Integrated Addiction Service (North Lanarkshire)

Alcohol Related Brain Damage Resource Pack
Important Message

Alcohol related brain damage can be prevented
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Alcohol Related Brain Damage

Scotland is experiencing an increase in rates of Alcohol Related Brain Damage (ARBD). It has some of the highest rates of this condition in the United Kingdom. The diagnosis is associated with deprivation and is occurring in younger people. Long-term excessive consumption of alcohol has a harmful effect on most of the body’s organs, particularly the brain and the gastro-intestinal system (gut and liver). The effect of protracted excessive consumption on the brain varies from person to person, for reasons which are not yet clear. Theories include the style of alcohol consumption, for example, binge versus chronic consumption, and differences in an individual’s susceptibility, usually thought to be genetic. There is no single cause of ARBD, which usually results from a combination of factors. These include the direct toxic effects of alcohol on brain cells, the effects of dehydration on the brain, vitamin and nutritional deficiencies, head injury and disturbances to the blood supply of the brain usually incurred whilst intoxicated. (Hillman, A. Ravenscraig Hospital, Greenock. Institute of Alcohol Studies, 2007).

ARBD is not dementia. It is a term used to cover a spectrum of conditions and disorders (including alcohol related dementia) and alcohol related brain injury, conditions which have been induced by chronic alcohol consumption resulting in some degree of brain damage. Data from the Scottish Public Health Observatory (ScotPHO) indicates that Scotland has the highest incidence of ARBD in Western Europe. Important risk factors for developing ARBD include:-

• Consuming regular harmful levels of alcohol for 10 years or more (less for women)
• Regular binge pattern of drinking
• Poor nutrition and absorption of essential vitamins
• 40 years of age (evidence that this age range is reducing even further)
Some functions of the brain

**Brain-Stem:**

The brain-stem functions as our own private ‘auto-pilot’. It is located at the lower part of the brain, directly connected to the spinal cord and provides a ‘highway’ for nerve connections (motor and sensory systems) to the rest of the body, including breathing, digestion, heart rate, blood pressure and arousal.

**Cerebellum:**

The cerebellum (meaning little brain) plays an important part in motor control. The cerebellum lies on top of the pons (an area that contains the respiratory centre. It links different parts of the brain and serves as a relay station from the medulla to the higher cortical structures of the brain, behind the brain stem). Although it does not initiate movement, it contributes to coordination, precision and accurate timing. Damage to the cerebellum may result in problems with fine movement, equilibrium, and posture. The cerebellum may also be involved in some cognitive functions.

**Mammillary Body:**

The Mammillary body forms a crucial component of the brain’s circuitry and is involved with the processing of recognition memory. It is really two structures; the medial mammillary nucleus and the lateral mammillary nucleus, found on the underside of the brain and considered part of the limbic system. Both are linked to the hypothalamus via a nerve path called the fornix and feature a series of neural projections that connect to other parts of the brain. Damage (due to thiamine deficiency) may result in impaired memory (anterograde amnesia). The mammillary body is also believed to add the element of ‘smell’ to memories.
**Thalamus:**

The thalamus is perched on top of the brainstem, near the centre of the brain. It is in a position to relay sensory and motor signals to the cerebral cortex, which in turn sends information to other systems in the body. The thalamus also plays an important role in regulating sleep and wakefulness, arousal, level of awareness and activity.

**Hypothalamus:**

The hypothalamus is a small cone-shaped structure about the size of an almond and is located below the thalamus just above the brain stem. Its main function is to link the nervous system to the endocrine system via the pituitary gland, but it also functions as a type of thermostat controlling body temperature, hunger, thirst, fatigue and sleep.

**Frontal Lobe(s):**

These are involved in motor functions, problem solving, memory, language, judgement, impulse control, social and sexual behaviour, therefore any of these functions may be affected by frontal lobe damage.

There are four lobes in the brain. The frontal lobe is the largest, and is located behind the forehead, near rough bony ridges. The frontal lobe is prone to injury because of its location. It is involved in planning, organising, problem solving, memory, impulse control, decision making, behaviour and emotions.

The frontal lobe is involved in high mental functioning and is responsible for the ability to recognise future consequences resulting from actions and the ability to choose between good and bad action (or better and best), and to override or suppress unacceptable social responses/behaviour.

Damage to the frontal lobe may result in disinhibition and behaviour changes, judgement difficulties and poor reasoning skills.
Associated conditions and effects

The frontal lobe is the largest lobe in the brain, yet it is often not specifically evaluated in routine neurologic examinations. This may in part be due to the attention to detail and rigorous testing strategies required to probe frontal lobe functions. As successful completion of any cognitive task considered a frontal lobe function requires multiple brain regions both within and outside the frontal lobe, some prefer the term ‘frontal systems disease’. In any case, dysfunctions of the frontal lobe can give rise to relatively specific clinical syndromes. When an individual’s history suggests frontal lobe dysfunction, detailed neurobehavioural evaluation is necessary.

Traditional classification systems divide the frontal lobes into the precentral cortex (the strip immediately anterior to the central or Sylvian fissure), prefrontal cortex (extending from the frontal poles to the precentral cortex and including the frontal operculum, dorsolateral, and superior mesial regions), orbitofrontal cortex (including the orbitobasal or ventromedial and the inferior mesial regions), and superior mesial regions (containing, primarily, the anterior cingulate gyrus). Each of these areas has widespread connectivity.

Given the unique connectivity between the frontal regions and deeper brain structures, lesions of these areas or their connections generate relatively distinctive clinical behaviours.

- The dorsolateral frontal cortex is concerned with planning, strategy formation, and executive function. Patients with dorsolateral frontal lesions tend to have apathy, personality changes, abulia, and lack of ability to plan or to sequence actions or tasks. These patients have poor working memory for verbal information (if the left hemisphere is predominantly affected) or spatial information (if the right hemisphere bears the lesion brunt).

- The frontal operculum contains the center for expression of language. Patients with left frontal operculum lesions may demonstrate, Broca aphasia and defective verb retrieval, whereas patients with exclusively right opercular lesions tend to develop expressive aprosodia.

- The orbitofrontal cortex is concerned with response inhibition. Patients with orbitofrontal lesions tend to have difficulty with disinhibition, emotional lability, and memory disorders. Patients with such acquired sociopathy, or pseudo-psychopathic disorder, are said to have an orbital personality. Personality changes from orbital damage include impulsiveness, puerility, a jocular attitude, sexual disinhibition, and complete lack of concern for others.

- Patients with superior mesial lesions affecting the cingulate cortex typically develop akinetic mutism.

- Patients with inferior mesial (basal forebrain) lesions tend to manifest anterograde and retrograde amnesia and confabulation.

Broca aphasia from a lesion in areas 44 and 45 on the left hemisphere leads to non-fluent speech, agrammatism, paraphasias, anomia, and poor repetition. Lesions anterior, superior, and deep to (but sparing) the Broca area produce abnormal syntax and grammar but repetition and automatic language are preserved. This disorder is known as transcortical motor aphasia and uninhibited echolalia is common. Memory disturbances only develop with lesion extension into the septal nucleus of the basal forebrain. Appreciation of verbal humour is most impaired in right frontal polar pathology.
Peripheral neuropathy:

Peripheral neuropathy is a term for the damage to nerves of the peripheral nervous system.

The nervous system consists of two main parts:

- The central nervous system, which includes the brain and spinal cord.
- The peripheral nervous system, which includes all parts of the nervous system which lie outside the central nervous system, including the motor nerves used by the brain to control muscle action.

The peripheral nervous system consists of three main types of nerves, each with its own specific function.

- Automatic nerves help regulate the automatic functions of the body, such as blood pressure, bladder and sexual function and sweat levels.
- Motor nerves control the muscles of the body.
- Sensory nerves pass sensations, such as cold, heat or pain, from the affected area of the body to the brain.

Peripheral neuropathy can cause one or more of the following systems.

- Numbness and tingling in the feet and hands.
- Burning, stabbing or shooting pain.
- Loss of co-ordination in the affect body parts.
- Muscle weakness, cramps, spasms. Loss of balance and co-ordination may also occur.

Peripheral neuropathy is a relatively common condition which may be caused by heavy alcohol use but may also be a result of diabetes.

The outlook for peripheral neuropathy can vary widely depending on the underlying cause and what sort of peripheral nerves have been damaged.

For example, if the sensory nerves of the hands or feet are affected, the outlook is generally good. However, it is important that the underlying cause (diabetes) is aggressively treated.

This is because over time, diabetic polyneuropathy can cause a diabetic foot ulcer; an open sore that develops in the foot. If the ulcer becomes infected, there is a risk that the foot tissue will begin to die and it may be necessary to amputate the foot.

The outlook is not so good in cases where neuropathy affects the automatic functions of the heart and circulation system (cardiovascular automatic neuropathy). This is because the condition can increase the risk of sudden death as a result of the heart suddenly stopping beating (cardiac arrest).
**Wernicke’s encephalopathy:**

Wernicke’s encephalopathy is a disorder affecting the brain. It is caused by lesions in the medial thalamic nuclei, mammillary bodies, periaqueductal and periventricular brainstem nuclei, and superior cerebellar vermis, often resulting from inadequate intake or absorption of thiamine (vitamin B1), especially in conjunction with carbohydrate ingestion. It is commonly correlated with prolonged alcohol consumption resulting in thiamine deficiency, but may also occur with thiamine deficiency states arising from other causes, particularly in individuals with such gastric disorders as carcinoma, chronic gastritis, Crohn’s disease and repetitive vomiting, particularly after bariatric surgery.

Wernicke’s encephalopathy usually develops suddenly. There are three main symptoms, though these are not always present, so diagnosis may be difficult. The condition is characterised by:-

- Involuntary, jerky eye movements or paralysis of muscles moving the eyes.
- Poor balance, staggering gait or inability to walk.
- Drowsiness, confusion, short term memory impairment.

Treatment begins with intravenous or intramuscular injection of thiamine. This is usually followed by assessment of the central nervous system and metabolic conditions.

Prognosis depends on severity. When treated early, recovery is normally rapid and complete.

If Wernicke’s encephalopathy is left untreated, or is not treated in time, Korsakoff’s syndrome may follow.

If Wernicke’s encephalopathy becomes established there is a likelihood of serious long term consequences with the individual requiring permanent in-patient care. In some cases the person may die.
**Korsakoff’s syndrome:**

Korsakoff’s syndrome is a brain disorder usually associated with heavy alcohol consumption over a long period of time. Historically it has also been called ‘Korsakoff’s psychosis’, although this can be confusing, as there are no true psychotic symptoms, in the medical sense. It is sometimes referred to as ‘alcohol amnestic syndrome’, meaning loss of memory. Although Korsakoff’s syndrome is not strictly speaking ‘dementia’, people with the condition experience loss of short-term memory.

Korsakoff’s syndrome is caused by a lack of thiamine (vitamin B1), which affects the brain and nervous system. Thiamine deficiency is often seen in people who consume excessive amounts of alcohol.

This is because:-

- Many heavy drinkers have poor eating habits. Their nutrition is often inadequate and may lack essential vitamins.
- Alcohol can inflame the stomach lining thereby impeding the body’s ability to absorb the key vitamins it does receive.

The condition tends to affect men between the ages of 45 and 65 who have a long history of alcohol use, although this trend is changing with a lowering of the age range. As women tend to be vulnerable to the impact of alcohol they tend to develop the condition at a younger age – for example, whereas it may take up to 20 years for a man to develop Korsakoffs, this could be reduced to half that time for a woman. It is not clear why some heavy drinkers develop Korsakoff’s syndrome and others do not. Diet may be a contributing factor.

Korsakoff’s syndrome may develop in individuals with a diagnosis of Wernicke’s encephalopathy, which has not been treated or treated soon enough.

Korsakoff’s syndrome cannot be diagnosed until the person has abstained from alcohol for at least four to five weeks, to enable the acute symptoms of alcohol withdrawal to subside. Diagnosis of the condition is by a medically trained individual who will employ psychological testing, looking at the person’s memory and other abilities. It is possible to have both Korsakoff’s syndrome and dementia.

The progression of Korsakoff’s syndrome can be halted if the person:-

- Completely abstains from alcohol.
- Adopts a healthy diet and takes vitamin supplements.

While it remains unclear whether additional thiamine helps people improve once the brain damage has already occurred, it may prevent further damage from occurring.

Improvement in the condition usually occurs within a period of up to two years. About a quarter of those affected make a very good recovery. About half make a partial recovery and need support to manage their lives. The remaining quarter make no recovery and may require long-term care. Ongoing heavy drinking coupled with poor nutrition is likely to result in the progression of the condition.
Prevention – the way forward

Diet, nutrition and alcohol

Good nutrition plays an important part in our lives. Digestion begins in the mouth and continues in the stomach and intestines, with help from the pancreas. The nutrients from digested food are absorbed from the intestines into the blood and carried to the liver, where it is prepared for use, or stored for future use, including the provision of energy, and for replacing worn or damaged cells.

Heavy drinkers often obtain at least half of their daily calories from alcohol, neglecting important food groups, such as those containing thiamine. In addition, alcohol inhibits the breakdown of nutrients by decreasing the secretion of digestive enzymes from the pancreas. Alcohol impairs the body’s ability to absorb these nutrients by damaging the cells lining the stomach and intestines and by disabling the transport of some nutrients into the blood. Even those nutrients which are digested and absorbed are often prevented from being fully utilised as alcohol can prevent their transport, storage and efficient excretion.

Although the absorption of thiamine (vitamin B1), an essential nutrient required by all tissues, is crucial in avoiding the onset of Korsakoff’s, other vitamins are also important; for example, vitamins A, C, D, E and K are all involved in wound healing and cell maintenance, with vitamin K being essential for blood clotting.

Foods rich in thiamine (vitamin B1)

As the human body cannot produce thiamine it must be obtained from certain food groups i.e. Asparagus; avocado; baked potato (with skin); beans (especially black beans, pinto beans, navy beans, split peas); Bovril; brown rice; brussel sprouts; cheese; coffee; Cola; eggs (in the yolks); fish (particularly yellow-fish tuna, mackerel, sardines); fruit (tinned or fresh); gravy; hot chocolate; leafy green vegetables; liver; Marmite; meat (particularly pork chops); mushrooms; nuts (particularly pine nuts, pistachios, macadamia, pecans); olives; peas; pickles; poultry; quorn; brown rice and bran; spinach; sunflower seeds; tea; wheat germ; whole grains; veggie burgers/veggie meatballs (containing soya).

Parenteral thiamine (Pabrinex intramuscular* high potency solution for injection)

Pabrinex contains the water-soluble vitamins C (ascorbic acid), B1 (thiamine), B2 (riboflavin), B3 (nicotinamide) and B6 (pyridoxine).

Vitamins are required by the body in small amounts in order to maintain healthy growth and development. They are involved in numerous biological activities in the body. Group B vitamins are necessary in the development and maintenance of the nervous system and the formation of blood cells. Vitamin C is an antioxidant and is responsible for maintaining healthy cell structure and collagen, which is found in skin, bone, cartilage and tendons. These vitamins cannot be produced by the body and are therefore obtained solely from the diet. Deficiencies in these vitamin groups may arise from poor nutrition or mal-absorption of nutrients from the gut. Heavy and sustained alcohol use and can cause gastric inflammation of the stomach, which in turn will often result in the individual vomiting up food before it can be digested,
thereby contributing to malnutrition and low levels of Vitamin B1. A severe depletion of thiamine may result in Wernicke’s encephalopathy therefore the administration of Pabrinex is vital in order to alleviate this condition. However, as heavy drinking is the commonest cause of thiamine deficiency in the West, preventative measures through effective alcohol education, which includes nutritional information, is an important area of work for addiction staff.

*Pabrinex can be administered intravenously but only if individual is an in-patient.

**Key elements**

Alcohol related brain damage can no longer be seen as an “older person’s” condition. Statistical information is available which highlights that the age range onset of ARBD is decreasing and that more females are being affected.

The important message about ARBD is that it can be prevented, but the issue needs to be tackled as early as possible. This means that every individual who attends the Integrated Addiction Service should be provided with good alcohol education, which is clear about the health risks of heavy or binge drinking, in a manner which is factual, but not scare-mongering. Service users should be advised of the importance of good nutrition and encouraged to view this as part of their general well-being.

- Early diagnosis: Increased use of oral vitamins and intramuscular Pabrinex.
- Person centred approach:
- Abstinence: Aiming for 6 weeks abstinence from alcohol. This may include detoxification / rehabilitation
- Attitudes: Changing attitudes and practices in relation to people with ARBD
- Increase knowledge: Nutrition / physical and mental health problems / neurological process of assessment.
- Assessment: Continuous assessment and review across professional boundaries. Assessment of Adults with Incapacity (Scotland Act) 2000

Additional information is available in:-

“A Fuller Life Report” of the Expert Group on ARBD.
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<th>Deficiency Symptoms</th>
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<tr>
<td>A:</td>
<td>Retinol Carotene (5,000 IU)</td>
<td>Growth and repair of body tissues (resist infection), bone and tooth formation, visual purple productions (necessary for night vision)</td>
<td>Night blindness, drying of eyes, rough skin, impaired bone growth</td>
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<td></td>
<td>Liver, eggs, dark green and deep orange fruits and dairy products</td>
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<td>B-1</td>
<td>Thiamine (1.5mg)</td>
<td>Carbohydrate metabolism, appetite maintenance, nerve function, growth and muscle tone</td>
<td>Mental confusion, muscle weakness, oedema, fatigue, loss of appetite</td>
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<td>Wheatgerm, liver, pork, whole grains and enriched grains, dried beans</td>
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<td>B-2</td>
<td>Riboflavin (1.7mg)</td>
<td>Necessary for fat, carbohydrate and protein metabolism cell respiration, formation of antibodies and red blood cells.</td>
<td>Sensitivity of eyes to light, cracks in corners of mouth, dermatitis around nose and lips</td>
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<td>Dairy products, green leafy vegetables, whole grains and enriched grains</td>
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<tr>
<td>B-6</td>
<td>Pyridoxine (2.0mg)</td>
<td>Necessary for fat, carbohydrate and protein metabolism</td>
<td>Dermatitis, anaemia, nausea, smooth tongue</td>
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<td>Fish, poultry, lean meats, whole grains</td>
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<tr>
<td>B-12</td>
<td>Cobalamin (6mcg)</td>
<td>Carbohydrate, fat and protein metabolism, maintains healthy nervous system, blood cells formation</td>
<td>Pernicious anaemia, numbness and tingling in fingers and toes</td>
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<td>Organ meats, lean meat, fish and poultry, eggs, dairy products</td>
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<tr>
<td>Biotin</td>
<td>(300mcg)</td>
<td>Carbohydrate, fat and protein metabolism, formation of fatty acids, helps utilise B vitamins</td>
<td>Not seen under normal circumstances; pale, dry, scaly skin, depression, poor appetite</td>
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<td></td>
<td>Egg yolks, organ meats, dark green vegetables; also made by micro-organisms in the intestinal tract</td>
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<td>Folic Acid</td>
<td>(400mcg)</td>
<td>Red blood cell formation, protein metabolism, growth and cell division</td>
<td>Anaemia, diarrhoea, smooth tongue, poor growth</td>
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<td>Green leafy vegetables, organ meats, dried beans</td>
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<td>Niacin</td>
<td>(20mg)</td>
<td>Fat, carbohydrate and protein metabolism, health of skin, tongue and digestive system, blood circulation</td>
<td>General fatigue, digestive disorders, irritability, loss of appetite, skin disorders</td>
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<td>Meat, poultry, fish, nuts, whole grains and enriched grains, dried beans</td>
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<td>Panto-thenic Acid</td>
<td>(10mg)</td>
<td>Converts nutrients into energy, formation of some fats, vitamin utilisation</td>
<td>Not seen under normal circumstances; vomiting, severe abdominal cramps, fatigue, tingling hands and feet</td>
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<td>Lean meats, whole grains, legumes</td>
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<td>C</td>
<td>Ascorbic Acid (60mg)</td>
<td>Helps heal wounds, strengthens blood vessels, collagen maintenance, resistance to infection</td>
<td>Bleeding gums, slow healing wounds, bruising, aching joints, nosebleeds, anaemia</td>
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<td></td>
<td>Citrus fruits, melon, berries, vegetables</td>
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<td>B Vitamin</td>
<td>Consequence of deficiency on the nervous system</td>
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<td>Nicotinic acid</td>
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<td>Ataxia</td>
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<td>Convulsions</td>
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<td>Neurasthenia</td>
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<td>Psychosis</td>
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<td>Organic brain damage</td>
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<td>Folate</td>
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<td>Subacute combined degeneration of the cord</td>
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Source: Adapted from Meyer (1958) and Lishman (1987)
Supporting individuals with ARBD

Daily Structure

Variety and stimulation are important but too many changes will be confusing. A weekly schedule with a regular routine and activities will help the person feel more secure and make it easier for the person to remember what usually happens at different times of the day. It is therefore helpful to:

- Leave things in the same place so the person can find them easily, e.g. keys.
- Encourage independence by not doing too much for the person. If you do need to offer assistance, try to do things with them rather than for them.
- Any changes which need to be made should be done gradually, preferably in small stages.
- Aim to build routine into all aspects of the person’s life e.g. household chores, personal hygiene, shopping, social activities, employment, appointments.
- Surround the person with familiar objects and people as much as possible.
- Consider having a container by the door which the person can check for keys, wallet, letters for posting, shopping lists etc.

Memory aids

Memory aids include things like

Lists:-
- Use notes on ‘post-its’
- Leave in visible places around the home
- Keep information short and concise

Diaries:-
- Set out a routine on a daily basis e.g. lunch at 1.00pm
- Break information down into small chunks of information e.g. doctor at 10.00am

Written instructions:-
- Confirm any information in writing as well as visually
- Keep information short
- Use familiar language – avoid new words or jargon

Visual aids:-
- Images such as a picture of coffee cups/sugar/plates might be helpful if attached to a cupboard door
- Photographs or videos of recent events may also help to trigger memories.

People with ARBD may find people’s faces easier to recall than their names. It’s a good idea to explain this to friends or relatives so that they will understand if the person doesn’t recognise them by name. Understandably, recognising somebody’s face but not remembering their name can cause the person with ARBD a lot of distress and anxiety and can be upsetting for their visitor. One way of dealing with this is to encourage the ‘visitor’ to introduce themselves each time they speak.
Communication

Communicating with friends and acquaintances is important to us all, particularly as a way of reducing isolation. Reducing isolation is also important for people with ARBD, therefore it is essential that they are not ignored, but you need to take into consideration that they often find it difficult to take in new information, and remember it. There are some simple steps which can be taken to alleviate this:

- Keep information simple and be prepared to repeat it, frequently, if necessary.
- Use familiar language – avoid jargon.
- Look at the person when talking to them.
- Slow down when you talk, but remember if the person didn’t have a hearing problem before, the ARBD won’t cause this, therefore there is no need to shout.
- Only ask questions which need a ‘yes’ or ‘no’ answer, e.g. would you like a biscuit? This should be accompanied by showing the person what you mean, e.g. showing them the packet of biscuits.
- New activities, and sometimes even those which the person has done recently, are best broken down into small steps or stages.
- Try not to communicate with the person while there is background noise, such as the television or radio.

Community living

Components of successful community living include:

- **Community integration:** Individuals should be encouraged and provided with the opportunity to fully participate in their local community thus promoting a sense of well being and recovery. Involvement in activity should be meaningful to the individual and may include leisure and work related activity.

- **Activity based rehabilitation strategies:** This includes the development or re-development of skills in daily living. Learning should be encouraged through “doing” rather than “showing” or “telling”. The skills should be trained repeatedly and frequently and in the context that it will be used.

- **Social and cultural stimulation:** It is important to assist people to continue with everyday activities and encourage the maintenance or development of positive social networks.

Source: Elaine Wakefield
**Global Picture**

- Global increase in alcohol related brain impairments.

- Attributed to: greater alcohol consumption amongst women and younger people, increased access to cheaper alcohol, liberalisation in the social context of alcohol and changes in vitamin prescribing practice (e.g. Ramayya and Jauhar, 1997; Gupta and Warner, 2008).

- Gupta and Warner (2008) stated: “the current prevalence of alcohol related (brain damage) is manifest in a cohort whose alcohol consumption was half the current levels of today’s younger and middle aged generations”, implying that alcohol related neurological impairments will increase further in coming years.

**Prevalence of ARBD in Lanarkshire**

<table>
<thead>
<tr>
<th>Area</th>
<th>2000</th>
<th>2001</th>
<th>2002</th>
<th>2003</th>
<th>2004</th>
<th>2005</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scotland</td>
<td>408</td>
<td>412</td>
<td>486</td>
<td>462</td>
<td>513</td>
<td>535</td>
<td>542</td>
<td>538</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>North Lan’shire</td>
<td></td>
<td></td>
<td></td>
<td>19</td>
<td>38</td>
<td>34</td>
<td>29</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>South Lan’shire</td>
<td></td>
<td></td>
<td></td>
<td>13</td>
<td>21</td>
<td>23</td>
<td>26</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total for Lan’shire</td>
<td>34</td>
<td>17</td>
<td>30</td>
<td>31</td>
<td>44</td>
<td>55</td>
<td>32 (31)*</td>
<td>59 (52)*</td>
<td>57 (50)*</td>
<td>75 (63)*</td>
</tr>
</tbody>
</table>

*denotes actual number of individual patients discharged

- Numbers of patients discharged with ARBD have more than doubled in last 3 years
- Estimated increase of 1/3\textsuperscript{rd} each year since 2003
- Increasing number from South Lanarkshire area

<table>
<thead>
<tr>
<th>Gender</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>21</td>
<td>41</td>
<td>44</td>
<td>46</td>
</tr>
<tr>
<td>Female*</td>
<td>10</td>
<td>9</td>
<td>12</td>
<td>17</td>
</tr>
</tbody>
</table>

*Increasing proportion of females in last two years

<table>
<thead>
<tr>
<th>Age</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
</tr>
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<tbody>
<tr>
<td>29</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>38-49</td>
<td>2</td>
<td>6</td>
<td>7</td>
<td>9</td>
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<td>50-59</td>
<td>13</td>
<td>7</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>60-69</td>
<td>11</td>
<td>19</td>
<td>28</td>
<td>28</td>
</tr>
<tr>
<td>70-79</td>
<td>5</td>
<td>8</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td>80</td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
</tbody>
</table>

*Proportion of younger patients increased by 400% since 2006 (youngest was 29)*
Cultural Factors

- ARBD in areas such as the West of Scotland, which include high levels of poly-substance misuse, social deprivation, non-fatal overdose and repeated traumatic brain injuries, complicating the picture of ARBD (Scottish Executive, 2004).

- Cognitive impairment resulting from alcohol misuse has been documented amongst marginalised groups, including amongst Aboriginal communities in Australia (Cairney et al, 2007).
Conditions can overlap / interact
**Diagnosis of ARBD**

There has been a recent proposal that a category of mild neurocognitive disorder be added to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5; American Psychiatric Association, 2012), for several neuromedical conditions, including substance use–related brain disorders.

Proposed ARBD criteria (adapted from Oslin’s criteria, 1998)

- Principal; the main issue is to establish that excessive and long term alcohol ingestion significantly contributes towards the development of cognitive dysfunction. Clinicians may well be presented with patients with alcohol related brain damage, complicated by vascular change and/or trauma. In these situations, provided alcohol is recognised as the major cause and vascular disease or trauma is considered as secondary then the primary diagnosis should be ARBD.

A: Criteria for the clinical diagnosis of probable ARBD include the following:

1. Evidence of cognitive impairment. (as demonstrated by clinical examination or use of appropriate instruments; e.g ACE-R).
2. Significant alcohol use as defined by the minimum average of 35 standard drinks per week for men and 28 for women, for a period of greater than 5 years. The period of significant alcohol use must occur within three years of clinical onset of the cognitive deficits.

B: The diagnosis of ARBD is supported by the presence of the following:

1. Alcohol related hepatic, pancreatic, gastrointestinal, cardiovascular or renal disease or other end-organ damage.
2. Ataxia or peripheral polyneuropathy (not attributable to other non alcohol related causes).
3. Neuroimaging evidence of cerebella atrophy, especially of the vermis
4. Cognitive damage and evidence of ventricular or sulcal dilatation are likely to improve within the first 60 days, residual damage will be slower to improve and may be permanent.

C: The following clinical presentation indicates that there may be complicating conditions such as vascular or traumatic lesions

1. The presence of language impairment, especially dysnomia or anomia.
2. The presence of focal neurological signs or symptoms (except ataxia or peripheral sensory polyneuropathy).
3. Neuroimaging evidence of cortical or subcortical infarction, subdural haematoma or other focal brain pathology.
4. Elevated Hachinski Ischemia scale score

(Reference: Professor Kenneth Wilson, University of Liverpool)
**Neuropathology**

The frontal lobes, the limbic system, the cerebellum, and related cognitive functions including attention, memory, and executive function domains are particularly vulnerable to alcohol-related damage and impairment (Hildebrandt et al., 2006; Noël et al., 2001; Ratti et al., 2002; Rupp et al., 2005, 2006; Sullivan et al., 2000, 2002).

White matter changes have been found in brain regions including the corpus callosum (Pfefferbaum et al, 2006) and cerebellum.

The cerebellar changes are important when considering executive dysfunction impairments and are now recognised as a part of ARBD (Van Oort and Kessels, 2009).

The fronto-striatal-cerebellar pathways support frontal-executive functions, and are vulnerable to alcohol misuse (Sullivan, 2003; Chanraud et al, 2010).

Damage to the mammillary bodies, gray matter, collicular bodies and thalamus occurs through the impact of thiamine deficiency in Wernicke’s Encephalopathy (Sullivan and Pfefferbaum, 2009).

If acute Wernicke’s Encephalopathy progresses to Korsakoff’s syndrome, additional changes are seen including bilateral volume deficits in the hippocampus i.e. shrinkage (Sullivan and Pfefferbaum, 2009).

**Neuropsychological deficits : Executive dysfunction**

Executive impairments may not only be an early sign of damage, but an intrinsic part of ARBD.

**Executive function processes**

- Response Inhibition
- Divided attention/supervisory attention
- Concept shift/formation
- Planning and organisation (rules and prioritising).

**Executive problems**

Difficulties with:-

- Attention and concentration
- Planning, organising, problem solving
- Complex, abstract, and flexible thinking
- Initiative
- Emotional and behavioural change
- Self awareness and insight

This area can be particularly important when dealing with someone who is trying to remain abstinent from alcohol. Using their initiative to plan activities and alternative solutions to previous drinking behaviour may be difficult, and they may not be able to see beyond previous behaviours.
Anterograde and retrograde amnesia

- Difficulty remembering recent events or recently learned information.
- Can’t remember the order of events (e.g. can’t remember if they moved house prior to giving up work).
- Difficulty finding information stored in memory (recognition v recall).
- Tendency to make up memories (confabulation).
- Preserved learned behaviour (perseveration).
Recovery assumes abstinence. With abstinence from alcohol and good nutrition:-

- 1 in 4 people with ARBD will recover completely
- 1 in 4 people will recover significantly
- 1 in 4 people will make some recovery
- 1 in 4 people will not recover but should not get any worse

(Cox 2004)

25% rate can improve with intervention e.g. Cognitive remediation Rupp et al. 2012.

Emerging evidence base shows recovery can take >2 years and up to 5 years.

Neuropathological studies have allowed differentiation between permanent neuronal loss and transient demyelination, and suggested mechanisms for restoration and recovery (reviewed in Harper, 2009; Crews and Nixon, 2009).
Chronic Alcohol Use - Schematic

- Direct effects of ethanol
  - Reduced thiamine intake
  - Acetaldehyde formation
  - Reduced thiamine phosphorylation in brain

- Poor diet
  - Reduced thiamine intake

- Gastro-intestinal disorders
  - Reduced thiamine uptake

- Liver disease
  - Reduced thiamine stores

- Hyperammonaemia
  - Astrocytic pathology
  - NMDA-receptor mediated excitotoxicity
  - Loss of neuron/astrocyte metabolic trafficking

- Alcohol Related Brain Damage
  - Other mechanisms
  - Cerebral energy deficit
  - Focal lactic acidosis

Source: Butterworth 1995 (adapted)
The Adult Support and Protection (Scotland) Act 2007
(A short introduction to Part 1 of the Act)

The Adult Support and Protection (Scotland) Act 2007. What’s it all about? The Adult Support and Protection (Scotland) Act 2007 was passed by the Scottish Parliament in February 2007 and received royal assent on 22 March 2007. Part 1 of the Act deals with the protection of adults at risk of harm. It is scheduled to come into effect in the autumn of 2008. This booklet is a brief summary of the provisions of Part 1 of the Act. You should refer to the Act itself if you need more detailed information and seek legal advice if you have any queries on its interpretation.

What does Part 1 of the Act do?

Part 1 introduces new measures to identify and protect individuals who fall into the category of ‘adults at risk’. These measures include:

• Placing a duty on councils to make the necessary inquiries and investigations to establish whether or not further action is required to stop or prevent harm occurring;
• A requirement for specified public bodies to co-operate with local councils and each other about adult protection investigations;
• A range of protection orders including assessment orders, removal orders and banning orders; and
• The establishment of multi-disciplinary Adult Protection Committees.

Who are ‘adults at risk’?

The Act, defines ‘adults at risk’ as individuals, aged 16 years or over, who:

• Are unable to safeguard themselves, their property, rights or other interests; are at risk of harm; and

• Because they are affected by disability, mental disorder, illness or physical or mental infirmity, are more vulnerable to being harmed than others who are not so affected.

The presence of a particular condition does not automatically mean an adult is an “adult at risk”. Someone could have a disability but be able to safeguard their well-being etc. It is important to stress that all three elements of this definition must be met. It is the whole of an adult’s particular circumstances which can combine to make them more susceptible to harm than others.

What is meant by ‘harm’?

For the purposes of the Act, ‘harm’ includes all harmful conduct and, in particular, includes:

• Conduct which causes physical harm;
• Conduct which causes psychological harm (e.g. by causing fear, alarm or distress);
• Unlawful conduct which appropriates or adversely affects property, rights or interests (e.g. theft, fraud, embezzlement or extortion); and conduct which causes self-harm.
Principles underlying the Act

The overarching principle underlying Part 1 of the Act is that any intervention in an individual’s affairs should provide benefit to the individual, and should be the least restrictive option of those that are available which will meet the purpose of the intervention.

This is supported by a set of guiding principles which, together with the overarching principle, must be taken account of when performing functions under Part 1 of the Act.

These are:

- The wishes and feelings of the adult at risk (past and present);
- The views of other significant individuals, such as the adult’s nearest relative; their primary carer, guardian, or attorney; or any other person with an interest in the adult’s well-being or property;
- The importance of the adult taking an active part in the performance of the function under the Act;
- Providing the adult with the relevant information and support to enable them to participate as fully as possible;
- The importance of ensuring that the adult is not treated less favourably than another adult in a comparable situation; and the adult’s abilities, background and characteristics (including their age, sex, sexual orientation, religious persuasion, racial origin, ethnic group and cultural and linguistic heritage).

What duties and powers does the Act contain?

Inquiries: The Act places a duty on councils to make inquiries about an individual’s well-being, property or financial affairs where the council knows or believes that the person is an adult at risk and that it may need to intervene to protect him or her from being harmed.

Independent Advocacy and other support services: The council has a duty to consider providing appropriate services, including independent advocacy, to support adults where an intervention under the Act is considered to be necessary.

Investigations: In order to make inquiries, the Act authorises council officers to carry out visits, conduct interviews or require health, financial or other records to be produced in respect of an adult at risk. The Act also allows a health professional (e.g. doctor or nurse) to conduct a medical examination.

However, a person is not obliged to answer any questions put to them in an interview, and must be informed of their right to refuse to be examined before a medical examination is carried out.

Co-operation: The Act requires the following public bodies to co-operate with local councils and with each other, where harm is known or suspected:

- The Mental Welfare Commission for Scotland;
- The Care Commission;
- The Public Guardian;
- All councils;
• Chief constables of police forces;
• The relevant Health Board; and
• Any other public body or office holder that Scottish Ministers specify.

The public bodies or officers must advise the relevant council if they know or believe that a person is an adult at risk and that action needs to be taken in order to protect that person from harm.

**Protection orders:** Part 1 of the Act allows a council to apply to the sheriff for a protection order. This can take one of three forms:

- An assessment order;
- A removal order; or
- A banning or temporary banning order.

The sheriff may grant an order only if satisfied that certain criteria are met.

**Assessment order:** This allows a council officer to take the adult from a place visited by the officer in the course of their investigations to conduct a private interview and for a health professional to conduct a medical examination in private. An application for an assessment order can only be made where it is necessary to establish if the person is an adult at risk and, if so, to establish whether further action is required to protect them from harm. An assessment order will only be necessary where it would not be possible to carry out a private interview or medical examination within the place being visited. Assessment orders are valid for up to seven days, but the assessment itself should be undertaken in the shortest time possible. It should be borne in mind that the assessment order does not have the power to detain the adult at risk in the place they are taken to and that the adult may choose to leave at any time.

**Removal order:** This authorises a council officer to remove an adult at risk to a specified place where there is a likelihood of serious harm if they are not moved. This type of order may be varied or recalled by the sheriff where this is justified by a change in facts or circumstance of the case. Removal orders are effective up to a maximum of seven days. Again, a removal order does not authorise the adult’s detention therefore the adult may leave the place they have been removed to if they wish.

**Banning order:** This bans the subject of the order from being in a specified place, for up to six months. It can only be granted where an adult at risk is being, or is likely to be, seriously harmed by another person and the sheriff is satisfied that banning the subject of the order from the place will better safeguard the adult at risk’s well-being or property than by moving the adult. The sheriff can also grant a temporary banning order pending the determination of a full banning order.

**When might these measures be appropriate?**

The fact that council officers will be given powers to visit and make inquiries where it is believed an adult may be at risk of harm should allow early intervention where necessary, with the emphasis on prevention of harm. By virtue of the power to get through the door for a visit, it may become clear what support or other actions would be beneficial for an individual’s particular situation, for example, by providing practical or emotional support or by taking measures under Adults with Incapacity or Mental Health legislation. It is anticipated that protection orders will be used sparingly. In most situations, and in line with the guiding principles of the
Act, other less restrictive measures will be sufficient to protect the person concerned. However, in those circumstances where firmer action is required, this legislation puts in place sufficient powers to ensure those who need support or protection can have it.

**Undue pressure:** A sheriff must not make a protection order if the sheriff knows that the affected adult at risk has refused to consent to the granting of the order, unless the sheriff reasonably believes that the affected adult at risk has been unduly pressurised to refuse consent and there are no steps which could reasonably be taken with the adult’s consent which would protect the adult from harm. An example of undue pressurise is where it appears that harm is being, or is likely to be, inflicted by a person in whom the adult at risk has confidence and trust, and that the adult at risk would consent if the adult did not have confidence and trust in that person. Another example of undue pressure would be where the adult is afraid of or being threatened by another person. However, this does not authorise a council officer or a health professional or other council nominee to ignore a refusal by a person to consent to participation in an interview, or a medical examination.

**What safeguards are in place to protect the rights of individuals?**

- The principles underlying the Act emphasise the importance of striking the balance between an individual’s right to freedom of choice and the risk of harm. These must always be taken into account when an intervention under Part 1 of the Act is being considered.
- A sheriff must not make a protection order if they know that the affected adult at risk has refused to consent to the granting of the order, except where the adult at risk is found to be under undue pressure to refuse to consent. The adult is still entitled to refuse to be medically examined or interviewed.
- Applications for all protection orders will be heard before a sheriff, unless the sheriff decides that by not holding a hearing the adult will be protected from serious harm and that it will not prejudice any other person affected by the application.
- The adult at risk may apply for a banning order to ban a person from a specified place (e.g. their home).
- The relevant parties may appeal against the granting of, or refusal to grant, a banning or temporary banning order.
- Statements expressed in advance about an individual’s preferred care or treatment must be taken into account in line with the guiding principles.

**Adult Protection Committees**

Part 1 of the Act creates an obligation on councils to establish multi-agency Adult Protection Committees. These committees are responsible for overseeing local adult protection polices in their area and will each produce a biennial report on the exercise of the Committee’s functions. They will also provide advice and information to those involved in adult protection work.

**Membership:** Councils are responsible for appointing the convener and committee members. While they may also appoint members to the Committee based on their relevant knowledge and skills, each committee must include nominated representatives from the relevant Health Board and police force. The Care Commission also has the option to nominate a representative. Committee procedures must also allow representatives of the following bodies to attend meetings:
- The Mental Welfare Commission for Scotland;
- The Public Guardian;
- The Care Commission (where a representative has not already been nominated to be a member); or
- Any other public body or office holder that Scottish Ministers may specify by order.

**More information about the Act**

To keep up to date with progress on implementation of Part 1 of the Act, you can sign up to receive a copy of the quarterly newsletter. Please contact:

Adult Protection Legislation Team
2 East Rear, St Andrew’s House
Regent Road
Edinburgh
EH1 3DG
Telephone: 0131 244 3633
Email: ASPunit@scotland.gsi.gov.uk
Website: [www.scotland.gov.uk/topics/health/care/VAUnit](http://www.scotland.gov.uk/topics/health/care/VAUnit)

**Source:** The Scottish Government
**Glossary**

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abulia</td>
<td>Defined as the lack of motivation or desire to perform a task. An example of abulia is when a stroke survivor fails to move an arm or a leg, even though the part of the brain required to carry out the movement is intact.</td>
</tr>
<tr>
<td>Agrammatism</td>
<td>Refers to the inability to speak in a grammatically correct fashion.</td>
</tr>
<tr>
<td>Anomia</td>
<td>A problem with word finding. Impaired recall of words with no impairment of comprehension or the capacity to repeat the words.</td>
</tr>
<tr>
<td>Anterior cingulated cortex</td>
<td>Also known as ‘area 25’ – involved in decision making and emotional regulation as well as vital to the regulation of physiological processes, such as blood pressure and heart rate.</td>
</tr>
<tr>
<td>Astrocytic</td>
<td>A star-shaped cell, especially a neuroglial cell of nervous tissue.</td>
</tr>
<tr>
<td>A-ketoglutarate</td>
<td>(alpha-ketoglutarate) is a complex enzyme which:</td>
</tr>
<tr>
<td></td>
<td>- Provides a key step in the citric acid cycle (part of energy production).</td>
</tr>
<tr>
<td></td>
<td>- Provides a way of controlling how fast the citric acid cycle goes.</td>
</tr>
<tr>
<td></td>
<td>- Links the citric acid cycle to amino acid (protein) synthesis and breakdown.</td>
</tr>
<tr>
<td></td>
<td>- Links sugar metabolism to amino acid metabolism.</td>
</tr>
<tr>
<td>Akinetic mutism</td>
<td>Term relates to inability to move (akinesia) or speak (mutism).</td>
</tr>
<tr>
<td>Ataxia</td>
<td>Wide based gait due to poor balance.</td>
</tr>
<tr>
<td>Broca aphasia</td>
<td>Characterised by non-fluent speech, few words, short sentences and many pauses – speaking appears to require great effort.</td>
</tr>
<tr>
<td>Demyelination</td>
<td>A condition of the nervous system in which the myelin sheath of neurons is damaged. This impairs the conduction of signals in the affected nerves, causing impairment in sensation, movement, cognition, or other functions depending on which nerves are involved.</td>
</tr>
<tr>
<td>Dorsolateral</td>
<td>(meaning back and side) – known fully as prefrontal cortex – important in working memory and executive function, including the regulation of thinking and action.</td>
</tr>
<tr>
<td>Echolalia</td>
<td>Repeating or ‘echoing’ what another person has said.</td>
</tr>
<tr>
<td>Excitotoxicity</td>
<td>Pathological process by which nerve cells are damaged and killed by excessive stimulation by neurotransmitters.</td>
</tr>
<tr>
<td>Hyperammonaemia</td>
<td>Metabolic disturbance characterised by an excess of ammonia in the blood – may lead to encephalopathy and death.</td>
</tr>
<tr>
<td>Medulla Oblongata</td>
<td>Lower half of the brainstem – contains the cardiac, respiratory, vomiting and vasomotor centres and deals with breathing, heart rate and blood pressure.</td>
</tr>
<tr>
<td>Mesial</td>
<td>Meaning – ‘of, in, near, or toward the middle’</td>
</tr>
<tr>
<td>Operculum</td>
<td>Meaning – ‘little lid’ – part of the cerebral cortex.</td>
</tr>
<tr>
<td>Orbitofrontal</td>
<td>Relates to ‘taste’ and ‘sight’ – damage can impair the learning and reversal of stimulus reinforcement associations.</td>
</tr>
<tr>
<td>Paraphasia</td>
<td>Partial aphasia in which the individual employs wrong words or uses words in wrong and senseless combinations.</td>
</tr>
<tr>
<td>Phosphorylation</td>
<td>A biochemical process that involves the addition of phosphate to an organic compound.</td>
</tr>
<tr>
<td>Polyneuropathy</td>
<td>Neurological disorder that occurs when many nerves throughout the body malfunction simultaneously.</td>
</tr>
<tr>
<td>Transketolase</td>
<td>An enzyme that participates in the transfer of ketal groups. Determination of activity in the red blood cell is an indirect indicator of thiamine deficiency.</td>
</tr>
</tbody>
</table>