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**Clinical  
Reviews**

## GLUCOSE BEFORE THIAMINE FOR WERNICKE ENCEPHALOPATHY: A LITERATURE REVIEW

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□ **Abstract—Background:** The prevailing teaching in medical school curricula and in medical textbooks is that if thiamine deficiency is suspected, thiamine supplementation should be given before administering glucose. **Objective:** We sought to evaluate the published evidence describing the commonly held belief that thiamine supplementation must be given before glucose in hypoglycemic patients to prevent Wernicke encephalopathy. **Methods:** Articles were identified through computerized searches of MEDLINE and other online sources. Pertinent references were traced back to their sources and also included in the literature review. The quality and content of each article was evaluated by the authors using the American Academy of Emergency Medicine literature review guidelines. **Results:** Nineteen papers were ultimately identified and evaluated. No evidence rose above the level of case report/series. There were 13 case reports/series, 4 animal studies, and 2 expert opinion articles. True clinical research about the question of whether or not a glucose load can precipitate acute onset of Wernicke encephalopathy is lacking. **Conclusions:** Mounting case report evidence suggests that *prolonged* glucose supplementation without the addition of thiamine can be a risk factor for the development of Wernicke encephalopathy. Based on our findings, a delay in giving glucose to hypoglycemic patients cannot be recommended at this time, although prompt thiamine supplementation after or concurrent with a return to normoglycemia is recommended. © 2012 Elsevier Inc.

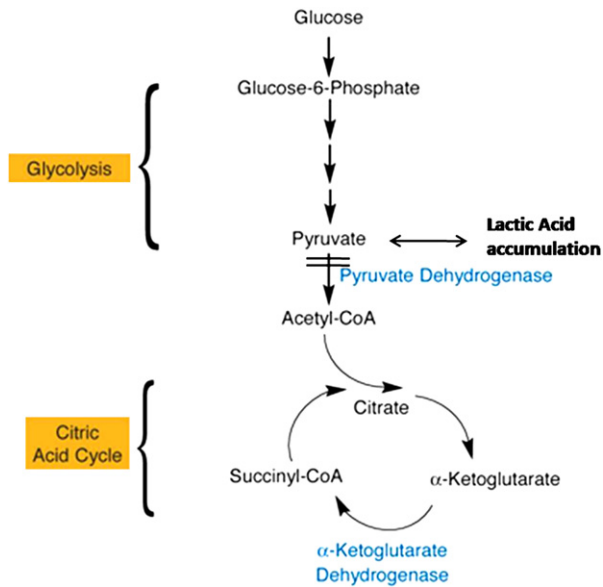
□ **Keywords—Wernicke encephalopathy; glucose; thiamine; medical myths**

### INTRODUCTION

#### *Wernicke Encephalopathy*

Wernicke encephalopathy is an uncommonly recognized neurological disorder caused by prolonged thiamine (vitamin B1) deficiency. In the United States, it is most commonly seen in alcoholics, but can also be found in any malnourished state; patients with hyperemesis gravidarum, intestinal obstruction, acquired immunodeficiency syndrome, gastric bypass, and malignancies are commonly cited (1–5). The typical symptoms include confusion, ocular abnormalities, and ataxia. Prolonged thiamine deficiency can lead to the development of Korsakoff syndrome, characterized by permanent mental impairment with confabulation and memory deficits.

Wernicke encephalopathy was first described in 1881 by Carl Wernicke and was originally named “polioencephalitis hemorrhagica superioris” due to its appearance on autopsy (6). It took 60 years for the link to be made to nutritional deficiency by case reports and confirmed by animal models (7,8).



**Figure 1. Thiamine in glucose metabolism.** The thiamine-dependent enzymes pyruvate dehydrogenase (PDH) and  $\alpha$ -ketoglutarate dehydrogenase ( $\alpha$ -KGDH) participate in the metabolism of glucose through two biochemical reactions, glycolysis and the citric acid cycle. The main function of these two sets of reactions is to generate adenosine triphosphate (ATP), which provides energy for the cells. Reduced PDH and  $\alpha$ -KGDH activity resulting from thiamine deficiency can lead to less ATP synthesis and build-up of the toxic intermediate, lactic acid. adapted from (21).

### *Glucose before Thiamine: Physiology and History*

Thiamine acts as a coenzyme for the decarboxylation of pyruvate to acetyl coenzyme A; this bridges anaerobic glycolysis and the Krebs cycle. Thiamine is also a coenzyme within the Krebs cycle and in the hexose monophosphate shunt (9). A lack of thiamine causes inhibition of anaerobic glycolysis, the process by which glucose is converted into usable energy in the form of adenosine triphosphate (ATP); in starvation, when glucose stores are depleted, the brain suffers most acutely as it relies almost entirely on glucose for energy and there is little thiamine available to act as cofactor for the conversion of any small amount of remaining glucose to ATP. Some damage may also be due to an accumulation in the brain of toxic intermediates, such as lactate (Figure 1) (1,5). Alcoholics are particularly prone to this deficiency due to decreased intake of thiamine, decreased absorption from the gastrointestinal tract in the presence of alcohol, and inability to use thiamine effectively secondary to lack of magnesium (also from malnutrition) as a cofactor for the binding of thiamine to thiamine-dependent enzymes (6).

The prevailing teaching in medical school curricula and in medical textbooks is that if thiamine deficiency is suspected, thiamine supplementation should be

given before administering glucose (10,11). The theory behind this is that if a thiamine-deficient patient is given a glucose load, meager thiamine stores would rapidly be exhausted, glycolysis further limited, and Wernicke encephalopathy would promptly ensue (9,12–14).

This is an important topic, as many alcoholics present to the emergency department with a change in mental status; this can be commonly due to alcohol intoxication, alcoholic ketoacidosis, or thiamine deficiency, as well as other non-alcohol-related causes of acute mental status change. Alcoholic ketoacidosis frequently presents with low blood glucose, and low blood glucose itself could be the cause of an acute change in mental status as the brain is starved of its main source of fuel. Many physicians would assume, due to their instruction in medical school and understanding of thiamine-deficiency pathophysiology, that patients with low blood glucose and suspicion for malnutrition should be given thiamine before glucose. However, because the risks of prolonged hypoglycemia include coma and death, we conducted a literature search to evaluate the source of this medical dogma and to investigate whether or not thiamine needs to be given before glucose in all situations to prevent precipitation or worsening of Wernicke encephalopathy.

## METHODS

A literature search of MEDLINE (1950–present) was performed and limited to studies published in English. The search term “(dextrose OR glucose) AND (thiamin\* or B1) AND (alcohol\* OR ethanol)” yielded 74 references. The search term “(dextrose OR glucose) AND (thiamin\* or B1) AND (Wernicke\*)” yielded 45 references. Searches were combined and the abstracts were assessed for relevance independently by two physicians. If either physician felt the article was relevant, it was included in this literature review. The references of these articles were evaluated, as were the references of subsequent generations of papers in an attempt to find the sentinel citation. Additionally, a search of the Internet search engine Google was performed using the keywords (without quotes) “glucose before thiamine.” This search was repeated on Google Scholar. Multiple articles, papers, and texts from this 248,000-hit search stated that thiamine should be given before glucose upon suspicion of Wernicke encephalopathy; this assertion was evaluated in approximately 50 articles that were deemed relevant to the issue, and any references cited were checked for peer-reviewed publications. No additional peer-reviewed literature was found with this method.

The included articles were evaluated for their level of evidence and methodology using the American Academy of Emergency Medicine (AAEM) literature review guidelines (Tables 1, 2).

**Table 1. AAEM Literature Review Guidelines Evidence Grades**

|         |  |
|---------|--|
| Grade A | Randomized clinical trials or meta-analyses (multiple clinical trials) or randomized clinical trials (smaller trials), <i>directly</i> addressing the review issue   |
| Grade B | Randomized clinical trials or meta-analyses (multiple clinical trials) or randomized clinical trials (smaller trials), <i>indirectly</i> addressing the review issue |
| Grade C | Prospective, controlled, non-randomized, cohort studies  |
| Grade D | Retrospective, non-randomized, cohort or case-control studies  |
| Grade E | Case series, animal/model scientific investigations, theoretical analyses, or case reports   |
| Grade F | Rational conjecture, extrapolations, unreferenced opinion in literature, or common practice  |

AAEM = American Academy of Emergency Medicine.

## RESULTS

The MEDLINE searches, when combined, resulted in 98 unique articles, which were reviewed independently by two physicians. Fourteen total articles were considered relevant by either physician. Examination of the references for the original 14 articles and the references of subsequent generations of papers found 5 additional articles that were selected for review. In total, 19 articles were included.

Google and Google Scholar search identified 1 additional non-peer-reviewed report (Gussow, 2007) and one general overview of Wernicke encephalopathy (Donnino et al., 2007), which were used as reference but not evaluated in the literature review (6,15).

The 19 articles reviewed included expert opinion (n = 2), case reports (n = 13), and animal models (n = 4). Several of the case report articles were written in literature review format; however, the literature reviewed by them never included any evidence of a level higher than case reports. To our knowledge, no randomized trials, cohort studies, or case-control studies exist regarding the use of glucose before thiamine to prevent acute worsening of Wernicke encephalopathy.

The animal studies were rated the best overall, with outstanding quality, and papers involving animal models have illustrated changes in the brains of thiamine-

depleted rats (acidosis in the medial thalamus, hyperintensity on magnetic resonance imaging in the thalamus, hypothalamus, and hippocampus, impairment of blood-brain barrier), within 40 min after glucose infusion, and posit several mechanisms for these changes (16–19). These papers also note worsening of symptoms in the rats after glucose loading. However, the amount and rate of glucose given is variable and cannot be extrapolated to human subjects. The 40-min effect time, likewise, is not generalizable to human patients.

Table 3 summarizes the level of evidence in the 19 articles included in this review (4,9,10,12–14,17–29).

## DISCUSSION

The most often cited source claiming a link between glucose loading and acute onset of Wernicke encephalopathy in thiamine-deficient patients is a four-case series by Watson et al. from 1981 (9). However, as pointed out by Hack and Hoffman in 1998, none of these cases involved the acute administration of glucose (20).

The first patient was a 27-year-old woman with anorexia from gastritis. She received 3 L of 5% dextrose over 24 h and developed Wernicke encephalopathy that resolved partially with thiamine administration. The second case was a 79-year-old woman with sepsis and anorexia from bowel pseudo-obstruction, who was given 2 L of 5% dextrose and developed symptoms of Wernicke that partially resolved with thiamine administration. The third case was a 45-year-old woman with end-stage renal disease and anorexia who was started on peritoneal dialysis using hypertonic glucose solutions due to fluid retention. Forty-eight hours later she developed symptoms consistent with Wernicke that resolved over the course of a month with thiamine administration. The last case was a 36-year-old man who was started on dialysis after myoglobinuric renal failure from a motor vehicle collision. After 5 days, while being given an infusion of 20% dextrose, he developed symptoms typical of Wernicke that partially resolved with administration of thiamine (9).

The 1981 Watson article uses a single-case report by Drenick et al. in 1966 as a reference for evidence of acute precipitation of Wernicke encephalopathy by

**Table 2. AAEM Literature Review Guidelines Rank based on Design and Methods**

| Ranking        | Design Consideration Present | Methodology Consideration Present | Both Considerations Present |
|----------------|------------------------------|-----------------------------------|-----------------------------|
| Outstanding    | Appropriate                  | Appropriate                       | Yes, both present           |
| Good           | Appropriate                  | Appropriate                       | No, either present          |
| Adequate       | Adequate with possible bias  | Adequate                          | No, either present          |
| Poor           | Limited or biased            | Limited                           | No, either present          |
| Unsatisfactory | Questionable/none            | Questionable/none                 | No, either present          |

AAEM = American Academy of Emergency Medicine.

**Table 3. Level of Evidence**

| Article Information  | Type                        | Grade | Quality        |
|--|-----------------------------|-------|----------------|
| Aasheim ET, 2008 (4)   | Case series (N84)           | E     | Good           |
| Drenick EJ, Joven CB, Swendseid ME, 1966 (21)                            | Case report                 | E     | Adequate       |
| Gibb WR, Gorsuch AN, Lees AJ, Yudkin JS, 1985 (12)                       | Case report                 | E     | Adequate       |
| Hack JB, Hoffman RS, 1998 (20)   | Expert opinion              | F     | Adequate       |
| Jordan LR, Zelaya FO, Rose SE, et al., 1998 (17)                         | Animal model                | E     | Outstanding    |
| Koguchi K, Nakatsuji Y, Abe K, Sakoda S, 2004 (13)                       | Case report                 | E     | Unsatisfactory |
| Kramer J, Goodwin JA, 1977 (23)  | Case report                 | E     | Adequate       |
| Lindberg MC, Oyler RA, 1990 (24)   | Case report/common practice | E/F   | Poor           |
| Mayes GA, 1989 (14)  | Expert opinion              | F     | Unsatisfactory |
| Miyajima Y, Fukuda M, Kojima S, Matsuyama T, Shylaja N, Aso K, 1993 (25) | Case report                 | E     | Adequate       |
| Nadel AM, Burger PC, 1976 (26)   | Case report (N2)            | E     | Adequate       |
| Navarro D, Zwingmann C, Chatauret N, Butterworth RF, 2008 (16)           | Animal model                | E     | Outstanding    |
| Phillips GB, Victor M, Adams RD, Davidson CS, 1952 (22)                  | Case series* (N9)           | E     | Unsatisfactory |
| Selitsky T, Chandra P, Schiavello HJ, 2006 (27)                          | Case report                 | E     | Poor           |
| Togay-Isikay C, Yigit A, Mutluer N, 2001 (28)                            | Case report                 | E     | Adequate       |
| Wallis WE, Willoughby E, Baker P, 1978 (29)                              | Case series (N4)            | E     | Adequate       |
| Watson AJ, Walker JF, Tomkin GH, Finn MM, Keogh JA, 1981 (9)             | Case series (N4)            | E     | Poor           |
| Zelaya FO, Rose SE, Nixon PF, et al., 1995 (18)                          | Animal model                | E     | Outstanding    |
| Zimitat C, Nixon PF, 1999 (19)   | Animal model                | E     | Outstanding    |

\* This study does not list methods. It is unclear whether patients were followed prospectively or retrospectively, or whether these are case reports.

glucose supplementation (21). The Drenick article discusses the worsening of Wernicke encephalopathy in an obese male undergoing a prolonged starvation diet. Upon development of nausea, he was started on intravenous glucose and later supplementation with orange juice alone. After 12 days of this regimen, he developed what appeared to be Wernicke encephalopathy that reversed with thiamine and reintroduction of oral alimentation (21). The Drenick article cites only one relevant reference, by Phillips et al. from 1952, for its assertion that glucose loading can precipitate Wernicke encephalopathy (21,22).

The Phillips study bears mentioning in detail as it will likely not be repeated due to ethical issues (thiamine was apparently withheld from patients with Wernicke for what appears to be arbitrary periods of time). This study seems to be an observational study, but the design is so problematic that it cannot be used to answer our question about the effects of acute glucose loads on Wernicke encephalopathy. In 1952, Phillips et al. attempted to define the effects of bed rest, alcohol cessation, and vitamin supplements on Wernicke encephalopathy (22). No controls were used and the Methods section makes it unclear as to whether the study was prospective, retrospective, or simply case reports of current therapy at the time. Nine alcoholic patients with presumed Wernicke encephalopathy (based on ophthalmoplegia) were given intravenous saline and glucose (200 g/L) without oral nutrition for varying amounts of time; it seems that this was done to eliminate the confounding effects of oral

nutrition with vitamin ingestion. The rate of infusion is not noted, but patients apparently received 1–2 L a day of the infusion. All patients had worsening of their external rectus paralysis, and 2 patients had worsening confusion. The shortest period of time that deterioration was noted was 2 days. Patients were given non-standardized doses of various vitamins at various days after infusion of saline and glucose. All patients showed improvement in ophthalmoplegia after the administration of thiamine and not other vitamins. Five of 9 patients showed improvement in nystagmus. Of the 7 patients evaluated for ataxia, all 7 showed some improvement in ataxia, and 6 of 9 appeared to have improvement in their confusion (22).

At best, this study could be considered an observational study. Due to the methodological problems, and the fact that a cohort in a cohort study needs to be identified *before* the appearance of the disease under investigation, this study is considered a case series for the purposes of this article. No references relevant to our question are found in this article, and this may very well be the first article to link glucose supplementation and Wernicke encephalopathy—although no claims to that effect are made until Drenick et al.'s article, citing this study, in 1966.

No evidence that we found rose above the level of case reports/series. Only one case appears to show *acute* degradation in mental status after glucose administration, but the timeline and dosage of glucose is not clear in the case report, which reads, almost in its entirety:

"A 61-year-old man was admitted with slight confusion. He had been a heavy drinker for several years and had general fatigue. A doctor... gave him an infusion including electrolytes and glucose. After starting IV injection, the patient fell into a deep coma... Although thiamine was started 5 days after admission, the patient died of pneumonia without recovery after 3 months" (13).

All other cases clearly showed deterioration in mental status after prolonged or massive (>2 L of 5%) glucose infusion, or showed evidence of Wernicke encephalopathy before glucose administration.

A review of the case reports suggests that a study on long-term glucose supplementation without thiamine would be unethical; however, due to lack of evidence of acute benefit or harm, a study evaluating the effects of a glucose load (1 or 2 ampules of dextrose 50%) in hypoglycemic patients before the administration of thiamine may be viable.

### Limitations

In reviewing the case reports, it is difficult to ascertain whether patients' conditions worsened due to the progression of their disease or due to the supposed effects of glucose on the depletion of thiamine stores. Overall, the case reports do not provide sufficient information to appropriately assess the time course of disease progression in a cause and effect manner. So although the evidence does not support rapid deterioration after glucose administration, it likewise does not seem to support the lack of rapid deterioration.

It is possible that a relevant reference was missed in the literature review, but the authors feel their efforts were fairly extensive and made their search in good faith. The rating scales were applied by the authors in discussion and not independently, and thus, rating scales were not measured for inter-rater reliability. The AAEM rating scales are used by the AAEM clinical practice committee and are used routinely to develop the AAEM clinical practice guidelines, which are readily available from their website. These have not been externally validated, but in this literature review, no evidence ultimately rose above the level of case series.

### CONCLUSION

True clinical research about the question of whether or not a glucose load can precipitate acute onset of Wernicke encephalopathy is lacking, and the exact time period for administration of thiamine to prevent worsening or development of Wernicke cannot be determined from the existing literature. Mounting evidence from case reports does seem to show that *prolonged* glucose supplementation without the addition of thiamine can be a risk

factor for the development or worsening of Wernicke encephalopathy. Based on our review of the literature, a delay in giving glucose to hypoglycemic patients cannot be recommended at this time, although prompt thiamine supplementation after or concurrent with a return to normoglycemia is recommended.

### Recommendations

All patients with a change in mental status should have their blood glucose level checked on arrival.

Patients with hypoglycemia should be restored to normoglycemia as quickly as possible (repeated dosing of dextrose 50% in adults until normoglycemia is achieved).

All patients at risk for malnutrition should be given thiamine intravenously or intramuscularly as soon as possible after restoration of normoglycemia; intravenous thiamine is less well proven than intramuscular thiamine, but seems to be safe and effective (6,30,31). One hundred milligrams seems to be an adequate and traditional dose chosen arbitrarily in the 1950s, although up to 500 mg has been advocated by some (6,30,32).

Patients with suspected or confirmed nutritional deficiency should receive daily thiamine supplementation while their nutritional status is improved either orally or with intravenous glucose supplementation. The optimal dosing and duration of treatment is unclear at this time (6,30).

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### ARTICLE SUMMARY

#### **1. Why is this topic important?**

Low blood glucose itself could be the cause of an acute change in mental status in alcoholics. Many physicians would assume that patients with low blood glucose and suspicion for malnutrition should be given thiamine before glucose, but prompt administration of glucose is more important.

#### **2. What does this review attempt to show?**

Glucose does not need to be held until thiamine is given in the emergency department (ED) setting.

#### **3. What are the key findings?**

Patients with hypoglycemia should be restored to normoglycemia as quickly as possible. Thiamine can be replaced on a non-emergent basis to prevent Wernicke encephalopathy and its sequelae.

#### **4. How is patient care impacted?**

There is no need delay glucose administration in a hypoglycemic patient to await thiamine absorption in the ED.