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ARBD Network and Alcohol Change UK Conference  
*"Issues in the Support and Management of ARBD"*

Warning signs, recognition and diagnosis

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Why are signs so often missed or undetected?

"Precisely because, as Kiper shows, the healthy brain has evolved to automatically attribute to other people the existence of a self that is sustained over time, has self-reflective capacities, and is capable of learning and absorbing new information. This attribution is the brain's unconscious default position, or cognitive-emotional bias, and does not simply disappear when we become caregivers for people whose own brains begin to falter. It is the invisible projection upon which each human encounter begins, a projection that is implicit in our every conversation and even in the structure of human language itself."

Dodge, Norman [Foreword to] Kiper, D. (2023) "Travelers to Unimagable Land", London: Profile Books

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A Methodological Approach

- Awareness of possible warning signs - things do not always mean what we think!
- Collateral information is invaluable – physical, psychological, social
- History of contacts with services (if any) and what do they tell us?
- What other factors may be impacting cognition (differentials)?
- Demographics – what can they tell us?
- Nutrition, gait problems, neuropathy, thiamine?
- Possible Impacts of alcohol across the life course.
- The role of thiamine and the complications of thiamine deficiency
- Wernicke's encephalopathy and the Caine Criteria
- Brain Imaging
- Cognitive assessment

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ARBD? – Possible Early Indicators

Psychological	Physical	Social
<ul style="list-style-type: none"> <li>• Poor concentration</li> <li>• Difficulty in coping with change</li> <li>• Apathy</li> <li>• Impaired cognitive functioning</li> <li>• Anxiety</li> <li>• Depression</li> <li>• Suspicion</li> <li>• Confabulation</li> <li>• Confusion and disorientation</li> </ul>	<ul style="list-style-type: none"> <li>• Poor co-ordination</li> <li>• Sensory impairment</li> <li>• General 'clumsiness'</li> <li>• Parasthesia (pins and needles)</li> <li>• Eye problems</li> <li>• unsteady gait</li> <li>• Signs of malnutrition</li> </ul>	<ul style="list-style-type: none"> <li>• History of heavy drinking (&gt;10years+)</li> <li>• Relationship difficulties</li> <li>• Lost contact with family</li> <li>• Isolation</li> <li>• Financial problems</li> <li>• Tenancy problems</li> <li>• Unemployment</li> <li>• Behaviour changes</li> </ul>

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ARBD? – Gathering Collateral Information

Psychological	Physical	Social
<ul style="list-style-type: none"> <li>• History of mental health problems or traumas?</li> <li>• Pre-morbid coping?</li> <li>• Awareness of drinking risks and need to change?</li> <li>• Does the person feel they have any difficulties or problems?</li> <li>• Do they know why we have been asked to speak to them?</li> <li>• How is their mood?</li> <li>• Describe their memory problems?</li> </ul>	<ul style="list-style-type: none"> <li>• History of seizures?</li> <li>• If diagnosed with epilepsy - compliant with meds?</li> <li>• Current prescribed meds.?</li> <li>• History of admission to acute (if yes – why)?</li> <li>• Any falls, accidents, specifically head injuries?</li> <li>• History of overdose or periods of unconsciousness?</li> <li>• Brain imaging recent or past?</li> </ul>	<ul style="list-style-type: none"> <li>• Who supports at this time and what types of relationships are they?</li> <li>• Who can we contact?</li> <li>• Have they ever been known to services - and if so to who?</li> <li>• Does he manage his money, ADL's, tenancy?</li> <li>• What is their diet like?</li> <li>• Any illicit or prescribed drug use?</li> <li>• Work/school history?</li> </ul>

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Glasgow and Scotland– Demographics

- **Gender:** 76% of referral to the Glasgow ARBD Team are male, 24% of the referrals are female.
- In 2022, there were alcohol-specific deaths in Scotland – (866 men and 440 women) (2)
- In 2021 men were 2.3 times more likely than women to be admitted to acute hospitals (2)
- **Post codes :** in 2022 Alcohol-specific death rates were 4.3 times higher in the most deprived areas than those in the least deprived (2)
- Hospital admission rates were six times higher for those living in the most deprived areas as compared with the least deprived areas. (2)
- 1 in 15 of the total deaths in Scotland in 2015 were caused by alcohol (2)

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### History of Service Contacts?

- If there is a history of “poor engagement”, it is important to look into the reason(s) for this.
- Thiamine deficiency alone can cause apathy and low mood, but is there any chance that poor engagement is about **frontal pathology**, as opposed to not being motivated?
- *“This has major implications for treatment outcome, given the emphasis of many treatment programs on motivation to change, and the possibility that impaired brain functioning as a result of alcohol use may prevent some from engaging with standard treatment programs” (Svanberg et al, 2015) (1)*
- *“It may be thought that they are “poorly motivated” or are “pre contemplative” about their addiction.” (Bell & Craig, 2013)*

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### Other possible causes of Cognitive Problems?

- Misuse of benzodiazepines can cause cognitive impairment, and benzo’s are a complicating factor for accurate cognitive assessment.
- Brain injury (e.g. falls / fights / injuries / road traffic accidents) and stroke can cause a range of cognitive *and* physical difficulties
- Hypoxic brain injury can result after short or long term loss of consciousness – may not have been treated (e.g. overdose / assault) or may have been part of treatment (e.g. intubation in ICU)
- Any history of seizures should be investigated
- Diagnosis of ALD, cirrhosis, hepatic encephalopathy?

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### Glasgow ARBD Team – Age Demographics

Total Referrals		Males by Decade		Females by Decade	
20's	0.5%	20's	0.166%	20's	0.56%
30's	5.28%	30's	4.49%	30's	7.26%
40's	19.05%	40's	18.136%	40's	22.23%
50's	36.95%	50's	37.853%	50's	33.98%
60's	30.95%	60's	30.948%	60's	30.85%
70's	7.01%	70's	7.826%	70's	4.78%
80's	0.45%	80's	0.499%	80's	0.275%
90's	0.06%	90's	0.08%	90's	0.00%

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### Nutrition / Gait Problems / Neuropathy

- BMI is helpful as an indicator of nutritional intake, but also provides a strong indicator in relation to harm reduction and Pabrinex intervention.
- Asking questions about pins and needles, numbness in feet or fingers, or established neuropathy provides an opportunity for harm reduction and intervention, as well as an indicator of CNS damage that is often associated with ARBD.
- Disturbance in gait or poor balance is often an indicator of cerebellar damage, but this may be attributable to other possible causes. In the context of these questions and this service user population, it should be an indicator for further assessment / discussion.

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### Alcohol and Risks across the Life Course

Pre-natal exposure:

- “Individuals exposed to alcohol during pregnancy exhibit a wide range of long-lasting impairments in neuropsychological and behavioural domains. Deficits include **diminished intellectual function, poor learning and memory, impaired executive and visual-spatial function**, delayed motor and language development, and attention difficulties.” (p16) (3)
- “Alcohol abuse in pregnancy is frequently associated with poor nutrition that can potentially increase brain damage.” (p5). (4)

Alcohol and Adolescence:

- “Deficits in **attention, memory, and executive functioning** are apparent in adolescent substance users, and are associated with alterations in **prefrontal, hippocampal, and cerebellar** structure and function as well as poor white matter integrity.” P207 (5)

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### Alcohol and Risks across the Life Course

Alcohol and Adolescence:

- “Nevertheless, we can cautiously conclude that elevated impulsivity and **poor executive function precede the onset of alcohol involvement**, and they place the individual at increased risk of the development of alcohol or other substance problems later on in life.” (6)

Alcohol into Adulthood:

- “Even people close to an individual who is alcohol dependent may not recognize changes in cognitive functioning, which develop gradually. Insidious development of disability could thus make estimation of baseline or maximal functioning difficult, if not impossible, for treatment providers.” (p195) (7)
- “Thus, a long history of exposure to alcohol or chronic and heavy alcohol consumption may decrease the brain’s plasticity (and thus its capacities for reversibility).” (p7) (8)
- “While abstinence is shown to reverse some of the structural damage and cognitive decline within a year of sobriety, residual impairments are

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### Accumulating Risk of Cognitive Damage

- Alcohol helps to reduce thiamine intake
- Alcohol impacts on organs which process and absorb thiamine
- Alcohol also uses up any available thiamine to metabolise
- Thiamine is water-soluble / alcohol has a diuretic effect
- Alcohol keeps therefore causes and maintains thiamine deficiency
- Continued dependent drinking therefore increases risk of Wernicke's encephalopathy and cognitive damage

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### Wernicke's Encephalopathy

*"Wernicke's encephalopathy (WE) is an acute neuropsychiatric condition due to an initially reversible biochemical brain lesion caused by overwhelming metabolic demands on brain cells that have depleted intracellular thiamine (vitamin B1)." (p151) (10)*

The Caine Criteria (11)

- Traditionally, there were a triad of signs recognised for a diagnosis of WE – altered mental status, ataxic gait, and ophthalmoplegia. (All three signs less commonly seen simultaneously)
- Caine et al (1997) then modified the triad "...so that the diagnosis only required any two of the following signs; eye signs, cerebellar dysfunction, altered mental state, and dietary deficiencies." (p57)
- Caine et al found that the sensitivity of these criteria for the accurate diagnosis was very significantly increased

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### Thiamine and the CNS

- Thiamine enters the body in many of the foodstuffs we consume
- From the intestine it makes it way into the cells which line the intestine
- Those cells facilitate it's transport into the blood stream
- Blood then transports thiamine to tissues and cells – such thiamine as reaches the brain thus crosses the blood brain barrier
- Within the cells, thiamine is transported to mitochondria (energy centres) and to the nucleus where it actually regulates gene activity

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### Thiamine and the Cerebellum (12)

- Studies have shown that the cerebellum is particularly sensitive to thiamine deficiency
- Causes a reduction in size and number of Purkinje cells in the **cerebellar vermis**
- Neural pathways lead from the cerebellum through the basal ganglia and thalamus to the frontal lobe
- Alcohol damage to the cerebellar vermis **could indirectly affect certain frontal lobe functions**, possibly even before damage is detectable

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### Brain Regions Impacted by Alcohol and TD

Region/system	Impact	function
Cerebrum	Frontal and parietal atrophy	Planning, problem solving, self reflection etc
Limbic system, e.g. hippocampus	Reduces connections	Memory and emotion
Cerebellum	Atrophy	Movement, gait, also cognition (link to frontal lobes)
Neurotransmitter systems	Several systems affected	Involved in communication throughout brain

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### Brain Imaging (M.R.I.)

Healthy Control

Alcohol Damage

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## Lesions Caused by B1 Deficiency (13)

Lesion- "a region in an organ or tissue which has suffered damage through injury or disease, such as a wound, ulcer, abscess, or tumour"

- Found in notable patterns around mammillary bodies, hypothalamus, thalamus, periaqueductal grey matter, colliculi and floor of fourth ventricle
- Lesions to colliculi and floor of fourth ventricle are seen to cause eye and brainstem signs
- Lesion to the medial dorsal nucleus of the thalamus can cause memory problems
- Bilateral hippocampal damage directly causes Korsakoff's amnesia

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## A Process to a Diagnosis of Exclusion

- Assemble all collateral information and speak to relatives / carers / friends
- Assemble preliminary investigative assessment – records / service history / details of neuro-imaging (MRI; CT Brain)
- Cognitive Screen (ACE-111)
- Psychiatric assessment
- Occupational Therapy
- Neuropsychological assessment

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## Changing our Thinking?

- "Between 50% and 80% of individuals with alcohol use disorders experience mild to severe neurocognitive impairment." (Bates et al, 2002) (7)
- Damage to cognition, and to executive functions caused by alcohol and thiamine deficiency (although not exclusively) across the life course results in poor treatment outcomes and engagement (7) (14) (15)
- Subtle cognitive problems can appear up to ten years before the detection of any neurological disorder (16)
- What are the potential costs (personal, family, unsuccessful periods in treatment or rehab, relapse and further harm, societal, economy) of not adapting our service models to take this into account?

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## Are Services Person Centred?

"You never really understand a person until you consider things from his point of view...Until you climb inside of his skin and walk around in it."

Lee, Harper. (2010). *To Kill A Mockingbird*. London, England: Arrow Books

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