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What is ARBD?

ACUK in collaboration with the ARBD Network

1

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What causes ARBD

The direct effects of alcohol on the brain as a consequence of long term heavy consumption of alcohol

The consequences of thiamine deficiency (Vitamin B1)

- Poor diet
- Poor absorption of thiamine through gut wall
- Alcohol related problems in the use of thiamine in brain cells

Nutrition

Genetics[1,2]

Gender

2

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To Score mild-moderate cognitive impairment (MMSE)

Women: 30 units a week
Men : 50 units a week

•At least 5 years +[1]
•Against a background of heavy social drinking
•Dependent on: Nutrition/ thiamine, genetics

Men: 3 pints a night
Women 2 .3 pints a night

Men 8.5 G&Ts per night
Women: 7 G&Ts per night

Men: 4 glasses of wine per night
Women: 3 glasses of wine per night

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Prevalence
RCP report (Torvik 1982,Harper 1989)

- Post mortem studies:
  - 40,000 post mortem studies from community: Europe, Australia and USA
  - 0.5~1.5% of patients of the general population at post mortem[2]
  - 35% of alcohol misusers have ARBD or cerebella damage[1]
- Diagnostic problems
  - 18% of patients diagnosed during their life time (PM studies)
  - 10% of WK patients have classical signs[2]
  - Estimated under diagnosis of 80-90%
- Severe ARBD:
  - 3 referrals a month from acute hospital
  - Population 310,000

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ARBD: (a practical classification)

1. Acute effects of alcohol
   - Drunk
   - Withdrawal
   - Encephalopathies (Wernicke Korsakoffs) : delirium (IV thiamine)
2. Medium term effects of alcohol (transient ARBD)[1] (wet brain)
   - In heavy, long term abusers: may last 3-4 months
   - Often dramatic improvement
3. Long term effects of alcohol/thiamine deficiency[2,3]
   - Brain cells take at least a year to grow
   - Deficits usually show some improvement over 2-3 years.
4. Permanent alcohol/ thiamine related damage[3]
   - Lasts indefinitely
   - May be some improvement in terms of social and behavioural adaptation

5

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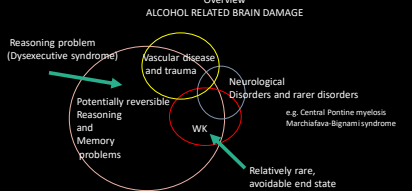
Overview
ALCOHOL RELATED BRAIN DAMAGE



6

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Cognitive profile of heavy drinkers

Cognitive function in heavy drinkers[1,2]

A comprehensive review (and meta-analyses) of the literature (143 papers from 250 countries covering 1997-2011) identified four profiles of cognitive impairment in heavy drinkers

- **No cognitive impairment**
- **Isolated reasoning deficits** with normal memory and global cognitive efficiency
- **Mild reasoning dysfunction with memory impairment** and preserved global efficiency
- **Global impairment** (problems with reasoning function, memory impairment and impaired cognitive efficiency)

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Warning signs for ARBD

- In people drinking 30 (women), 50 (men) units for at least five years[1]
- Cognitive problems[2] as reported by friends, family or obvious in conversation or testing (ACE 111 or mini-ACE)
- Physical signs[2]
  - Fatigue, insomnia, anxiety, apathy
  - Loss of weight, reduced BMI
  - Recurrent vomiting in last month
  - Relative increase in carbohydrate intake
  - Early neurological problems: Giddiness, double vision, pins and needles

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Groups with probable high prevalence rates

- People attending alcohol treatment units[1]
  - Poor compliance with alcohol treatment programs and engagement[2]
- Frequent hospital attenders contacts and delayed discharges due to alcohol related problems[3,4,5]
- Homelessness[6,7,8]
- Prison populations[9]

Despite this, ARBD remains underdiagnosed[10]

9

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In a non-acute setting

N=641 patients referred to an ATU[1]

- Reasoning and thinking problems in 58%

Subtle: May precede alcohol related neurological disorders by ten years

- Memory loss in 32%
  - Short term (Anterograde episodic) (the ability to learn new information)
  - Long term (Retrograde episodic)
  - Confabulations
  - Suggestibility
- NB: working and visual memory intact

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In the acute setting;

Wernicke's Encephalopathy

- Ophthalmoplegia
- Ataxia
- Confusion
- Also irritability, drowsiness, fits, and other signs

(Not all signs need to be presenting)

Hospital admission

Use IV thiamine as an emergency treatment if suspicious

Korsakoff's Syndrome

- Short (anterograde) and long term (retrograde) memory problems
- False memories (confabulations)
- Apathy, Reasoning problems
- Neurological problems

11

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The cognitive presentation

12

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Short term memory problem (anterograde amnesia)

Q: So what I would like to ask you is a little bit about your memories of the last three or four years

A: Because I forgot what was going on , I turned to John thinking he knows the answer and he understood me. He understood what I was going through and he would suggest something, that's how I managed.

Q: Do you remember the issue with alcohol those years ago.

A. I remember drinking. But even though he told me I was drinking too much, I could not remember drinking the previous time and I was sure I was not, even though he told me, everybody told me. I thought the last drink was the night before and I was having after effects. I don't remember doing it on the actual day.

Q. Does that mean you don't remember the drink before the one that you were drinking at the time? [correct] Did that make you think you were drinking less than you were because you could not remember it?

A. Yes, I was thinking I had it the night before [um] and the effects of it made me go blank

- (Consent to use as teaching aid provided by patient and carer)

13

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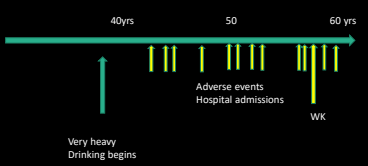
Long term memory: architecture (personal interpretation)



14

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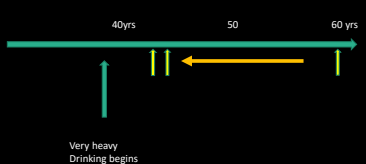
Long term memory: architecture (personal interpretation)



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Confabulations

- A falsification of memory occurring in clear consciousness, in association with organically derived amnesia.
  - Spontaneous/Fantastic type:
    - Far-fetched adventures, grandiose in theme, spontaneous (probably frontal in nature)
    - Brief in content , Reference to recent past, Is usually provoked
  - Momentary / Contextual type

16

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Spontaneous/Fantastic confabulation

- 60 year old male presented with Wernicke's encephalopathy. Developed ARBD:
  - No eye signs
  - No peripheral neuropathies
  - No ataxic gait
  - No disorientation
- Significant retrograde episodic memory difficulties (long term memory)

17

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Spontaneous/Fantastic confabulation

- Well known background from heavy drinking culture in local area.
- Known to have been in Scotland for a year or two during the Falkland's war, working on a building site. Has family in Scotland
- Believes that he was part of the secret Special Services operation, invaded the Hebrides to release children from capture by terrorists.
- Believes that there are 'security people' who he has infrequent contact with regarding his experiences
- His recall of these events are somewhat vague and he shrouds them in mystery because of 'security' issues.

18

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Momentary/contextual type

interviewed on ward, just after visitors to the ward had left

Q. How are you today?
A. Fine.
Q. I hope I did not interrupt any visitors?
A. Its OK; my wife has just left.
Q. How long have you been married
A. Many years.
Q. Are you still living with her?
A. Yes
Q. Are you going to live with her when you are discharged ?
A. Of course.
Q Are you going back to your own flat when you leave hospital?
A Yes
Q You will be living alone then for a while?
A Yes
Q Will it be Ok if I come and visit you?
A Yes

The reality
No visitors
No contact with divorced wife for many years
Has no one living with him

What's going on?
Short term memory loss: contradicting himself
Long term memory loss; thinks he still lived with his wife
Highly suggestible
False belief about visitors; cued by other visitors and leading questions

19

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Lack of insight

- There may be a whole lot of understandable reasons…e.g.
  - Does not think it is anyone else's business
  - Perhaps she is embarrassed
  - Does not want strangers in the house
  - Does not like attending alcohol treatment service
- She might be in psychological 'denial' of her alcohol problems
  - Admitting to the problem may be too much of an emotional challenge
- It may be because the part of the brain that enables her understanding of her problem is not working…..

20

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anosognosia

Q. Do you think you have any problems at all?
A No
Q. Are you physically well?
A. Yes
Q. Do you have any memory problems?
A. No
Q. Can you walk perfectly well?
A. Yes
Q. Are you ill?
A. No
Q. You have a Zimmer?
A. Yes…stops people walking into me.
Q. Can you run?
A Yes
Q Why do you use a Zimmer?
A Lots of old people around and it pushes them out of the way.

Conversation extracted from notes taken at interview

43 year old women in a nursing home
Extremely frail
Walks with a zimmer and help of two nurses
Severe cognitive disturbance

21

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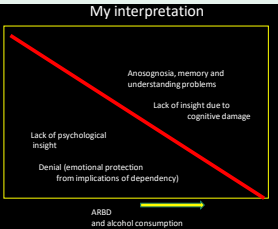
My interpretation



22

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If we don't have a pathway of care…..

23

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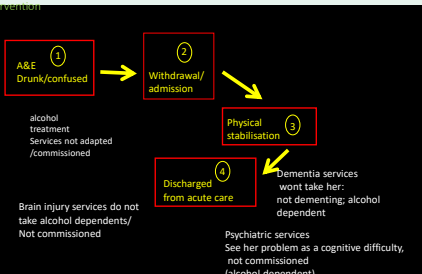
Steps in non-intervention



24

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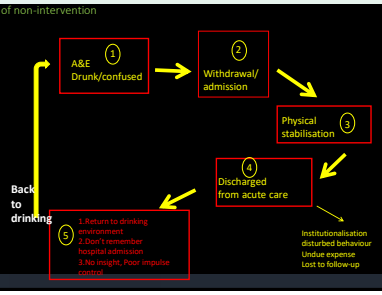
steps of non-intervention



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25

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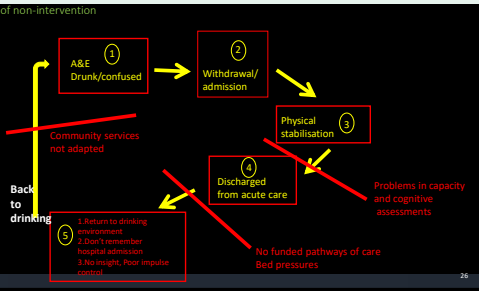
steps of non-intervention



26

26

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Managing ARBD

Is it worth the effort?

27

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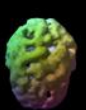
SPECT scans



Brain during alcohol misuse

28

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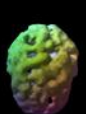
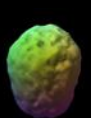
SPECT scans





Brain during alcohol abuse

Brain after one year of abstinence

Duration of abstinence is significantly associated with cognitive recovery[1]

29

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Routine assessment of people with alcohol problems[1]

- NICE guidelines (National Institute for Health and Clinical Excellence, 2011 England and Wales) recommend that all patients referred to alcohol treatment services should have a cognitive assessment. e.g. MoCA
- Therapists should gain experience in mental capacity and decision-making assessment
- When there is a likelihood of Wernicke's encephalopathy and related symptoms, in-patient referral and i.v. thiamine should be considered.
- All patients are assessed for risk of thiamine deficiency in the absence of Wernicke's encephalopathy-related symptoms and if there are significant risk factors then i.m. thiamine treatment is indicated.
- All patients having received parenteral thiamine should be continued on oral thiamine.
- Anyone receiving medically supported withdrawal should be prescribed oral thiamine even when no risk factors of Wernicke's encephalopathy are present.

1. Royal College of [illegible]

30

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Clinical phases of rehab programme[1]

- Phase 1 **Physical stabilisation** (variable time)
  - acute hospital management of encephalopathy, delirium tremens and withdrawal in the context of physical stabilisation and appropriate thiamine therapy (NICE 2010).
- Phase 2 **Psycho-social assessment** (usually 2-3 months:)
  - Calm environment
  - Early initiation of rehab programme
  - Regularisation of sleep, appropriate nutritional maintenance and mood stabilisation
  - Development of therapeutic relationships
  - Early engagement with family and carers
- Phase 3 **Therapeutic rehabilitation** (lasts up to 2-3 years[1])
  - Ecologically relevant, Milieu-based approach, Adaptable environment
  - 1. Dairy keeping 2. Activity scheduling 3. Graded task assignment
  - memory and orientation cuing
- Phase 4 **Adaptive rehabilitation** (variable)
  - adapting the environment so as to compensate for residual cognitive and functional deficits

Phase 5 **Social integration and relapse prevention** (on-going)

31

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It is worth the effort

Secondary care settings: N=72 acutely ill

- Treatment/management duration: average of 2.27 years (range: 0.18-6.54 years).
- Mortality: 8%
- Of the 66 surviving patients: 77% remained abstinent
- In abstinent patients there was an average of a 7-point improvement in HoNOs scores across the management period (physical, social and mental health (apart from depression)
- ACE (cognitive scores) in the subset of 38 for which we have initial and end scores: Average improvement from 67/100 to 81/100.
- Residential end point
  - 19 (29%) were settled in nursing or residential homes,
  - 13 (19%) were settled in supported living
  - 32 (48%) were settled in their own property
  - 2 homeless
- 85% reduction in acute hospital bed days during this period[1]

32

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overview

ARBD is

- Common in at-risk populations
- Is associated with considerable mortality and morbidity including damaging families and loved ones
- Has a significant and impact on health and social services
- Is likely to interfere with treatment plans, care plans and outcome of concurrent diseases/conditions
- Most people will respond to interventions

Rarely identified or treated

33

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Useful info:

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34