriting, difficulty assembling things (e.g., flat pack furniture) and navigational problems (e.g., finding a new location using a map)
IQ	So called 'crystallised abilities' such as vocabulary (verbal skills), over-learned motor skills, implicit/procedural memory processes and general IQ remain largely intact	

INFLUENCING FACTORS

The nature and extent of alcohol-related CI depends on the amount, duration and frequency of use, as well as age of use onset, health (mental and physical), nutrition and environmental/socio-demographic factors (i.e., housing instability) [49, 68]. Alcohol has been shown to have a 'dose-dependent' effect on cognition, whereby a higher 'dose' and greater duration of time spent drinking is associated with greater impairment compared to abstinence or very light drinking. However, there is little consensus on the precise level or duration of alcohol use necessary to cause cognitive decline [69]. Recent research indicates that in older adults, lower levels of drinking are associated with higher rates of CI compared to other age-groups [58, 69, 70]. A longitudinal study of older adults found that heavy drinking (>30 units a week) increased the risk of hippocampal atrophy (loss of neurons in the hippocampus) 6-fold, and moderate drinking (14-21 units per week) 3-fold relative to abstainers [58]. In terms of biological sex, there is evidence that females, because of their metabolic and physiological make-up, may be more vulnerable to the neurotoxic (brain cell-death) effects of alcohol and resulting CI [71].

COGNITIVE RECOVERY

AOD clients with insight into their CI may be concerned about permanent damage. Educating clients that CI is at least partially reversible with abstinence may help build motivation for behaviour change and encourage treatment engagement and retention. Although some cognitive recovery occurs after cessation of drinking (time-dependent recovery), the speed and degree of recovery varies across cognitive domains and individuals. Research suggests there is some recovery of CI within days [72] or weeks following abstinence [73] (particularly in attention and verbal memory) while other studies suggest that it may take more than 6-months for recovery to manifest [74]. However, there is a general consensus that executive functioning, problem solving, processing speed, visuospatial ability and perceptual motor deficits require a year or more of abstinence to recover [39]. A recent meta-analysis that synthesised the findings of multiple studies concluded that only attention shows recovery in the intermediate term (2-12 months), with all other domains showing persistent and significant deficits one-year post-abstinence [39]. Whilst complete cessation of alcohol is the best way of achieving improved cognitive functioning, the benefits of reducing alcohol use (i.e., harm reduction) should not be downplayed [75].

Supporting neuropsychological research on cognitive recovery, brain imaging studies show increased brain volumes with abstinence, potentially due to white matter regenerating [49, 76]. Individual rates of recovery depend on a number of factors including age, (with younger people experiencing greater improvements in a smaller time frame), severity of dependence/history, nutrition, health, and environmental factors. When interpreting the literature it is important to note that clients who are able to maintain abstinence over the long term may have had better cognitive functuining, pre-abstinence than those who continue to drink at harmful levels.

WHEN TO REFER FOR NEUROPSYCHOLOGICAL ASSESSMENT?

Neuropsychological assessment should ideally occur after an extended period (at least four weeks) of abstinence, as CI can be confused with the temporary effects of intoxication and withdrawal. For clients who are unable to maintain extended abstinence, a neuropsychological assessment can still provide an indication of an individual's cognitive strengths and weaknesses, but will not provide a reliable diagnosis (which might be needed to secure social and financial support). **See Appendix E** for information on neuropsychological assessment services located in Victoria.

Case Study: Alcohol Related Brain Injury

This case study illustrates some of the issues facing older people with an AOD history, and the role of seeking supports to improve cognitive functioning. In addition, it provides some examples of the typical features seen in Alcohol Related Brain Injury (ARBI).

Isabelle is a 60-year-old woman with a 15-year history of drinking approximately 1-2 bottles of wine per night. She has gone through multiple detoxifications – some supervised and some unsupervised. She resides in a supported accommodation service (SRS), and regularly has altercations with staff about her drinking – placing her at risk of losing her accommodation. Since moving to the SRS she had been eating regularly and taking her medication and her brother Tom reported that her cognition had improved somewhat as had her daily living skills.

Isabelle's brother accompanied her to the assessment. She was appropriately dressed for the weather, although her top appeared slightly stained. She was wearing glasses. Isabelle was friendly and polite throughout the assessment, maintained good eye contact and displayed appropriate conversational turn-taking behaviour. Some dressing difficulties were observed during the assessment, where she was unable to put on a jumper without assistance. She was impulsive at times, attempting to commence tasks before having been given complete instructions. Isabelle repeated a story during the assessment, and required a number of questions and instructions to be repeated. She reported memory complaints and feelings of confusion, and reported regularly leaving personal belongings on public transport. She reported no other cognitive complaints.

Her history was disorganised and contradictory at times; she appeared to have particular difficulty with time lines, reporting many events in her life as taking place 12 months ago. Isabelle appeared to have limited insight to her difficulties, describing herself as having a photographic memory. On the morning of the assessment Isabelle reported taking two diazepam tablets, but drinking no alcohol. The night before the assessment she reported taking two diazepam tablets and drinking two glasses of wine; she did not appear to be intoxicated.

On assessment Isabelle had marked impairments in her executive skills, visuo-spatial skills and her attention, while her memory was intact. Given her history of heavy alcohol consumption, functional impairments and impaired neuropsychological profile, a diagnosis of Alcohol Related Brain Injury was made. It was recommended that she be supported to access social and community based activities to occupy her time. She would also likely benefit from structure and routine in her day which could be displayed on a whiteboard at home to minimise confusion and help her keep track of things. It was also recommended she regularly attend her GP and specialist appointments to ensure her medical needs are well managed.

DRUG RELATED COGNITIVE IMPAIRMENT

Heavy use of drugs besides alcohol can result in short or long term CI, although the extent and duration of these deficits are not as well understood. One factor complicating the analysis of specific drug associated impairments is the high incidence of co-morbidity and polysubstance use. However, excluding participants with comorbidity/polysubstance use in studies leads to an inflated estimate of cognitive performance, which means that studied samples are not always representative of treatment seekers. The following sections outline the CIs associated with the use of specific drugs. It is noted that this material is intended as a guide, and individual differences in premorbid abilities, history or presence of trauma or PTSD, mental health diagnoses and other disorders known to affect brain function will influence a client's presentation.

This section details the cognitive effects of:

- **Cannabis**
- **Stimulants**
- **Opioids & Opioid Replacement Therapies**
- **Benzodiazepines**
- **Novel Psychoactive Substances &**
- **Other drugs**

CANNABIS RELATED COGNITIVE IMPAIRMENT

Cannabis is the most commonly used illicit substance [77]. In 2019, 36% of Australians had reported use of cannabis in their lifetime, with 11.6% having used in the previous 12 months [77]. Cannabis is made up of over 70 compounds known collectively as 'cannabinoids'. The potency of cannabis varies according to the strain (chemical composition), gender, growing environment and age of the plant [78, 79]. The primary cannabinoids are delta-9-tetrahydrocannabinol (THC) and cannabidiol (CBD), and they appear to exert different, and at times opposing effects on the brain **(see Table 6)**.

Table 6. Common THC & CBD Effects

FUNCTION	THC	CBD
Learning	↓	↑
Anxiety	↑	↓
Psychotic symptoms	↑	↓
Brain hippocampal volume changes	↓	–

THC use is responsible for the euphoric 'high' that cannabis users experience; however it is also associated with many of the negative cognitive and psychological effects of cannabis (i.e., anxiety, paranoia and even psychosis). CBD on the other hand exerts a pain-relieving and calming effect [80, 81], leading to its use in self-medication and formal medicinal use (it is the primary component of so-called 'medicinal cannabis'). Recently, CBD has been recognised as being neuroprotective in that it helps preserve neuronal structure and function and protects against oxidative stress (the breakdown of brain cells) [82].

The ratios of THC and CBD in recreational cannabis has changed over recent decades, with THC increasing and CBD decreasing over time (leading to a higher incidence of adverse effects) [83]. In addition, there are significant variations in the potency of these across studies [84], which can make it difficult to generalise earlier research findings to those seeking treatment for cannabis use disorders today.

CANNABIS AND MENTAL HEALTH

PSYCHOSIS

Cannabis use is associated with an increased risk of developing psychosis or a psychotic disorder, with age of onset, frequency of use and the potency of cannabis used influencing this risk [85]. For example, one European multicentre study found that daily cannabis users were 3.2 times more likely to develop a psychotic disorder compared to non-users, and this increased to 4.8 among those using high potency cannabis (i.e. THC › 10%) [85]. Cannabis (specifically THC) use can also exacerbate existing psychotic disorders, by increasing the incidence of 'positive' symptoms (i.e., manic symptoms in bipolar disorder, paranoia, hallucinations, delusions) and heightening the risk of relapse to substance use [86, 87]. Since schizophrenia and other psychotic disorders are associated with moderate to severe CI, it is important to screen for their presence when assessing cognition in people with cannabis use disorder [88].

AFFECTIVE AND ANXIETY DISORDERS

There is a high prevalence of depression and anxiety disorders among cannabis users. An Australian population study indicated that among those with cannabis dependence, the prevalence of affective disorders was 14%, compared to 6% in non-users [89]. Similarly, the rate of anxiety disorders was 17% compared to 6% in non-users. However much of this increased prevalence was likely due to demographic and other substance use [89]. These findings highlight the importance of a patient-centred approach in managing substance use and mental health issues. Since moderate cognitive deficits in executive function, memory and attention often exist in people with depression, cognitive assessment must consider the impact of co-morbid affective disorders when assessing cognition in people who use cannabis regularly [90].

As with most substances, the impact of cannabis on cognition during periods of acute intoxication may differ from the residual or long-term effects.

EFFECTS OF CANNABIS ON COGNITION

Acute cannabis intoxication has been associated with widespread alterations in cognitive functioning, most notably psychomotor function (movement, co-ordination, etc.), attention and concentration, speed of information processing, learning and memory (primarily episodic memory), for examples of behavioural manifestations, see **Table 2**. In comparison, we know far less about the residual effects of cannabis use (i.e., those that linger post cessation, with research assessing cognition from as little as 7 hours after last use, and up to 20 days after last use [91, 92]. Nonetheless the residual impairments most consistently observed occur in verbal learning and memory (e.g., difficulty recalling information discussed in previous treatment sessions) [91-93]. This is because the hippocampus and prefrontal cortex (largely responsible for learning and memory) has a high density of cannabinoid receptors and is therefore particularly vulnerable to THC exposure (primarily through reductions in brain volume) [92-94]. Whilst deficits have also been detected in executive functioning (working memory) [93], the long-term effects of cannabis on inhibition and decision-making are less clear. Whilst some cognitive deficits are often reversed after the first month of abstinence [95, 96], there is some research evidence suggesting that impairment in verbal memory, attention, and some executive functions (e.g., cognitive flexibility) may persist after prolonged abstinence [91, 97].

INFLUENCING FACTORS

Research suggests that an earlier age of onset of cannabis use is associated with poorer cognitive performance, and that regular/heavy (daily or almost daily) cannabis use during adolescence can have significant negative lasting impacts on cognition [98-100]. This is consistent with the 'brain maturation hypothesis', which suggests that consumption of drugs during critical periods of brain development interrupts normal development, causing long term changes to neural circuitry and cognitive function [101]. As the endocannabinoid system plays an important role in brain development, it is plausible that prolonged use during adolescence results in a disruption in the neural growth that occurs during this period [102]. Other factors effecting the extent of CI are the amount, frequency, dose, potency and duration of cannabis use [93]. Recent research suggests that genetic and environmental factors (e.g., adverse chilhood events) may play a more dominant role [103]. In summary, the current evidence suggests that declines in cognitive functioning are most likely to manifest among daily (or near daily) users among those who have been using for an extended period (i.e., more than one year) and that the magnitude of these declines are relatively modest, compared to alcohol for example.

COGNITIVE RECOVERY

Most of the CI associated with cannabis appears to dissipate within a few weeks of abstinence [104], although the extent of recovery may be more modest among heavier users and when cannabis has higher THC to CBD ratios [105]. However, some studies have detected CI on specific domains even after a month of abstinence, though again this tends to be in heavy users [106].

MOST CI RECOVERS WITHIN 1-YEAR OF ABSTINENCE, THOUGH SOME RESIDUAL DEFICITS IN EXECUTIVE FUNCTIONING MAY PERSIST BEYOND THAT TIME

STIMULANT RELATED COGNITIVE IMPAIRMENT

Illicit stimulants include compounds such as amphetamine ('speed'), methamphetamine ('ice'), 3,4-methylene dioxymethamphetamine (MDMA, 'ecstasy'), and cocaine [77]. Cocaine is the second most commonly used illicit drug in Australia, with 4.2% of individuals reporting use in the previous 12 months [77]. Use of meth/amphetamines has fallen over recent years, with 1.3% of individuals reporting use in the previous 12 months [77]. However, individuals who use methamphetamine tend to use the drug frequently (i.e. 17% use it weekly) [77], and this can lead to more pronounced CI. Adverse side-effects of long-term heavy stimulant use or high levels of intoxication, whilst rare can include stroke, cardiac problems and neurodegenerative conditions which are all associated with CI.

STROKE

Stimulant use is associated with an increased risk of stroke. The risk of haemorrhagic stroke (a bleed in the brain) is 5-times greater in users of meth/amphetamines compared to healthy controls, and 2.5 times greater in cocaine users; whilst ischaemic stroke (a blocked artery) risk is 2 times greater in cocaine users [107]. Methamphetamine increases the risk of stroke among people under 45 years [108]. Long-term health outcomes following stroke are also impacted by stimulant use; for example, methamphetamine use is associated with higher mortality rates and poorer health outcomes 1- and 3-years post-discharge from hospital [108].

CARDIAC ISSUES

Long-term stimulant use is significantly associated with an increased risk of acute cardiac events, including acute coronary syndrome, acute myocardial infarction (heart attack), acute aortic dissection and sudden cardiac death, hypertension and heart failure [109]. Whilst rates of CI are highly variable among survivors of cardiac arrest, memory problems are most common, followed by impairments in attention and executive functioning [110].

NEURODEGENERATIVE DISEASE

Limited findings suggest a potentially increased risk of developing Parkinson's Disease (PD) in persons using methamphetamine; specifically, enduring psychomotor deficits and dopaminergic dysfunction due to long-term use are associated with a higher rate of PD [111]. Epidemiological studies have also identified a significantly younger age of PD onset in people who use methamphetamine when compared with population norms [112].

METHAMPHETAMINE RELATED COGNITIVE IMPAIRMENT

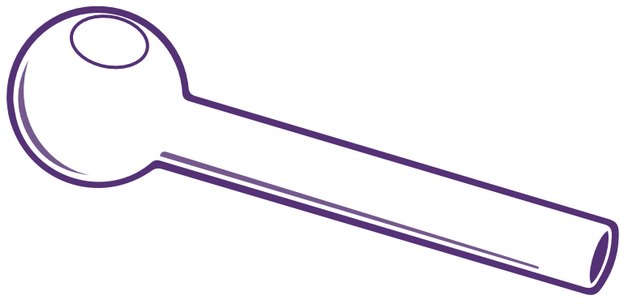
When taken in low doses, stimulants can actually enhance some cognitive functions (e.g., attention) [113]. However among people presenting to treatment for methamphetamine problems, a decline in cognitive functioning is common. Evidence from meta-analyses* of all the studies points towards reduced neurocognitive performance across most cognitive domains including [114, 115];

- Severe deficits in impulsivity and reward processing (delay discounting**)
- Moderate deficits in attention, shifting cognitive set (mental flexibility), verbal fluency, verbal learning and memory visual memory and working memory
- Modest deficits in speed of processing and visuospatial abilities.
- Deficits in social cognition (facial emotion recognition);

Methamphetamine dependence has been associated with self-reported deficits in everyday functioning [116]. Methamphetamine use appears to have an adverse impact on behavioural control – characterised by agitation, irritability, impulsivity, reduced mental flexibility, and impaired decision making [114, 117]. Indeed, the high rates of treatment drop-out, relapse, and other high-risk behaviours and related adverse outcomes observed with this population may reflect impaired cognition. Many of these cognitive functions are important for recovery from methamphetamine use disorder. High impulsivity and poor cognitive control can exacerbate drug-seeking behaviours e.g., by making it harder to resist urges to use [114]. However, even small deficits may hinder a client's ability to follow instructions for self-care, or adhere to treatment plans. Nonetheless, it is important to keep in mind that these deficits may precede the onset of methamphetamine use and have accelerated the progression to methamphetamine use disorder [118].

COGNITIVE RECOVERY

Many of the CIs (mental flexibility, attention, processing speed, verbal memory, fine motor functioning, and verbal fluency) associated with methamphetamine use appear to improve following a period of abstinence [119], sometimes after as little as one-month [21, 120, 121]. Individuals assessed after 6 months of continuous abstinence demonstrate significant improvements in areas of verbal memory, problem solving and social emotion processing, that are broadly equivalent to healthy controls [122]. In contrast however, one study indicated that poor cognitive control could be detected after 18 months continuous abstinence, despite unimpaired set shifting, attention and working memory, suggesting that some executive functions take longer than others to recover. However, often these persistent deficits are within a range of normal variation and unlikely to reflect functional difficulties in day-to-day life [123].



*A combined effect size from multiple independent studies.

** A preference for smaller sooner rewards over larger later rewards.

COCAINE RELATED COGNITIVE IMPAIRMENTS

Cocaine use disorder is associated with deficits in attention, working memory, verbal learning, memory and impulsivity [114]. CI has also been observed across those meeting criteria for cocaine dependence [114], as well as non-dependent recreational users [124]. One study suggested that an early age of onset of cocaine use (<18 years) is associated with more pronounced cognitive deficits in working memory (and general executive functioning), sustained attention, and declarative memory [125].

COGNITIVE RECOVERY

Cognitive functioning appears to improve with prolonged abstinence. One longitudinal study examined cognitive functioning of cocaine users and healthy controls at baseline and at one year follow-up [124]. Users who were at least 6-months abstinent at follow-up showed significant improvements in the domains of attention and memory, with test scores equivalent to the control group. Nonetheless, others have reported a broadly similar cognitive performance to non-using healthy controls among those substantially reducing their cocaine use (by around 75%) [126].

ECSTASY RELATED COGNITIVE IMPAIRMENTS

'Ecstasy' is a colloquial term commonly used to refer to substances containing illicit ±3,4-methylenedioxymethamphetamine (MDMA). MDMA causes the brain to release serotonin, and to a lesser extent dopamine and noradrenaline [127]. MDMA is known as an 'entactogen', a pro-social psychoactive substance that can induce feelings of euphoria, empathy and increased self-awareness [128]. In 2019, approximately 3% of Australians reported using ecstasy [77]. Repeated use can cause tolerance, and use of the drug is self-limiting, in that some individuals decrease use due to decreased positive effects [129]. There are a number of significant health risks associated with the use of ecstasy, which can include a mild or temporary decline in cognitive performance. Due to its primary action on serotonergic neurons, ecstasy is associated with an extended period of residual effects on anxiety, depression and sleep, known as a 'come down' [129].

RESIDUAL OR SUBACUTE COGNITIVE EFFECTS

Studies examining the subacute effects of ecstasy use (5-25 days following consumption) have highlighted that users may experience difficulties in learning and memory [130, 131]. Several studies also point to poorer performance relative to controls in executive functioning [132], including planning, cognitive inhibition, and cognitive flexibility [133-135]. As most ecstasy users engage in poly-substance use (i.e., combine it with alcohol, other illicit drugs, cigarettes) poly-drug use could account for some of the inconsistent findings in the literature.

COGNITIVE RECOVERY

With continued abstinence, limited neurocognitive effects have been noted in the literature. For example, Halpern et al (2011) found little evidence of decreased cognitive performance in ecstasy users (who did not use any other drugs) who were abstinent for at least 60 days [136].

OPIOID RELATED COGNITIVE IMPAIRMENT

Opioids refer to substances (both synthetic and plant-based) derived from opium, whether licit (i.e., morphine, buprenorphine and codeine) or illicit (i.e., heroin). Less than 0.1% of Australians aged over 14 years reported heroin use in 2019, with 4.2% reporting having misused a pharmaceutical drug [77]. Prescription opioid dependence is becoming increasingly prevalent throughout Australia. In 2016, pharmaceuticals were the principal drug of concern in 5% of all AOD treatment episodes [46].

ACUTE USE AND INTOXICATION

Opioids have a sedating effect which is magnified when combined with other sedating drugs (i.e., alcohol, diazepam). Verbal working memory, flexibility and impulsivity are generally poorer among those using heroin compared to healthy controls [137]. However, it is important to keep in mind that many studies fail to account for differences in pre-morbid IQ, age, education etc in their comparison between those using opioids and healthy controls [137].

LONG TERM EFFECTS

Chronic long-term opioid use may be associated with difficulties in attention, speed of information processing, memory, executive functioning, inhibition and decision-making in a dose-dependent matter [138-142]. Illicit and prescription opiates (particularly high purity opioids like fentanyl, morphine and oxycodone) carry a high risk of overdose [143]. During overdose opioid-induced respiratory depression can cause respiratory arrest, and even coma or death. Prolonged respiratory depression can cause hypoxia and result in brain injury [143].

COGNITIVE RECOVERY

After one year of abstinence from heroin, most cognitive functions are comparable to healthy controls, suggesting that cognition is restored [41]. Indeed, an Australian study found that residents of a therapeutic community who had stopped using opioids for at least 3-months had equivalent cognitive performance to people who had never used opioids [142]. This was the case across all domains assessed, and included cognitive inhibition, reasoning, mental flexability, verbal memory and visuospatial functioning [142].

OPIOID REPLACEMENT THERAPY: METHADONE & BUPRENORPHINE

There is often a concern among patients that the sedating effects of agonist/partial-agnonist medications prescribed for the management of opioid use disorders (i.e., opioid replacement therapies such methadone and buprenorphine) may impair cognitive performance. A recent meta-analysis found methadone users and users with less than 12-months abstinence displayed global impairments, with poorer psychomotor speed, attention, executive functioning and memory relative to healthy controls [144], albeit less pronounced impairment than among people with active heroin dependence [145].

OTHER DRUGS

KETAMINE

Ketamine is a dissociative anaesthetic with hallucinogenic properties, and is used in clinical settings for pain management and specialist anaesthesia (i.e., paediatrics, veterinary procedures and field medicine) [146]. In terms of cognitive effects, infrequent or recreational ketamine use does not appear to be associated with long term CI [147-149]. In the short term, following heavy use, impairments in memory (including difficulties in encoding, retrieval and semantic processing), working memory and planning have been detected [148]. Frequent ketamine use over a 12-month period has been associated with impairments in visual recognition and spatial working memory, with greater impairments among more frequent users [149]. However, the cognitive performance after periods of abstinence (matching that of healthy controls) suggests CIs may be reversible [148, 149].

GHB

GHB (gamma-hydroxybutyrate), is a central nervous system depressant with biphasic effects (low and high doses produce opposite effects) characterised by initial stimulant-like and euphoric effects at low doses, and sedative effects at higher doses. The difference between a high and low dose is measured in just millilitres, heightening the risk of overdose/ adverse effects. High doses can cause sudden unconsciousness ('blow out', a short lasting coma), followed by sudden awakening [150]. Emerging evidence shows that acute use of GHB is associated with CI in working memory, learning and memory [151]. Acute psychomotor impairments have also been reported in the literature, though these rarely persist beyond 3 hours post ingestion [152]. Concerns regarding long-lasting neurocognitive effects in association with repeated coma have been raised, as a result of cerebral hypoxia [153].

INHALANTS & SOLVENTS

Inhalant or solvent use (also known as volatile substance abuse), colloquially known as chroming, huffing, bagging or sniffing, is particularly common among minority and disadvantaged groups [154]. Common substances inhaled include volatile solvents (e.g. glues, paint thinners or petrol), aerosols (e.g. hairsprays, or spray paints) and gasses (e.g. propane, butane, nitrous oxide). The cognitive effects of solvent use depend on the dosage, type of chemical used, and degree of inhalation over time [155]. Toluene (present in spray paints, paint thinners and petrol) has significant abuse potential and is associated with a range of impairments in verbal skills, speed of information processing, attention, learning, memory, psychomotor coordination and some aspects of executive functioning [156]. Chronic inhalant use has additional health risks with potential for significant damage to neurological, renal, hepatic and pulmonary systems with persistent effects noted [155]. In terms of recovery, some impairments abate in the weeks following abstinence, with memory and executive function requiring months to years of abstinence to fully recover [157].

BENZODIAZEPINES

Licit Benzodiazepines (i.e., Alprazolam, Clonazepam, Diazepam, Temazepam, Oxazepam) are the most frequently prescribed class of psychoactive pharmaceutical drug in the world [158]. They are minor tranquilisers with sedative effects, typically prescribed to manage seizures, anxiety, withdrawal, sleep disturbances, and spasticity. However, these drugs pose significant risks when used long-term (i.e., for longer than a couple months) [158]. The cognitive effects of benzodiazepines are significant. In the short term, sedative effects are evident (i.e., decreased concentration, reduced psychomotor and memory performance) [159], and in the long-term, current users exhibit global CI, with deficits observed in working memory, processing speed, divided attention, visuospatial, memory and expressive language [160, 161]. These impairments appear to persist after withdrawal and even after several months of abstinence [161].

NITROUS OXIDE

Nitrous oxide (N_2O) is a legal gas typically used in the automotive and food-service industries (i.e., in whipped cream canisters), as well as clinically as a dissociative anaesthetic (for pain management in medical industries). Nitrous oxide used illicitly is often referred to as 'nangs' or 'laughing gas' and is currently popular in night club and festival scenes (inhaled directly from the canister or transferred into a balloon or other air-tight container). Inhalation of nitrous oxide produces an immediate and short term high (often seconds at most), therefore limiting its abuse potential. However, there is a subpopulation of users who consume nitrous oxide heavily, with side effects including hypoxia or asphyxia from lack of oxygen or vitamin B12 deficiency leading to peripheral neuropathy [162]. However, it is important to note that research on the cognitive effects of nitrous oxide to date has been limited to animal studies to date [163].

NOVEL PSYCHOACTIVE SUBSTANCES (NPS)

Novel psychoactive substances - termed 'legal highs' or 'designer drugs' - are a group of highly unregulated drugs synthesised with the aim of mimicking the effect of typical illicit drugs such as stimulants, hallucinogens, dissociative agents or cannabis. Compared to other drugs, the potential for NPS neurotoxicity is dramatically increased - each batch is produced from harmful synthetic materials, varying each time in terms of content, concentration, potency and effect, therefore increasing the risk of overdose - particularly if used in conjunction with other illicit substances [164, 165]. Common NPS substances are described below.

SYNTHETIC CANNABINOIDS

Synthetic cannabinoids ('spice', 'K2', 'kronic') are chemically and structurally different to THC, but also activate the endocannabinoid system [166]. Compared to THC, synthetic cannabinoids bind more strongly to receptors in the brain (they are full agonists) [167] and as such they have been associated with more negative effects and elevated health risks [168, 169]. Both users and ambulance data report increased adverse effects arising from synthetic cannabis intoxication, with symptoms including increased paranoia, suicide attempts and ideation, overdose, acute kidney and liver failure, seizures, psychosis, harmful effects on the lungs, and impairments in memory [169]. Compared to natural cannabis, synthetic cannabis has been shown to be more harmful in terms of executive functioning [170], however the long term clinical effects of synthetic cannabis on cognitive functioning are currently unknown.

SYNTHETIC CATHINONES

Most synthetic stimulants are derivatives of cathinone - the most common being mephadrone - colloquially known as 'bath salts' or 'meow-meow'. The long-term effects of synthetic cathinone use in humans are unestablished. A recent study in rats supplied with a binge-like regimen of synthetic cathinones over a 4-day period found reduced working memory performance in the rats 2-weeks later [171]. Substantially more research is necessary to determine the long-term effect of synthetic cathinones in humans.

SUMMARY

CI in the context of alcohol use disorders has been subjected to the most research to date, and hence is most established. While examination of the cognitive profiles associated with drugs other than alcohol are slowly emerging, it remains heavily confounded by limitations in study design, variable lengths of abstinence, poly-drug use and the failure to consider premorbid differences. The only way to understand the nature and extent of CI and what improvements are likely to occur over time, is through individual, comprehensive neuropsychological assessment.

ASSESSMENT AND DIAGNOSIS OF COGNITIVE IMPAIRMENT

NEUROPSYCHOLOGICAL ASSESSMENT

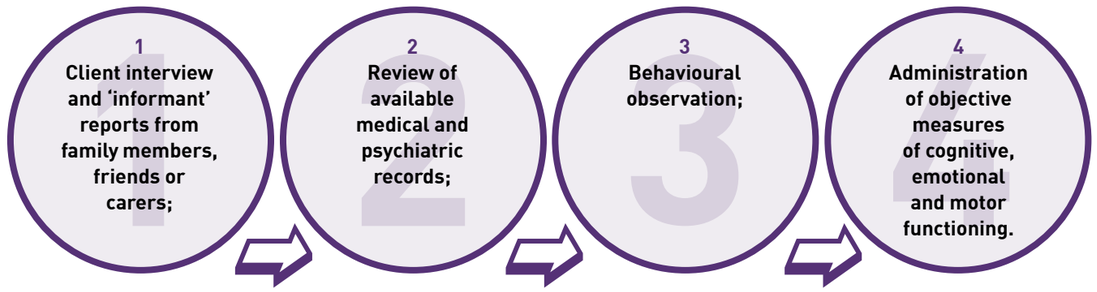
Clinical neuropsychologists are specialists in evaluating cognitive functioning and as such, neuropsychological assessment is considered the 'gold standard' means of evaluating a client's cognitive, emotional and behavioural functioning. In screening for CI, client interviews alone may not be sufficient to determine the extent or severity of CI. Clients are known to be poor reporters of their own cognitive functioning [172, 173], and clinicians who rely exclusively on informal unstructured clinical interviews without formal neuropsychological testing have been shown to severely under- or over-estimate the extent of their clients CI [174]. Neuropsychological testing thus serves an integral function in evaluating the extent and nature of any difficulties a client may experience.

ADVANTAGES OF NEUROPSYCHOLOGICAL ASSESSMENT

- Allows for detection, characterisation and longitudinal tracking of cognitive function (through repeat testing) with sensitivity and accuracy that exceeds self-report screening tools;
- Can determine whether a client meet the diagnostic criteria for clinical disorders;
- Used to guide neuro-rehabilitation programs;
- Used to track/monitor outcomes and response to treatment (medication, rehabilitation, surgery, etc.);
- Used to generate information regarding prognosis;
- Indicates the functional impact of damage observed from brain imaging;
- Provides recommendations on how the delivery of AOD treatment can be tailored to individual needs;
- Can distinguish true impairment from malingering/fictitious disorders (particularly important in justice system contexts) [175].

THE ASSESSMENT PROCESS

As part of an assessment, neuropsychologists will collate detailed information from:



The assessment process is undertaken over several hours or multiple days depending on the service. Assessments are completed using standardised age and sometimes gender and education normed assessment tools. Following the assessment, information will then be integrated and formulated to provide a diagnosis and prognosis (likelihood of improvement over time) where appropriate, in addition to a summary of the client's strengths and weaknesses and associated recommendations for further care and rehabilitation. These findings are conveyed through feedback sessions with clients, referring agencies, carers and supports (i.e., family and friends). A formal report is also provided to the referring agency and additional care providers if requested.

What Types of Referral Questions can be Answered?

Neuropsychologists can respond to a range of referrals questions including the following:

- Does the client have an ABI, TBI, a neurodegenerative disorder or an intellectual or learning difficulty?
- What is the clients level of functioning now and how will it change over time?
- What are the clients' strengths and weaknesses?
- How can care providers better support their client and help them compensate for their difficulties?

In the AOD sector, it is often difficult to provide diagnostic formulations when there is active - or a recent history of substance use, co-occurring mental health, or psychosocial difficulties. Many clients who are referred to neuropsychological assessment are not capable of maintaining a period of abstinence sufficient to provide an accurate diagnosis. However, assessment in these scenarios can still yield a summary and formulation of a client's strengths and weaknesses that can assist care providers in supporting their clients.

What Information is Provided in a Neuropsychological Report?

Neuropsychological reports vary according to referral questions and the setting in which they are completed. For instance, medico-legal reports are often highly detailed and focussed on specific referral questions relating to the matter before the court, whereas reports in rehabilitation settings focus more on providing recommendations and support for further care. The following information should be available from most neuropsychological reports:

- A summary of the presenting issue and referral question;
- A summary of the clients' history and presentation;
- A description of cognitive deficits identified on formal assessment;
- A description of preserved skills or relative strengths;
- When appropriate, a diagnosis or opinion regarding the aetiology of any difficulties;
- Recommendations for referrals / additional support and compensatory strategies for identified weaknesses.

When should neuropsychological assessment take place?

Ideally, assessment of neuropsychological functioning should take place after the client has abstained from substance use for several weeks, however in many instances this is unrealistic. Assessing cognitive functioning when the client is using alcohol/drugs is still worthwhile, as it will provide an strong indication of how they are functioning cognitively in their day to day life. Nonetheless, it is important to remind clients not to be drug-affected/intoxicated on the day of testing, ideally avoiding substance use in the 2-3 days prior to assessment.

Important Steps/Considerations before Making a Neuropsychological Referral

- Formulate a clear referral question; ✓
- Does the client understand why they are being referred and why the assessment is being done? ✓
- Do they know that they must be abstinent for the assessment? ✓
- Do they know what they must bring with them to the assessment? ✓
- Do they know how to get to the assessment and what time it is - are there reminders in place? ✓
- Is a support person attending the appointment with them? ✓
- Are the results part of a client's involvement with other agencies? Will a report need to be provided to that agency? ✓
- Who will be explaining results to support persons and other agencies? ✓
- Do you have an accurate history – have you determined whether a neuropsychological assessment was recently conducted? ✓

Clinicians may find it helpful to consider the clients everyday functioning (both strengths and limitations) when considering referral for neuropsychological assessment. You may like to use the 'Activities for Daily Living' (ADL) or 'Instrumental Activities of Daily Living' (IADLs) questionnaire, supplemented with the questions outlined in Table 7. It is recommended that clinicians complete Optional Module 2 of the current Victorian Assessment "ABI Referral Tool for Neuropsychology Assessment" see **https://www2.health.vic.gov.au/about/publications/formsandtemplates/Optional-AOD-modules-11-modules**

Table 7. Indicators of Functional Impairment

FUNCTIONAL DOMAIN	NORMAL FUNCTIONING	INDICATION OF POSSIBLE DEFICIT
Accommodation & domestic activities	☐ Stable accommodation into the near future ☐ Keeps accommodation clean ☐ Manages food preparation / shopping	☐ Fails to keep accommodation ☐ Disorganised/messy living arrangements ☐ Unable to cook or is unsafe in handling food ☐ Guardianship Order
Self-care	☐ Manages hygiene ☐ Manages adequate nutrition ☐ Wears appropriate clothing	☐ Body odour and unsuitable/dirty clothing ☐ Appears malnourished ☐ Toileting accidents

Community activities	☐ Manages appointments ☐ Can communicate by phone or letter ☐ Negotiates services (e.g., Centrelink)	☐ Difficulty with communication (e.g., understanding and/or expression) ☐ Requires assistance to access services
Finances	☐ Sufficient finances to pay rent/food/bills ☐ Manages own finances/banking	☐ In debt or unable to recall extent of finances/assets ☐ Has an Administration Order
Mobility/ Transport	☐ Mobile within the community ☐ Has driver's license	☐ Unable to reliably catch public transport / gets lost ☐ Multiple driving accidents
Social	☐ Maintains supportive relationships ☐ Appropriately assertive in getting needs met ☐ Mindful of the rights of others ☐ Avoids risky situations ☐ Practices safe sex	☐ Socially inappropriate behaviour ☐ Socially anxious ☐ Lacking cooperation/stubborn Verbal/physical aggression ☐ Ignores others' needs ☐ Puts self into unsafe circumstances
Occupational/ educational	☐ Employment status (full-time, part-time, casual?) ☐ Has difficulty keeping regular employment ☐ Has meaningful hobbies/activities	☐ Unable to obtain or keep employment ☐ Poor attendance record
Mental health	☐ Resilient in response to everyday stressors and pressures ☐ Mood is stable ☐ Compliant with prescribed medications	☐ Appears depressed ☐ Reports feelings of panic / anxiety / stress ☐ Irritable or rapid mood changes ☐ Unable to manage anger ☐ Overly concerned with bodily function/ health ☐ Symptoms of psychosis ☐ Becomes very elevated ☐ Delusional beliefs

COGNITIVE SCREENING

While in-depth neuropsychological assessment is the gold-standard for cognitive evaluation, in some circumstances brief cognitive screening tools – whether clinician administered or self-report - can be useful in acute or time limited settings, or where referral for in-depth assessment may not be feasible (i.e., the client may not be willing or able to undertake further assessment). In these contexts, brief screening tools can provide an overview of cognitive functioning and guide further decision-making/treatment [176]. **Appendix A** lists commonly used brief (15-20 minute long) cognitive screening tools, their cost, training requirements and suitability to AOD clients. When choosing a screening instrument, it is important to consider the validity, reliability, standardised sample (population norms), administration time, and the skills/training required to administer each screening tool, its appropriateness for certain cultural groups and those for whom English is a second language, and any stimulus materials needed (timer, paper, etc.).

Many formal measures, including self-reported measures of cognitive functioning, have user level restrictions, where an individual is required to be from a specific profession or hold an appropriate qualification prior to use. To determine user restrictions for particular measure refer to the test publisher.

WHICH SCREENING TOOLS SHOULD BE USED?

While the Mini-Mental State Examination (MMSE) [177] is widely used, it was originally developed as a test for dementia. It is not sensitive to the types of CI common in AOD treatment seekers, as it lacks items that assess executive functioning. Unlike the MMSE, the Montreal Cognitive Assessment (MoCA) [178] and the Addenbrooke's Cognitive Examination (ACE-R) [179] have at least some items screening for EF deficits, making them more appropriate to an AOD population. Both can be administered within 10-15 minutes and provide a total score of overall cognitive functioning with education-adjusted cut-offs for impairment. Importantly, both of these tools have been validated for use with AOD treatment populations including in Australian treatment settings [32, 180, 181]. When working with distinct groups, such as Indigenous clients or clients with criminal justice involvement, specific validated tools should be used such as the Kimberley Indigenous Cognitive Assessment (KICA), which has been specially developed for indigenous clients living in rural settings [182]. A recent systematic review of screening tools that have been validated in AOD populations identified the MOCA as the most studied tool, with consistent diagnostic classification accuracy [183]. Newer measures such as the Brief Executive Function Assessment Tool (BEAT) are promising, however they require further validation prior to widespread use [184].

WHEN SHOULD YOU SCREEN?

As with neuropsychological assessment, the optimal time for screening is after several weeks of abstinence. Due to acute fatigue, medical issues or medication use that may compromise performance, an active/unstable psychiatric condition or psychosis, AOD intoxication, sensory issues, illiteracy, severe stress or a severe depressive episode, or even shyness or low motivation can be misinterpreted as CI. As such, the treatment or elimination of these factors should ideally be attempted occur prior to screening, though in reality, this is often not feasible.

SCREENING TOOLS FOR CI MUST BE INTERPRETED IN THE CONTEXT OF OTHER FACTORS KNOWN TO INFLUENCE COGNITIVE FUNCTIONING

NEUROPSYCHOLOGICAL TEST BATTERIES

There are a range of validated screening batteries (collections of discreet neuropsychological tests) which take longer to administer, cost more, require more extensive training than a brief screening tool, and are limited to certain professionals (e.g., psychologists). These tools include the 'Behavioural Assessment of the Dysexecutive Syndrome' (BADS) [315], the 'Repeatable Battery for the Assessment of Neuropsychological Status' (RBANS) [316], and the 'Neuropsychological Assessment Battery – Screening Module' (NAB) [317]. The NAB and the RBANS have both been used with AOD treatment populations. These neuropsychological test batteries provide total scores with age and education-adjusted cut-offs for impairment and are often used as part of formal neuropsychological assessment.

COMMON SELF-REPORT MEASURES OF CI

As well as performance based screening tools, there are a number of self-report scales that assess subjective (and collateral) cognitive functioning. An example is the 'Frontal Systems Behavior Scale' (FrSBe) [318], a 46-item scale assessing several of the broad daily social and occupational functions mediated by the frontal lobes (apathy, disinhibition, executive dysfunction, working memory, planning, or awareness deficits). The scale includes a self-report and a collateral report (e.g., from a family member or friend). Both reports have shown adequate reliability indices, but the use of the collateral report is recommended when the patient's insight is questionable. Verdejo-Garcia (2006) examined the effects of different drugs on apathy, disinhibition and executive dysfunction by administering the FrSBe to 36 dependent poly-substance users [185]. While the FrSBe can therefore feasibly be used in AOD populations, it has not yet been specifically validated for use with an AOD treatment-seeking population.

When using self-report measures, it is important to consider your client's insight into their own CI, and potential validity and reliability issues. On their own, these measures are not diagnostic, but being quick and easy to administer, they can serve as a useful tool for identifying where neuropsychological assessment may be warranted.

BEHAVIOR RATING INVENTORY OF EXECUTIVE FUNCTION – ADULT VERSION (BRIEF-A)

The BRIEF-A [186] assesses the views of an adult's executive functioning/self-regulation in their everyday environment and was designed for people with mild CI [187]. It comprises 75 items that load on to nine independent theoretically and empirically derived clinical scales measuring inhibition, self-monitoring, planning/organisation, shifting, initiating, task monitoring, emotional control, working memory and organisation of materials. This paper and pencil test takes around 10-15 minutes to administer, and there is both a self-report and an informant report version available. The BRIEF-A has been shown to be the most sensitive measure of executive functioning in patients with substance use disorders [188]. The BRIEF-A has also been shown to be sensitive to changes in executive functioning following cognitive remediation, in an Australian therapeutic community [189].

COGNITIVE FAILURES QUESTIONNAIRE (CFQ)

The 'Cognitive Failures Questionnaire' (CFQ) [190] captures everyday functional cognitive difficulties (e.g. cognitive lapses, absent-mindedness, and memory slips). It is a 25-item scale with total scores ranging from 0-100, in which higher scores indicate greater problem severity (no cut-off for poor cognitive functioning). A study by Rast et al. (2009) [191] indicated that the CFQ items assess forgetfulness, distractibility and proneness to making cognitive slips and errors in everyday tasks. The CFQ has been used to assess cognition in recreational ecstasy users [192], and alcohol-dependent individuals in treatment but has not yet been specifically validated among AOD treatment-seekers [193].

CLIENT INTERVIEW & HISTORY TAKING

Taking a thorough 'history' (understanding your clients' 'life story') will help you understand your clients' complaints in context, including how they might have arisen, and how you can best assist. An appropriate history will inform future assessment and referral (notes should be provided to neuropsychologists if a referral is made). During the interview, elicit information regarding the client's family history, social circumstances, employment, medical history and wellbeing, keeping in mind that this will be influenced by the client's memory and insight. It may be useful to ask clients to self-monitor and record any problems they face in daily life for a while prior to screening (prospective screening) or complete a self-reported measure of cognitive functioning. The client may be able to provide developmental (school reports), medical (hospital, prior assessments) or forensic records they have which might help elucidate impairments.

You can use your organisation's intake tool or the Victorian AOD tool :
www2.health.vic.gov.au/about/publications/FormsAndTemplates/victorian-aod-intake-tool
and supplement it with responses to the questions listed below in **Table 9**. The following section will cover interview/history-taking techniques specific to people in AOD treatment.

If your client reports cognitive concerns, try asking the questions below to get a sense of the nature, extent and likely impact of any CI:

Table 9. Assessing Cognitive Concerns

DOMAIN	SPECIFIC QUESTIONS YOU CAN ASK
Description of the problem	What is/are the problem(s)? Can you give some examples of the problem?
Onset and duration of the problem	When did it/they start? Did the problem(s) begin suddenly or escalate gradually? How long has/have the problem(s) existed?
Variability of the problem	Have there been any improvements in functioning at any time? Have you noticed fluctuations? When does the problem flare up and when does it improve?
Emotions	Have you been having trouble regulating your emotions? Do you get angry about things you wouldn't get angry about before? 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?
Daily living skills	Do you have trouble with self-care, managing bills, cleaning, mobility? Do you get any help with these things? Can you give some examples?
Vocations/employment	What jobs have you done? Have you had trouble maintaining or gaining employment?
Social functioning	Have you been having issues with people around you at work or at home? (i.e., difficulties initiating, developing, or maintaining healthy relationships?)
Offending behaviour	Effect on offending behaviour: Do you have trouble stopping yourself from doing things that are illegal?

COLLABORATIVE HISTORY TAKING & PSYCHOSOCIAL HISTORY

Occasionally a family member or significant other may be present at the interview (with the clients' consent), and this can help corroborate, supplement or clarify any information supplied. However, collateral informants may have derived their information from the client, may have limited exposure to the client during times when their impairments may be most noticeable (during times of high-cognitive load), may have a motive to under-represent their deficits (i.e., to keep them working), or over-represent deficits (particularly in forensic contexts, or to seek compensation), or may simply be in denial about the extent of their loved ones' impairments. Assessing the impact of cognitive deficits may be further complicated by ongoing substance use. It is important to remember when conducting an interview that many clients can present as relatively unimpaired (language functions are often preserved, as are autobiographical memories as are well-learned skills such as driving, activities of daily living, knowledge of facts about the world). Therefore, it is important to focus on the client's objective symptoms and complaints (i.e., occupational difficulties, breakdown in relationships). Some clients will not report noticeable problems in daily life, but might still report significant distress, whilst others will exhibit significant problems or impairments but lack awareness of them.

CLIENT SUBSTANCE USE, DEPENDENCE AND TREATMENT HISTORY

One of the most useful places to start is getting a full substance use and dependence history e.g., using the 'Victorian AOD comprehensive assessment form' available online via the health.vic website (see sample below).

In addition to information about an individual's substance use, the planning of ongoing treatment is informed by knowledge of what treatment has been attempted in the past, and in particular the reasons for any successes or setbacks.

- **Withdrawal history:** Date, setting (outpatient, home based withdrawal, inpatient), substances, any complications with withdrawal (i.e. seizures, behavioural dysregulation, absconding), medications taken, 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 treatment/courses conducted (including 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 (medical officer), setting (e.g. community pharmacotherapy, specialist clinic) and how effectiuve it has been.
- **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?

More information about recognising signs of dependence, as well as identifying and managing withdrawal, intoxication and overdose can be found in: Manning, V., Arunogiri, S., Frei, M., Ridley, K., Mroz, K., Campbell, S., Lubman, D. (2018). Alcohol and other Drug Withdrawal: Practice Guidelines, 3rd ed. Richmond, Victoria: Turning Point (freely available online).

PRESCRIPTION MEDICATIONS

Some medications can have side effects that impact cognition, cause fatigue or reduce alertness. It is not uncommon for AOD clients to be prescribed multiple medications that in combination may have additive and/or sedating effects. Clients may have several providers who are unaware of one another's prescribing. Sometimes, prescription medications may be overprescribed or prescribed for too long. For clients who frequently move around, or who have been involved in the justice system, the lack of a regular GP who they trust and feel comfortable with may be a complicating factor. If a client is taking multiple medications and you are concerned that this may be impacting them negatively, then consider asking their GP for a medication review. Referral to an addiction medicine specialist may be useful for some clients to gain a complete picture of their prescription medication use. Distinguishing between 'therapeutic' and illicit use of prescription medications can be difficult, although common indicators are having multiple prescribers ('doctor shopping') or pharmacies, maintaining secrecy around relationships with prescribers, avoiding discussions of medication effects and reasons for the prescription as well as hoarding/stockpiling medications **(see case study on page 53)**.

SAFESCRIPT

Safescript is Victoria's real-time prescription program for high risk medicines including opioids such as codeine, methadone; benzodiazepines such as alprazolam, diazepam; "z"-class sleeping tablets such as zolpidem; stimulants such as dexamphetamine; and other high risk medications such as ketamine and quetiapine. Information is contained on a centralised database which can be accessed by doctors and pharmacists during a consultation. It is a clinical tool which assists GP's and pharmacists in making safer decisions about whether to prescribe or dispense a medicine. It facilitates the early identification, treatment and support for patients who are developing signs of dependence. For more information visit **www2.health.vic.gov.au/public-health/drugs-and-poisons/safescript.**

When considering CI it is advisable to get a full understanding of your clients presciption medicine use, e.g. using the 'Victorian AOD comprehensive assessment form' prescription medication module (page 12), available online via the health.vic website **(see case study on page 52)**.

Case Study: Potential Prescription Opioid Abuse

Joshua – a 39-year-old Caucasian male – attended his appointment slightly late having lost his way. He was well groomed and neatly dressed.

He was alert throughout the appointment although slightly fatigued towards the end of the session. He tolerated the session with a short break for lunch, and returned on time. Joshua had good eye contact and his speech was clear and fluent. His mood appeared slightly anxious with restricted affect initially, but settled as session progressed, becoming more reactive, and he smiled and laughed appropriately. He rated his mood as a 5/10. He was a somewhat vague historian. In terms of cognitive complaints, Joshua reported that he forgot things frequently and often-lost items (phone, wallet, bankcards). He reported that he often gets frustrated over little things and feels like his thoughts are "all over the place" and that he "can't think."

After a short time, Joshua reported currently using prescribed oxycodone and codeine on a daily basis "to manage pain" in his shoulder sustained after falling while jumping to catch a Frisbee. Joshua stated that did not have a regular doctor, and instead attended bulk-billing centres near his home and work. When asked about his oxycodone dosage, Joshua became withdrawn and avoided the question.

Joshua was referred to detoxification where he went through supervised withdrawal and received a medication review to help him deal with his pain.

DEVELOPMENTAL HISTORY

Assessing childhood developmental history is one way to understand whether your client may have an undiagnosed learning disability, language disorder, intellectual disability or Attention Deficit Hyperactivity Disorder (ADHD). Many clients have elements in their history that could indicate a neurodevelopmental cognition which could still be affecting their cognition. The questions in **Table 10** can help uncover potentially undiagnosed developmental disorders.

Table 10. Assessing Developmental History

DOMAIN	SPECIFIC QUESTIONS YOU CAN ASK
Pregnancy	Did your mother use/have an AOD use disorder, or go through withdrawal while pregnant?
Birth	Were you born prematurely (early)? Where you underweight at birth?
Motor Milestones	At what age did you start walking? Did you have any trouble learning to walk? If yes, did you undertake occupational therapy or physiotherapy?
Speech and Language Milestones	At what age did you start talking? Did you experience difficulties communicating or require speech therapy?
Social Skills	Did you form strong connections in school? Were you bullied?
Literacy and Numeracy	Did you have any problems with reading, writing or mathematics? Did you repeat any grades/years at school?
Early Behavioural Problems	Did you need to see anyone for help with your emotions or behaviours? Were you very shy? Were you able to concentrate in school?
Educational Attainment	When did you leave school and why? What was your favourite subject? Did you complete/attempt further education after secondary school?
Family history of learning difficulties	Did anyone else in your immediate and extended family have similar problems?

Tips for assessing clients who may have neurodevelopmental disorders:

- **Learning disabilities:** Indicators of a learning disability include being behind relative to peers in literacy or numeracy, or attending special classes or specific interventions at school. Explore how comfortable the client is with reading now (e.g., can they read consent forms or appointment letters?).
- **Language difficulties:** If the client was slow to begin speaking their first-language as a child, find out if they still experience problems in speaking their first-language – this can be in either expressive language (how they talk) or receptive language (their ability to understand spoken language). A referral to a speech therapist may be useful.
- **Intellectual disability:** This is typically diagnosed in childhood and if so there ought to be existing assessments available to consult. Some clients do, however, 'fall through the cracks' and may not have previously accessed services. If an intellectual disability is suspected, formal assessment by a psychologist or neuropsychologist is required where the client's intellectual functioning will be reviewed in addition to their adaptive skills (ability to perform everyday functional tasks).

MEDICAL HISTORY

Clinicians should explore any risk factors for ABI stemming from past head injuries, hypoxic or anoxic brain injury, neurodegenerative diseases, or other medical conditions (i.e., epilepsy) in addition to any past assessments undergone, reviews or investigations by specialists (e.g. neurology, rehabilitation consultant, radiology). If appropriate - and with written client consent - hospital records can be obtained.

It is important to remember that the client may not fully or accurately recall their diagnoses, past medical issues, or admissions to hospital. They may neglect to report what was actually a severe illness/event, or alternatively portray an incident as unduly significant. Objective records should be relied on where possible. Alternately if the client has had the same GP for many years a GP health summary may be useful.

Table 11. Assessing Medical History

DOMAIN	SPECIFIC QUESTIONS YOU CAN ASK
Medical Condition/s (Past and Present)	What are your current and past medical diagnoses? Do you have any blood born viruses such as HIV, Hepatitis B or C? (more relevant for people who inject drugs). If yes, when was it diagnosed? Has it been treated? Have you had your liver function tested? If yes, what were the results?
Overdose History	Have you ever experienced an overdose? If yes, how long were you unconscious? Were you hospitalised/how long for? Did you experience any thinking difficulties after the overdose?
Periods of Hospitalisation	Have you ever been hospitalised? If yes, why and for how long? Did you self-discharge? Did you undertake any rehabilitation/other therapies? Did you experience any difficulties after discharge from hospital?
Prescription medications	What medications are you currently prescribed? What are they prescribed for? What dosage are you prescribed? Do you take them as prescribed? Have you noticed any changes in how you think or feel since you started taking them/upped/reduced your dosage?
Changes	Since the incident have you experienced any changes in your ability to think, feel, in your personality or daily life?
Family History	What kinds of serious disorders or illnesses (physical or psychological) have your family members experienced?

BRAIN IMAGING & ASSESSMENT

This section may be helpful if your client has had a brain scan in the past, or you have access to the results of that scan. Clients who are hospitalised with a suspected TBI, stroke or other significant event will often receive some form of brain imaging – together with the history and presentation of the client at the time, this is helpful in understanding what occurred. In this section, the most common forms of imaging together with what some commonly used terms are outlined [194].

Computerised Axial Tomography (CT or CAT)	Is one of the most readily available and quick neuroimaging techniques, utilising x-rays. It is typically used to detect bleeding and structural changes in the brain, often immediately following suspected stroke or TBI.
Magnetic Resonance Imaging (MRI)	Works using magnetic fields. It takes longer than a CT scan, and cannot be used with people who have metal elements in their body. MRI offers a more sophisticated and high resolution image of the brain, and if often used for more detailed investigation of neural and nerve tissue once damage has already been detected through CT. MRI can be used to assess: • Whether a certain area of the brain (structure) is the correct size/volume for their age; • Whether there are abnormal collections of blood, what the blood vessels are like (MR Angiogram); • All the connections between different parts of the brain (diffusion tensor imaging [DTI]); • And how the brain is working through changes in blood flow when performing certain tasks (e.g., talking, reading a map) (functional MRI [fMRI]). The presence of structural changes in specific areas of the brain are likely to correspond to distinct cognitive deficits. However, CT and to a lesser extent MRI, is not always capable of detecting smaller levels of damage. Importantly, these scans cannot be used to precisely predict what clinical problems or changes a person may experience after an injury. Consequently, to evaluate the severity and impact of brain injury on a person's functioning, further assessment is required.
Electroencephalogram (EEG)	Measures brain waves, and is usually used to investigate the presence of seizures.
Positron Emission Tomography (PET) & Single Photon Emission Computed Tomography (SPECT)	Are both ways of analysing a person's brain function. Both use radioactive tracers to see which parts of the brain are more or less active during certain tasks. These forms of brain imaging are typically used in the diagnosis of dementia, investigation of seizures and research.

Table 12. Common terms used in brain imaging reports

TERM	MEANING
Contusion	Bruise or damaged brain tissue
Infarct	Dead tissue caused by lack of oxygen to the brain
Lacunes / Lacunar Infarcts	Small strokes – again these can be common with increasing age
Haemorrhage	Bleeding in the brain
Subarachnoid Haemorrhage	Bleed in the space surrounding the brain
Diffuse axonal injury	Stretching and tearing of the white matter
Atrophy	Shrinking of brain volume, or thinning of a brain area
Volume	Relates to the size of the structure of interest
Lesion	Damaged brain tissue (wound, abscess, ulcer, tumour)
Space occupying lesion	Something is taking up space in the brain (i.e., a tumour or haemorrhage)
Mass effect	The surrounding brain tissue is being displaced by the lesion

EXPLORING RISK OF TBI

As noted, the majority of TBI injuries are mild, following which a complete recovery is typically expected [4]. For moderate to severe injuries, long term difficulties in sensory, motor, cognitive, psychological, or behavioural domains may occur [3]. Key medical indicators of moderate to severe TBI that can be explored in history taking include:

- A significant blow to the head (e.g. motor vehicle accident, assault or fall);
- A significant period of loss of consciousness (e.g., one hallmark estimate of injury severity is a loss of consciousness lasting longer than 30 minutes);
- Hospitalisation for treatment (e.g., taken by ambulance), brain imaging (CT or MRI) showing acute intracranial changes (i.e. bleeding or swelling of the brain);
- Post traumatic amnesia (PTA) of more than one day post injury (a state in which an individual is unable to form new memories with longer periods indicating a more severe injury).

At the time of injury, following a suspected TBI, immediate medical treatment and review is required. For past injuries, referral to a medical doctor and/or neuropsychologist can assist in clarifying a client's history. As part of such referrals, a client's injuries and hospital records including brain imaging reports and available measures of injury severity are reviewed, alongside formal neuropsychological assessment to determine the impact of the TBI on cognitive functioning. Clients will often experience PTA, and will therefore not recall the injury or events occurring following the injury. The degree of post-injury amnesia will vary according to the severity of the injury experienced – consequently, clinical documentation should be relied upon (ambulance and hospital data).

A recent systematic review on the global incidence of TBI examined severity of TBI injuries and found that 81% of them were mild, 11% were moderate and 8% were severe [195].

When it comes to predicting functional recovery following TBI, lower injury severity is typically associated with better and faster recovery. Older-age at time of injury is a strong predictor of poorer outcome. The broad range of physical, cognitive and behavioural issues associated with TBI are outside the scope of these guidelines. For more information, visit **www.racgp.org.au** and see **Appendix B** for information on how to access support for your client experiencing a TBI in the context of a workplace or road accident and **Appendix C** for information on general support for CI.

PSYCHOSOCIAL FUNCTIONING

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.

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 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 major injuries or traumatic events;
- 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.

MENTAL HEALTH & TRAUMA

Investigating a client's mental health is critical for a number of reasons. Firstly, the prevalence of mental health disorders is very high in the AOD sector [77]. Secondly, poor mental health is a risk factor for reduced cognitive functioning. Ensuring adequate treatment of mental health is an important step towards reducing CI, and improving overall quality of life.

What are the Statistics?

- Thirty-five percent of individuals with a substance use disorder are likely to exhibit at least one co-occurring affective (depression, bipolar or anxiety) disorder [196];
- Psychotic and schizoaffective disorders are also more prevalent among those with substance use issues compared to the general population [197-199].

Trauma experienced in childhood or adulthood has been associated with later substance use and dependence [200, 201]. Clients utilising treatment services are more likely to be experiencing, or to have experienced trauma - most commonly physical and sexual assault [201]. Giordano and colleagues (2016) surveyed attendees at 13 outpatient AOD centres in the US and found that 85% of their sample had experienced at least one traumatic incident, while 81% had experienced more than one traumatic experience [202]. Turning Point data supports this trend, with clients referred to the neuropsychology clinic reporting high rates of abuse, neglect and family violence as a child, as well as be experiencing family violence and abuse in adulthood [6].

Family Violence

ABI and CI is prevalent among perpetrators of family violence, as well as victims. Head injuries (caused by injury sustained to the head and neck, as well as falls) often associated with family violence can lead to brain injury. It is important to note that many women do not seek medical care after they sustain a head injury, and hence there may be limited documentation ([203]). Further, clients (most often women) subject to family violence will often present with a range of other issues (i.e., housing instability, trauma, poor nutrition, stress) which can often be confused with CI. Consequently, cognitive assessment should ideally occur after the client is safe, and these issues are somewhat resolved. Clients subject to family violence should be linked to appropriate services at first instance (i.e., social workers, shelters, legal aid, The Orange Door or other Family Violence Services).

HOW DOES MENTAL HEALTH IMPACT ON COGNITION?

Mood disorders	Mood disorders (anxiety, depression) are often associated with poorer cognitive functioning. Major depressive disorder (MDD) is consistently associated with deficits in attention, verbal and nonverbal learning, working memory, visual and auditory processing, processing speed, executive and motor functioning [204], while generalised anxiety disorder (GAD) has found effects on concentration and working memory [205].
Post-Traumatic Stress Disorder (PTSD)	PSTD has been associated with reduced processing speed, verbal learning, and executive functioning. In addition to the serious emotional and social/socio-cognitive deficits associated with PTSD, the hallmark cognitive changes observed among both children and adults with PTSD include deficits in declarative memory (often loss of memory/ recollection of the traumatic event or details of the event), processing speed, verbal learning, concentration, attention, planning, and problem solving [206-208]. Children who experience early & sustained abuse have in some cases also been shown to exhibit smaller brain volumes, reduced cognitive and academic achievement, with resultant IQ, and cognitive deficits persisting into adulthood [209, 210].
Schizophrenia	Those with schizophrenia often exhibit pronounced cognitive difficulties in areas of attention, working memory, verbal learning and memory, and executive functions - with added impairments in social cognition, affect recognition, and 'Theory of Mind' [211, 212].

The cognitive deficits associated with mental health issues can resolve if the underlying disorder is effectively treated (if possible). Consequently, clinicians can and should screen for mental health issues (see **Appendix D** for a rudimentary guide) – and further referrals should be made to the client's GP, with potential psychiatric intervention or follow up. Again, the Victorian AOD tool has measure and guides for taking a complete mental health history. In regards to risk assessment, clinicians are advised to use their own organisations risk assessment tools.

Case Study: Mental Health & Cognition

How complex trauma can impact on cognitive functioning.

Sam is a 28 year old woman who was referred due to concerns about her impaired attention in the context of 10 years of polysubstance use (marijuana, methamphetamine, alcohol and GHB), she had been abstinent for about 2 months after a 28 day extended detox admission. She had experienced trauma and violence throughout her life, including physical, verbal abuse and sexual assaults. She reported often waking up after having vivid nightmares and experiencing flashbacks. Sam noted that she had been given a referral to trauma counselling but did not want to go.

Presentation

Sam was late to her appointment and had to leave early. She reported having taken a Valium that morning after a stressful conversation. She appeared anxious and slightly jumpy although this settled as the session progressed. Her affect was appropriate and reactive to humor, with good eye contact. Her speech was clear, coherent and fluent. Sam was neatly dressed. She reported concerns about "her thinking" – noting that she often had trouble remembering certain words and important dates. She indicated that she thought she was organised and found her administrative job manageable. Sam put forth good effort throughout the assessment, and was willing to make educated guesses on difficult items.

Findings

Sam's neuropsychological profile was characterised by low-average intellectual ability, largely intact memory, some weaknesses in higher-level attention (mental flexibility) and variable processing speed. It was likely that these subtle weaknesses were related to her mental health difficulties and long history of trauma. Sam's profile was not suggestive of an ABI, although she did present with risk factors and was vulnerable to a reduction in cognition. As such it was recommended that she continue to remain abstinent and seek treatment for her mental health.

Resources:

- Hospital Emergency Departments/Psychiatric Departments;
- Direct Line: 1800 888 23 (can provide immediate psychiatric advice/care if necessary e.g., Crisis Assessment and Treatment Team [CATT]);
- Lifeline: 13 11 14 & Beyond Blue: 1300 22 4636;
- Arbias Ltd: Provide specialist services in Victoria tailored to substance-related brain impairment. They provide neuropsychological assessment, case management, housing, lifestyle support, information and training for AOD specialists

COMPLEX POPULATIONS

Some clients present with additional complexities that may cause or exacerbate CI. Clinicians should be aware of these complexities and how they can affect a client's functioning and treatment progression. Whilst not all complexities could be covered here, some of the more common scenarios will be addressed.

CORRECTIONS & FORENSIC SETTINGS

Clients presenting to AOD services are more likely to have a forensic history or corrections involvement. People with AOD issues are more likely to come into contact with the justice system due to a higher likelihood of drug-based offences (i.e., use or possession), behavioural factors such as impulsiveness or inappropriate, aggressive or anti-social behaviour.

Within the forensic setting, it has been suggested that almost 40% of the prison population have an ABI, potentially arising in part as a result of long-term substance use [213]. As no formal objective cognitive screening routinely occurs in Victorian prisons (rather, self-report is relied on), experts believe that this is a conservative estimate [213]. For example, a recent review of a new ABI self-report screening tool in the Victorian correctional system highlighted high rates (25%) of 'false negatives' (lack of appropriate diagnosis) among male inmates – noting that difficulties associated with memory and learning likely made their 'histories somewhat unreliable', with inmates frequently under-reporting ABI risk factors [213]. Moreover, the reality that substance use continues in prison means that any formal assessment could be hampered by the effects of intoxication or withdrawal [215].

As noted, there are considerable difficulties in distinguishing between an ABI and various other disabilities (i.e., intellectual disability, neurodevelopmental or degenerative disorders), personality disorders or behavioural traits. In regards to pre-existing learning or neurodevelopmental disorders, rates of intellectual disability in prisons are estimated at between 7-10% [216]. Moreover, 25% of youth with offending histories are thought to experience learning difficulties in reading or mathematics [217].

CI is becoming increasingly recognised as a key factor related to recidivism and violent/anti-social behaviour, particularly with comorbid substance use [2181].

IMPORTANT CONSIDERATIONS & RECOMMENDATIONS

- It is important to take into account level of education when screening for CI or querying an ABI with clients who have forensic involvement;
- Screening for CI should utilise objective measures of impairment (i.e., medical histories, standardised cognitive assessments) rather than relying on subjective reports;
- Clients may be hesitant to engage fully with a treatment provider when they have been mandated to treatment, or have concerns reporting their behaviours/issues (i.e., concerns with confidentiality);
- Entering into discussions of criminality and offending behaviour is frequently seen as potentially disengaging, particularly where the limitations of confidentiality become complicated by the existence of 'third parties' in the treatment relationship (such as the courts);
- Treatment and support for CI should focus on the ability to live independently and an eventual return to work (these being two stressors frequently associated with criminal recidivism).

Resources

- Jackson, M., & Hardy, G. (2011). Acquired Brain Injury Screening, Identification & Validation in the Victorian Correctional System. Arbias Ltd & La Trobe University.

  Available at: **www.corrections.vic.gov.au/utility/publications+manuals+and+statistics/acquired+brain+injury+in+the+victorian+prison+system**

Case Study: Learning Disabilities & Corrections Involvement

Hugh is a 31 year old man who was referred for an assessment by his corrections assessor who had observed that he had difficulties understanding and processing questions and could not completed a self-screening questionnaire.

His assessor reported that Hugh had told him that he had been struck by a vehicle as a child. At interview, Hugh stated he had experienced a brain injury as a result of this accident and since then experienced difficulties with memory, attention, reading and understanding concepts. He experienced occasional episodes of depressive mood and had a history of binge alcohol and cannabis use.

Luckily, hospital records were available for his injury, which documented a severe traumatic brain injury. He had been taken to hospital unconscious and was in a coma for four days – experiencing confusion and disorientation for a further 11 days after regaining consciousness.

On assessment, he was happy, engaged well and displayed no sensory, motor of behavioural impairments. He provided a clear personal history but asked for help to complete a self-reported measure of mood as he had trouble understanding the questions. The assessment showed normal performance on measures of nonverbal skills, speed of thinking, learning, memory and executive functioning while he struggled on measures of working memory and verbal skills. His word reading was equivalent to a grade 4 standard.

As a result of the assessment, Hugh was diagnosed with a learning disorder in the area of reading (dyslexia) which has not been previously identified. This accounted for why he struggled to complete any self-reported measures given to him by clinicians and struggled on verbally based tasks despite many of his other skills being normal for someone his age. Given the severity of this TBI at such a young age, he had made a substantial recovery and apart from his reading, was functioning quite well. It was recommended that he continue to use the various strategies he had developed, such as making notes in a diary to refer to later and writing down tasks he had to complete at work. The details of the reading and writing hotline were provided to him in the event he wished to participate in adult literacy classes to improve his reading abilities and confidence.

ABORIGINAL AND TORRES STRAIT ISLANDERS

Compared to the wider Australian population, Indigenous adults and youth face significant health inequities [219], and are at a higher risk of experiencing both CI and substance use disorders, and its associated health and social harms (i.e., Hepatitis C, HIV from injecting drug use, psychological disorders/distress, overdose, family violence, and head trauma resulting from falls/intoxication) [220].

While most Indigenous people abstain from alcohol, some evidence suggests that those who do drink, are more likely to do so at harmful levels, 34% drink at a risky level at least monthly in 2019 [77]. In 2019-2020, of those receiving treatment for alcohol as a principal drug of concern, 1 in 6 (or 18%) were indigenous Australians.[46] Higher rates of hospitalisation in Indigenous populations as a result of head trauma, stroke and overdose have been associated with increased rates of ABI [221], which is particularly prevalent among individuals who are incarcerated or living in regional and rural areas [222]. A study of CI in a rural Australian substance treatment in-patient population found that the proportion with scores on the ACE-R indicating possible CI was 82% among Indigenous clients versus 28% in non- Indigenous clients [223].

One challenge to assessing CI among clients from culturally and linguistically diverse (CALD) and Indigenous backgrounds is that assessment tools have been developed in a western framework for people from western cultures, and hence available normative data is likely inappropriate [182, 224]. If there are concerns around potential ABI or CI then consider making a referral to neuropsychological services. It is important to involve the clients support people (family, carers, key community members, i.e., elders, Aboriginal Liaison Officer [ALO]) in the assessment and treatment process, and use an interpreter where appropriate. Client retention, accurate information and use of compensatory strategies are unlikely to occur unless supports are involved at all stages and culturally-specific tools with appropriate normative data should be used where available.

Resources

- The Victorian Aboriginal Community Controlled Health Organisation (VACCHO) provides various resources to clinicians working with Indigenous clients. Available at **www.vaccho.org.au/wd/aod/**.
- Three rural Aboriginal AOD nursing teams in Mildura, Shepparton and Bairnsdale provide clinical support to clients, and links to Aboriginal AOD workers. Along with social and emotional wellbeing workers, these teams provide Aboriginal clients with holistic, culturally appropriate care.
- Aboriginal-specific services accept referrals from catchment-based intake and assessment as well as self-referrals and direct referrals from other services, and are expected to prioritise access for Indigenous clients. Further advice on accessing these services is available at DirectLine (1800 888 236).

CULTURALLY AND LINGUISTICALLY DIVERSE (CALD) POPULATIONS

Refugees and asylum seekers are at higher risk of experiencing psychiatric issues (i.e., anxiety, depression, trauma, PTSD) and reduced social and educational opportunities associated with their pre-migration, detention and resettlement experiences [225]. Whether migrants from CALD backgrounds experience higher rates of AOD use and mental health issues appears to depend on the rates of AOD consumption in one's country of origin, their health literacy, migration and settlement experience, social-support, and their mental health [226].

People from CALD backgrounds appear to be under-represented in AOD treatment, and take-up fewer referrals to professional support services. Barriers to help-seeking include reduced access to Medicare and support, as well as concerns about confidentiality (fears that the clinician or interpreter who speaks their language may be a member of their community) and culturally-related shame. Additionally, some CALD clients may harbour a distrust for those they perceive to hold authority (i.e., doctors, other medical staff). If assessing cognitive functioning, be aware that cognitive performance may be reduced when fasting (i.e., Ramadan, Lent, Yom Kippur). A major limitation in assessment of CI with CALD populations, is that neuropsychological tests are often available in only a handful of languages, they do not take into consideration different cultural or educational backgrounds and there is an absence of normative data.

Resources

- **NADA:** Working with Diversity in Alcohol & Other Drug Settings. **http://www.nada.org.au/**;
- **DAMEC (NSW)**: Drug and Alcohol Multicultural Education Centre. **http://www.damec.org.au/**

COMPENSATORY STRATEGIES FOR COGNITIVELY IMPAIRED CLIENTS

We all use compensatory strategies in our daily life. These strategies may be particularly useful for clients with CI depending on their specific presenting difficulties and unique circumstances. When developing compensatory techniques for clients with CI, it is important to tailor the strategies to the individual, help them practice the strategy, and to normalise strategy use [227]. It helps if the client is aware of their difficulties, as they may be more motivated to use strategies.

Strategies can be divided into internal ('in mind') strategies, and external strategies. Using internal strategies is generally more effortful, which may limit their uptake.

General Principles: Memory

1) Information that is frequently used is easier to remember: Using information creates greater and stronger associations to related information allowing for easier retrieval at a later time.

2) Recognition is easier than recall: We can make it easier to recall information by using cues and prompts.

3) Forgetting is adaptive: We cannot and are simply not meant to remember everything, and therefore more salient or important information is prioritised.

INTERNAL STRATEGIES

Internal strategies require mental effort and enhance information retention using mental strategies such as rehearsing content in our heads or intentionally creating associations with other related or similar information. It should be noted that many clients with CI will not be able to use these strategies without ongoing support and structure [3].

Useful strategies that can be taught in sessions include:

- **Repetition:** This enhances attention to information and strengthens the neuronal pathways, it can be helpful to try repeat information by presenting it in different mediums.
  - Repeat key information to clients in session.
  - Ask the client to repeat the information back to you, using their own words.
  - Ask your clients to write the information down in a notebook.
  - Repeat the information at subsequent sessions.

- **Spaced repetition and retrieval:** The information is rehearsed over increasing time intervals.
  - Repeat the key information in session, at increasing intervals (e.g., after 2 minutes, again after 10 minutes and then again after an hour).
  - Prompt the person to retrieve the information giving specific cues and gradually fade out these cues.

- **Association:** Our brain relies on networks of neurons to create, store and retrieve information. The more associations we make with a new memory the better we remember it. Associations that are familiar are easier to remember (i.e., milk and tea). Associations that are novel or unusual with no clear link are much harder to recall (i.e., a car and tea). Names are a perfect example, there is usually no physical feature that associates us with our names.
  - Ask the client to think about the unique features associated with a piece of information. For example, to remember a person's name, you can associate that name with a characteristic or something you have previously learned (i.e., her name is Susan, and my sister's name is Susan). To remember the Spanish word for cat ('gato'), a common example is to think of a gate and imagine a cat sitting on top of the gate (also called keyword mnemonics).

- **Chunking:** Chunking reduces the load on memory by grouping similar items together, making it one unit.
  - For example, recalling a phone number – 0446 567 589 - is easier to recall than 044657589. Another example is to group items on a list according to the number of letters each item has (bread, chips).

- **Self-prompting:** This involves associating context-based cues with the central piece of information (what they were doing, where they were, when it was learned etc.). This is a good method to use when you are trying to retrieve a piece of information such as where you left your keys, or what happened several days before.
  - Ask the client to retrace their steps i.e., what room were you in when you learned that information? Who were you with?

- **Musical mnemonics:** Singing or rhyming information has been found to assist in information recall, especially for long lists of things (e.g., the alphabet song).
  - Try replacing the words from a popular song with information you need to remember.

- **Acronyms and acrostics:** These are helpful in recalling key information that is unlikely to change.
  - Acronyms reduce the load placed on memory. For example': WHO 'World Health Organisation, NDIS, National Disability Insurance Scheme '. Client's may like to generate some useful acronyms applicable to their own lives.
  - Acrostics are similar to acronyms, except that instead of forming a 'word', they generate a sentence that helps to recall information (i.e., Eat An Apple After A Night-time Snack stands for the continents- Europe, Antarctica, Asia, Africa, Australia, North America, South America).

- **Method of loci:** This is a visualisation method that people may use to remember vast amounts of unrelated information, however, in daily life it is usually easier to write a list!
  - Ask your client to visualise a room or a familiar path through a building and mentally associate particular pieces of information with objects along the way.

- **Narrative mnemonics:** Ask the client to develop a short story out of important information (i.e., all the items they need to take to work with them, like glasses, gym shoes, lunch, medication).
  - For example, the client may generate a story about their glasses running away with their gym shoes to have a nice lunch but then they had to come back because the shoes needed their medication.

EXTERNAL STRATEGIES

These strategies utilise external aids to assist in completing everyday activities - such as diaries, alarms, phone calendars and notes. Overall, these are the most utilised and useful strategies for people with memory deficits [3]. Compared to internal strategies, external strategies are quicker and easier to adopt.

Smartphones are an ideal memory aide. Some clients are able to use them for day-to-day support, provided they receive adequate training and assistance, do not easily misplace their smartphone, remember to keep their phone charged, and have adequate credit/internet access. Taking photos of important information, or making detailed notes (i.e., through dictation) throughout the day, can be a helpful memory aid for some clients. Text reminders are used by many organisations to improve appointment attendance, and may be helpful for your clients as well [228]. Clients may benefit from a reminder a week before, a few days before and then again the day before an appointment.

A study by McDonald et al. (2011) compared 'Google Calendar' to using a paper/hardcopy diary as a memory aid among those diagnosed with an ABI [229]. They demonstrated that Google calendar was more effective in improving memory performance than a diary, with a 24% increase in remembering to do things in the future (prospective memory). They noted that the key difference between the strategies was that google calendar provided active reminders (e.g., phone notifications) which reduced pressure, stress, and the need for self-monitoring in order to accurately remember an activity. Nevertheless, it is important to keep in mind that clients, particularly those in an AOD setting, may not have access to technology or a regular internet connection. In these circumstances, it may be more beneficial for clients to set up predictable routines that require less self-regulation (i.e., appointments at the same time or day each week), and to seek help/reminders from others.

OTHER TECHNIQUES:

- Clients less familiar with, or with reduced access to technology may benefit from keeping a physical diary or calendar in which they can record important dates, and a to do list;
- Assist the client in setting regular alarms for key tasks (i.e., an alarm reminder to put the bins out on a specific day each week, or take medication);
- Assist the client in setting reminders on their phone a day and hour before an important event; e.g., organise for bills to be paid automatically (i.e., 'centrepay');
- Record your sessions (on a piece of paper, phone or other digital recording device) for your client to have access to later; or allow them to take notes
- Teach the client to keep important things (i.e., things they need to take with them to their next appointment) in a designated place – see the 'memory station' below.

Medications

- Link taking medications to regular events (e.g., taking medications after you brush your teeth or eat dinner (something they are unlikely to forget to do);
- Keep medications in a prominent, designated place that is difficult to miss (but out of the reach of children);
- If people have trouble recalling whether they have actually taken their medications, or have a lot of medications to take at different times, a 'dossett box' may be helpful (a dossett box is a pill organiser in which medications are deposited into seven daily segmented containers);
- For people who really need to take medications on specific days at specific times, and cannot self-manage using a dossett box, a 'Webster-pak' from the pharmacy may be useful. A multi-dose Webster-pak is a sealed weekly calendar pack designed to assist people in taking their medications correctly as prescribed.

Memory Stations

A 'memory station' can be set up within the clients' home/room (see example below). Memory stations usually include a white board or pin board, a small table to keep important items (i.e., keys, medications, wallet, watch or glasses), and other things that need to be taken with the person the following day (i.e., a phone, important documents), a calendar for appointments, important dates (bills that need to be paid), and tasks that have and have not been completed

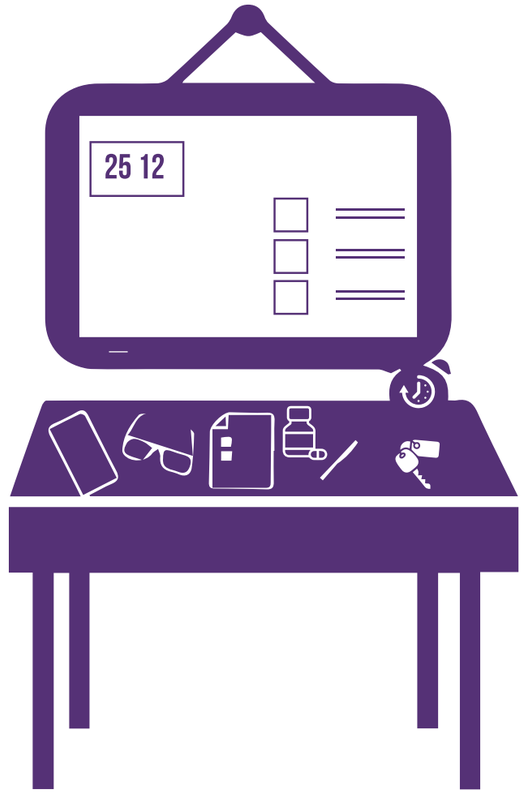


Figure 6. Example of a Memory Station

LIFESTYLE RECOMMENDATIONS

DIET & NUTRITION

Good nutrition is key to having an efficient and healthy brain. Poor diet and nutrition is a common side effect of substance use, particularly alcohol use [230]. In particular, Thiamine deficiency is often associated with alcohol dependence. Thiamine, or vitamin B1, is naturally present in many foods including whole grain products, meat and eggs. A deficiency can occur in individuals who consume high amounts of alcohol due to poor nutrition and poor absorption of vitamins associated with inflammation of the stomach lining. Treatment with thiamine has, however, been shown to improve cognitive functioning in alcohol-dependant individuals [231]. As such, it is important to discuss daily nutritional habits with clients and explore their dietary routines.

SLEEP

Sleep plays a key role in immune and heart function, metabolism, emotional processing, mood, creativity, and cognition. Sleep is also crucial for learning and memory. Sleep is necessary for the consolidation of newly learned information – that is, strengthening of important memories and pruning of rarely remembered memories [232]. Sleep deprivation impairs memory formation and appears to particularly impact the retention of emotional material [233]. Learning and memory formation can be affected by the quality and length of sleep. In the AOD population there is a high incidence of mood and anxiety issues, which are often associated with sleep disturbance [234]. In addition, psychoactive substances influence sleep quality and architecture (the different stages that the brain cycles through during sleep), leading to less restorative sleep, an interruption to overnight learning and memory consolidation, and impacting other processes that occur overnight such as emotional processing. Interestingly however, a short period of controlled sleep-deprivation can 'reset' and ameliorate some depressive symptoms [235].

SLEEP & ALCOHOL

Alcohol has significant impact on sleep architecture, with different effects across the two halves of the night, the first half being associated with reduced time to fall asleep and suppressed REM (rapid eye movement) sleep, followed in the latter half by increased wakefulness and excess REM. Therefore, whilst alcohol can help us fall asleep more quickly, we often wake up frequently during the second half of the night. Altered sleep disrupts memory consolidation, cell regeneration, and causes next day fatigue and slowed reaction time, as well as impacting mental and physical health.

Recommendations for achieving better sleep:
Adapting sleep recommendations to the client's needs are important. Sleep changes need to be achievable and sustainable. Those clients who use substances to aid sleep may benefit from an addiction medicine review, or even an admission for detoxification and rehabilitation.

Tips for improved sleep quality include:

- Reduce variability in sleep day to day – go to bed & get up at a similar time each day;
- Cut down on using caffeine and other stimulants in the afternoon/before bed;
- Keep away from bright screens/lights in the last two hours before bed (download a blue light filter on any technology used);
- Try to keep electronics out of the bedroom (i.e., TV or phone, use an analogue alarm clock instead);
- Seek sunlight exposure in the mornings (e.g., a morning walk or outdoor meditation);
- Wind down and relax before bed (e.g., music, bath, reading, meditation);
- Keep the bedroom dark and relatively cool before attempting sleep;
- If you're not asleep within about 20 minutes of trying to sleep, get up and do something quiet and calm for a short time;
- Avoid use of recreational substances as a sleep aid;
- Avoid spending too much time in bed before attempting sleep.

TREATMENT PLANNING

Depending on the nature and severity of CI, some clients may be better suited to receive treatment, or at least commence a treatment episode in certain settings. Where CI is severe and compromises independent living, more structured and supportive environments are preferable. A client with severe memory deficits and requiring withdrawal for example may be better suited to inpatient withdrawal than non-residential withdrawal settings. Since full recovery from CI requires around one-year of abstinence, it is important to emphasise the importance of abstaining from substance use and to offer treatment modalities and interventions that are likely to maximise their chances of achieving this. This includes residential rehabilitation or dayhab, which offers a structured environment with individual and group therapy where people learn and practise skills to control substance use, triggers and cravings.

Clients should be referred to their GP or an Addiction Medicine Specialist if pharmacotherapy (e.g., Buprenorphine (Subutex, Suboxone), Methadone, etc.) or anti-craving/relapse prevention medications (naltrexone, acamprosate, Disulfiram, Baclofen, Topirmate, etc.) could help them reduce or cease their substance use. However, it is important to note that CI, particularly memory deficits, may restrict adherence to medication regimens. Ideally, assessment of mental health co-morbidity and the need for pharmacological and psychological intervention (e.g. to a dual diagnosis clinician) should take place before comprehensive neuropsychological assessment to achieve an accurate picture of an individuals level of cognitive functioinng.

With CI as an additional complexity factor, clients in the Victorian AOD system may be eligible to receive Care and Recovery Co-ordination (i.e., case-management), which could facilitate referral to GPs, neuropsychology, family, mental health and specialist services, etc. However, it is important to highlight that whilst there is some evidence suggesting people with CI derive less benefit from treatment, there is no evidence to suggest they do not benefit from mainstream approaches like CBT and MI [236]. Nonetheless, psychological approaches with fewer cognitive demands such as contingency management (incentivising abstinence with vouchers or other benefits) may be more effective for clients with severe deficits in executive functioning [237], though this continues to have limited availability as a treatment approach in Australia. It is likely that a client will profit more from counselling after a period of time-dependent recovery has occurred, when they are in a better position to attend to information, understand what the clinician is asking of them, engage in reflective thinking and express their thoughts and feelings more clearly. For this reason, it may pay to focus on psychoeducation in the first 1-2 weeks of treatment engagement and to introduce more cognitively demanding interventions a few weeks later, when at least partial cognitive recovery has taken place. For more information on how to adapt psychological interventions see the chapter on adapting the delivery of psychosocial interventions located on the following page.

Aftercare in the form of peer support/mutual groups such as AA/NA or SMART Recovery has also been shown to improve outcomes for clients one-year after treatment [238]. It can provide a way of expanding social networks to include individuals who share similar challenges and goals. Indeed, engaging in pro-abstinent social networks had greater association with improved drinking outcomes among cognitively impaired outpatients than cognitively-intact outpatients in project MATCH [239].

Importantly there is no evidence to suggest people with CIs cannot not benefit from the currently available suite of treatment settings and types. Nonetheless, clients with CI are more likely to benefit from the more extended, intensive and integrated packages of care, including services that assertively connect clients to health, social and welfare services to meet their multiple and often complex needs. In Victoria, the current AOD Pathways helpline **https://www.directline.org.au/aod-pathways** is a dedicated service to help clients with the most complex needs to navigate and access AOD treatment. Finally, whilst client-centred care and choice should always be prioritised, clinicians should aim to adhere to established evidence-based guidelines to ensure safe and effective practice wherever possible.

ADAPTING PSYCHOSOCIAL INTERVENTIONS

CI can interfere with AOD treatment in a number of ways. Not only can it lead to ongoing or increased substance use, but it can also hamper treatment engagement, adherence, duration, and outcomes [43, 44, 240]. Higher rates of treatment non-completion among clients with CI has been found across multiple settings [241]. Research suggests that clients with poor inhibition and poor decision-making skills, particularly those with high rates of delated-discounting (the tendency to seek immediate rewards over larger rewards delivered later) are more likely to relapse after treatment [242, 243]. The cognitive functions impaired in AOD clients, are those needed to fully engage in psychosocial treatments like Cognitive Behaviour Therapy (CBT) and to facilitate sustain behaviour change [242]. For example, one study suggests that CI indirectly influences substance use outcomes by reducing the quality of coping skills acquired in CBT [244]. Other commonly used evidence-based treatments include Motivational Interviewing (MI), Mindfulness Based Relapse prevention (MBRP) often require relatively high levels of cognitive functioning. For example, they require an ability to attend to, learn, and recall information, keep track of things, plan, make decisions, consider multiple perspectives (cognitive flexibility) and inhibit responses. Executive functioning, especially working memory, is necessary to maintain and manipulate conscious, goal-relevant information [242]. It is important to note that the relationship between CI and outcomes may be indirect in that they are mediated by other factors such as motivation, self-efficacy, therapeutic alliance and treatment compliance [27]. As such, adapting the delivery of psychological interventions to an individual's relative cognitive strengths and weaknesses may help clients to derive maximum benefit from AOD treatment. Indeed adapting a residential rehabiltation program in NSW enabled clients with CI to achieve similar treatment completion and retention rates as clients without CI [245].

ADAPTING DELIVERY

To accommodate CI, the way in which psychological interventions are delivered can be modified (e.g., by having shorter, more frequent sessions with regular breaks to consolidate learning, and incorporating regular reviews of important content). When conveying key concepts it may help to draw on different methods (i.e., use of diagrams, mind-maps, infographics, pictures, stories, or videos). It can help to write down key points for the client, have them take notes or make session summaries (which you should photocopy) and retain in a 'treatment log book'. It is also helpful to give homework (e.g., as part of CBT) in the presence of family, friends or key supports alongside clear written instructions. The following section outlines practical strategies that may assist in the delivery of treatment.

IMPAIRED ATTENTION & INFORMATION PROCESSING SPEED

- Rule out any acute medical causes such as delirium;
- Schedule important assessments at a time your client is usually alert (avoid early morning, medication times or late in the evening); and encourage the them to stick to a routine (i.e., sleeping, eating) at regular times;
- Reduce environmental distractions (i.e., noise) and consider where the client sits so they are not distracted by posters or people walking past the office;
- Present key infomation slowly, focus on only one key area/theme per session to avoid confusion;
- Use short, simple sentences;
- Link new information back to information or ideas already discussed;
- Check the clients understands instructions, ask them to explain the instructions back to you;
- Frequently orient the person (note the time, place, what you're doing and why);
- Take breaks if needed, especially for long tedious tasks like filling out forms;
- Encourage the client to slowly check over their work once they believe they have finished.
- Coach your clients in strategies that will allow them more time to decide what to do/actions to take – this may include asking others to repeat questions, speak more slowly to give them more time to think, and rehearse responses or behaviours to common situations;
- Help the client develop a script or strategies they can use to let others know they have difficulties e.g., "I'm more likely to remember what you are saying if you speak more slowly".
- Encourage the client to follow their routine (i.e., sleeping, eating) at regular times.

LANGUAGE DIFFICULTIES

- Consider referral to a speech pathologist/speech evaluation;
- Ask simple yes/no questions;
- Avoid long sentences;
- Emphasise visual communication/ alternative communication forms.

VISUOSPATIAL IMPAIRMENT

- Rule out problems with vision;
- Determine whether the client should be operating a motor vehicle or heavy machinery;
- Set-up safety measures within a client's home, or contingency plans in case they get lost;

MEMORY IMPAIRMENT

- Ensure key messages and strategies are presented on multiple occasions;
- Use visual aids and pictures;
- Encourage the client to practice & utilise rehearsal strategies;
- Simplify written instructions;
- Ask the client to summarise key information using their own words
- Provide information using mnemonics, chunking and imagery.
- Assist the client & their supports in providing structure to the clients' day by setting up a daily routine (including self-care activities) and avoiding long periods of inactivity. Utilise several reminder systems (i.e., alarms) to prompt initiation of these activities throughout the day;
- Be aware they may function well in a structured environment (i.e., prison or rehabilitation) but struggle when returning to the community, where they may require more support, or even supported accommodation;

EXECUTIVE FUNCTIONING: ABSTRACT THINKING

Clients who experience difficulties thinking abstractly and responding to open-ended questions may do better with a more directive therapeutic style [246]. This stylistic change can subsume 'how' a session is organised and guided by the clinician, but does not necessarily mean the clinician unilaterally determines treatment goals:

- Avoid conveying multiple abstract concepts or asking multiple questions in one sentence or in a short time-period (avoid 'run-on' or 'two-pronged' sentences), for example, when challenging distortions, negative automatic thoughts, cognitive restructuring it may be helpful to get the clients to consider and note which CBT methods help them most with perspective taking (e.g., examining the evidence, thinking in shades of grey, experimental technique, countering distortions etc.);
- Avoid use of complex analogies and use real-life, concrete examples (drawing on the clients own experiences e.g., with decisional-balance exercises within an MI framework, or when exploring core beliefs within a CBT framework;
- Use role play to practise strategies for coping with triggers/high-risk situations/relapse, for example when practising drug refusal skills in CBT);
- Use figures, diagrams, cartoons, stories or other media to convey ideas and concepts

IMPAIRED MENTAL FLEXIBILITY

- Avoid changes to routine but if disruptions are inevitable, remind the client of any changes several times, send text message reminders, and explain why it has occurred;
- If a client is repeating themselves or perseverating, interject with a quick summary of what the client has expressed, and then assertively direct the conversation;

PROBLEM SOLVING PROBLEMS

- Teach the client to approach novel tasks in a systematic manner (e.g., break the task into smaller parts, make a list of those parts and tick them off once they are completed); A 'Plan, Do, Review' framework can support problem-solving;
- Encourage the use of concrete problem-solving strategies: Identify the problem, set measurable and realistic goals, generate solutions, & monitor attempts/success;
- When generating solutions to a problem, ask the client to brainstorm all possible solutions and help them to work through the simplest, most workable option for them, using pros/cons lists, or 'mind maps' if necessary;
- Assist others working with the client to adopt the same approaches for problem-solving - this will aid in strategy uptake and learning;
- Show the client what to do as well as tell them;
- Practice new skills in lots of different situations to encourage generalisation to novel environments.

IMPULSIVITY

Clients may display impulsive, disinhibited or inappropriate behaviours, particularly if they are intoxicated, frustrated or mentally unwell. Highly impulsive clients can experience difficulties with self-reflection and monitoring, and are less likely to consider the long-term consequences of their behaviour (sharing personal information inappropriately, sexual promiscuity, risky behaviour, outward expression of emotion including anger, frustration etc. Reducing inhibition and emotional reactivity has also been shown to improve retention and completion of therapeutic programs [44].**The following are some management strategies that may help. If the difficulties are persistent and preventing them from engaging in treatment seek psychiatric, psychological or neuropsychological assessment. Always follow your organisation's specific guidelines for staff and client safety.**

- Set clear therapeutic boundaries and behavioural expectations and ensure they understand the consequences of a breach;
- Establish consistent self-monitoring techniques such as 'stop, think, check'
- Do role-play exercises and train the client to recognise which behaviours are problematic, which scenarios may trigger certain behaviours and when to apply compensatory strategies.
- If agitated by the presence of others, schedule appointments at quiet times when fewer clients are around;
- Use neutral language where possible, assume a calm and sympathetic tone;
- If the client becomes frustrated, distract them with neutral questions, change the subject, or take a break;
- It may be appropriate to return to a problem behaviour either later or at another session. You can ask the client to explain their feelings (perhaps using an emotion wheel), and identify what upset them;
- For clients who see a number of different professionals it can be helpful to have a team meeting to discuss a consistent approach to managing behaviours of concern;
- If the behaviours are very problematic and impacting on the client's accommodation, ability to access services or daily functioning (i.e., dress or feed themselves) a referral to a specialist service may be necessary;
- Sometimes ignoring inappropriate behaviours will be appropriate (try not to take them too personally, but do not place yourself at risk). Do not hesitate to debrief with your support people or team about how these behaviours may be effecting you.

POOR INSIGHT

A client may lack insight into their cognitive limitations, or be aware of them but unable to recognise when they influence behaviour (e.g., making decisions)

- If a client lacks insight into their difficulties, it can be helpful to re-direct them back to their goals or the situation that led them to you;
- If they have unrealistic goals then setting smaller goals in line with their longer-term goal can be helpful.
- Be aware that for some clients with a CI who lack insight, this is a core deficit that you may not be able to change easily Gently work around these deficits and avoid confrontation.

POOR SOCIAL COGNITION

Clients may present with difficulties reading social cues, conversational turn-taking, may misinterpret the actions of others, and may struggle to identify and regulate their own emotions. It may help to:

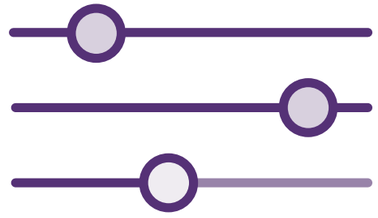
- Provide a clear agreement on the structure of therapy sessions at treatment commencement (e.g. indicating that it might be necessary to interrupt the client at times in order to keep progressing);
- Be aware that clients may be sensitive to criticism or misinterpret suggestions from the therapist as criticism. Openness to discussing the therapist-client dynamic can be flagged in the first session, and close monitoring of the therapeutic relationship and process is essential;
- Role-play social interactions and practice assertiveness skills. (e.g., ask them how they would feel if someone acted in ("X") way towards them – ask them to put themselves in that persons 'shoes';
- Teach specific strategies (i.e., maintaining eye-contact, empathetic language, tonal moderation, active-listening, asking questions/showing interest in others);
- Practice emotion-recognition using emotive faces;
- Encourage attention to other people's reactions – ask the client to document when someone has reacted in a confusing or negative way to them, and deconstruct the situation at the next session;
- Encourage the client to check in with others/supports about their behaviour and what they should adapt;
- Teach the client the 'STOP-THINK' technique - when they feel themselves getting angry or emotional, encourage them to stop and think about whether a reaction is appropriate or helpful before reacting.

THERAPEUTIC APPROACH

Whilst there is some evidence to suggest that clients with CI achieve poorer outcomes than those without CI, there remains insufficient evidence to suggest that talking therapies such as CBT and MI are ineffective or inappropriate for people with CI. Unlike CBT and MI, more insight-oriented psychotherapy approaches such as gestalt, narrative, and psychodynamic therapies may be more challenging for people with CI. Focusing on behavioural strategies to build skills and extend areas of competence is generally more effective than focusing on emotional processes, although recognition of areas of emotional impact and assisting with emotional adjustment to CI is important. It is worth noting that clients with a pre-injury/CI history of success with one approach may not achieve the same successes after injury/CI. Clinicians should encourage clients to regularly review progress towards achieving their goals, and offer praise for attaining or working successfully towards them.

Clients with CI can be more resistant to change, often due to a lack of awareness of their cognitive difficulties or denial. Managing resistance may involve including family and significant others in assessment, treatment, education and prevention efforts. Resistance may also be countered by assisting the client to recognise how their substance use is affecting their goal attainment, what the potential benefits of treatment are and to consider what barriers to treatment may exist.

ADAPT THE WAY THERAPY IS DELIVERED; USE PRACTICAL DEMONSTRATIONS, CONCRETE EXAMPLES WITH OPPORTUNITIES TO ROLE-PLAY AND PRACTICE WITH AND WITHOUT PROMPTS. PRESENT SIMPLE, STRUCTURED INFORMATION VIA WRITTEN MATERIAL AND VISUAL AIDS, USE REPETITION, SELF-MONITORING AND FREQUENT SUMMARIES.



SESSION STRUCTURE

Clients with CI may be more inclined to complete and benefit from a therapeutic session if they have a clear understanding of its duration and format. A useful strategy for conveying this information involves using a diagram of a clock with discrete sections representing different session components (see Figure 7). The total length of the session can be set, with sections indicating what will be discussed, the setting of homework or scheduling for the upcoming week, rehearsal of a behaviour, and a wrap-up segment. If the client has pressing questions or things to discuss, allocate time at the beginning or end of the session within the clock and alert the client when the time is up.

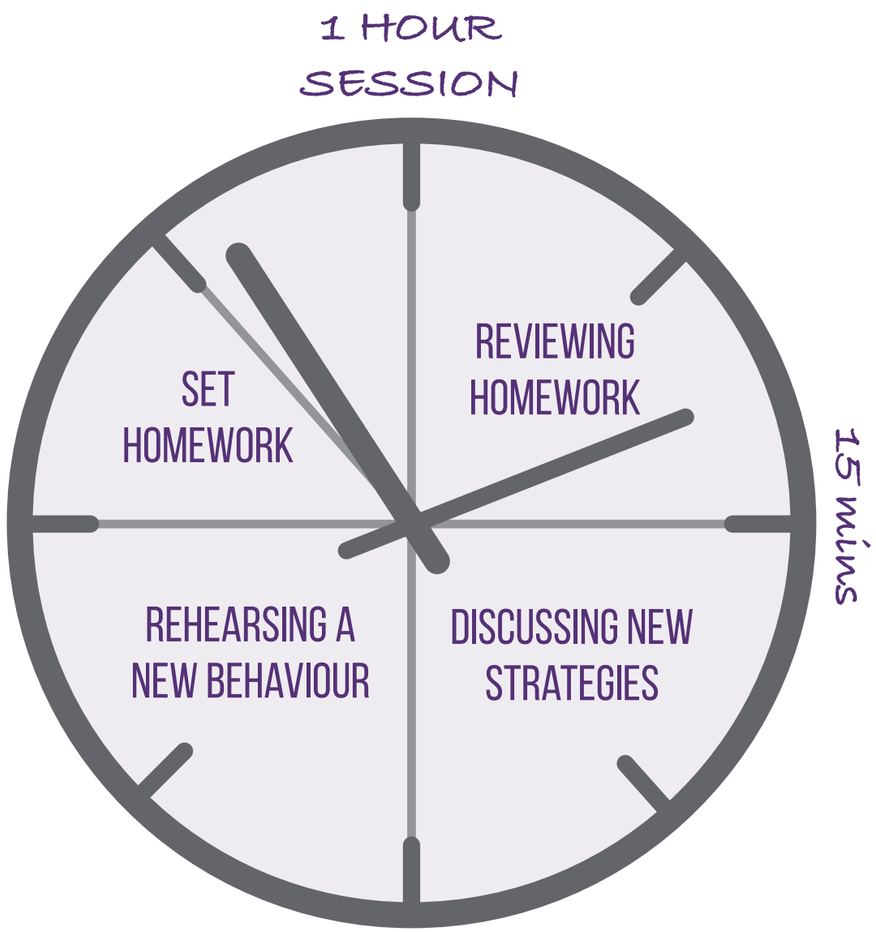


Figure 7. Example of a Session-Planning Clock

COGNITIVE REHABILITATION

The recovery of CI can be time-dependent. It can occur naturally following the cessation of drinking in the absence of targeted cognitive remediation, or it can be experience-dependent (i.e. practicing cognitive tasks to strengthen cognitive function). Two decades of cognitive neuroscience in addiction has identified numerous altered cognitive functions and processes that may be suitable targets in addiction treatment [247]. This has led to a proliferation of studies trialling cognitive rehabilitation or training interventions which aim to restore or strengthen cognitive functioning. According to the 'dual process model', addiction arises as a result of two independent but inter-related information processing systems; an underactive 'top down' reflective or executive control system and an overactive impulsive 'bottom-up' motivational system [67, 248, 249]. Over time, as top-down, reflecive or executive control processess weaken as a result of neurotoxic damage to the pre-frontal cortex, behaviour becomes increasingly governed by automatic, impusive (bottom-up) processess. Various cognitive training techniques have been developed to correct these altered processes, by strengthening executive functioning (cognitive control) and more recently, by dampening automatic impulsive responding to alcohol or drug cues. However, despite promising results, their implementation in clinical practice has yet to occur. This is partly due to the fact that whilst improvements in cognitive functioning are often observed, improved substance use outcomes are often not reported or even assessed [250]. Nonetheless, as evidence for their effectiveness continues to build, it is likely that cognitive training will become more widely available as an adjunctive approach in addiction treatment.

Traditionally cognitive rehabilitation has taken two theoretical approaches; compensatory and restorative.

Compensatory techniques draw on preserved cognitive skills, using external cognitive aids (e.g., electronic diaries) and tend to be used more in populations with more severe CI or TBI. In contrast, cognitive rehabilitation has the goal of restoring impaired cognition, at least partially. Within a mental health context, cognitive rehabilitation (CR) is also known as cognitive enhancement therapy (CET) and cognitive remediation therapy (CRT), and were first applied to patients with schizophrenia with the aim of ameliorating associated attention, memory and executive functioning (EF) deficits [251]. CR techniques often entail a 'drill and practice' approach, with repetitive and adaptive cognitive exercises targeting multiple domains analysed in neuropsychological assessment. Current CR approaches can be broadly divided into the following categories: working memory training, inhibitory control training, cognitive bias modification, and those training procedures targeting multiple cognitive processes such as executive functioning (EF) and self-regulation (e.g., Goal Management Training).

In a therapeutic community in Australia, women who received CR showed significantly better inhibition, as well as ratings on a self-reported assessment of executive functioning (BRIEF-A), self-control and quality of life, relative to a control group (treatment as usual). CR was delivered over 12×2-hour group sessions across 4 weeks and combined strategy training, (modelling, exercises to demonstrate concepts, and role-plays), computerised working memory training and self-awareness training, goal management training and multifaceted treatment of executive dysfunction [189].

WORKING MEMORY TRAINING

Computerised working memory training (WMT), an example of a 'top-down' training, requires participants to repeatedly manipulate and recall sequences of shapes or numbers which increase in difficulty over time (i.e., adapt to the individual's performance level). WMT aims to extend WM capacity, so individuals can better integrate, manipulate, and prioritize important information, enabling them to make healthier, goal-aligned decisions (e.g., relating to their substance use). Training usually takes place several times a week over several weeks (on average 25 weeks) during which multiple WM domains are trained (i.e., N-back, mental arithmetic, letter number updating, complex WM span). Commercial computerised working memory packages (Cogmed, Cogpack, Lumosity, BrainHQ, mHealth, etc.) are available for purchase online, many in app-form, but the evidence of their impact on substance use outcomes is limited. Whilst research suggests that WMT has near-transfer effects (i.e., improves performance on similar WM tasks), it rarely generalises to other measures of WM or EF (far-transfer effects), or substance-use outcomes [252]. One study found that among heavy drinkers, WMT reduced alcohol use 1-month after training [253], but these reductions were not clinically significant. Another study found that WMT reduced delay-discounting among people using alcohol [254], although this finding has not been replicated in studies of clients receiving treatment for illicit drug use disorders (including methadone maintenance cannabis, methamphetamine, poly-drug use), and most importantly, has been found to improve substance use outcomes [255-258]. Whilst there is insufficient evidence to recommend WMT as an adjunctive approach at present (particularly given their cost and intensity), clients may enjoy training and feel they are actively engaged in restoring their brain health as a pathway to recovery.

RESPONSE INHIBITION TRAINING

Poor inhibitory control is associated with heavier substance use, and therefore inhibitory control training (ICT) aims to strengthen inhibitory control by repeatedly practicing inhibiting a previously learned motor responses to specific stimuli. The most commonly used training tasks are the go/no-go (GNG) and the stop signal task (SST) where participants are trained to make a response ('Go') e.g., by pressing a certain key on a keypad every time they view a non-AOD cue and do nothing ('No Go' or 'STOP') (i.e., inhibit a 'Go' response) when they view an AOD cue/stop-signal. In one study, a beer GNG training task significantly reduced weekly alcohol intake relative to those trained towards 'beer' stimuli in a university non-treatment seeking student sample, but there have been only a few studies on treatment-seekers with AUDs, with mixed findings [259-261].

COGNITIVE BIAS MODIFICATION

Cognitive biases are thought to arise from the pairing of the rewarding effects of alcohol and drugs with sensory cues [262]. These automatic cognitive biases can include 'attentional bias', the tendency for our attention to be specifically captured by drug-related environmental cues [263], and 'approach bias', the automatic action tendency to move towards (approach) drug cues [264]. Cognitive Bias Modification (CBM) is a computerised training approach that aims to re-train these automatic responses to appetitive cues. Attentional bias re-training requires participants to repeatedly shift their attention to neutral or positive (non-drug) cues and away from drug-related cues. Among treatment seekers with AUDs, five or more sessions of AtBM has been shown to significantly decrease relapse rates, or time to relapse relative to a control condition [265, 266]. However AtBM appears to be less effective at reducing attentional bias and reducing substance use in people seeking treatment for heroin, methamphetamine and nicotine use disorders and cocaine use disorders [267-269].

During approach bias re-training (ApBM), clients repeatedly practice "avoiding" substance-related images displayed on a computer screen by "pushing" these images away (i.e., beer, wine) and "pulling" healthy alternatives towards themselves (i.e., images of non-alcoholic drinks). Research with treatment-seekers has shown that doing this over multiple trials across 4 or more sessions can change an alcohol approach bias into an alcohol avoidance bias. ApBM has been shown to significantly reduce early relapse rates following inpatient alcohol withdrawal in a multi-site Australian RCT [270], and by 8-13% one-year after training during rehabilitation treatment in international research [265, 271, 272]. ApBM has also shown promise in a number of tobacco smoking cessation trials [273-275], and in a recent pilot RCT on individuals with cannabis use disorder [276], and may be a useful adjunctive intervention during treatment for methamphetamine use disorders [277].

GOAL MANAGEMENT TRAINING

In contrast to self-administered computerised training programs, goal management training (GMT) focuses on improving goal-directed behaviours using a therapist assisted one-on-one or group intervention. GMT addresses executive functioning deficits (i.e., failure to stick to intentions and enact plans), and aims to emphasise the link between decisions/actions and their outcomes [278]. It trains executive function skills and attention over approximately 8 x 2-hour sessions. Importantly, the focus of GMT is on the transfer of these skills to goal-directed tasks in the context of real-life situations. In a study by Alfonso et al. (2011) combined GMT and mindfulness led to improvements in WM (letter-number sequencing task), decision-making (Iowa Gambling Task) and response-inhibition/cognitive control (Stroop Task) in polysubstance 'abusers' [279]. It is currently unclear whether GMT is effective at reducing AOD use or relapse rates among those with substance dependence, whether improvements in cognitive functions as observed on cognitive test generalise to every-day functioning (far-transfer), or whether these changes persist long-term. GMT is an emerging approach in the addictions field with some preliminary positive findings that may lead to future research on its effectiveness.

The GMT procedure comprises 5 steps:

1 **STOP**
Orient awareness toward the facts of the situation

2 **DEFINE**
The goal of the task

3 **LIST**
The task into sub-steps

4 **LEARN**
The steps

5 **CHECK**
If the results of the present action correspond to the stated goal

BRAIN STIMULATION METHODS

Observed changes in consumption behaviours among patients treated for neurological disorders has led to an interest in the role of brain stimulation in the treatment of substance use disorders, particularly in terms of reducing impulsivity. Transcranial direct current stimulation (tDCS) is a well-tolerated, non-invasive brain stimulation technique in which a weak electrical current is applied for 10-30 mins through electrodes placed on the skull, which then alters brain activity [280]. Although the mechanisms underlying the treatment are not well understood, it is thought that stimulation of the dorsolateral prefrontal cortex (DLPFC) enhances working memory [281], reduces impulsivity, and may reduce craving for drugs [280, 282].

A recent systematic review concluded that tDCS may be a promising intervention to help reduce craving in response to drug cues [283]. Stimulation of the anterior cingulate cortex has been shown to significantly reduce cocaine cue-elicited activation relative to a sham-stimulation control condition [284], whilst tDCS stimulation significantly reduced cue-elicited heroin craving [285]. Importantly, tDCS of the DLPFC with tobacco smokers over a 5-day period significantly decreased the number of cigarettes smoked relative to sham-stimulation [286]. Despite these promising findings, there has been little research on the long-term effectiveness of tDCS and negative findings have been reported (e.g., with no reductions in alcohol 3 months after the treatment) [287]. Researchers have started examining the impact of combining tDCS with computer-administered cognitive training tasks, with evidence suggesting that this approach may lead to improvements in attention and working memory [288]. Consequently, future cognitive training interventions may include tDCS to enhance the effectiveness of standard cognitive training approaches.

HEART RATE VARIABILITY BIOFEEDBACK

Heart rate variability biofeedback (HRV-BFB) training involves rhythmic stimulation of the cardiovascular system through paced breathing at a rate of approximately 6 breaths per minute [289, 290]. HRV-BFB has been shown to improve symptom severity in disorders characterised by autonomic nervous system dysfunction (i.e., anxiety, depression, post-traumatic stress disorder) [291-293]. Research has found that those with mild to severe TBI often display decreased HRV [294, 295], and those with lower levels of HRV often display poorer functioning on cognitive tests, particularly sustained attention and working memory [296]. These studies suggest that HRV-BFB may be an effective neurocognitive enhancement mechanism [297]. HRV-BFB has been shown to improve neurocognitive performance by enhancing focus and visual acuity, and by promoting emotional regulation needed for peak performance [298]. A recent study found that 30-minute biofeedback sessions administered to healthy older adults daily, over a three-week period significantly increased attentional skills, however long-term effects were not studied [299].

PHYSICAL ACTIVITY

Physical activity, especially aerobic exercise (cardio intensive activities such as brisk walking, running, dancing, yoga, pilates, etc.) has been associated with increased cognitive functioning in healthy and older adults, particularly in areas of memory and learning, with some evidence that it leads to improved cognitive control, inhibition and cognitive flexibility [300]. Physical exercise can also cause a range of structural changes particularly in the hippocampus and pre-frontal cortex (through neurogenesis, increased gray matter volume, enhanced cerebral blood flow & oxygenation, etc.) which has been associated with improvements in daily functioning, wellbeing and sleep quality [301-303].

There is some evidence that physical exercise may also be an effective intervention among those with mild to moderate TBI [302]. A recent systematic review suggests there is a positive effect of physical exercise on global cognitive functioning with an exercise program of 8 or more weeks producing better and more sustained improvements [302]. A recent single-blind RCT assessed the efficacy of a daily 12-week yoga intervention compared to other forms of physical exercise among 96 male participants (aged 18-40 years) in a residential treatment unit [304]. Significant improvements in memory performance were observed in both the yoga and exercise group 12-weeks post-intervention though inhibition/cognitive control was significantly better in the yoga compared to the exercise group. Importantly reductions in substance use have been observed following physical exercise during an AOD treatment program [304].

COGNITIVE ENHANCING DRUGS

Cognitive enhancers are drugs that are associated with alertness, concentration, wakefulness, energy levels, executive functioning and creativity, also known as nootropics, or 'smart drugs'). This includes stimulant-type medications, prescription medications (such as Modafinil); used to treat narcolepsy, and Ritalin (methylphenidate) used to treat attention deficit disorders as well as non-stimulant (non-prescription) agents such as Ginkgo biloba. The hypothesized mechanisms of action include increased cerebral blood flow, increased adrenalin or a direct impact on neurotransmitters (e.g., acetylcholine and norepinephrine). Whilst there are some studies suggesting these medications may reduce substance use [305], there is limted evidence of a direct beneft on cognition [306, 307]. This, combined with their short-lived effects, risk of dependence and potential side-effects, means cognitive enhancers are not recommended as an approach to addressing CI in people with SUDs. Nonetheless, a diet rich in B vitamins, fish oil and herbal supplements may offer a safer approach to improved cognition, though again, research in this area remains limited.

APPENDICES

APPENDIX A: COMMON SCREENING TOOLS – ARE THEY APPROPRIATE?

COGNITIVE SCREEN	SUMMARY OF THE SCREEN	APPROPRIATE TO USE IN AN AOD SETTING?	COST & TRAINING	TIME (MINS)
Mini-Mental State Examination (MMSE) ⚠	The MMSE [177] is the most widely used test for CI. However, it was originally developed as a screen for dementia in older adults. A score on the MMSE <25 indicates CI, 21-24 mild impairment, 10-20 moderate impairment, and <10 severe impairment. The MMSE consists of 30 questions taking approximately 10-15 minutes to administer, and 5 minutes to score.	Insensitive to the type of CI common among AOD treatment seekers (due to the absence of items assessing executive functioning impairment) and is not recommended [238, 308].	$80-$100 Does not require specialised training or equipment	10-15
Addenbrooke's Cognitive Examination (ACE-R) ✓	The Addenbrooke's Cognitive Examination (ACE) was originally developed to assess the omissions of the MMSE (with items assessing visuo-spatial functioning, memory and verbal fluency). The revised version of the ACE, the Addenbrooke's Cognitive Examination-Revised [179], includes the provision of three alternate forms and allows derivation of five sub-scores (orientation, registration, attention and concentration, recall, verbal fluency, language, visuospatial abilities, perceptual abilities) each representing a specific cognitive domain. The total score of the ACE-R is 100, with higher scores indicating better cognitive functioning. A score less than 88 (94% sensitivity and 89% specificity) or 82 (84% sensitivity and 100% specificity) is indicative of dementia.	The ACE–R has not been validated as a screen for CI for AOD clients. However, a recent study of older (50+) service users reported that 65% scored below the cut-off score [309], with significant correlations between ACE–R total score and functional outcomes (i.e., mental health). Together, these findings suggest that the ACE–R is a viable option as a screening measure for CI in SUD populations [181].	Free Does not require specialised training or equipment	

Montreal Cognitive Assessment (MoCA) 	The MoCA is a one-page paper-based tool specifically developed for the quick assessment of mild CI, with tasks assessing a range of cognitive abilities including memory, visuospatial abilities, executive function, attention and concentration, language, and orientation. The MoCA has been shown to have high reliability and good internal consistency [310]. Total scores are derived from adding subscale scores and adding one point for less than 12 years of education. The MoCA recommends that a score equal to or more than 26 indicates normal cognitive functioning (with a maximum score of 30), while a score of 25 or less indicates impairment. However, some studies suggest that lower cut-off scores may be more appropriate in ethnically diverse/populations with English as a second language [311].	Copersino et al. (2009) [32] found that the MoCA showed acceptable sensitivity (83.3%) and specificity (72.9%) for the identification of CI in substance dependent adults (18- 65 years) with a cut-off of 25 or less indicating impairment. Wester et al. (2013) [180] reported good sensitivity (91%) and specificity (88%) at detecting alcohol-related CI at scores of equal to or less than 24.The MoCA has been shown to be superior to the MMSE among people undergoing inpatient withdrawal treatment [238].	The tool is free and easily available online. From September 1st 2020, clinicians undertaking formal diagnosis will be required to undergo a 1 hour long training and certification program available online at www.mocatest.org/	10
Kimberley Indigenous Cognitive Assessment (KICA) & Urban Modified KICA 	The KICA is validated with Indigenous Australians older than 45 years. Items on the KICA-Cog assess orientation, recognition and naming, registration, verbal comprehension, verbal fluency, visual naming, recall, executive function (only one item), and praxis. Emotional wellbeing and medical history are also assessed, and family and community members are involved in the process. A score of 33/39 and below on the KICA-Cog indicates possible dementia. The KICA is validated on Indigenous Australians from the Kimberley and Northern Territory, while the Urban Modified KICA was developed for Indigenous Australians living in urban and regional settings [182].	The KICA includes a section on 'Smoking and Alcohol History' – acknowledging the high incidence of AoD problems in some indigenous communities, and contextualises any impairments that might be found.	Free Formal training is not required, some equipment is needed (a comb, cup, matches, plastic bottle, timer) but an instruction booklet is provided and cultural awareness training is recommended. A qualified interpreter will be necessary for some clients, which may increase the cost.	20

Brief Executive Function Assessment Tool (BEAT)	The BEAT is a 20-item measure designed to specifically assess cognitive impairment in substance use disorder populations [184]. Items assess domains such as visuoconstruction, learning and memory, attention, working memory, motor sequencing, reading, naming and aspects of executive functioning including self-reported concerns. A total score is calculated out of 60, with cut-off scores between 17 and 30 indiccative if mild impairment, whilst a cut-off score of 16 or below is indicative of severe impairment.	The BEAT has been specifically developed and validated for use in AOD-using populations and has demonstrated excellent discriminative ability and good sensitivity and specificity as a screening tool for detecting cognitive impairment in these populations.	Free Training and professional supervision with a psychologist is highly recommended to ensure appropriate administration and interpretation.	20

APPENDIX B: ACCESSING SUPPORT FOR TBI

TBI AS A RESULT OF A ROAD/MOTOR-VEHICLE ACCIDENT

Road related accidents in Victoria are covered by the Transport Accident Commission's (TAC's) no fault insurance. This means clients may be eligible to receive funded rehabilitation and services through private agencies and may be eligible to receive financial compensation. This is an important consideration as hospital/rehabilitation admissions due to head injury in this context would generally trigger referral for neuropsychological assessment, and so clients may have already had an assessment, if not several, over the course of their recovery. Clients should be referred to a solicitor who can advocate for them if there are any outstanding matters to be addressed relevant to compensation.

TBI RESULTING FROM A WORK RELATED INCIDENT

Work related injuries should be insured by WorkCover, including any relevant investigations or necessary rehabilitation. Again, it is recommended that a clinician explore any prior assessments undertaken in this context and clients should be referred to a solicitor if there are any outstanding matters to be addressed relating to these entitlements.

APPENDIX C: ACCESSING FUNDING & SUPPORT FOR CI

NATIONAL DISABILITY INSURANCE SCHEME (NDIS)

The NDIS is an Australia-wide Government scheme which provides funding directly to people with disability (including CI) to enable them to access various support services. For more information, call 1800 800 110 (free call). The following information is derived from the National Disability Insurance Scheme (Becoming a Participant) Rules 2016 (these requirements may be subject to change).

WHO IS ELIGIBLE?

To eligible for support your client must establish that their CI constitutes 'a permanent condition requiring support (i.e., assistive technology, or support from other people) for daily functioning throughout their lifetime (demonstrated reduced functional capacity). Whether or not your client meets this require will be informed by a thorough assessment.
Your client must also:

- Live in an area where the NDIS is available (available online);
- Be an Australian citizen, permanent resident or hold a protected special category visa;
- Be aged under 65 (at time of request);

HOW CAN YOUR CLIENT ESTABLISH A PERMANENT CONDITION?

According to the NDIS, "an individual may be eligible to become a participant of the NDIS where CI affects their ability to participate at home, at school, at work and/or in social situations".

A CONDITION

The client must meet all of the following:

- One or more intellectual, cognitive, neurological, sensory or physical impairments, or one or more impairments attributable to a psychiatric condition; &
- The person's impairment or impairments are, or are likely to be, permanent (see below); &
- The impairment or impairments result in substantially reduced functional capacity to undertake, or psychosocial functioning in undertaking, one or more of the following activities: communication, social interaction, learning, mobility, self-care, self-management; &
- The impairment or impairments affect the person's capacity for social and economic participation; &
- The person is likely to require support under the NDIS for the person's lifetime.

REDUCED FUNCTIONAL CAPACITY

An impairment is evident when a person has reduced functional capacity to undertake one or more relevant activities necessary for daily life i.e., communication, social interaction, learning, mobility, self-care, self-management. To establish a reduced functional capacity, the client must establish one of the following:

- An inability to participate effectively or completely in the activity or perform tasks or actions required to undertake or participate effectively or completely in the activity without assistive technology or equipment or home modifications; or
- Requires assistance from others to participate or perform tasks associated in participation; or
- The person is unable to participate or perform tasks even with assistance of any kind.

PERMANENCY: A DIFFICULT CRITERION?

In order for a condition to be considered permanent, there are specific criteria that must be met. Examples include:

- The impairment must persist to a hindering degree for the duration of the persons' natural life
- There must be no known, available or appropriate evidence-based clinical, medical or other treatments that would be likely to remedy the impairment
- Specialist consultation as well as medical and neuropsychological assessments will be required before a determination on permanency can be made. One difficulty in establishing permanency in the context of CI is that a condition will only be considered permanent if "the impairment does not require further medical treatment or review in order for its permanency or likely permanency to be demonstrated". It is commonly (but not always) the case that clients with CIs will require repeat assessment and treatment over time in order to determine whether impairments are likely to be permanent.

EARLY INTERVENTION SUPPORT: MOST RELEVANT TO COGNITIVE IMPAIRMENT & AOD

Even if it is difficult for you and your client to establish that your clients' impairments are likely to be permanent, your client may still be eligible for 'early intervention support' if they are able to demonstrate that this support is likely to benefit the person by reducing the persons' future needs for support in relation to disability in terms of any of the following:

- Mitigating or alleviating the impact of the persons' impairment upon the functional capacity of the person;
- Preventing further deterioration of functional capacity;
- Improving functional capacity;
- Strengthening the sustainability of informal supports available to the person, including through building the capacity of the persons' carer.

REGISTERED NDIS PROVIDERS: SUPPORT SERVICES FOR CLIENTS WITH ABI AND THEIR CARERS

Melbourne City Mission

MCM provides a variety of support services for those with ABI and their carers. Support services include assistance in performing every-day activities and skill-building, independent living support, family assistance, support coordination and community linkages.

For more information visit: **www.melbournecitymission.org.au/services/disability-services/acquired-brain-injury.**

BrainLink

BrainLink Services is a Victorian based service that provides a first point of call for people and their families living with ABI, peer support programs, specialist case management, comprehensive information and resources and referral services to other disability services. For more information visit: **http://www.brainlink.org.au**

APPENDIX D: SCREENING FOR MENTAL/PSYCHIATRIC HEALTH ISSUES

DOMAIN	SPECIFIC QUESTIONS
Psychiatric condition/s	Are you currently or have you previously been diagnosed with a psychiatric disorder?
Current mood	Could you rate your current mood between 1-10, with 1 being awful and 10 fantastic? • Administer the Kessler 10 [312], a simple measure of psychological distress (available online). • Administer the Depression, Anxiety and Stress Scale (DASS-21) [313], a 21-item self-report scale designed to measure the emotional states of depression, anxiety and stress (cut-off scores included).
Periods of hospitalisation / psychiatric care	Have you ever been admitted into psychiatric care/hospital? Have you ever seen a psychiatrist/ psychologist/mental health counsellor? If yes when, why and how often? What therapeutic interventions are you currently receiving?
Presence of any Hallucinations/ delusions/ paranoia	Have you ever experienced delusions or hallucinations (auditory or visual)? • Have you/do you hear voices when no one else is around? • Have you/do you have ideas that other people might think/have thought were concerning or unusual? Does your client think they have special powers/are in a positon of power, or are being persecuted (when they definitely do not or could not be)?
Risk to oneself or others	Use your organisation's risk assessment tool, administer a suicide risk assessment – for example, the Australian PsyCheck screening tool [314] which is designed for use by non-mental health specialists (available online). **The Australian PsyCheck Screening Tool advises that a client is at high risk of self-harm if some of the following are true:** • A history of repeated attempts at self-harm • A recent attempt at self-harm • A clear plan of action and access to lethal means • A close friend or family member who has attempted or completed suicide • Concurrent depression and/or hopelessness • Concurrent untreated psychotic symptoms • Significant losses and/or stressors (past or present) • Recent discharge from hospital or separation from a trusted practitioner **A client is more likely to be at risk to others if they are:** • Have a criminal history, particularly involving weapons • Have a history of child abuse or mistreatment by others • Have expressed a verbal intent to harm others • Have paranoid thoughts about others • Have a preoccupation with violent images and thoughts • Have access to lethal means • Have high levels of anger, frustration or agitation • Lack of problem-solving skills • Display current role instability (for example changes in work, relationship or accommodation circumstances)

APPENDIX E: VICTORIAN NEUROPSYCHOLOGY AND DISABILITY SERVICES

The Turning Point Neuropsychology Service is located in Richmond, Victoria and provides secondary consultation services to assist clinicians in identifying the support needs for clients and any recommendations that could be immediately implemented in addition to whether formal neuropsychological assessment is required. If formal assessment is indicated, the service offers funded neuropsychological assessments and feedback for clients.

Visit **www.turningpoint.org.au/treatment/clinicians/neuropsychology** for more information and instructions on how to make a referral.

Other neuropsychology services include:

CBDATS (the Community Brain Disorder's Assessment and Treatment Service at Austin Health).

- www.austin.org.au/bdp/bdpcs/
- Telephone: (03) 9490 7366
- Email: BDPV@austin.org.au
- Fax: (03) 9490 7358

Diverge is a private Neuropsychology service that specialises in behavioural support.

- Ph 9329 4330 https://diverge.org.au/

The university based clinics below also offer neuropsychology services for nominal fees:

Monash Psychology Centre

- www.monash.edu/turner-institute/research-clinics/mpc
- Telephone: +61 3 9902 4480
- Fax: +61 3 9905 0605
- Email: mpc@monash.edu

La Trobe Psychology Clinic

- www.latrobe.edu.au/psychology-clinic
- Telephone: (03) 9479 2150
- Email: psych-clinic@latrobe.edu.au

The University of Melbourne Psychology Clinic

- psychologicalsciences.unimelb.edu.au/psychology-clinic/home
- Telephone: (03) 9035 5180

If your client is a current hospital patient, they may be able to access hospital based neuropsychology services. Otherwise, APS find a psychologist www.psychology.org.au/Find-a-Psychologist is a resource for locating private providers if the client has funding.

Other disability services include:

- If your client has an Intellectual Disability and Mental Health problems The Victorian Dual Disability Service (VDDS)
- www.svhm.org.au/our-services/departments-and-services/v/victorian-dual-disability-service
- Where: St Vincent's Hospital Melbourne
- Telephone: (03) 9231 1988
- Email:vdds@svha.org.au

The Multiple and Complex Needs Initiative is a government funded service for people with multiple and complex needs including Mental illness, substance abuse, intellectual impairment, ABI or Forensic issues.

- Disability services intake and response: 1800 783 783
- Email disability.services@dhhs.vic.gov.au

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