Delirium

November 3, 2016 by Josh Farkas


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definition

- Delirium is acute, generalized brain dysfunction (“cerebral insufficiency”). Key features:
  - acute (e.g. not dementia)
  - causes inattention (e.g. disorientation, inability to perform complex tasks)
  - tends to wax and wane: may have periods of lucidity in between periods of confusion.
- Diagnosis is made clinically, based on examination and history.
common features

- Commonly classified as follows:
  - Hyperactive delirium: agitation
  - Hypoactive delirium: patient is withdrawn, mute, drowsy. This can often fly under the radar because it's not overtly problematic.
  - Mixed delirium: periods of hyperactive delirium & hypoactive delirium
- Features can involve:
  - Psychotic symptoms (hallucinations, delusions, paranoia).
  - Emotional symptoms (fear, anxiety, irritability, anger).
  - Day-night reversal with hyperactivity at night ("sundowning").
  - Increased sympathetic activity (hypertension, tachycardia).

epidemiology

- Extremely common among critically ill patients (>50% of patients).
- Risk correlates with illness severity, intubation, and low cognitive reserve function (e.g. baseline dementia or minimal cognitive impairment).

delirium screening? (CAM-ICU)

- CAM-ICU ([https://www.mdcalc.com/confusion-assessment-method-icu-cam-icu](https://www.mdcalc.com/confusion-assessment-method-icu-cam-icu)) is a bedside test to detect delirium among ICU patients which is often used in clinical research.
- It has become popular to test CAM-ICU routinely for ICU patients. However, there is no solid evidence that this improves outcomes.
- The 2018 SCCM guidelines concluded that evidence doesn't establish whether delirium screening is beneficial, and thus they made no recommendation regarding this.¹
- Application of the CAM-ICU test several times a day represents a significant workload for the bedside nurse.
- Rather than screening for delirium, the best global strategy to reduce delirium in the ICU might be to apply delirium prevention strategies to all patients in the ICU (more on this below).
- Evidence supporting primary prevention of delirium is more robust than evidence supporting early detection and intervention.²

causes

Delirium can reflect a broad range of neurologic or systemic aberrations. In elderly patients, delirium can be the manifestation of systemic illness (e.g. sepsis or respiratory failure). All contributory factors should be sought out and managed. Common causes are listed here, but this list isn't exhaustive.

- CVA
- Hemorrhage (e.g., parenchymal hemorrhage, subdural hematoma)
- Ischemic stroke

### Infection
- Meningitis, encephalitis
- Sepsis arising from any focus of infection (e.g. urinary tract infection, pneumonia)

### Medications: The biggest offenders are listed here, but dozens can cause confusion. Review the entire medication list carefully, focusing on new medications and medications acting on the nervous system. Use an application such as Epocrates to look for drug-drug interactions and search for medications that cause delirium.
- GABAergics (benzodiazepines, muscle relaxants)
- Antihistamines (e.g. diphenhydramine, promethazine)
- Opioids
- Antimicrobials (esp fluoroquinolones, cefepime)
- Anticonvulsants (carbamazepine, phenytoin, valproate)
- Parkinson's medications
- Metoclopramide
- Zolpidem and related sleep medications
- Steroid

### Metabolic
- Hypoglycemia
- Fever/hyperthermia
- Electrolyte abnormality (especially abnormal sodium, hypercalcemia)
- Wernicke's encephalopathy
- Hyperammonemia not due to cirrhosis (rare, but occasionally seen)

### Organ failure
- Cardiovascular: shock, hypertensive encephalopathy
- Pulmonary: hypoxemia, hypercapnia
- Liver: Hepatic encephalopathy
- Renal: Uremia
- Endocrine: Thyroid storm/myxedema coma
- Heme: Thrombotic thrombocytopenia purpura (TTP)

### Seizure

### Toxicologic
- Withdrawal from EtOH, benzodiazepines, gabapentin, baclofen, opioid, serotonin-norepinephrine receptor inhibitors
- Intoxication/poisoning (e.g. carbon monoxide, lithium, digoxin)

### Sleep deprivation
- Noisy ICU environment
- Frequent blood pressure or neurologic checks
- Uncontrolled pain
This is tricky. Delirium can be due to life-threatening conditions, so a thorough evaluation is required for a patient with new-onset delirium. However, a patient with repeated episodes of delirium doesn’t need a big workup for each exacerbation. The most concerning feature is when the patient’s current mental status is an abrupt and major departure from baseline.

**evaluation**

Vital sign abnormalities (may reflect shock or respiratory insufficiency)

Neurologic exam (evaluate for any focal neurologic findings)

Foci of infection (e.g. site of any invasive lines/tubes)

**labs**

- **Basics**
  - Fingerstick glucose (STAT)
  - Electrolytes, including Ca/Mg/Phos
  - CBC with differential

- Consider, depending on context:
  - LFTs
  - Ammonia
  - TSH
  - ABG/VBG if patient somnolent and hypercarbia suspected
  - Infectious workup (e.g. urinalysis, blood cultures)
  - Pertinent drug levels (e.g. digoxin, lithium, theophylline)
  - Additional toxicologic workup, depending on context (e.g. carboxyhemoglobin level for community-onset delirium during winter).

**neuroimaging**

- Nonenhanced CT head may be considered for:
  - Patients presenting with delirium, especially if history is unclear
- CNS trauma
- Significant anticoagulation
- Neurologic exam showing focal signs (note though that subdural hematoma can depress mental status without focal findings)
- Substantially reduced level of consciousness
- MRI may provide additional information about a variety of conditions (especially CVA).

**lumbar puncture**

- Primarily useful for patients presenting to the hospital with delirium.
- In the absence of a specific precipitating factor (e.g. neurosurgery, endocarditis), it’s uncommon for a patient to develop meningitis de novo within the hospital. Thus, lumbar puncture is generally low-yield for someone who develops delirium while in the ICU.

**EEG**

- Indicated if there is a suspicion for seizure or nonconvulsive status epilepticus:
  - Seizure history
  - Unusual facial twitching or automatisms (e.g. chewing or lip-smacking movements)
  - Nystagmoid eye movements or hippus (spontaneous pupillary fluctuations; video below)

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**delirium prevention**

The rate of delirium in the ICU is extremely high, so delirium prevention measures should be employed for all patients. This is a fundamental component of high-quality critical care for every patient.

**avoid deliriogenic medications**

- Medications with a strong tendency to cause delirium should be avoided if possible.
- Probably the worst offenders here are benzodiazepines. They have consistently been associated with delirium in correlational studies and RCTs. Ideally benzodiazepines should be avoided entirely (with some exceptions, for example patients chronically on benzodiazepines).
- Other particularly deliriogenic medications which should generally be avoided in the ICU include:
  - Fluoroquinolones (https://emcrit.org/pulmcrit/fluoroquinolone-critical-illness/)
  - Zolpidem (AMBIEN) and related hypnotic agents
  - Anticholinergics (e.g. diphenhydramine)

**thoughtful pain management strategy**

- Delirium can be closely interrelated with pain:
  - Uncontrolled pain may interfere with sleep and thereby promote delirium.
  - Excessive use of opioids can cause delirium. This is mostly problematic when opioids are being used for their sedative properties (i.e. as a nonspecific calm-down medication for the agitated patient, instead of actually titrating the opioid against pain).
  - If the patient has substantial pain, a multimodal pain strategy (https://emcrit.org/pulmcrit/analgesic-ladder/) may help control pain while avoiding delirium. This often involves the following components:
    - Scheduled acetaminophen (e.g. 1 gram Q6hrs)
- Low-dose ketamine infusion for pain (e.g. 0.1-0.3 mg/kg/hr)
- Low doses of PRN opioid titrated against pain
- Other non-opioid therapies as applicable (e.g. nerve blocks, lidocaine patches).

**preserve sleep quality**

- Sleep deprivation is an important cause of delirium.
- Limit stimulation at night (close the door, shut off the TV, turn off the lights, avoid unnecessary examinations).
- Ear plug use at night has been shown to avoid delirium. This should probably be done for all ICU patients if tolerated.
- Eye shades may similarly be used if tolerated.
- Remove unnecessary invasive devices.
- Decrease the frequency of vital sign monitoring at night if possible (especially blood pressure cuff cycling).

**melatonin**

- Several RCTs have emerged recently suggesting that melatonin agonism may prevent delirium (reviewed [here](https://emcrit.org/pulmcrit/melatonin/)). Ongoing studies will hopefully clarify this further. Given that melatonin agonists are extremely safe, this is currently a very reasonable intervention.
- Depending on your hospital's pharmacy, options may include melatonin (~3 mg) or ramelteon (8 mg). This should be given on a scheduled basis prior to sleep.

**pharmacologic insomnia treatment**

- Common errors when treating insomnia:
  - Use of deliriogenic medications (e.g. benzodiazepine, diphenhydramine, or zolpidem)
  - Administration of medications early in the morning (e.g. between 2-5 AM), which causes patients to sleep during the day.
- If medication is going to be used for insomnia, it should be employed *early* in the night (ideally before ~11 PM).
- Preferred medications are:
  - Quetiapine 25-50 mg PO.
  - Trazodone 50 mg (regarded as a delirium-friendly treatment for insomnia, but little evidence available).

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**treatment overview**

Treatment begins with basic anti-delirium strategies listed in the last section on *delirium prevention*.

**basics**

- Evaluate for and treat any specific cause(s).
- Exclude hypoglycemia and fix electrolyte abnormalities (especially hypernatremia).
- Treat fever if that may be contributing.
- Give thiamine if Wernicke's encephalopathy is possible (a treatment dose for Wernicke's encephalopathy is high, e.g. 500 mg IV q8hr).
- Review the medication list and discontinue drugs that may be aggravating delirium.
- Treat pain adequately and relieve other sources of discomfort (e.g. constipation, unnecessary invasive devices).
- Provide patient's eyeglasses and hearing aids.

**philosophy of medication therapy for delirium**

- Medications don't really fix delirium (the only thing that seems to do that is sleep).
- The goals of medication are:
  - (a) Render the patient safe and manageable (if severely agitated).
  - (b) Promote sleep and normal circadian cycle.

**entrainment of circadian rhythm**

- Delirious patients often lose their normal day-night cycle. They may benefit from efforts to promote sleep at night.

https://emcrit.org/ibcc/delirium/
Simple measures may be effective:
- During the day: Open the curtains, use aggressive physical therapy, stimulate patient.
- At night: Use ear plugs, avoid frequent blood pressure measurement, turn off the television, and limit stimulation.
- Medications can help promote sleep at night, particularly QHS melatonin or ramelteon (a synthetic melatonin agonist). Some evidence suggests that these agents may prevent or treat delirium.
- For patients who are frequently requiring antipsychotics, it may be helpful to schedule these before sleep (e.g. quetiapine 50 mg QHS).
  Scheduling antipsychotics at ~9 PM before sleep may allow the patient's getting PRN doses at 3-5 AM, which causes the patient to sleep all day and then be agitated the following night.

**dexmedetomidine**

- Emerging as a useful and extremely safe therapy for agitated delirium. Advantages include lack of respiratory depression and ability to titrate.
- Great choice for dealing with nocturnal agitation (sundowning). Titratability allows this drug to be used to re-entrain a circadian rhythm:
  - During night: titrate dexmedetomidine to promote sleep.
  - During day: aggressively titrate dexmedetomidine OFF, promoting wakefulness during the day.
- The short half-life of dexmedetomidine avoids the sundowning cycle wherein patient gets tons of medications for sundowning around 2 AM, sleeps for ~18 hours, and then wakes up and gets agitated the following evening.
- Main complication is bradycardia. If dexmedetomidine is truly needed, this may be managed with a very low-dose infusion of peripheral epinephrine (e.g. 0-4 mcg/min).

**antipsychotics**

**role of antipsychotics in treatment of delirium?**

- (1) Treatment of agitation: May avoid inadvertent tube removal and facilitate cooperation with care.
- (2) Treatment of insomnia.
- Antipsychotics are not beneficial in patients with hypoactive delirium.\(^4\)
- Antipsychotics are contraindicated in patients with Parkinson's disease or Lewy Body Dementia, as these patients will be at increased risk for extrapyramidal side-effects.

**how are these drugs actually working?**

- Antipsychotics are “dirty drugs” that hit a lot of different receptors. It's debatable exactly how they are working.
- At the low doses usually used in the ICU, some atypical antipsychotics (especially quetiapine) may be acting predominantly as sedatives rather than anti-psychotics.\(^5\) They may function as non-deleriogenic sedatives:
  - The primary effect is sedation (largely via inhibition of histamine; olanzapine also causes sedation via anticholinergic activity).
  - Normally, blockade of these receptors would promote delirium. However, atypical antipsychotics also have built-in antipsychotic activity (due to dopamine blockade) which prevents exacerbation of the delirium.
  - This explains why the atypical antipsychotics most useful in the ICU are the most sedating ones (compared to, for example, less sedating agents such as risperidone).
  - Other sedatives are typically superior (e.g. propofol, dexmedetomidine). However, occasionally an atypical antipsychotic may be useful as a sedative-sparing agent (e.g. a patient is having difficulty tolerating propofol due to hypotension or hyperlipidemia).
For critically ill patients, evidentiary support is strongest regarding three agents: haloperidol, quetiapine, and olanzapine.\(^5\)

### haloperidol

- Traditional mainstay of therapy for severe agitation.
- Main advantage is that it's universally available and can be provided in a variety of IV doses, which allows for dose-titration in a patient with agitation.
  - Start with a dose of 2-5 mg IV (1)
  - Wait 20 minutes to observe full effect, then administer additional dose as needed.
  - Max dose is unclear; historically astronomical doses have been used. However, if the patient doesn't have a favorable response after 10-20 mg IV then it's probably better to try a different class of agents (e.g. dexmedetomidine).
- Two main contraindications:
  1. Prolonged QTc (may increase risk of haloperidol-induced torsade de pointes).
  2. Parkinson's disease or Lewy Body dementia.

- Intravenous administration is preferred, as this may reduce the risk of extrapyramidal symptoms.
- Not a good agent for ongoing therapy, due to a relatively high risk of extrapyramidal symptoms (e.g. tardive dyskinesia). An atypical antipsychotic (quetiapine or olanzapine) is generally preferable if ongoing maintenance therapy is needed, with IV haloperidol reserved for PRN break-through agitation.
- Pitfall: The doses of haloperidol which are typically listed in pharmacopeias (e.g. 10 mg IV) are with reference to young, psychotic patients. For most elderly ICU patients, 10 mg can be far too high. When in doubt, if the patient isn't dangerously agitated start with lower doses (e.g. 2 mg IV).
- Haloperidol was ineffective in the MINDS-USA trial, likely because 89% of patients in this trial had hypoactive delirium.\(^4\) This emphasizes that haloperidol is unhelpful for hypoactive delirium (more on this [here](https://emcrit.org/pulmcrit/antipsychotics-delirium/)).

### commonly used antipsychotics

<table>
<thead>
<tr>
<th>Haloperidol (HALDOL)</th>
<th>Quetiapine (SEROQUEL)</th>
<th>Olanzapine (ZYPREXA)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Route</strong></td>
<td>IV preferred</td>
<td>PO only</td>
</tr>
<tr>
<td></td>
<td>IM possible</td>
<td>Rapidly absorbed (within 1 hr)</td>
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<td></td>
<td>PO discouraged</td>
<td>PO (slowly absorbed)</td>
</tr>
<tr>
<td><strong>Half-life</strong></td>
<td>~22 hours</td>
<td>~7 hours</td>
</tr>
<tr>
<td></td>
<td></td>
<td>~30 hours</td>
</tr>
<tr>
<td><strong>Major advantages</strong></td>
<td>Most titratable agent.</td>
<td>- Shorter half-life makes this a reasonable drug for insomnia.</td>
</tr>
<tr>
<td></td>
<td>Immediately available in most units.</td>
<td>- High max daily dose (800 mg) can make this a good option for patients with difficult-to-control agitation on the ventilator.</td>
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<tr>
<td></td>
<td></td>
<td>- No risk of Torsades de Pointes.</td>
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<td></td>
<td></td>
<td>- Longer half-life makes this a good choice for patients on ventilation (provides some degree of daytime sedation).</td>
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<tr>
<td><strong>Disadvantages</strong></td>
<td>Highest risk of extrapyramidal symptoms, especially if continued over time.</td>
<td>Limited routes of use Onset too slow for acute agitation.</td>
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<tr>
<td></td>
<td></td>
<td>Relatively low dose ceiling (max 30 mg/day).</td>
</tr>
<tr>
<td><strong>Effect on QTc</strong></td>
<td>Highest (But absolute risk still very low)</td>
<td>Low</td>
</tr>
<tr>
<td><strong>Torsades Risk</strong></td>
<td></td>
<td>No risk</td>
</tr>
<tr>
<td><strong>Dose</strong></td>
<td>- Start with 2-5 mg IV</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Wait 20 min before re-dosing.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- If no response to cumulative dose of 10-20 mg try different drug class.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Insomnia: 25-50 mg QHS</td>
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<tr>
<td></td>
<td>- Delirium in ventilated patient: 50 mg q12 hours, up-titr. Usually don't go higher than ~200 mg BID, but this can occasionally be necessary. May give higher PM dose than AM dose to promote sleep/wake cycle.</td>
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<tr>
<td></td>
<td>- In general, twice as powerful as haloperidol (e.g. 5 mg IM olanzapine is equivalent to 10 mg IM haloperidol).</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Delirium in ventilated patient: 5-20 mg QHS.</td>
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<tr>
<td><strong>Major roles</strong></td>
<td>- Acutely agitated patient.</td>
<td></td>
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<tr>
<td></td>
<td>- Test dose can be used to see how patient will respond to an antipsychotic.</td>
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<td></td>
<td>- Insomnia</td>
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<td></td>
<td>- Agitation/delirium on ventilator</td>
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<td></td>
<td>- Agitation/delirium on ventilator</td>
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<td></td>
<td>- Acutely agitated patient.</td>
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<tr>
<td></td>
<td>- Patient with prolonged QTc.</td>
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</tbody>
</table>

\(^5\) [For critically ill patients, evidentiary support is strongest regarding three agents: haloperidol, quetiapine, and olanzapine.](https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/antipsychotict.jpg)
quetiapine

- Good agent for insomnia in non-intubated patient:
  - Relatively rapid oral bioavailability (peak serum levels within an hour).
  - Half-life is relatively short, so that the patient will wake up the following morning.
- May be used for agitation in the intubated patient as well:
  - Should be dosed q12 hours. However, it may be preferable to give higher doses at night to preserve day-night cycles (e.g. 100 mg qPM & 50 mg qAM).
  - Very high dose ceiling (~800 mg total daily dose) can occasionally be useful for the extremely difficult-to-sedate patient on the ventilator. Rapid up-titration seems to be safe among critically ill patients.6
- The most encouraging data for ICU delirium was obtained using quetiapine (infographic above). Use of quetiapine did cause patients to receive less fentanyl, so it’s possible that benefits don’t reflect a positive effect of quetiapine (but rather may reflect a benefit from less fentanyl). However, this mechanism of benefit likely occurs in real life as well, so it doesn’t necessarily invalidate the study’s conclusions.
olanzapine

Olanzapine, like haloperidol, is only FDA approved for IM administration. However, it can be given intravenously.

Olanzapine

Some unique advantages:

- Doesn’t cause Torsade de Pointes (so you don’t have to worry about the damn QTc interval).
- Can be given via numerous routes.
- Olanzapine isn’t FDA approved for IV administration (but neither is haloperidol). This has led to some concerns regarding IV administration. However, an increasing body of literature shows that IV administration is safe and effective.
  - Advantages of IV administration include: ability to give smaller doses and titrate to effect, painless administration, elimination of risk of needle-stick injury to staff.
- When given parenterally, olanzapine is roughly twice as powerful as haloperidol.
  - 10 mg olanzapine is a hefty dose (equivalent to ~20 mg haloperidol). This is too high for most elderly delirious ICU patients. A starting dose of ~2.5-5 mg may be more appropriate.
- Oral olanzapine can take some hours to reach peak effect. If prompt effect is desired, better options include oral quetiapine or IV olanzapine.
- One small RCT found that olanzapine was equally effective compared to haloperidol, while causing fewer extrapyramidal side-effects.
Evaluation for new-onset delirium

- **Initial assessment**
  - Vital signs, neuro exam
  - STAT fingerstick glucose
  - Review medication list
  - Review recent events/procedures

- **Labs: consider depending on context***
  - Electrolytes including Ca/Mg/Phos
  - CBC with differential
  - ABG/VBG only if hypercapnic encephalopathy likely
  - TSH
  - LFTs, ammonia
  - Relevant drug levels (e.g. lithium, digoxin, lidocaine)
  - Carboxyhemoglobin level for new presentation in winter
  - Additional tox/infectious workup as appropriate

- **Consider CT head especially if***
  - Anticoagulated
  - Trauma
  - Acute/severe headache
  - Focal abnormality on exam
  - Substantially reduced level of consciousness

- **Consider LP especially if***
  - Delirium is chief complaint on admission
  - Evidence of infection (fever, leukocytosis, nuchal rigidity)

- **Consider EEG especially if***
  - History of seizure
  - Exam concerning for nonconvulsive status

*Delirium is very common in the ICU and not every patient needs a ton of tests. More aggressive workup needed if: rapid & major change in mental status, delirium is the presenting problem to hospital, no obvious cause for delirium, no prior history of delirium/dementia.

The internet birth of critical care by @emcrit


**treatment algorithm**
Delirium treatment

- Remove causative factors
  - Exclude hypoglycemia if this is possible
  - Review the medication list and d/c deliriogenic meds if possible.
  - Treat hypernatremia if present.
  - Remove unnecessary invasive devices/tubes & restraints.
  - Consider scheduled acetaminophen if uncontrolled pain or persistent fevers.
  - If Wernicke's encephalopathy possible, empiric thiamine 500 mg IV Q8hr
  - If cirrhotic, consider empiric treatment for hepatic encephalopathy

- Sleep maintenance
  - Scheduled melatonin agonist before sleep (melatonin ~3 mg or ramelteon 8 mg).
  - Earplugs & eye shades at night if tolerated.
  - Avoid unnecessary sleep interruption (e.g. frequent 8p cuff monitoring).
  - If difficulty sleeping, administer pharmacologic therapy early in the night (e.g. quetiapine 50 mg qhs)

- Nocturnal dexmedetomidine
  - May be useful in patients with severe nocturnal agitation, especially if this is refractory to antipsychotic therapy.
  - Use dexmedetomidine at night, titrate to light sleep.
  - Discontinue dexmedetomidine during the day to maintain circadian rhythm.

- Reorientation during the day
  - Use patient's glasses & hearing aides if needed
  - Physical therapy and early mobilization.

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podcast


The Podcast Episode

Want to Download the Episode?

[Right Click Here and Choose Save-As](http://traffic.libsyn.com/ibccpodcast/IBCC_EP10_Delirium_final.mp3)

questions & discussion

To keep this page small and fast, questions & discussion about this post can be found on another page [here](https://emcrit.org/pulmcrit/delirium/).
DO NOT treat agitated delirium with a benzodiazepine. This will suppress symptoms temporarily, but will eventually make the delirium worse.

Avoid benzodiazepines in general, with certain very specific exceptions (chronic benzodiazepine use, status epilepticus). The practice of using lorazepam for insomnia needs to be abolished.

Don’t overlook the cause of delirium, especially when it represents a dramatic neurologic change in a patient without any neurologic history. It may be the manifestation of a severe disease process (e.g. subdural hematoma, sepsis).

Don’t treat elderly, multimorbid ICU patients with the same doses of antipsychotic that you would use for a young psychotic patient. When feasible, start low and titrate to effect.

Going further:

- **Delirium** ([https://lifeinthefastlane.com/ccc/delirium/](https://lifeinthefastlane.com/ccc/delirium/)) (Chris Nickson, LITFL)
- **Antipsychotics**
  - MINDS-USA & role of antipsychotics in delirium ([https://emcrit.org/pulmcrit/antipsychotics-delirium/](https://emcrit.org/pulmcrit/antipsychotics-delirium/)) (PulmCrit)
  - Olanzapine ([https://emcrit.org/pulmcrit/intravenous-olanzapine-haloperidol/](https://emcrit.org/pulmcrit/intravenous-olanzapine-haloperidol/)) is ok for IV administration. And doubling down on IV olanzapine again [here](https://emcrit.org/pulmcrit/iv-olanzapine/)
- Other sub-types


