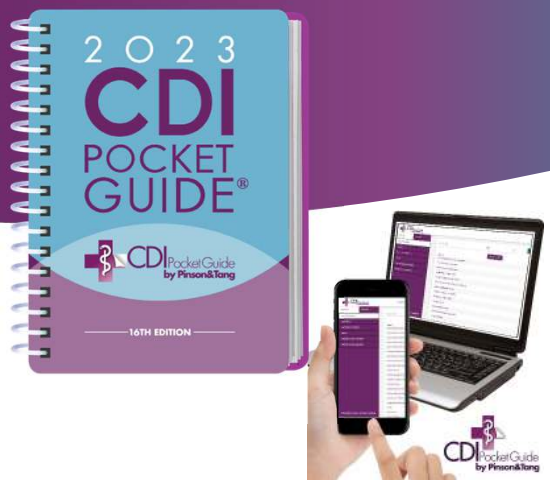
July 2023



CDI Pocket Guide®

Encephalopathy

Pinson&Tang

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About Us

Richard Pinson

MD, FACP, CCS, CDIP

Dr. Richard Pinson is a physician, educator, administrator, and healthcare consultant. He practiced Internal Medicine and Emergency Medicine in Tennessee for over 20 years having board certification in both.

Cynthia Tang

RHIA, CCS, CRC

Cynthia brings over 35 years of experience in coding and clinical documentation integrity, and health information management. For over 30 years she has traveled across the country implementing successful and sustainable coding and CDI programs in hundreds of hospitals.

CDI Pocket Guide by Pinson&Tang

We created the **CDI Pocket Guide®** in 2008 because we wanted to provide this information to all hospitals, large or small. At the time, the only way to receive training in this field was with large-scale, expensive consulting projects. We thought we could bring this pocketful of information with the clinical criteria to identify important diagnoses to any individual who was interested in working in the CDI and coding field. Our CDI Pocket Guide® quickly became a best-selling book and an industry standard, and many consider it to be their CDI "bible".

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Encephalopathy

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Agenda

2023 CDI Pocket Guide
Pages 136-141

- ICD-10 Classification
- Definition and Characteristics of Encephalopathy
- Acute vs Chronic Encephalopathy
- Encephalopathy with Dementia, Alcohol, Delirium, Hepatic
- Case Studies

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ICD-10 Classification

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Encephalopathy

MCC (G92):
- Toxic (G92.9)
- Toxic-metabolic (G92.8)
- Drug-induced (G92.8)

MCC (G93.41):
- Metabolic
- Septic
- DM hypoglycemic

CC:
- Unspecified (G93.40)
- Other NEC (G93.49)

Other Specified Types

Non-CC
- Alcoholic (G31.2)
- Arteriosclerotic (I67.2)
- Congenital (Q07.9)
- Degenerative in specified disease NEC (G32.89)
- In diseases classified elsewhere (G94)
- Hepatic (K76.82)
- Influenzal (J11.81)
- Korsakoff's (F10.96)
- Lead (T56.0-)
- Non-DM hypoglycemic (E16.2)
- Trauma (F07.81)
- Vit B deficiency (G32.89)

CC:
- Hypertensive (I67.4)
- Anoxic, hypoxic (G93.1)
- Wernicke's (E51.2)
- Chronic static (G93.49)

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Encephalopathy as Principal Diagnosis

Two Circumstances

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Primary reason for admission

Patients admitted with encephalopathy due to UTI (or electrolyte imbalance, etc.) are usually admitted primarily for the AMS/ encephalopathy.

Most patients are not admitted to the hospital for a UTI, which is typically treated as an outpatient. Antibiotics are administered IV for the UTI because they're hospitalized and would likely be treated with PO as an outpatient.

Encephalopathy is not a symptom code or included in the E/M rule.

Adverse drug effect

When toxic encephalopathy is due to an adverse effect of a drug, G92 is sequenced first followed by the adverse effect code (T36-T50).

DRGs 70-72 Nonspecific cerebrovascular disorders
DRGs 91-93 Other disorders of nervous system

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Encephalopathy

Definition

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National Institute of Neurologic Disorders and Stroke (NINDS):

"*Any **diffuse** disease of the brain that alters brain function or structure*."

Can be further classified as:

- Acute (functional) or
- Chronic (structural)

diffuse:
generalized

functional:
affected brain function temporarily

structural:
affected brain structure usually permanently

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Acute vs. Chronic Encephalopathy

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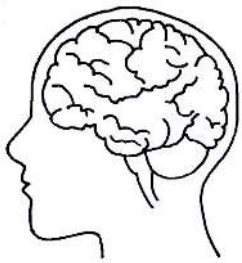
Acute	Chronic
Acute or subacute diffuse (generalized) alteration in mental status	Chronic diffuse (generalized) or focal alteration in mental status
Functional	Structural
Reversible	Irreversible
Resolves – when underlying cause is corrected	Permanent
Metabolic disorders like dehydration, infection, effects of drugs and toxins, hypertension, liver failure, hypoxemia	*Traumatic brain injury, anoxic, cumulative exposure to toxins/ solvents (chronic lead poisoning), Korsakoff (alcohol), Spongiform (viral)*



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Components of Mental Status

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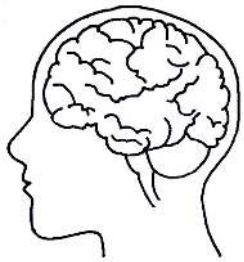
- Alertness
- Orientation
- Attention
- Behavior
- Judgement
- Memory
- Perception of Reality
- Thought content



Acute encephalopathy: all/most are affected

Isolated changes in some components but not others is <u>not</u> acute encephalopathy.

Chronic encephalopathy can be focal (some) or diffuse (all).

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Acute Encephalopathy Causes

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Metabolic

- Fever
- Any infection
- Dehydration or electrolyte imbalance
- Hypoxemia (e.g., respiratory failure)
- DM hypoglycemia/ hyperglycemia
- Organ dysfunction (liver, kidney, etc.)

Toxic

- Drugs
- Toxins (non-drugs)
- Acute alcohol intoxication

Other

- Hypertension

Toxic-Metabolic: combination of toxic and metabolic factors

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Examples

Acute vs. Chronic Encephalopathy

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72-year-old female with PMHx of Type 2 DM and hypertension admitted with aspiration pneumonia, fever, confusion, disorientation, agitation. Blood sugar 320, WBC 16,000, sodium 128.	60-year-old male with history of seizure disorder taking Dilantin admitted with nystagmus, ataxia, slurred speech, progressive alteration in mental status and lethargy. Dilantin level 45 mg/L (Therapeutic range 10-20).	45-year-old female with 25 years of chronic alcohol dependence is admitted for a fractured hip after a fall. She has no recollection of what happened. She is noted to have poor short-term memory, good long-term memory, apathetic affect, and confabulation.	22-year-old male with history of Fentanyl OD two years ago resulting in prolonged respiratory arrest and 60-day hospitalization. Admitted for RLE cellulitis. Neuro exam showed poor long- and short-term memory, labile mood, disorientation and minimal verbal response.
Clinically indicates: Acute metabolic encephalopathy due to infection, hyperglycemia, low sodium	Clinically indicates: Acute toxic encephalopathy due to dilantin	Clinically indicates: Chronic encephalopathy characteristic of Korsakoff syndrome	Clinically indicates: Chronic encephalopathy—Anoxic brain damage (G93.1)

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Dementia vs. Encephalopathy

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Dementia

Dementia: Significant loss of intellectual abilities, such as memory or decision-making, that is severe enough to interfere with activities of daily living.

Dementia without encephalopathy:

1. Acute mental status change ambiguous or unverified
2. Admission mental status does not improve during hospitalization

Dementia with Encephalopathy

Patients with dementia are vulnerable to acute encephalopathic changes.

When dementia is complicated by encephalopathy:

1. Acute mental status change is substantiated
2. Is associated with demonstrable metabolic or toxic disorders
3. Mental status returns to baseline when causative factors corrected

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Examples

Dementia vs. Encephalopathy

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78-year-old female with dementia admitted with fever 101.5, UTI, and dehydration. Family complains of altered mental status.

Nursing notes indicate confused but cooperative, oriented only to self, poor memory, able to perform ADLs, agitated at night requiring sedation. At discharge, confusion and agitation had resolved.

Clinically indicates:
Dementia with sundowning
Encephalopathy unsupported

95-year-old admitted with NSTEMI, UTI and acutely altered mental status compared with her baseline state of dementia. BP 110/70, SpO2 93%, Temp 100.6, BUN 24, creatinine 1.8.

Her mental status was evaluated daily and returned to her usual baseline with IV fluids, antibiotics and two days of supplemental oxygen.

Progress notes and DS: NSTEMI, encephalopathy resolving with conservative management and treatment of underlying UTI.

Clinically indicates:
Acute metabolic encephalopathy due to UTI, possible AKI, low grade fever

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Examples

Dementia vs. Encephalopathy

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Patient arrives at hospital from home via EMS. Reported last seen normal late last night. She apparently is normally alert, oriented, GCS of 15. Family found patient altered with decreased responsiveness. Patient is bedbound and has a caretaker. Patient also has a history of dementia.

Exam reveals patient responds to name, follows commands intermittently, and lethargic. Reported patient is usually more alert than is currently. Diagnosed with multiple embolic strokes and dehydration. "Metabolic encephalopathy" is noted. CT negative.

Patient refusing to eat or drink. Family does not want to pursue further tests or medical treatment and desires patient to go home with hospice.

GCS 14 on arrival and remains 14 upon discharge. Discharged in slightly less than 48 hours. Started to receive IVF's but appears to have been stopped according to MAR. Serum creatinine 1.3 down to 0.9 and Na 146 to 141.

Metabolic encephalopathy is not supported. Did not have diffuse/generalized AMS and did not return to baseline when systemic factors corrected.

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Encephalopathy due to Alcohol

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Acute alcohol intoxication

Acute **toxic encephalopathy due to alcohol intoxication** is coded as T51.0X1A, toxic effect of ethanol, with G92, toxic encephalopathy.

Alcohol withdrawal

Withdrawal delirium (delirium tremens) is not a toxic encephalopathy since the toxin has been withdrawn.

Correct code for this situation is F10.231, alcohol dependence with withdrawal delirium.

Alcoholic encephalopathy

Alcoholic encephalopathy is coded to G31.2, degeneration of nervous system due to alcohol.

Wernicke's: acute encephalopathy, oculomotor dysfunction, and ataxia caused by thiamine deficiency in alcoholics.

Korsakoff syndrome: late manifestation of Wernicke's.

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Examples

Toxic Encephalopathy due to Alcohol?

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19-year-old female college student drank one liter of Vodka in 30 minutes on a dare.

Admitted with obtundation, combativeness, incoherent speech. Blood alcohol 485 mg/dL.

Clinically indicates:
Acute alcohol intoxication with toxic encephalopathy due to alcohol

41-year-old male admitted with compound fracture of the humerus from a fall down the stairs. Patient was drinking heavily at a bachelor's party.

Alert, uncooperative, oriented to person and place, slurred speech, ataxic gait.

CT of brain unremarkable. Blood alcohol 350 mg/dL.

Clinically indicates:
Acute alcohol intoxication without encephalopathy (drunk)

48-year-old with 25-year history of alcohol abuse and dependence admitted for chest pain, r/o MI. Last drink one hour ago. Blood alcohol 150 mg/dL.

Neuro exam: alert, dysarthric, marked ataxia, course tremor of arms and fingers, failed heal-to-toe walk test.

Clinically indicates:
Alcoholic cerebellar degeneration (G31.2)

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Delirium vs. Encephalopathy

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Delirium

Delirium: Disturbance in attention and awareness that develops over a short time; acute confusional state.

Delirium codes to R41.0, disorientation, confusion. If due to alcohol or drugs, it is coded to the appropriate F-code for substance use/abuse.

Delirium with Encephalopathy

Delirium is a common manifestation of encephalopathy.

DSM-5 defines delirium as a disturbance in attention and awareness that develops over a short time and may be attributable to drugs/chemicals or "to the physiological consequences of another medical condition."

"The other condition should also be coded and listed separately immediately before delirium…"

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Examples

Delirium vs. Encephalopathy

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80-year-old male admitted with severe diabetic hyperglycemia and mild ketosis. Blood sugar 750, sodium 128, BUN 40, creatinine 1.3.

Physician exam: agitated, disoriented, confused, resisting treatment. Required IM Haldol for sedation.

Diagnosis:

(1) Diabetic hyperglycemia

(2) Dehydration

(3) Delirium due to # 1 and 2

Clinically indicates:
Acute metabolic encephalopathy due to hyperglycemia and dehydration

78-year-old female admitted with severe abdominal pain and found to have nephrolithiasis. Treated with IV Toradol.

On the second day of admission, the patient was confused and hallucinating at night. Toradol changed to tramadol and symptoms resolved.

Clinically indicates:
Adverse effect of drug
Not an encephalopathy

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Hepatic Encephalopathy

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Hepatic encephalopathy is altered brain function caused by the liver's inability to eliminate toxins from the blood.

Associated with decompensated cirrhosis.

It describes a spectrum of neurologic impairment in patients with liver failure such as altered mental status, confusion, disorientation, inappropriate behavior, combativeness, gait disturbances, and/or altered level of consciousness ranging from drowsiness to deep coma.

Caused by **elevated blood ammonia**. Treated with lactulose.

Can be acute (overt), chronic, or acute on chronic.

See CDI Pocket Guide® topic and webinar:
Liver Disease and Failure

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Hepatic Encephalopathy with TME

Coding Clinics 2021 and 2022

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Toxic Metabolic Encephalopathy and Hepatic Encephalopathy

- **Question**: A 70-year-old patient with a history of **nonalcoholic steatohepatitis (NASH) cirrhosis** complicated by hepatic encephalopathy and diabetes presented to the emergency department (ED) secondary to altered mental status. The patient was admitted for a full work-up and was diagnosed with **toxic metabolic encephalopathy (TME) secondary to acute on chronic hepatic encephalopathy**.

  Is it appropriate to separately report TME when due to hepatic encephalopathy? Would TME be considered inherent to hepatic encephalopathy? How should toxic metabolic encephalopathy due to acute on chronic hepatic encephalopathy be coded?

- **Answer:** Assign codes [K76.82, hepatic encephalopathy] and [G92.8], Toxic encephalopathy, for toxic metabolic encephalopathy due to acute on chronic hepatic encephalopathy. Code G92 is assigned separately to specifically capture the toxic metabolic encephalopathy. All three codes are needed to capture the patient's diagnoses.

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Encephalopathy due to CVA

Coding Clinic 2017 Second Quarter, page 9

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Encephalopathy Associated with Cerebrovascular Accident

- **Question:** A patient is admitted to the hospital due to altered mental status and is diagnosed with an acute lacunar infarct and encephalopathy secondary to the lacunar infarction. Would the encephalopathy be coded separately or is it considered inherent to the acute lacunar infarct?

- **Answer:** Assign code G93.49, Other encephalopathy, for encephalopathy that occurs secondary to an acute cerebrovascular accident/stroke. Although the encephalopathy is associated with an acute lacunar infarct, it is not inherent, and therefore is coded when it occurs.

Clinically valid? Does the patient have a diffuse alteration in mental status?

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Key Takeaways

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1) Confirm diffuse (generalized) nature of mental status alteration
2) Identifying the cause is important (confirmed or suspected)
3) Encephalopathy can be PDX if primary reason for admission or adverse drug effect
4) Dementia patients: Verify change from and return to baseline
5) Alcohol: Distinguish alcohol intoxication with and without toxic encephalopathy; alcoholic encephalopathy which is a chronic encephalopathy (degeneration). Withdrawal delirium (delirium tremens) is not a toxic encephalopathy.
6) Delirium: Often a symptom of encephalopathy (DSM-5)
7) Do not query for metabolic encephalopathy each time there is hepatic encephalopathy.
8) CVA: Does the patient have a diffuse alteration in mental status?

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Case Study #1

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49-year-old patient with history of type 2 diabetes on insulin and CKD III presents to the ED with AMS. ED physician noted, "she seems a little sedated but is following commands" with physical exam positive for lethargy. Initial total Glasgow Coma Scale was 9 with a verbal response of 1. Patient was noted to be in DKA with glucose 487 and started on DKA protocol with fluids, electrolyte replacement and insulin drip.

Patient was admitted to the ICU for DKA with acute kidney injury and toxic-metabolic encephalopathy. GCS scores were reported while the patient was in the ICU with total scores ranging from 6 to 15 on hospital days 1 and 2. Patient returned to baseline mental status by hospital day #3, weaned off insulin drip and started on routine insulin with hyperglycemic protocol. The diagnosis of toxic metabolic encephalopathy was consistently documented throughout the entire record from admission to discharge.

Is toxic metabolic encephalopathy integral to diabetic ketoacidosis (DKA)?

Encephalopathy is not integral to DKA.

Principal Diagnosis:

Diabetic ketoacidosis

Secondary Diagnosis:

Metabolic encephalopathy
(does not indicate "toxic" encephalopathy)

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Case Study #2

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Patient who presented to the ED on 4/6 from jail for confusion after being there for one day. Admitted with a diagnosis of toxic encephalopathy and possible alcohol withdrawal. He was also found to have an elevated CK. He was treated with IV fluids and benzos.

H&P: Patient admitted with alcohol withdrawal syndrome with perceptual disturbance. Complete current banana bag. Monitor for seizures, tremors. On Librium for ETOH withdrawal. Urine drug screen is positive for opiates, methamphetamines, benzodiazepines. Pt also admits to drinking alcohol. Received Ativan in ER.

Progress Notes: Alcohol withdrawal, toxic encephalopathy due to drugs and ETOH

Discharge Summary: Alcohol withdrawal, toxic encephalopathy due to drugs/ETOH, rhabdomyolysis.

Principal Diagnosis:

Alcohol withdrawal syndrome F10.232

Secondary Diagnosis:

Poisoning, methamphetamines T43.621A

Poisoning, opiates T40.2X1A

? Toxic encephalopathy G92

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Case Study #3

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72-year-old female admitted on 1/14 following observation with catatonic depression, not eating x one week, postobstructive uropathy, suspected sepsis, metabolic encephalopathy, dehydration, AKI on CKD (Cr 2.68 -> 1.58). WBC 16.9K, Temp 99, RR 18, P 82.

1/15 Hospitalist progress note: "Postobstructive uropathy manageable with Foley. Will continue IV fluids." All subsequent progress notes same (copied and pasted). Seen daily by Psych with plans to transfer to Psych unit when medically stable. 1/19 Psych PN: "At this point the clinical picture looks a lot more like her psychotic episodes of the past" – will plan to transfer to psych unit once a bed is available.

Patient transferred to psych unit on 1/25. No source of infection identified or documented.

Metabolic encephalopathy is not supported.

Principal Diagnosis:

Catatonic depression F33.3

Secondary Diagnosis:

AKI N17.9

Postobstructive uropathy T43.621A

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Case Study #4

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We keep getting denials for toxic encephalopathy in the cases where the patient has taken medications or had an overdose.

The denials state that the AMS was due to a drug overdose thus the alteration was inherent to T44.3X2A, T50.992A and T43.222A, and G92 should be removed.

They include as references Dr. Pinson's ACP Hospitalist, Encephalopathy 2015 article and UpToDate Acute Toxic-Metabolic Encephalopathy.

Example 1: Patient took unknown number of Benztropine, Melatonin and Celexa and said he wanted to die. Obtunded, speech garbled. Unable to obtain ROS due to mental status.

Treatment: NG tube, administration of charcoal-sorbitol, hold sedating medications. Patient awake, alert, oriented the following day. Provider documented: Acute toxic encephalopathy.

Example 2: Patient found altered by husband, brought to ED. Found empty Klonopin bottle at bedside. General appearance: lethargic, disoriented, confused. GCS 7, 12-14 after Narcan admin—AMS resolved and GCS 15 at discharge.

Treatment: Narcan, Acetadote, MRI to rule out other underlying causes.

Provider documented: Toxic encephalopathy due to overdose

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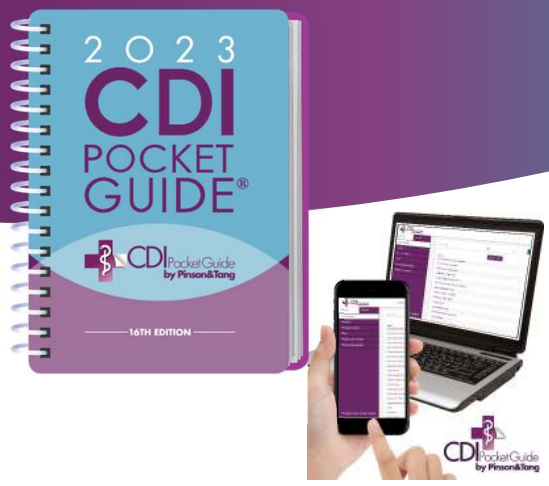

Thank you for attending!

Questions? Please contact us at
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