

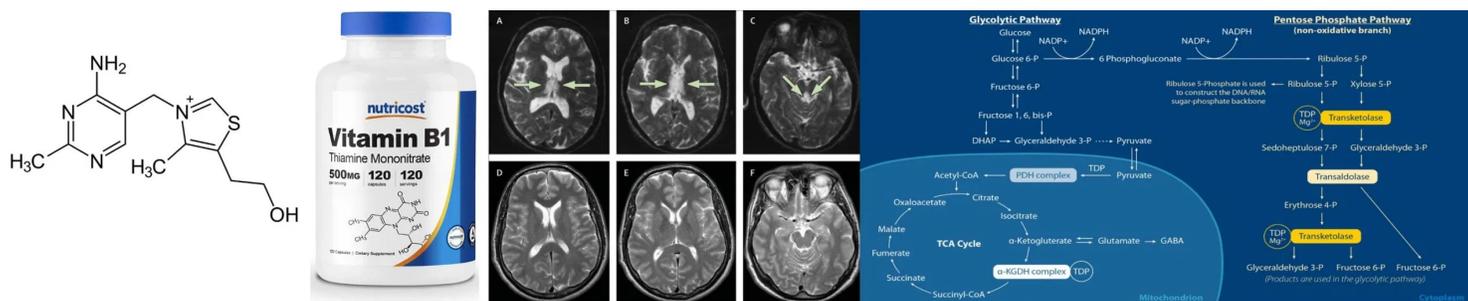
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# The Internet Book of Critical Care

## Wernicke encephalopathy

October 14, 2020 by [Josh Farkas](#)



(<https://emcrit.org/ibcc/wernicke/attachment/wernicketop/>)

## CONTENTS

- [Pathophysiology \(#pathophysiology\)](#).
- [Signs & Symptoms \(#signs\\_&\\_symptoms\)](#).
- [Risk factors \(#risk\\_factors\)](#).
- [Epidemiology \(#epidemiology\)](#).
- [Laboratory studies \(#laboratory\\_studies\)](#).
- [Imaging \(#imaging\)](#).
- [Differential diagnosis & securing the diagnosis \(#differential\\_diagnosis\\_&\\_securing\\_the\\_diagnosis\)](#).
- [Prevention \(#prevention\)](#).
- [Treatment \(#treatment\)](#).
- [Prognosis \(#prognosis\)](#).
- [Summary \(#summary\)](#).
- [Podcast \(#podcast\)](#).
- [Questions & discussion \(#questions\\_&\\_discussion\)](#).
- [Pitfalls \(#pitfalls\)](#).
- [PDF of this chapter \(https://emcrit.org/wp-content/uploads/2017/01/hyperthermia.pdf\)](#). (or [create customized PDF \(https://emcrit.org/ibcc/about-guide/#pdf\)](#).)

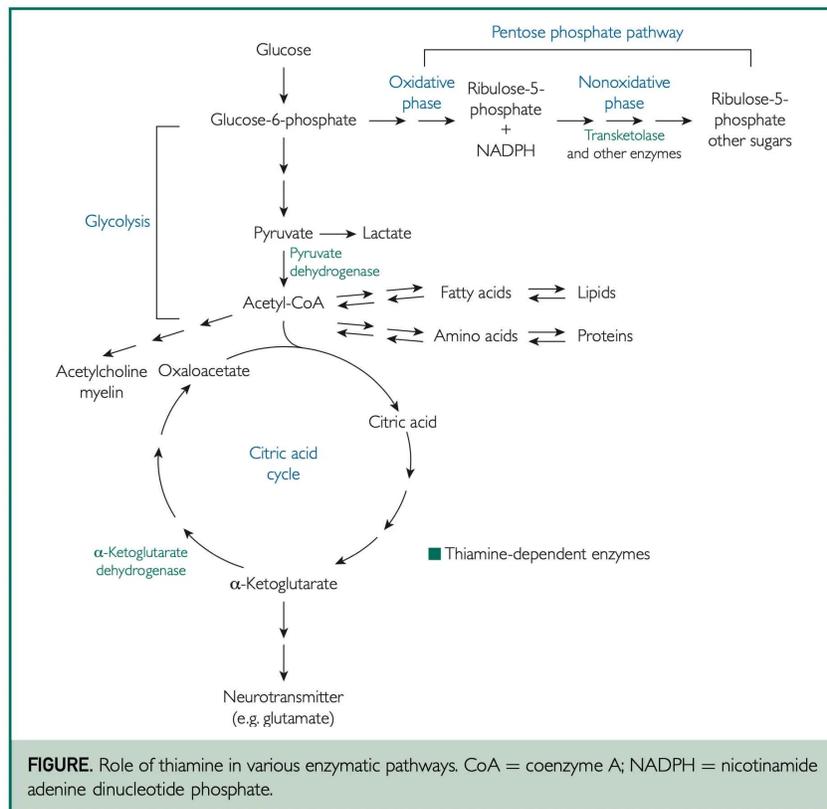
## pathophysiology

([back to contents](#)) (#top)

### biochemistry

- Thiamine is a co-factor in numerous enzymatic reactions involved in energy metabolism, as shown below. Thiamine deficiency has consequences including the following:
  - (1) Accumulation of lactate
  - (2) Excess production of glutamate, which may lead to excessive excitatory neurotransmission in the brain

- This can cause vasogenic and cytotoxic edema.

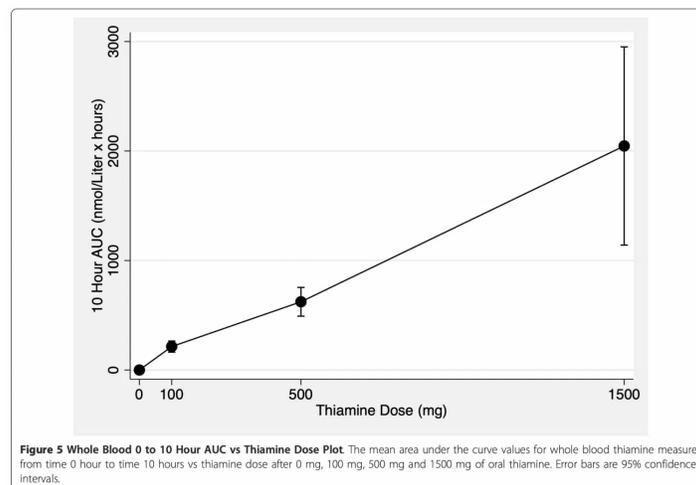


Sinha S et al. PMID 31171116

(<https://emcrit.org/ibcc/wernicke/attachment/wernickepath/>.)

## pharmacology of thiamine

- The daily requirement of thiamine is ~1-2 mg. Thiamine is absorbed in the jejunum and stored in the liver. Absorption is relatively *inefficient*, for example, one study of healthy volunteers found that only 5% of a 50-mg oral dose was absorbed. (30146080 (<https://pubmed.ncbi.nlm.nih.gov/30146080/>)). However, a recent study suggests that this might be overcome with the use of very high doses of oral thiamine, as shown in the figure below. (22305197 (<https://pubmed.ncbi.nlm.nih.gov/22305197/>)).
- Total body stores of thiamine are ~30 mg (but potentially lower in liver disease).
- Inadequate thiamine intake can cause a previously *healthy* person to become deficient within ~2-4 weeks. (32551830 (<https://pubmed.ncbi.nlm.nih.gov/32551830/>)). In the context of acute or chronic illness, deficiency can occur more rapidly.



There appears to be a linear relationship between oral thiamine dose and absorption, which extends out to high doses. This suggests that oral "mega-doses" of thiamine could be used in lieu of intravenous thiamine, in situations of IV thiamine deficiency (similar to high-dose oral B12 supplementation). Oral doses up to ~8,000 mg daily seem to be well tolerated.

-Smithline HA et al. 2012 PMID 22305197

- Legs are affected more than arms (because the ataxia is due to vestibular dysfunction *combined* with cerebellar dysfunction).
- This affects gait primarily (arms and speech are usually unaffected).

### other potential clues to Wernicke encephalopathy

- Hypothermia (due to hypothalamic involvement).
- Hypotension.

### other clinical features of thiamine deficiency

- Peripheral neuropathy ("dry beriberi")
  - May cause *pain*, paresthesias, weakness, and/or sensory loss.
- High-output heart failure ("wet beriberi")
- Abdominal pain, emesis, and lactic acidosis ("gastrointestinal beriberi")

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## risk factors

[\(back to contents\) \(#top\)](#)

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 *Alcoholism is the cause of only ~50% of Wernicke encephalopathy. Other cases may occur due to a variety of factors, frequently in a multifactorial fashion. Factors predisposing to Wernicke encephalopathy may be divided into roughly four groups:*

### #1/4: decreased thiamine intake

- Malnutrition, for example due to:
  - Substance use disorder, alcoholism
  - Dementia, poverty
  - Schizophrenia
  - Anorexia nervosa
  - Cachexia (e.g., associated with AIDS, malignancy)
  - Fad diets
- Hyperemesis
  - Hyperemesis gravidarum
  - Cannabinoid hyperemesis syndrome
- Total parenteral nutrition lacking thiamine

### #2/4: decreased gastrointestinal absorption

- Crohn's disease
- Peptic ulceration, chronic gastritis
- Chronic diarrhea
- Gastrointestinal surgery (especially bariatric surgery)

### #3/4: decreased hepatic storage capacity for thiamine

- Cirrhosis of any etiology
- Alcoholism

### #4/4: increased use or removal of thiamine

- Large dietary carbohydrate load (especially refeeding syndrome)
- Hypermetabolic states (e.g., sepsis, systemic inflammation, hyperthyroidism)
- Certain rapidly-growing malignancies
- Hemodialysis or peritoneal dialysis
- Diuresis (especially heart failure patients on chronic diuretics)
- Metronidazole can function as a thiamine antagonist. ([32390125](https://pubmed.ncbi.nlm.nih.gov/32390125/) (<https://pubmed.ncbi.nlm.nih.gov/32390125/>))

## epidemiology

[\(back to contents\) \(#top\)](#)

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### Wernicke encephalopathy may be more common than generally perceived

- ~1% prevalence in the general population.
- ~12.5% prevalence among people with alcoholism.
- ~8% prevalence among patients status post bariatric surgery. [\(30364782 \(https://pubmed.ncbi.nlm.nih.gov/30364782/\)\)](https://pubmed.ncbi.nlm.nih.gov/30364782/)

### thiamine deficiency as an iatrogenic complication of hospitalization

- Causative factors:
  - Pre-existing inadequacy of thiamine stores.
  - Inadequate nutrition (e.g., prolonged periods of time with NPO).
  - Hypermetabolic states (e.g., infection, inflammation).
  - Increased urinary excretion (e.g., diuresis or dialysis).
- The prevalence of thiamine deficiency among critically ill patients may be high, with some articles reporting ~20%. [\(30146080 \(https://pubmed.ncbi.nlm.nih.gov/30146080/\)\)](https://pubmed.ncbi.nlm.nih.gov/30146080/)
- Wernicke encephalopathy may manifest initially *within* the hospital, following admission for another illness. This will be extremely difficult to diagnose, as it will tend to be attributed to multi-factorial delirium (previously termed "ICU psychosis").
- Thiamine deficiency might also contribute to critical care neuropathy or gastrointestinal dysfunction in some patients. [\(30146080 \(https://pubmed.ncbi.nlm.nih.gov/30146080/\)\)](https://pubmed.ncbi.nlm.nih.gov/30146080/) One RCT investigating the utility of thiamine in septic shock found no benefit overall, but a reduced mortality among the subgroup of patients with thiamine deficiency. [\(26771781 \(https://pubmed.ncbi.nlm.nih.gov/26771781/\)\)](https://pubmed.ncbi.nlm.nih.gov/26771781/)

[\(https://emcrit.org/ibcc/wernicke/attachment/wernickecase/\)](https://emcrit.org/ibcc/wernicke/attachment/wernickecase/)

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## laboratory studies

[\(back to contents\) \(#top\)](#)

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### whole blood thiamine diphosphate level

- Whole blood thiamine diphosphate level is largely a reflection of thiamine levels within erythrocytes. This may be more accurate than *serum* or *plasma* thiamine measurement, since whole blood thiamine is a more accurate reflection of *intracellular* thiamine stores.
- The normal value is generally taken to be 70-180 nM/L, although there is no universally defined cutoff. [\(30151974 \(https://pubmed.ncbi.nlm.nih.gov/30151974/\)\)](https://pubmed.ncbi.nlm.nih.gov/30151974/)
- Whole blood thiamine diphosphate levels seem to be superior to other tests of thiamine levels. [\(25564426 \(https://pubmed.ncbi.nlm.nih.gov/25564426/\)\)](https://pubmed.ncbi.nlm.nih.gov/25564426/) However, its sensitivity and specificity remain unclear.

## imaging

[\(back to contents\) \(#top\)](#)

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### CT scan

- CT scan usually won't reveal Wernicke encephalopathy. The primary role is to *exclude* alternative etiologies.

([https://emcrit.org/?attachment\\_id=478706](https://emcrit.org/?attachment_id=478706))

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## signs & symptoms

([back to contents](#)) (#top)

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*Signs and symptoms are generally unreliable in the diagnosis. The classic triad of encephalopathy, ophthalmoplegia, and ataxia is present in only ~15% of cases. Among non-alcoholic patients with Wernicke encephalopathy, the triad is even less common.*([31171116](#)

(<https://pubmed.ncbi.nlm.nih.gov/31171116/>.)

### #1 = encephalopathy

- Delirium is the most consistent clinical characteristic.
- This may progress to coma.
- Isolated encephalopathy without ocular or cerebellar signs may be more common in nonalcoholic Wernicke encephalopathy.([32390125](#)

(<https://pubmed.ncbi.nlm.nih.gov/32390125/>.)

#### Eye motor abnormalities in Wernicke encephalopathy, case 2



### #2 = ophthalmoplegias

- **Horizontal nystagmus** is the most common finding (although both horizontal and vertical nystagmus may occur).
- Cranial nerve palsies may include:
  - Abducens nerve (CN6) – usually bilateral.
  - Conjugate gaze palsies.
  - These may progress to complete ophthalmoplegia.

#### Eye motor abnormalities in Wernicke encephalopathy, case 1



### #3 = ataxia

- CT could show reduced attenuation at the periaqueductal gray matter and medial portion of the thalami. (32390125 (<https://pubmed.ncbi.nlm.nih.gov/32390125/>)) There may also be contrast-enhancement.

(<https://emcrit.org/ibcc/wernicke/attachment/fujikawa/>)

### **MRI scan**

- MRI has a sensitivity of ~50% with a specificity of ~90%. (32390125 (<https://pubmed.ncbi.nlm.nih.gov/32390125/>)) However, MRI may normalize within days of thiamine initiation.
- Distribution of abnormalities:
  - Symmetric
  - Typical location: Paraventricular regions of the thalamus, hypothalamus, mammillary bodies, periaqueductal region, and floor of the fourth ventricle. (32390125 (<https://pubmed.ncbi.nlm.nih.gov/32390125/>))
  - Additional abnormalities can be seen in the putamen, caudate, splenium of the corpus callosum, dorsal medulla, pons, red nucleus, substantia nigra of the midbrain, cranial nerve nucleus, vermis, dentate nucleus, paravermian region of the cerebellum, fornix, and the precentral and postcentral gyri. (32390125 (<https://pubmed.ncbi.nlm.nih.gov/32390125/>))
- Findings:
  - Increased T2 signal and fluid-attenuated inversion recovery signals (T2/FLARE).
  - Decreased T1 signal.
  - Diffusion abnormality.
  - Contrast enhancement may result from blood-brain barrier disruption.
- Note: abnormalities in the corpus callosum should raise concern for Marchiafava-Bignami disease (due to alcoholism plus vitamin deficiencies).

(<https://emcrit.org/ibcc/wernicke/attachment/otawernicke/>)

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## differential diagnosis & securing the diagnosis

([back to contents](#)) (#top)

The differential diagnosis will depend on how the patient presents, so this may vary quite a bit. However, some common considerations may include:

- Alcohol withdrawal
- [Refeeding syndrome](https://emcrit.org/ibcc/refeeding/) (which may involve Wernicke encephalopathy *plus* superimposed electrolyte abnormalities)
- Hepatic encephalopathy
- Normal pressure hydrocephalus
- Posterior circulation ischemic stroke
- Septic shock (thiamine deficiency causing Wernicke encephalopathy plus high-output heart failure and lactic acidosis)

### securing the diagnosis of Wernicke encephalopathy

- No diagnostic criteria exist which are both sensitive and specific. Therefore, clinical judgement is required.
- Empiric thiamine supplementation should be started *while* the diagnostic process is underway (prior to definitive diagnosis).
- Consideration should be given to the following components:
  - (1) Risk factors for thiamine deficiency.
  - (2) Exclusion of alternative diagnostic possibilities.
  - (3) Neuroimaging (especially MRI) may sometimes be strongly supportive.
  - (4) Clinical response to IV thiamine (in some cases, ophthalmoplegia can resolve within hours of starting therapy).
  - (5) Whole blood thiamine levels (this may be useful to adjudicate the diagnosis retrospectively).
- Attempts should be made to reach diagnostic certainty, since Wernicke's Encephalopathy requires a *protracted* course of IV thiamine.

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## prevention

([back to contents](#)) (#top)

### conventional approaches to preventing Wernicke encephalopathy

- Thiamine supplementation is a sensible intervention for at-risk patients (e.g., patients with alcoholism or malnutrition). More on the risk factors for thiamine deficiency [above](#) (#risk\_factors).
- 100 mg IV/IM thiamine daily is often used for this purpose (in the absence of any high-level evidence).
- Oral thiamine is probably adequate for this purpose as well (with advantages regarding reduced cost and ease of administration). One small study found that in a population of patients with alcoholism, 250 mg thiamine IM daily generated similar thiamine levels after three days when compared to a regimen of 50 mg PO five times daily. (3358822 (<https://pubmed.ncbi.nlm.nih.gov/3358822/>)). This suggests that an oral regimen of ~200 mg twice daily might be adequate to prevent deficiency in at-risk patients.
  - In clinical practice, at-risk patients are often treated with IV thiamine for a few days and then thiamine supplementation is stopped entirely. Rather than discontinuing thiamine supplementation altogether, it might be more sensible to transition from intravenous thiamine supplementation to high-dose oral supplementation, which could be continued for the duration of the patient's hospital course.
  - However, oral thiamine may *not* be a viable strategy for patients with impaired gastrointestinal absorption of thiamine.

([https://emcrit.org/?attachment\\_id=478707](https://emcrit.org/?attachment_id=478707))

### could more aggressive prevention be useful among critically ill patients?

- Nearly all critically ill patients will have at least one risk factor for thiamine deficiency (e.g., reduced nutritional intake, diuresis, systemic inflammation, dialysis). Consequently, thiamine deficiency is very common among critically ill patients (as explored [above \(#epidemiology\)](#)).
- Oral thiamine administration is probably effective, safe, and cheap (costing roughly ~\$10 per patient for an entire ICU stay).
- ICU delirium is very expensive, with estimates that a single episode of ICU delirium may cost ~\$17,000. ([30179988](https://pubmed.ncbi.nlm.nih.gov/30179988/) (<https://pubmed.ncbi.nlm.nih.gov/30179988/>))
- This suggests that if the incidence of Wernicke encephalopathy is higher than one in ~1,700 patients (0.05%), then routine thiamine administration could be both clinically beneficial *and* cost-saving. Available data shows that the incidence of Wernicke encephalopathy is well above 0.05%, which could make prophylaxis a logical intervention. ([24666443](https://pubmed.ncbi.nlm.nih.gov/24666443/) (<https://pubmed.ncbi.nlm.nih.gov/24666443/>))

(<https://emcrit.org/ibcc/wernicke/attachment/thiamine-ppx/>)

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## treatment

[\(back to contents\) \(#top\)](#)

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### thiamine

- When in doubt about the diagnosis, empiric treatment should be initiated.
  - IV thiamine is entirely safe.
  - Failure to provide therapy may promulgate chronic neurologic injury.
  - Thiamine should ideally be given simultaneously or before administration of carbohydrates (e.g., food or IV dextrose).
- Parenteral thiamine is generally required, since oral absorption is inefficient. ([28680171](https://pubmed.ncbi.nlm.nih.gov/28680171/) (<https://pubmed.ncbi.nlm.nih.gov/28680171/>)) If intravenous thiamine is *unavailable*, then using very high-dose oral thiamine may be attempted to overcome low bioavailability (e.g., perhaps 500-1,500 mg PO q8hr). ([22305197](https://pubmed.ncbi.nlm.nih.gov/22305197/) (<https://pubmed.ncbi.nlm.nih.gov/22305197/>))
- There is no well-defined dosing regimen, nor high-quality evidence. Articles often recommend the following: ([32551830](https://pubmed.ncbi.nlm.nih.gov/32551830/) (<https://pubmed.ncbi.nlm.nih.gov/32551830/>))

- i) 500 mg IV TID for 2-3 days, followed by
- ii) 200-500 mg IV daily for ~5 days or until clinical improvement ceases, followed by
- iii) oral thiamine (e.g., 100-500 mg PO TID)
- Ophthalmoparesis may improve within hours of receiving IV thiamine.

## magnesium

- Rationale for ensuring adequate magnesium levels:
  - Magnesium is a cofactor for thiamine activity and promotes cellular uptake of thiamine.
  - Disorders which lead to thiamine deficiency often also cause magnesium deficiency (especially alcoholism).
- Check and aggressively replete magnesium (more on that [here \(https://emcrit.org/ibcc/hypomagnesemia/\)](https://emcrit.org/ibcc/hypomagnesemia/)).

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## prognosis

[\(back to contents\) \(#top\)](#)

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- Ocular abnormalities usually begin to improve within about one day.
  - Ataxia and confusion usually begin to improve within a week. Confusion will generally resolve over a month.
  - Chronic Korsakoff's psychosis (marked by confabulation) may occur in patients with delayed treatment.

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## summary

[\(back to contents\) \(#top\)](#)

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[\(https://emcrit.org/ibcc/wernicke/attachment/wernickebox/\)](https://emcrit.org/ibcc/wernicke/attachment/wernickebox/)

## podcast

[\(back to contents\) \(#top\)](#)

<https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/apps.40518.14127333176902609.7be7b901-15fe-4c27-863c-7c0dbfc26c5c.5c278f58-912b-4af9-88f8-a65fff2da477.jpg>

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## questions & discussion

[\(back to contents\) \(#top\)](#)

To keep this page small and fast, questions & discussion about this post can be found on another page [here](https://emcrit.org/pulmcrit/wernicke/).

<https://i0.wp.com/emcrit.org/wp-content/uploads/2016/11/pitfalls2.gif>

- Failure to consider Wernicke encephalopathy in patients with atypical risk factors (e.g., ICU patients, hyperemesis gravidarum, chronic diuretic use for heart failure).
- Inadequate dose and duration of thiamine used for patients with Wernicke encephalopathy. One or two days of IV thiamine isn't sufficient!
- Incorrectly excluding the diagnosis of Wernicke encephalopathy because patients lack the classic triad of symptoms (confusion, ataxia, ophthalmoplegia). In fact, very *rarely* will patients with Wernicke encephalopathy display all of these components.
- Inadequate use of thiamine prophylaxis against Wernicke encephalopathy among at-risk critically ill patients. Oral thiamine is probably adequate for this purpose, making the expense of prophylaxis negligible.

### Going further:

- [Don't Wernicke's, B\(1\) Happy](https://emcrit.org/toxhound/wernickes/) (Tox & Hound, by Meghan Spyres)
- [Wernicke Encephalopathy](https://rebelem.com/wernicke-encephalopathy/) (RebeLEM, by Anand Swaminathan) and also a [podcast](https://rebelem.com/rebel-core-cast-40-0-wernicke-encephalopathy/)
- [Wernicke encephalopathy](https://radiopaedia.org/articles/wernicke-encephalopathy/) (Radiopaedia, by Craig Hacking and Frank Gaillard)

5-minute summary by Anna Pickens (EM in 5)



### references

- **03358822** Baines M, Bligh JG, Madden JS. Tissue thiamin levels of hospitalised alcoholics before and after oral or parenteral vitamins. *Alcohol Alcohol*. 1988;23(1):49-52 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/3358822/\)](https://pubmed.ncbi.nlm.nih.gov/3358822/)]
- **22305197** Smithline HA, Donnino M, Greenblatt DJ. Pharmacokinetics of high-dose oral thiamine hydrochloride in healthy subjects. *BMC Clin Pharmacol*. 2012 Feb 4;12:4. doi: 10.1186/1472-6904-12-4 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/22305197/\)](https://pubmed.ncbi.nlm.nih.gov/22305197/)]
- **24666443** Blackmore C, Ouellet JF, Niven D, Kirkpatrick AW, Ball CG. Prevention of delirium in trauma patients: are we giving thiamine prophylaxis a fair chance? *Can J Surg*. 2014 Apr;57(2):78-81. doi: 10.1503/cjs.020112 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/24666443/\)](https://pubmed.ncbi.nlm.nih.gov/24666443/)]
- **25564426** Frank LL. Thiamin in Clinical Practice. *JPEN J Parenter Enteral Nutr*. 2015 Jul;39(5):503-20. doi: 10.1177/0148607114565245 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/25564426/\)](https://pubmed.ncbi.nlm.nih.gov/25564426/)]
- **25985980** Welsh A, Rogers P, Clift F. Nonalcoholic Wernicke's encephalopathy. *CJEM*. 2016 Jul;18(4):309-12. doi: 10.1017/cem.2015.27 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/25985980/\)](https://pubmed.ncbi.nlm.nih.gov/25985980/)]
- **26771781** Donnino MW, Andersen LW, Chase M, Berg KM, Tidswell M, Giberson T, Wolfe R, Moskowitz A, Smithline H, Ngo L, Cocchi MN; Center for Resuscitation Science Research Group. Randomized, Double-Blind, Placebo-Controlled Trial of Thiamine as a Metabolic Resuscitator in Septic Shock: A Pilot Study. *Crit Care Med*. 2016 Feb;44(2):360-7. doi: 10.1097/CCM.0000000000001572 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/26771781/\)](https://pubmed.ncbi.nlm.nih.gov/26771781/)]
- **26869612** Infante MT, Fancelli R, Murialdo A, Barletta L, Castellan L, Serrati C. Challenges in Diagnosis and Treatment of Wernicke Encephalopathy: Report of 2 Cases. *Nutr Clin Pract*. 2016 Apr;31(2):186-90. doi: 10.1177/0884533615621753 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/26869612/\)](https://pubmed.ncbi.nlm.nih.gov/26869612/)]
- **28680171** Alim U, Bates D, Langevin A, Werry D, Dersch-Mills D, Herman RJ, Mintz M, Ghosh S. Thiamine Prescribing Practices for Adult Patients Admitted to an Internal Medicine Service. *Can J Hosp Pharm*. 2017 May-Jun;70(3):179-187. doi: 10.4212/cjhp.v70i3.1657 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/28680171/\)](https://pubmed.ncbi.nlm.nih.gov/28680171/)]
- **30146080** Attaluri P, Castillo A, Edriss H, Nugent K. Thiamine Deficiency: An Important Consideration in Critically Ill Patients. *Am J Med Sci*. 2018 Oct;356(4):382-390. doi: 10.1016/j.amjms.2018.06.015 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/30146080/\)](https://pubmed.ncbi.nlm.nih.gov/30146080/)]
- **30151974** Whitfield KC, Bourassa MW, Adamolekun B, Bergeron G, Bettendorff L, Brown KH, Cox L, Fattal-Valevski A, Fischer PR, Frank EL, Hiffler L, Hlaing LM, Jefferds ME, Kapner H, Kounnavong S, Mousavi MPS, Roth DE, Tsaloglou MN, Wieringa F, Combs GF Jr. Thiamine deficiency disorders: diagnosis, prevalence, and a roadmap for global control programs. *Ann N Y Acad Sci*. 2018 Oct;1430(1):3-43. doi: 10.1111/nyas.13919 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/30151974/\)](https://pubmed.ncbi.nlm.nih.gov/30151974/)]
- **30179988** Vasilevskis EE, Chandrasekhar R, Holtze CH, Graves J, Speroff T, Girard TD, Patel MB, Hughes CG, Cao A, Pandharipande PP, Ely EW. The Cost of ICU Delirium and Coma in the Intensive Care Unit Patient. *Med Care*. 2018 Oct;56(10):890-897. doi: 10.1097/MLR.0000000000000975 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/30179988/\)](https://pubmed.ncbi.nlm.nih.gov/30179988/)]
- **30364782** Patel S, Topiwala K, Hudson L. Wernicke's Encephalopathy. *Cureus*. 2018 Aug 22;10(8):e3187. doi: 10.7759/cureus.3187 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/30364782/\)](https://pubmed.ncbi.nlm.nih.gov/30364782/)]
- **31171116** Sinha S, Kataria A, Kolla BP, Thusius N, Loukianova LL. Wernicke Encephalopathy-Clinical Pearls. *Mayo Clin Proc*. 2019 Jun;94(6):1065-1072. doi: 10.1016/j.mayocp.2019.02.018 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/31171116/\)](https://pubmed.ncbi.nlm.nih.gov/31171116/)]
- **32041699** Fujikawa T, Sogabe Y. Wernicke encephalopathy. *CMAJ*. 2020 Feb 10;192(6):E143. doi: 10.1503/cmaj.190998 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/32041699/\)](https://pubmed.ncbi.nlm.nih.gov/32041699/)]
- **32551830** Kohnke S, Meek CL. Don't seek, don't find: The diagnostic challenge of Wernicke's encephalopathy. *Ann Clin Biochem*. 2020 Jul 13:4563220939604. doi: 10.1177/0004563220939604 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/32551830/\)](https://pubmed.ncbi.nlm.nih.gov/32551830/)]
- **32390125** Ota Y, Capizzano AA, Moritani T, Naganawa S, Kurokawa R, Srinivasan A. Comprehensive review of Wernicke encephalopathy: pathophysiology, clinical symptoms and imaging findings. *Jpn J Radiol*. 2020 Sep;38(9):809-820. doi: 10.1007/s11604-020-00989-3 [[PubMed \(https://pubmed.ncbi.nlm.nih.gov/32390125/\)](https://pubmed.ncbi.nlm.nih.gov/32390125/)]

The Internet Book of Critical Care is an online textbook written by Josh Farkas (@PulmCrit), an associate professor of Pulmonary and Critical Care Medicine at the University of Vermont.