Hi. I’m Navaz Karanjia. I’m a neurointensivist and the director of neurocritical care at UCSD. Today I’m going to give you a brief introduction to continuous EEG monitoring in the ICU.
We’re going to do a brief overview and introduction to the techniques used in ICU EEG monitoring, go through a number of clinical examples, then talk about challenges and future trends. First, a birds eye view of why continuous eeg monitoring is useful in icu patients.
It is a portable, continuous, noninvasive method of measuring cerebral function that is quantifiable, has been well studied for decades, and as such can be used as a vitals sign for the brain in the ICU.
It can identify neurologic complications, and what is usually a surprise to people is that it not only useful for identifying seizures, but is also very useful for identifying ongoing cerebral ischemia, expanding hematomas, and increased ICP, in time to intervene and alter outcome. It can also identify systemic metabolic complications, assess prognosis, and be used to monitor depth of sedation, for example, when we’re titrating to burst suppression, and regulate therapies like induced hypertension for vasospasm.
How is it done?
It can be done either by hardwired system—here's a pic of my old unit at UCLA prior to the hospital renovation, with every NCCU bed with its own hardwired EEG system, to little portable units like this one that can be wheeled from room to room. Systems should have full multichannel EEG capability, be easily accessible and visible because it is used like a vitals monitor, and are usually fully integrated into the electronic medical record because certain parameters, like alpha delta ratio and symmetry, are charted like vitals are in the EMR.
But since the EEG is just a bunch of squiggly lines to most people, and it takes a long time to read through 24 hours of continuous EEG, they had to come up with a way to make it trendable in a meaningful manner so it could actually be useful as a vitals sign of neurologic function. The major technique that is used to do that is the compressed spectral array, which takes the raw EEG data and plots it via fourier transform into an easy to read EEG trending diagram, like this.
This is 8 hours of EEG compressed through spectral analysis. The higher the band, the more EEG activity there was at that time. The reason why this is so powerful is that it can show you what’s been going on on the EEG, in a compressed time scale, so you can look back over 8 hrs and at a glance tell how many seizures the patient has had. The software also crossreferences all the frequencies against each other so we can identify when there is high amplitude synchronous activity, which is highly suspicious for seizure. It can also identify when the ratio of certain frequencies change, like when the alpha delta ratio decreases, which is highly sensitive and specific for vasospasm. It can show you clearly when asymmetry in the EEG starts to occur, which usually indicates something has started to go wrong on one side of the brain.
Here’s a demonstration of how easy it is to see that this patient had 4 seizures over a 6 hour period, using trending software.
The way it looks when all put together is you have a monitor with the raw eeg on one side, which can be reviewed at any time, and a number of trending screens that can be configured for nursing convenience. For instance, this one has been set up to show you when you have a seizure, which shows up as red here. It’s showing your compressed trending analyses on the R and L hemispheres right below that, so you can watch for asymmetry, your alpha variability is below that, it’s got a little diagram of the head on the bottom right there that shows which leads are falling off, and it has numeric values here of your percent alpha, your percent delta, which in many neuroICU’s are tracked as vitals parameters. All of these machines also generally backup to longterm storage and have remote access capability.
So that’s how it works. Why do we do it, and who do we do it to?
So the most obvious clinical use is for seizure identification. You think, why would we need an EEG to identify seizures? They’re clinically obvious, right?
The truth is, in the icu, most seizures have no outward signs, or are nonconvulsive. Nonconvulsive seizures are electrographic sz without obvious convulsions, they may be evidenced only by staring, twitching, or a change in vitals, so they can only be diagnosed if the pt is being monitored. And, they cause the same amount of neurologic injury as convulsive seizures.
And when I say common, what do I mean? From studies of many thousands of patients over the years, we’ve found out that 8-10% of micu patients without brain injury with unexplained

- 8-10% of MICU patients without known brain injury with unexplained AMS; 67-100% were NCSz only (Towne, Oddo)
- 21% of MICU patients with AMS due to toxic-metabolic cause, sepsis, or renal failure; 98% were NCSz only (Classen)
- 20-35% of all hypoxic-ischemic injury patients with AMS; most NCSz (Wijdicks, Krumholz, Wright)
- 48% of patients who present with status epilepticus with persistent AMS have NCSz when monitored for 24 hrs; 14% were in NCSE (DeLorenzo)
- 31-38% of ICH patients with AMS; 58% NCSz only (Claassen, Vespa)
- 22% of all moderate to severe TBI patients; 50% NCSz only; dilantin exposure not predictive of seizure (Vespa)
- 19% of all altered SAH patients; 70% were in NCSE (Claassen)
- 11% of all altered ischemic stroke patients; 80% were NCSz only (Claassen)
Why do we care about identifying seizures? It’s because seizures kill neurons, which independently worsen functional outcome and mortality. The mortality after convulsive status epilepticus is 25% at 30 days, 15% have severe neurologic sequelae, 40% have a poor outcome. However, 43% can have a good recovery IF you have treatment protocols that ensure adequate therapy and are adhered to – that reduces mortality from 45 to 8%.
Nonconvulsive seizures are even worse. At 30 days, 65% of patients who have nonconvulsive seizures are dead. Nonconvulsive sz increase ICP, cerebral ischemia, hemorrhage size, midline shift, morbidity, mortality and atrophy the brain. But like with convulsive seizures, we know that we can make a difference in these patients’ outcomes if the seizures are caught early—if patients are diagnosed and treated less than 30 minutes from seizure onset, mortality is 36%, versus 85% if patients are diagnosed and treated over 24h from seizure onset.
In order to understand why seizures are so bad for you, you’ve got to understand what a seizure is, first of all. Normally, your EEG should look like this. Each line is the electrical potential between 2 EEG leads, and they all look different and random because the neurons in your brain should all be popping off randomly depending on what you’re doing. A seizure is a synchronized firing of a group of neurons, if it’s generalized it’s generalized to the whole brain, if it’s focal its just in one part of the brain. This synchronized firing is bad because it requires a huge amount of metabolic energy to fire this big and this often. So these cells are using glucose and oxygen and other nutrients up like crazy. To meet this demand, when things are compensated
the body increases its cerebral blood flow, heart rate, blood pressure, releases its energy stores, and generally revs up the sympathetic system, so its able to meet the metabolic requirements of the brain.
Physiologic changes-decompensated

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But your body can only do this for 3-5 minutes, before the cerebral demand outmatches the supply, and you get cellular hypoxia and hypoglycemia. Neurons start to get ischemic and die, which is how you get cerebral edema and increased ICP. Your body runs out of glucose and electrolytes get imbalanced, and organs start to fail because of hypoperfusion and low energy stores, so you get hepatic failure, heart failure, etc. Now the cerebral decompensation always happens whether you have a convulsive or nonconvulsive seizure. The systemic changes only sometimes accompany a nonconvulsive seizure.
The way we know these things are happening are by looking at microdialysis numbers and PET scans during seizures. Microdialysis is a technique that’s used commonly in academic nccu’s where patients have a little catheter inserted into their brain tissue that measures levels of glucose, lactate, glutamate, etc. what’s important to see here is that you can see, when someone’s seizing, the brain glucose is basically 0, while the levels of glutamate, your brain’s excitotoxic neurotransmitter, is very high which causes calcium influx and a whole host of neurotoxic metabolic changes. The lactate to pyruvate ratio, which is basically a marker of whether you’re in anaerobic metabolism or not, spikes during the seizure, which shows the neurons have switched into anaerobic metabolism mode during the seizure. When they do this, we know that they are becoming ischemic. And you can see here on this pet scan that during a seizure the cortex is over metabolically active.
This leads to neuronal ischemia, which you can see this on MRI. The DWI sequence over here shows ischemic tissue as bright, after a pt had a focal seizure. This patient had a generalized seizure, and so has had a significant amount of necrosis everywhere. And here’s an MRI a few months after an ICU stay where you can see significant hippocampal atrophy after they had had nonconvulsive seizures during their icu stay.
When quantified, the atrophy of the hippocampus doubles in TBI patients if the patient was seizing during their ICU stay.
And that atrophy is only if you survive. Because icp doubles on average during a seizure,
And midline shift worsens by on average half a centimeter during a seizure.
This is why the national guidelines from the neurocritical care society on who should receive ICU eeg monitoring state that these populations of patients should be monitored: anyone who just had a seizure without returning to baseline over 10 minutes, anyone who is comatose in the ICU, including those post cardiac arrest, anyone who has an intracranial hemorrhage, including TBI, SAH, and ICH, and anybody who is altered where nonconvulsive seizure are suspected.
So that is the big baddie, seizure, which we all know we need to watch for and treat. What about those other changes I was talking about earlier, with asymmetry, alpha delta ratios, and all that? How is knowing about that useful?
First asymmetry. This was a pt I had, a 16yo after an aneurysm clipping, who became just slightly more drowsy than we were used to on postop day 3. But he had been woken up all night, so we didn’t know if he was just sleepy, or if it was because of the sedation he was on because he was intubated. His exam was nonfocal. However, his EEG was asymmetric, as you can see here, the top 4 leads are the R side, and the bottom 4 leads are the L side, and the R sided leads are much quieter than the bottom 4. For those of you that don’t see it, don’t despair, the software spits out a nice color coded picture for you to see asymmetry too. This isn’t his picture unfortunately, but it can show you when there are hot spots of activity like here where there’s a red spot, or in his case, it would have shown you the whole R side would be abnormally quiet or blue. So we got a stat CT, which found a large R subdural which was evacuated and the pt had a good recovery.
Next, variability.
This was a 40yo lady I took care of who was day 5 s/p a H&H4 F4 SAH, who had been persistently drowsy since admission, who had developed maybe less spontaneous L leg movement since 2am. TCD in ACA’s 90’s on the previous day. Her TCD’s the previous day were equivocal, in the 90’s. On our trending software, however, the R frontal region alpha variability, which is the variation in amplitude of the alpha frequency in the EEG and one of the markers we track for ischemia, had decreased focally in her R frontal region, which was very worrisome for new ischemia or vasospasm. We called in our neurointerventionalist immediately, they angioed her and found R ACA vasospasm, and were able to plasty it open and save her leg. A year later, she walked back into my clinic.
A number of QEEG parameters have been shown to correlate with delayed cerebral ischemia: alpha variability, which I mentioned in the last slide, as well as total power, and alpha-delta ratio, and have been studied for their sensitivity and specificity. A-D ratio, for example. If it decreases more than 10% below baseline, it has a sensitivity of 100% and specificity of 76% for cerebral ischemia. [If the 50% threshold is used, it has a 89% sensitivity and 84% specificity. Here’s an example of what happens to the spectral eeg during vasospasm. Pretty dramatic. The reason why continuous EEG is so useful for monitoring these patients is that vasospasm doesn’t happen at convenient times. It doesn’t pop up right when you happen to be doing your neurocheck, or right when that tcd is done. It may reach a critical point oh, 5 minutes after you’re done doing your neurocheck, and then your patient undergoes 55 minutes of stroking out before you’ll check them again and maybe see something. Having a continuous method of monitoring for vasospasm is critical to our patients.
Here was a 39yo lady who had an intracerebral hemorrhage due to a R parietal AVM bleed. She was awake with only some left sided neglect, and she never had a change in her exam, but her trending showed decreased alpha variability in her R parietal area, and when we got a stat CT, we found she had rebled and had new midline shift. When we did a stat recheck of her coags, which hadn’t been done for about 8 hours, we found that her platelets had suddenly dropped and later we found out she had HIT. So obviously, our course of management changed for that patient, and perhaps due to our early detection, she ended up doing fine.
We often play with shutting down patients’ cerebral metabolism in the ICU, whether it's to reduce their ICP or after a bout with status epilepticus, and continuous EEG monitoring is obviously a good method with which to track those therapies because you can titrate to a quiet EEG, or burst suppression, or what have you. And as patients’ metabolism of the drugs change, you can continually assess whether you’re giving them too much drug, or too little, etc.
Being able to assess prognosis with an EEG is something that’s not unique to continuous monitoring, but where it is helpful is
Being able to assess a change in prognosis in a timely fashion, and thus act on it sooner rather than days later when you get your next spot EEG. This becomes important in our patients who are organ donation candidates, where being able to move forward with those discussions in a timely fashion can mean the difference between donating viable organs or not.
Some challenges in continuous eeg monitoring
Teaching nurses to read the EEG interface takes time and effort. Creating protocols which have them notify a provider at the appropriate time can be challenging because it’s a lot of information, and can generate a lot of noise for the nurse and provider if it’s not very clear what’s clinically relevant and what’s not. The technical difficulties of off hours hookups and maintaining good electrode contact in a thrashing sweaty ICU patient are challenging but not insurmountable as we now have caps with non-paste requiring electrodes which can be put on by the nurse and stay put. Something I didn’t mention here but is equally important are the IT logistical challenges of having that much information that your streaming and storing.
How is icu eeg monitoring going to evolve in the future?
Well, it’s been shown that continuous EEG monitoring, when utilized by a knowledgeable provider, changes care decisively in about half of patients monitored. This results in a reduced length of stay by up to 40%, and a similar decrease in cost of hospitalization. This is why the majority of neurocritical care units in the United States use continuous EEG monitoring now, and are even setting up remote continuous EEG monitoring for those that can’t do their own; for example, the UCLA group is providing continuous EEG monitoring for all the TBI patients at Walter Reed. All these software packages are commercially available and have become pretty easy to use.
What’s really the future of neurocritical care monitoring is integration of continuous EEG monitoring with other neurologic vitals signs, like the patients GCS, ICP, CPP, cerebral blood flow, cerebral tissue oxygenation, and microdialysis numbers, not just the numbers, but the waveforms as well. Here’s one interface that’s used at the university of Cambridge. This has the awesome potential, by plotting all this continuous data against one another, for us to discover new relationships between physiologic parameters, and figure out how they all affect one another.
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So to recap, continuous ICU eeg is useful to monitor for seizures and changes in neurologic function which can be an indicator of ischemia or structural changes, can give us early enough warning to intervene, can assist us in prognosticating in a timely fashion and guide our therapies, can help us understand our patients’ physiology better, thereby reducing complications, hospital stay, and expenses.
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Here's who's seizing in the ICU