



Commentary

Short Takes

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Association of peripheral blood pressure with gray matter volume in 19- to 40-year-old adults. Schaare HL, Masouleh SK, Beyer F, et al. Neurology 2019;92:e756-e773, <https://doi.org/10.1212/WNL.0000000000006947>

Brief Summary: This study tested whether increased blood pressure is associated with lower regional gray matter volumes in young adults. Four hundred twenty-three individuals (mean age 27.7 years, standard deviation 5.3 years) were included. These individuals had no prior history of hypertension or of chronic or acute disease. The study measured continuous blood pressure levels. The subjects underwent a 3 T T1-weighted image to measure regional gray matter volumes.

Blood pressure levels greater than 120/80 mm Hg were associated with lower gray matter volumes in diverse brain regions including the hippocampus, amygdala, thalamus, and frontal and parietal structures. Schaare et al. conclude that increases in blood pressure are associated with diminished brain gray matter volumes, buttressing the concept that the brain is a potential target organ in individuals with any increase in blood pressure.

Bottom line: We have been concerned about hypertension and the brain for many years.¹ In children, hypertension is defined by a Bell curve, whereas in the adult it is defined by blood pressure increases that result in end organ damage. Over the past 10 years there is compelling evidence that childhood hypertension negatively affects executive function and cerebral vascular reactivity; now there is evidence that increase in blood pressure diminishes gray matter volumes even in young adults. Studies in hypertensive children are ongoing using magnetic resonance imaging tractography that aim to show disruption in white matter tracts. We have shown that there is an association between hypertension and pediatric stroke, albeit probably not as strong an association as in adults with stroke.

Here blood pressure greater than 120/80 is associated with decreased gray matter volume in the young adult. This observation further buttress the concept that increased blood pressure may affect the young brain with chronic volumetric changes.

Editor's note: Short Takes offers a brief analysis by Steven G. Pavlakis of selected articles that may be of interest to child neurologists. Articles that strike the fancy of the analyst or the editors are selected for inclusion, but we welcome suggestions.

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<https://doi.org/10.1016/j.pediatrneurol.2019.05.008>
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I think this is a call that hypertension in the child can now be defined by its impact on the brain, and brain changes can occur as early as age 19 years. Ongoing studies will likely find that brain changes start even earlier. Any impact might be defined as consistent with “hypertension.” Once pediatricians and pediatric neurologists become aware of this problem, which may be reaching epidemic proportions secondary to life style and obesity, definitions and interventions will likely change. This article provides more evidence that increased blood pressure might be bad for the brain. We have postulated that this may be secondary to impaired autoregulation, and the treatment is prevention of the risk factors of hypertension and aggressive intervention.

Postconvulsive central apnea and asystole A risk marker for sudden unexpected death in epilepsy (SUDEP), St Louis EK and Dworetzky BA. Neurology 2019;92:115-116, <https://doi.org/10.1212/WNL0000000000006756>

Brief Summary: This study characterized postictal apnea and postictal asystole in generalized convulsive seizures in intractable epilepsy. Postconvulsive central apnea (PCCA) occurs in both generalized and focal epilepsy, but ictal central apnea occurs only in individuals with focal epilepsy.

This study examined 87 adults with intractable epilepsy in monitoring units. Monitored generalized convulsive seizures were an inclusion criterion. Studies were classified as generalized or focal. The study included 148 generalized seizures. Ictal central apnea occurred only in patients with focal seizures, whereas PCCA occurred in patients with both focal and generalized seizures. PCCA occurred concurrent to asystole in two patients. St Louis et al. opine that this asystole would qualify for a near-SUDEP incidence of 10 per 1000 patient years.

Bottom line: When I was on service recently, a two-month old baby had a seizure and an episode of apnea and asystole, which required resuscitation. I have been intrigued by the recent increased attention of SUDEP.

When I first trained, pediatric neurologists were aggressive about treating epilepsies. Dr. Sidney Carter, one of the founders of our field, was even aggressive with febrile seizures based on an experience he had in a patient with febrile seizures who recurred with status epilepticus. By the late 1980s, on the basis of new data about recurrence risk after a first seizure, certain common practices developed regarding withholding antiepileptic drugs after a first seizure based on risk of recurrence. By the 1990s many neurologists

were refraining from starting antiepileptic drugs in many situations. Now with an increased awareness of SUDEP, my hunch is that the pendulum will swing back to a more aggressive approach to treatment of seizures.

My baby with asystole certainly is worrisome, but in a baby one must consider gastroesophageal reflux as an etiology of asystole with apnea. The episode might have been an independent reflux episode or a seizure or both because reflux may be aggravated by a seizure.

In general, the mechanism of SUDEP is not understood, but studies define one of two possibilities including airway obstruction, cardiac abnormalities, or a mixed cause.

In this article, the presence of asystole suggests a very high incidence of near-SUDEP in the population of adults with intractable epilepsy, which is certainly a cautionary tale. Furthermore, PCCA and asystole is likely a high-risk event in patients with postictal apnea and asystole. As such, this might ultimately be a biomarker for SUDEP risk. Further studies are needed to clarify this issue in both the adult and child.

Reference

1. Sharma M, Kupferman J, Brosgol Y, et al. The effects of hypertension on the paediatric brain: a justifiable concern. *Lancet Neurol.* 2010;9:933–940.