The nutritional management of a central venous incident

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Abstract

A central venous incident (CVI) is the second leading cause of death worldwide, and is associated with permanent disabilities.There are many nutrition and lifestyle modifiable risk factors for a CVI. These include diabetes, and hypercholesterolaemia and hypertension, all of which are largely preventable, and involve effective, low-cost treatment. Malnutrition in CVI patients ranges from 6-62%, and often worsens during hospitalisation owing to multiple factors, including dysphagia, the inadequate intake of food, inactivity and metabolic changes in the clinical setting. When malnutrition is present in patients who have an acute CVI, the increased risk of poor functional outcomes relates to complications such as gastrointestinal bleeding, pressure ulcers, and urinary tract and respiratory infections. These are associated with higher mortality and increased length of stay in hospital, and contribute to decreased quality of life and impaired rehabilitative outcomes. Screening and nutritional assessment is vital on admission. The Mini Nutritional Assessment and Patient-Generated Subjective Global Assessment have been validated in this patient population. The energy and protein requirements of stroke patients are poorly defined. There is some evidence for the supplementation of antioxidants, but the efficacy thereof depends on their ability to cross the blood-brain-barrier. Large-scale studies are necessary to assess the effect on neurocognitive recovery. Meeting requirements in this patient population is a challenge because of dysphagia, as well as neurological and cognitive deficiencies, and is best achieved with the support of a multidisciplinary team. Early enteral nutrition improves survival, while oral nutrition supplements improve nutrient intake and quality of life.

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Introduction

A central venous incident (CVI), also referred to as a central venous accident, stroke or brain attack, is caused by an interruption to the cerebral blood flow. The consequence may be a temporary or permanent injury, and loss of function of the brain tissue. There are two main types of CVIs, i.e. a ischaemic CVI (85% of cases), which is the result of an occluded blood vessel, and is subdivided into embolitic and thrombotic; and a haemorrhagic CVI (15% of cases), which occurs when a blood vessel in the brain bleeds or ruptures secondary to an aneurysm or arteriovenous malformation, and can be subdivided into intraparenchymal and subarachnoid. A transient ischaemic attack is caused by a temporary clot, and is often referred to as a mini stroke.[1]

Incidence, morbidity and mortality

A CVI is the second leading cause of death worldwide.[2] Fifteen million people suffer a CVI each year, approximately a third dies, and a third is permanently disabled.[3] The direct and indirect cost of CVIs in the USA in 2010 was $74 billion, and approximately €64 billion in Europe.[4,5] The World Health Organization predicted that disability-adjusted life years lost to CVIs would rise from 38 million in 1990, to 61 million in 2012.[3] More than 30% of CVI survivors have permanent disabilities, and approximately 20% require institutionalised care for three months.[6]

One in three deaths in the USA is caused by heart disease and CVIs, and at least 200 000 deaths are preventable.[7] There is strong evidence that the decline in CVI incidence in the USA can be attributed to a combination of interventions, of which improved control of hypertension is thought to be the most likely.[8] There are many nutrition and lifestyle modifiable risk factors for CVI, including diabetes, hypercholesterolaemia and hypertension, all of which are largely preventable and involve effective, low-cost treatment. [9,10] Overweight and obesity are also accepted as risk factors for a CVI. A weight reduction or a body mass index (BMI) of 18.5-25.0 kg/m^2 is often recommended after a stroke, but has never been confirmed in a clinical trial.[11] The benefit of overweight in the survival of a CVI is suggested by some evidence.[12-17] These data are referred to as the obesity paradox, but remain poorly understood.[11]

Malnutrition in central venous incident patients

From a nutritional point of view, knowing which part of the brain has been affected by a CVI assists with the anticipation of likely nutritional consequences in terms of formulating a nutrition care plan in order to ameliorate the risk of malnutrition. For example, a CVI may be associated with inadequate nutrient and energy intake, for instance due to hemianopia,[18] which may cause the patient to only see half of the food on the plate; or aphasia,[18] with the attendant inability of the patient to express food preferences.

Early nutrition support is essential in preventing malnutrition and improving outcomes after a CVI. Nutrition intervention is part of the multidisciplinary approach during the acute and rehabilitation phases of a CVI.[9] Malnutrition in CVI patients ranges from 6-62%,[19-37] and often worsens during hospitalisation,[38-45] depending on the type and severity of the CVI. Patients who experience a haemorrhagic CVI, for instance, are more likely to develop malnutrition than those who have an ischaemic stroke.[43] Females are also more prone to malnutrition than males, probably because they tend to have a CVI at a more advanced age.[46] The presence of pre-existing malnutrition and the development of malnutrition after a CVI has implications in terms of outcome.[9]

Malnutrition on admission

Fifteen to seventy per cent of patients are malnourished on admission to hospital,[9,19,47-52] with 8-49% of CVI patients showing signs of protein-energy malnutrition (PEM) at the time of the incident. The risk factors for and the reported wide variability in prevalence (Table I) are thought to be due to the variable definitions of malnutrition and the diagnostic criteria used.[19,20,41] The serum micronutrient levels (vitamins A, E and C, and riboflavin) may be decreased on admission, and can deteriorate further during hospitalisation either due to CVI-induced oxidative stress or pre-existing malnutrition.[53-58]

Table I: Risk factors for malnutrition in central venous incident patients[6,43,59,60]

On admission	After a central venous incident
• Being older • Being institutionalised • Poor social circumstances • Prior cognitive impairment • A physical disability • The presence of gastrointestinal disease • Chronic disease • Using polypharmacy • Functional disability • Diabetes mellitus • A history of strokes	• Experiencing a cognitive deficit, such as visual neglect (loss of the visual field on the side on which the stroke occurred) • The presence of upper extremity paresis • Depression and a poor appetite • Apraxia affects a stroke patient's ability to self-feed • Dysphagia

Malnutrition during hospitalisation

The weight loss, muscle and fat mass decreases which occur during hospitalisation in CVI patients in half[18,23,39-45] of the patients referred to a stroke rehabilitation clinic, as well as the increased malnutrition prevalence range during the hospital stay (16-26% after one week, up to 35% at two weeks), are equally concerning.

Geriatric patients who had suffered a severe stroke were reported to be at higher risk of malnutrition (56% among those with a hospital stay greater than three weeks),[61] and particularly those who required feeding assistance (32%).[27] This scenario is supported by more recent studies[33,36] in which dysphagia was also highlighted as a major risk factor for malnutrition in CVI patients.[6]

Mechanisms of malnutrition post a central venous incident

Risk factors which may contribute to the increased prevalence of malnutrition associated with a hospital stay include lack of early assessment and treatment, failure to recognise or treat dysphagia, an inadequate energy intake, feeding difficulties consequent to neurological, cognitive and motor impairment,[12,21,23,61] inactivity, paralysis, neuroendocrine sympathetic activation, fever, dysregulation of the appetite, proinflammatory cytokines and systemic hormonal imbalances.[11]

Dysphagia

Dysphagia is common, occurring in 22-65% of CVI patients,[20,62-71] and may persist in some patients for many months.[65,68,72] Although approximately 80% of CVI patients regain their ability to swallow within 2-4 weeks,[62,70,73] waiting for spontaneous improvement in the ability to swallow results in delays in the initiation of nutrition support, and increases the risk of the patient becoming malnourished.[74] Complications associated with dysphagia in acute CVI patients include a poor prognosis, pneumonia, malnutrition, dehydration, persistent disability, increased length of stay in hospital, increased rehabilitation time, institutionalisation after discharge, increased healthcare costs and mortality,[65,75] as well as a compromise to the quality of life of both the patient and caregiver.[71] In relation to pneumonia and its complications (poor nutritional status during hospitalisation,[69,75-77] increased costs associated with longer hospitalisation,[75,69] and greater disability at three and six months),[69,75,78] awareness of silent aspiration without signs of distress[65] is of critical importance, and is reported to be the leading cause of mortality after CVI, accounting for nearly 35% of post-CVI deaths.[75,76]

Energy intake

Inadequate energy intake contributes to escalating malnutrition in hospital, and may relate to dysphagia or other factors. In a study on a 100 consecutive patients, only 10% consumed ≥ 100% of the estimated average requirement (EAR) for energy within two weeks of admission, while 33% consumed < 50% of EAR before discharge.[79] In severely ill stroke patients admitted to a neurosurgical intensive care unit, the average daily energy intake of the non-survivors for the first seven days was significantly lower than that of the survivors (p-value 0.034).[80]

Bed rest and inactivity

Bed rest and inactivity are known to contribute to muscle loss which has been reported to occur within 10 days of bed rest commencing,

and shown to result in a 30% reduction in muscle protein synthesis in healthy adults. Furthermore, a 6% leg lean muscle loss has been shown to result in 16% reduced muscle strength,[58] which may contribute to the malnutrition, as well as to the functional progression and outcomes of CVI patients.

Sarcopenia also occurs after a stroke due to both paralysis and decreased physical activity. Disuse atrophy is not confined to the paralysed limb.[11,81] Thus, it is thought that other mechanisms, in addition to bed rest and inactivity, may play a role in muscle loss after a CVI.

Metabolic and neuroendocrine abnormalities

Rapid and dramatic weight loss in rodent models has been demonstrated after an experimental CVI which started immediately after the induction of ischaemia, and reached a maximum of up to 20% within five days. This indicates enhanced catabolic signalling beyond physical inactivity.[11] The contributing factors are not yet understood, but increased cytokines and sympathetic and neuroendocrine activation are believed to play a role.[11] Inflammatory cytokines induce tissue degradation and weight loss in humans. Tumour necrosis factor transcripts were found to be higher in both the paralysed and non-paralysed thigh muscles of subjects compared with those in the age-matched controls.[11,82]

Systemic neuroendocrine activation develops after a CVI, and includes upregulated local and systemic sympathetic activation, dysregulation in the thyroid and hypothalamus-pituitary-adrenal axis, as well as decreased vagal stimulation.[11,41,83,84] Upregulated sympathetic signalling may explain the overall catabolic stimulation.[11] Both catecholamines and natriuretic peptides (particularly A-type natriuretic peptide) have been shown to exert strong lipolytic signals.[11] Increased natriuretic peptides levels were observed in patients after a stroke, parallel stroke severity and infarction volume.[11,85] Hypothalamic damage results in dysregulation of the hypothalamus-pituitary-adrenal axis.[11,84,86] Consequently, both cortisol and corticotropin plasma levels increase early after the onset of symptoms and correlate with CVI severity.[11,84,87]

Malnutrition and outcomes

The relationship between PEM and poor outcomes, including impaired recovery, may relate to the influence of PEM on the mechanisms of ischaemic brain injury.[88] Animal studies indicate that PEM alters the expression of plasticity-associated genes (associated with the recovery mechanisms after global ischaemia), and induces changes in hippocampal plasticity-associated protein.[89] This suggests that PEM may induce abnormalities in function, plasticity and the structure of the hippocampal fibres.[89]

There is evidence of poor outcomes due to malnutrition with respect to both haemorrhagic and ischaemic strokes.[6,33,34,43] When malnutrition is present in acute CVI patients, there is an increased risk of poor functional outcome, gastrointestinal bleeding, pressure ulcers, urinary tract and respiratory infections, higher mortality and increased length of stay in hospital,[6,12,23,25,28,33,34,43,90-92] all of which contribute to an increased hospital stay, decreased quality of life and impaired rehabilitative outcomes.[90] A higher energy intake in the early stages of admission was one of the modifiable factors which enabled a prediction of the extent and rate of restoration of functional abilities at discharge.[79] In this regard, a multicentre randomised trial, the Feed or Ordinary Diet (FOOD) Trial 1 (n = 2 955), further documented that undernourished patients were significantly more likely to die during follow-up than patients of normal weight,[91] an association which has been confirmed by the findings in other studies.[23] Furthermore, dehydration, common in CVI patients (i.e. in 53% of patients on admission),[37] with the associated increase in haematocrit and blood viscosity, can worsen the ischaemic process. Furthermore, high plasma osmolality levels on admission have been reported to be associated with poorer survival at three months.[6] The early identification and treatment of malnutrition can affect the patient's ability to take part in rehabilitation, functional activities and complete daily living activities.

Screening and nutritional assessment

The high incidence of malnutrition on admission and the deterioration in nutritional status in hospital indicate that it is of utmost importance to screen and assess CVI patients early after admission, and to continue monitoring such patients throughout the hospitalisation period using age-specific screening tools. Although it has been argued[19] that these screening tools may not have been validated in CVI patients, some, such as the Mini Nutritional Assessment[93,94] and Patient-Generated Subjective Global Assessment, have been validated.[93,95] The involvement of a speech therapist in the management of the patient is also essential after admission, in order to assess the patient's ability to swallow using the Eating Assessment Tool-10 and/or the Minimal Eating Observation Form screening tools, which have been shown to be of benefit.[96,97] In the absence of a speech therapist, other, less reliable, general guidelines which can be applied to assess swallowing include the presence of a "wet" (gurgly) voice, a weak, voluntary cough, prolonged swallowing, coughing on swallowing, and hoarding of food in the cheeks.[65] However, aspiration, and particularly silent aspiration, cannot be eliminated with the use of any of these general guidelines.

A complete nutritional assessment should be carried out on all CVI patients after admission. A detailed nutrition history, including weight changes and intake, should be obtained on admission.[9] Since many of these patients are unable to speak or are cognitively impaired, it is often necessary to obtain the help of a family member or nurse if the patient is residing in a frail care facility.[9] Monitoring intake, dysphagia, depression and appetite will alert staff to the risk of malnutrition.[9] Pre-existing malnutrition can be identified with a clinical assessment, which provides a reference point for monitoring. Although anthropometry should be part of a complete nutrition assessment, it often difficult to obtain even basic measurements, such as weight and height, in CVI patients. However, a strong correlation between mid-upper-arm circumference (MUAC) and BMI has been reported, making MUAC an viable option in the long-term monitoring of the patient.[90]

The biochemical assessment varies with severity of illness. When an acute-phase response is present, nutritional status will not be reflected by most serum protein.[98,99] Nevertheless, decreased albumin levels in acute CVI patients are associated with impaired functional status, a greater risk of infectious complications, higher mortality and poor outcomes.[6,25,43,99] Albumin can be used as a predictor of mortality during hospitalisation and the need for institutionalised care. Pre-albumin in young ischaemic CVI patients is also an independent predictor of clinical outcome.[100] Albumin can be used serially as a marker of nutritional status in long-term patients in the absence of inflammation.[8] An assessment of the blood glucose should be included, since hyperglycaemia is associated with poor functional recovery or outcome,[101-103] higher mortality,[102,104-106] acute deterioration with respect to a minor CVI[107] and infarction expansion.[101,106-109]

Medication may impact on intake, decrease energy requirements and result in specific nutrient deficiencies (Table II). CVIs are more common in elderly patients who may also be on other drugs for pre-existing chronic diseases, which may influence their taste and smell, functions which may have already been altered consequent to the CVI. Other interactions in relation to weight loss or gain, taste alterations and smell may also occur.[110]

Table II: Nutrition-medication interactions and consequences

Medication	Nutrition impact
Propofol	Provides 1.1 kcal/ml as fat[9]
Phenytoin	• Decreased absorption when given with continuous enteral nutrition[9] • Folate and vitamin D depletion[9]
Narcotics	Constipation
Morphine	A decrease in calorie requirements, resulting in an 8% decrease in MEE in critically ill patients[111]
Neuromuscular blocker (pancuronium) plus morphine	A decrease in calorie , resulting in a 4.6 kcal/kg/day decrease in MEE, and a 5% below HBE prediction, using the actual weight[112]
Barbiturates	A decrease in calorie requirements, resulting in 14% decrease in below PEE (using the Harris Benedict equation),[113] and a 42% decrease in REE[114]

MEE: mean measured energy expenditure, PEE: predicted energy expenditure, REE: resting energy expenditure

Does nutrition support improve outcomes?

Despite the reported association of poor nutritional status with worse outcomes in CVI patients, good clinical data on outcomes are inadequate and are derived from small-scale studies. Although mortality is usually seen as the gold standard in terms of outcome, it needs to be debated whether nutrition support in this patient population can realistically alter the mortality rate, and/or whether quality of life, improved neurocognitive deficit and functional outcome are sufficient indications of the success of the nutrition support. Even in terms of functional outcomes, nutrition support alone is unlikely to result in improvements without active rehabilitation, involving medical, physiotherapy, occupational therapy and speech therapy participation. Differences in the indices of nutritional status were not shown between energy- and protein-rich sip feedings and standard nutritional care in randomised controlled trials.[90,115,116] The failure to demonstrate a positive response may be because of an inability to identify patients who are truly malnourished, and thus most likely to benefit from nutrition support.[19]

It was suggested in the FOOD trial 2 that early tube feeding may substantially reduce the risk of dying after a CVI, but that the improved survival may be at the expense of increasing the proportion of those who survive having poor outcomes.[30] On the other hand, improved survival with oral supplementation in well-nourished CVI patients was not documented in the FOOD trial 1.[29] However, the FOOD trial 1 was criticised because nutritional status was assessed only once, standardised assessment methods were not used at all of the trial sites, and the nutritional content of the diets and compliance with the supplements were not documented.[9]

Importantly, it was reported in a study on undernourished CVI patients that oral supplementation significantly improved nutritional intake, as well as nutritional status and survival.[117] Thus, it would appear that undernourished patients may be those who might benefit most from oral supplementation. Nutrition support has also been shown to be effective in terms of reducing the length of stay in hospital,[118] improving health-related quality of life and grip strength,[119] reducing pressure sores, increasing energy and protein intake,[120] and improving motor function measures, but not measuring cognition[116] in CVI patients.

Nutrient requirements

The overall aims of nutrition support in acute CVI patients are to avoid weight and muscle loss, to adapt the diet to the swallowing ability of individuals and to optimise functional recovery.

Table III: Eating difficulties observed in central venous incident patients

Rehabilitation unit[27]	General hospital[124]
• Eats ≤ ¾ of the served food (60%) • Has difficulty manipulating the food in the plate (56%) • Has a problem with the transportation of food to the mouth (46%) • Problems with intake in the sitting position (29%) • Has abnormal eating speed (slow or fast) (26%) • Has problems with manipulating food in the mouth, e.g. leakage, hoarding and chewing difficulties (24%) • Has swallowing difficulties (18% • Has problems opening and closing the mouth (16%) • Level of alertness (9%)	• Manages food on the plate (66%) • Problems with food consumption (55%) • Problems with intake in the sitting position (45)

Energy

Various factors contribute to the altered requirements of stroke patients, such as need for ventilation, and the presence of infections, pre-existing malnutrition and co-morbidities.[9] Medicinal treatment (Table III) in the acute period after a CVI, such as barbiturates, or induced hypothermia to reduce intracranial pressure, results in a decrease in energy requirements.[9,121,122] On the other hand, a neurological insult is associated with altered metabolic demands owing to increased peripheral plasma catecholamines, cortisol, glucagon, interleukin (IL)-6, IL-1RA, and acute-phase protein.[123] Paralysis or inactivity may decrease requirements.[9]

Unlike the well documented hypermetabolism and increased nutrient requirement needed for patients witha head injury,[125,126] no single formula for the calculation of energy requirements has been validated in CVI patients. Indeed, the presence of hyepremetabolism in stroke patients was recently challenged.[127] An evaluation of 91 CVI patients from the time of their stroke to 90 days indicated that energy expenditure increased by only 10-15% above the Harris Benedict equation and resting energy expenditure did not vary with the stroke characteristics.[123] Linear regression analysis in 34, sedated, mechanically ventilated patients with ischaemia and haemorrhagic stroke revealed a significant relationship between total energy expenditure (TEE) and Harris Benedict equation calculation without a stress or activity factor, suggesting that the Harris Benedict equation for basal energy needs appeared to most accurately reflect the TEE for a CVI patient.

The evidence from several studies supports the exclusion of a stress factor from the Harris Benedict equation in stroke patients in order to avoid the complications of overfeeding and its complications, especially hyperglycaemia.[127] On the basis of the available evidence, a daily protein intake >1 g/kg is recommended in clinically stable patients in the subacute phase of a stroke with normal renal function, in order to achieve a carbohydrate to protein ratio $< 2.5: 1$, an energy intake of ≥ 25 kcal/kg in non-obese subjects to maintain body weight, and < 25 kcal/kg in obese subjects to maintain a carbohydrate to protein ratio $< 2.5: 1$.[56] However, these patients are in an intensive care unit environment, so any complications, such as infections, may alter these recommendations. In addition, it is uncertain if active rehabilitation was taken into consideration in these studies; another factor which can lead to increased energy requirements. Close monitoring and clinical observation remain important indicators of energy sufficiency.

Protein

Adequate protein intake may be of greater importance than energy intake in the CVI patient. A negative nitrogen balance was reported in 44% of acute CVI patients on enteral feeds where the energy requirements were calculated with the Harris Benedict equation after 24 hours of goal feeding. The mean time to achieving the nitrogen balance was five days.[128] The synthesis of brain protein is essential for neuron survival.[129] Experimental studies have shown that acute ischaemia induces early and profound alterations of brain protein synthesis,[56] including suppressed protein synthesis in the ischaemic penumbra,[130] which, if not reversed, can result in cell death and the progression of the infarction zone.[56] Thus, the restoration of protein synthesis may assist the cells with repairing ischaemic damage and the recovery of function.[131-134] Additionally, since glucose utilisation in the ischaemic brain neurons is impaired, amino acid serves as an alternative source of aerobic energy production.[6] The low plasma levels of tyrosine, the amino acid precursor of the brain adrenergic neurotransmitters; epinephrine, norepinephrine and dopamine, has been found in patients who have had an ischaemic stroke.[135]

A series of studies have been supportive of the finding that a daily 20 g protein supplementation was associated with the better recovery of neurological deficit and improved cognitive recovery.[136-138] On the other hand, a negative correlation with the dietary carbohydrate to protein ratio in patients with subacute CVI was reported.[137] On the basis of the available evidence and personal experience, the use of a 20 g protein supplement to meet requirements and support improved cognitive recovery and neurotransmitter synthesis is recomended.[47] It would appear that there is little evidence to support a protein intake of 1-1.5 g/kg.[9,127]

Carbohydrates

Care should be taken not to overfeed carbohydrates to stroke patients. A carbohydrate intake in excess of protein has been shown to slow neurocognitive recovery.[56] Various alterations in the brain glucose metabolism occur in focal cerebral ischaemia, including hyperglycolysis in the penumbra cells, and reduced aerobic glucose metabolism in the cerebral region.[139,140] Excessive carbohydrate ingestion in this condition may result in increased lactate production, which may further impair brain structures.[56]

Lipids

Little is know about the ideal energy contribution made by lipids to total energy intake, and the effect of supplemental fatty acids, such as n-3 fatty acids, on acute stroke patients. A lower eicosapentaenoic acid concentration was reported to be one of the significant risk factors for an ischaemic stroke in a cross-sectional study.[141] It was suggested in another study on Japanese patients who had an acute ischaemic stroke that a low serum n-3 polyunsaturated fatty acid (PUFA) to n-6 PUFA ratio on admission predicted neurological deterioration.[142]

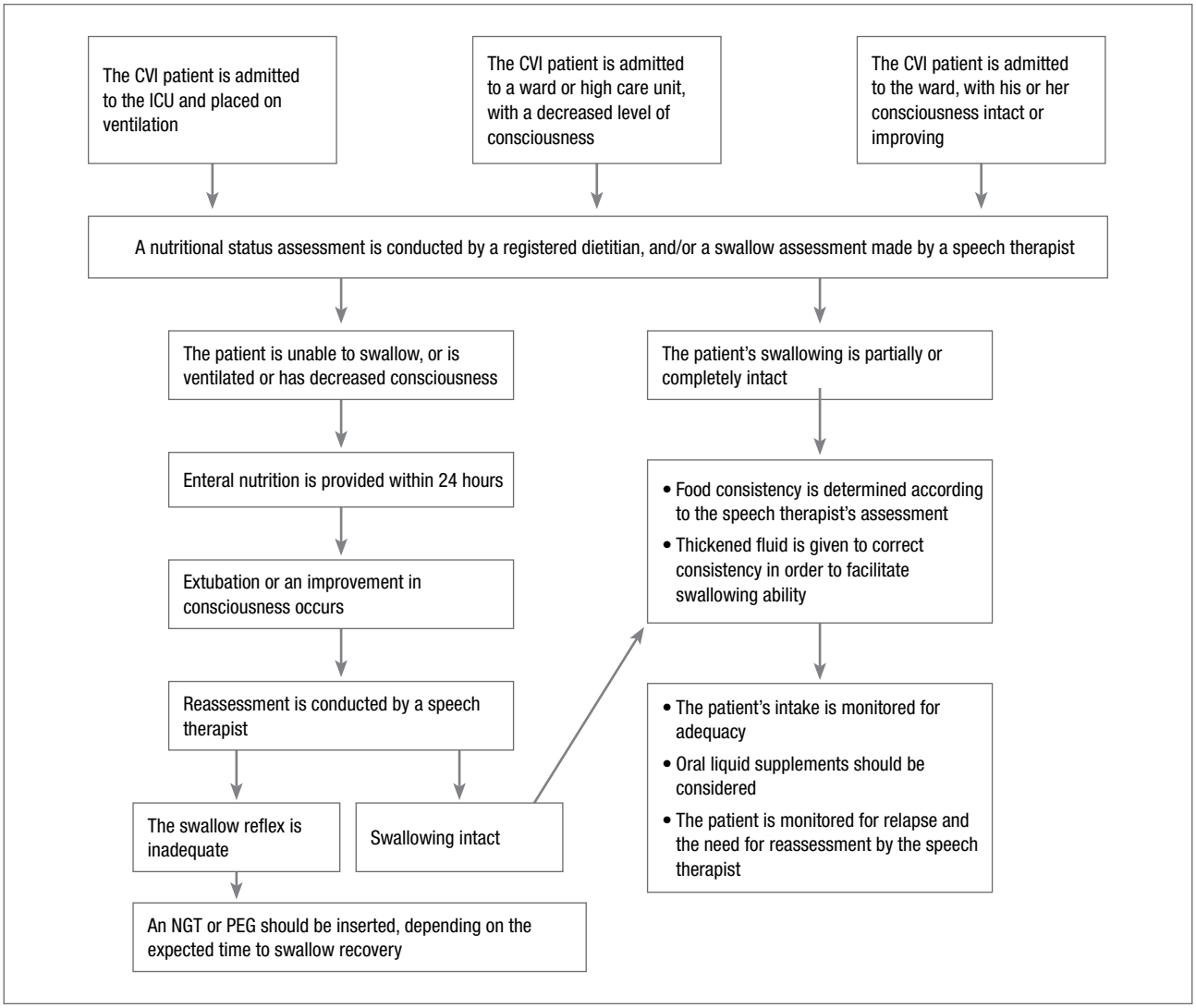
Antioxidants and other micronutrients

Cellular damage in cerebral ischaemia is partly caused by oxidative damage, secondary to free radical formation and lipid peroxidation.[143-147] Various indications of oxidative stress have been documented, including increased plasma levels of cholesteryl ester hydroperoxide, a maker of lipid peroxidation,[143] and a significantly higher prooxidant burden.[148] In addition to indications of heightened oxidative stress, the serum levels of various antioxidants have been shown to be decreased in CVI patients. Reduced vitamin A, E and C levels[53] in patients with acute ischaemic CVI,[54] reduced total antioxidant capacity in patients with acute CVI,[55] and also a

significant reduction in serum vitamin C[35] and zinc, have been reported.[53,149-152] Oxidative stress seems to be more pronounced in diabetic CVI patients than in non-diabetic patients who have experienced an acute CVI.[153] This redox imbalance has been shown to continue in the post-acute rehabilitation period.[154]

In terms of supplemental therapy, the potential benefits of such supplements depend upon the extent to which these substances can cross the blood-brain barrier.[145] Nevertheless, a low zinc intake has been reported in CVI patients,[155] and the administration of a daily 10 mg zinc supplementation for 30 days was associated with better recovery of neurological deficit, as determined by the National Institutes of Health Stroke Scale score, when compared to the control patients.[155] The mechanisms responsible for the positive effect on neurological recovery include the brain reactivation of protein synthesis, improved chemical neurotransmission and the repair of cerebral damage.[56] Both deficiency and excess zinc may affect brain recovery.[150] Thus, the zinc status of such patients needs to be normalised, rather than the patient being oversupplemented.

A combination of the B-group vitamins and folic acid may reduce oxidative damage after a CVI since it has been shown to significantly decrease serum malondialdehyde levels, a marker of lipid peroxidation, and result in significantly lower levels of C-reactive protein (CRP), a marker of tissue inflammation.[156] The use of oral vitamin E, C with B vitamins and folic acid, alone or in combination, was compared in acute ischaemic CVI patients, against a control group. Antioxidant capacity improved in the total antioxidant group, and declined in the control group. Interestingly, the changes were less marked in subjects who received B-group vitamins, with or without antioxidants. The plasma malondialdehyde levels decreased significantly in all three treatment groups, but increased in the control group.[157] Serum CRP was significantly lower in the treatment groups, compared with the control group.[157] These studies were conducted in patients who had an acute ischaemic stroke, but it can be expected that antioxidant capacity would have further declined during the rehabilitation period owing to the acute event and an inadequate diet in the first few weeks post CVI.[53] Additionally, four subgroups in a randomised double-blind, placebo-controlled trial



CVI: central venous incident, ICU: intensive care unit, NGT: nasogastric tube, PEG: percutaneous endoscopic gastrostomy

Figure 1: Nutrition support algorithm [9]

on CVI patients admitted to a rehabilitation facility, received either daily oral antioxidants or n-3 fatty acids, both, or a placebo, for 12 months. One year later, a trend for lower mortality was found in the subgroups who received n-3 fatty acids, but this did not improve the rehabilitation outcome.[44]

Clearly, there is need for further research of micronutrient supplementation in stroke patients in order to define safety, effectiveness, efficacy, supplementary dose, frequency and clinical outcomes.

Meeting nutrient requirements

The challenge in CVI patients is not so much meeting very high energy and nutrient requirements, but rather meeting the minimum requirements. Nutrition support should be started within 24 hours of the CVI. How the patient is fed is determined by the clinical setting, nutritional status, the presence of dysphagia and the severity of the CVI (Figure 1). Enteral feeding is the method of choice for patients who are unable to eat, if their gut works.[6,9] If the patient is experiencing increased intracranial pressure, gastric emptying may be delayed and post-pyloric feeding or prokinetics will need to be considered.[9]

Fine-bore enteric polyurethane nasogastric tubes (NGT) should be used in patients when enteral feeding is expected to occur for a considerable period.[9] To avoid aspiration, the bed should be elevated, and a clinical assessment carried out daily.[9] Confusion often results in the removal of the NGT by the patient, resulting in a loss of feeding time[9] as the use of a nasal bridle[158] is not available in South Africa. Instead, it is important to try and make up the loss in feeding time. as indicated by the Enhanced Protein-Energy Provision via the Enteral Route Feeding (PEP uP) protocol.[159] The position of the NGT should be assessed regularly. After approximately two weeks, the patient's swallowing function must be determined with videofluoroscopy by a speech therapist. If this indicates that enteral nutrition will be needed for an extended period, a percutaneous endoscopic gastrostomy (PEG) should be inserted. Most CVI patients tolerate a polymeric formula. Semi-elemental formula is generally only indicated in those with complications. A fibre-containing formula is necessary in long-term patients not on inotrope therapy since CVI patients are prone to constipation.[9] Parenteral nutrition is seldom used in CVI patients, and is reserved for those with an ileus, when there is the use of escalating doses of inotropes, increased intracranial pressure and in patients receiving paralysing agents.[9]

The transition from enteral feeding to oral intake is often a gradual one. Initially, it may include stopping the enteral feeding one hour before the controlled feeding is started by a speech therapist; then progressing to night feeds with small frequent meals, and eventually to a full oral intake with sip feeds.[6] During the transition to oral feeding, monitoring of the energy and protein intake, hydration status, weight, electrolytes and the development of any respiratory complications is important.[6] The transition from tube to oral feeding can be physically and mentally challenging and stressful as it often includes not only the challenge of improving the swallowing function, but also because some patients may have to learn to recognise food, and to learn to eat, again.[6] This is further complicated by physical disabilities, such as hemiplegia and loss of function in the dominant arm.

Table IV: The challenges associated with food intake after a central venous incident[6,59,161,162]

Challenges	Resultant behaviour
Medication	Poor appetite
Altered sense of taste and smell	
Depression	
Facial weakness	Drooling
	Spillage, i.e. taking in an inadequate amount of food
	Reluctant to eat owing to embarrassment
	Eats slowly, i.e. the food is removed before the meal is finished
Poor arm or hand function Haemiplegia on the dominant side	Cannot maintain head and body position
	Cannot manipulate food on the plate
	Eats slowly, i.e. the food is removed before the meal is finished
	The use of special utensils may improve intake
Communication	Is unable to communicate preferences or problems
Visual and perception disorders	Cannot see part of the plate
Cognitive deficit	May limit the ability to self-feed
Alteration in consciousness	Misses meal times or falls asleep during a meal
Drowsiness	
High levels of anxiety	Avoids eating in the presence of other people
Fatigue	Falls asleep during meals

Eating difficulties

Up to 50% of patients who have a CVI are usually unable to swallow safely, and more than 80% of patients hospitalised for > 21 days have been reported to have had difficulty eating.[21] Eating difficulties were observed in 80% of patients (Table III) admitted to a CVI rehabilitation unit, and 53% were unable to eat without assistance.[27] Along similar lines, it was reported in another study that 82% of acute stroke patients in a general hospital had eating difficulties[124] which clearly compromised energy and nutrient intake and quality of life (Table IV).

The management of dysphagia[9] depends on its nature (oropharyngeal and/or oesophageal, which is further divided into subtypes of mechanical and/or motor dysphagia and its severity) (Table V), and includes postural adjustment, swallow manoeuvres, pharyngeal electrical stimulation treatment and diet modification.[9,75]

Table V: The three levels of the National Dysphagia Diet and suggested liquid consistency[9,75]

Level	Examples
Level 1: Dysphagia (puréed)	Smooth, cooked porridge, puréed vegetables, puréed fruit (without the skin and pips), thickened juice, puréed legumes, puréed, strained and/or thickened soup, puréed meat, moist scrambled eggs, mashed potatoes, custard, yoghurt, and smooth puddings, i.e. cream caramel *Liquids:* Spoon thick, i.e. thick enough to coat a spoon
Level 2: Dysphagia (mechanically altered)	Between a puréed and a soft diet. Soft cereal with texture, e.g. oats, porridge, noodles and pasta in a sauce, soft canned or cooked fruit, ripe bananas, moist minced meat, steamed fish in cheese sauce, scrambled eggs, soup with small pieces in it, well-cooked soft vegetables, mashed or soft boiled potatoes, soft baked desserts, milk-based desserts, jelly and custard *Liquids:* Nectar thick, i.e. with the consistency of nectar
Level 3: Dysphagia (advanced)	A soft diet. Bread without seeds; moist cereal, rice and pasta; desserts without nuts, no dry biscuits, soft, peeled fruit; minced or soft meat with gravy; soft, cooked vegetables; boiled, mashed and baked potato, and sweet potato. *Liquids:* Honey thick, i.e. with the consistency of honey
Regular	No restrictions. May vary according to individual tolerance

Patients who are able to eat may require consistency changes, i.e. a puréed or soft diet, according to the level of disorder, and the addition of thickener to liquids. The American Dietetic Association, through the National Dysphagia Diet Task Force, developed the National Dysphagia Diet (NDD).[162] The NDD includes three levels of solid food, and four levels of fluid (thin, nectar thick, honey thick and spoon thick) (Table 5).[9,75]

Currently, there is no strong evidence for the use of thickened liquids, despite its inclusion in the guidelines.[75] Patient compliance with thickened liquids is often poor because of its low acceptability.[75] Reduced fluid intake may result owing to difficulties with swallowing liquids.[9,75] The aspiration of water is seen as a benign event.[9] The Frazier Water Protocol was developed to improve fluid intake, without the risk of aspiration, in patients restricted to no oral intake or thickened fluids only. According to the protocol water intake is permitted, according to specific guidelines.[9,75] This approach has not been objectively tested, but experience from the Frazier Rehabilitation Institute, USA, is of very low levels of aspiration, dehydration and chest infections.[75]

Modified diets may result in poor intake owing to poor acceptance and physical disability, and may contribute to malnutrition in this patient population.[71] This necessitates the use of oral liquid supplementation or even continuous or night enteral feeds until intake from food alone is optimal. It is essential that nursing staff are trained in proper feeding techniques with respect to patients with feeding dependence. Complications may occur with rapid and uncontrolled presentation of the food by the caregiver. Other factors which may help to improve intake include eating in an environment devoid of external distraction, and the use of adaptive equipment, such as angled utensils.[71]

Protocol

The burden on nursing personnel in a CVI unit is very high. Patients require assistance for nearly all daily living tasks. In terms of nutrition, of those who can eat orally, many require assistance, or may even have to be fed. A large proportion of patients are on bolus feeds, supplied via a NGT or PEG. The protocol ensures that personnel are familiar with the type of feeds used, and general progression and feeding times in order to prevent errors. Targets set for patients on enteral nutrition can be achieved and hospital-acquired malnutrition prevented if the PEP uP guidelines[159] are followed.

Conclusion

A CVI is a life-changing incident, and long-term quality of life depends upon recovery of the neurological and cognitive function. Early assessment and screening, as well as constant monitoring, are essential if malnutrition is to be detected upon admission, and to prevent its progression during hospitalisation. Meeting requirements in this patient population is also a challenge because of the presence of dysphagia, and neurological and cognitive deficiencies.

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