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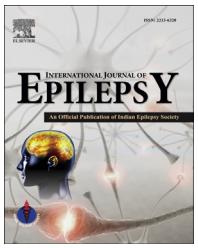

Answers

1. With refere..

   Answer: A = Zero

   Explanation/Comment: It is heartening to note that there seems to be a trend towards better knowledge, attitudes and practices about epilepsy in recent surveys.

   Ref: *M. Shaju et al, Knowledge, attitude and practice of parents regarding pediatric antiepileptic drug therapy, International Journal of Epilepsy, 1(2014)57–63.*

2. With..

   Answer: B = Alcohol Abuse

   Explanation: Alcohol use could be infrequent in the muslim population of Nigeria. In other developing countries too, the remaining alternatives cause are more fragment precipitating factor than alcohol abuse.

   Ref.: LF Owolabi et al, Status epilepticus in adults; A study from Nigeria, International Journal of Epilepsy 1(2014) 69–74.

3. While comparing..

   Answer: D = Abnormal neuro electrical activity

   Comment: The definition of neonatal seizures is purely clinical, hence entirely arbitrary and resulting in over as well as under diagnosis.

   Source: KP Vinayan, SL Moshe, Neonatal seizures and epilepsy, International Journal of Epilepsy 1(2014) 75–83.

4. Which of the following…

   Answer: A = Multifocal or fragmentary myoclonus

   Comment: Multifocal or 'fragmentary' myoclonus is characterized by brief asynchronous twitching of different muscle groups. Of the three types, fragmentary myoclonus is the type least commonly associated with EEG changes.

   Source: KP Vinayan, SL Moshe, Neonatal seizures and epilepsy, International Journal of Epilepsy 1(2014) 75–83.

5. Many factors contribute to..

   Answer: b = Low concentration of extracellular potassium in immature brain

   Explanation: Developmental imbalance between the maturation of excitatory and inhibitory circuits is supposed to be the most important factor for the increased seizures burden in the newborn. Immature brain has HIGH concentration of extracellular potassium, which may contribute to the increased excitability. The newborn brain is also vulnerable to a variety of insults like hypoxia and hypoglycemia, further increasing the chance of seizures.

   Source: KP Vinayan, SL Moshe, Neonatal seizures and epilepsy, International Journal of Epilepsy 1 (2014) 75–83.

6. Motor automatisms, …

   Answer: D = Sustained eye opening and fixation

   Explanation: Motor automatisms, previously called subtle seizures may usually occur in encephalopathic neonates. These are very difficult to characterize without simultaneous EEG. In preterm infants, the most common manifestation is sustained eye opening with unresponsiveness and eye fixation. In full term infants, horizontal sustained deviation of the eyes is usually seen.

   Slow roving eye movements and orofacial movements may be physiological and are very difficult to differentiate from subtle seizures. Jittery or tremulous movements are usually seen in metabolic and electrolyte anomalies and drug withdrawal. They are stimulus sensitive and may be modified by positioning. Hyperekplexia, a genetic disorder due to a mutation in glycine receptor gene, is characterized by stimulus sensitive myoclonus, muscle rigidity, episodes of tonic spasms and rare apnea.

   Source: KP Vinayan, SL Moshe, Neonatal seizures and epilepsy, International Journal of Epilepsy 1(2014) 75–83.

7. Bening Familial Neonatal Convulsions…

   Answer: A = Potassium Channel

   Explanation: Benign neonatal epilepsies are characterized by transient seizures and good neurodevelopmental outcome. There are two types; benign familial neonatal seizures and benign non familial neonatal seizures. Benign familial neonatal seizures otherwise called benign familial convulsions (BFNC) is an autosomal dominant potassium channelopathy affecting the KCNQ2 and KCNQ3 genes.

   Source: KP Vinayan, SL Moshe, Neonatal seizures and epilepsy, International Journal of Epilepsy 1(2014) 75–83.

8. Early Infantile Epileptic Encephalopathy (…

   Answer: D = Inborn errors of metabolism

   Explanation: Early Infantile Epileptic encephalopathy (EIEE, Ohtahara syndrome) and the early

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myoclonic epileptic encephalopathy (EMEE, Aicardi syndrome) are the two named epileptic encephalopathies with onset in the neonatal period. Both the syndromes are associated with resistant epilepsies and poor neuro-developmental outcome along with suppression burst pattern in the EEG. Ohtahara syndrome is usually seen with severe structural malformations of the brain and the main seizures type is tonic spasms from the beginning. On the other hand, EMEE is usually associated with inborn errors of metabolism. The interburst interval is longer in EIEE compared to EMEE. Some times, the burst suppression pattern is in EMEE may not be appreciated at disease onset, and follow up EEGs may be necessary to make the diagnosis. Seizures are typically refractory to currently available anticonvulsant medications. Outcome is uniformly poor for both the syndromes with a high rate of mortality in early infancy. Surviving children may develop hypsarrhythmia at around 3–6 months with classical features of west syndrome.

Source: KP Vinayan, SL Moshe, Neonatal seizures and epilepsy, International Journal of Epilepsy 1(2014) 75–83.

9. All True statement about Levetiracetam, …

Answer: C = Mostly metabolized in liver, with minimal renal elimination

Explanation: Levetiracetam (LEV) is a novel antiepileptic drug having a broad spectrum antiseizures activity in both generalized as well as focal onset seizures. It is a relatively well-tolerated antiepileptic drug (AED) in both adults and children. It is a new AED with renal elimination and no hepatic metabolism.

Source: R. Mahale et al, Possible Levetiracetam induced encephalopathy presenting as electrical status epilepticus: An unknown occurrence, International Journal of Epilepsy I(2014) 84–89.

10. Two types of GM2 Gangliosidosis (Tay-Sachs and…

Answer: A = Organomegaly

Explanation: Differentiation of the two subtypes (Tay-Sachs and Sandhoff's) of GM2 gangliosidosis is biochemically not possible if the facility for serum total hexosaminidase levels testing are not available. In GM2 gangliosidosis the radiological changes are described in three phases. Initially there is enlargement with abnormal signal changes of basal ganglia (particularly caudate) followed by features of hypomyelination with diffuse cerebral atrophy in the terminal stages.

Clinically patients with classic GM2 gangliosidosis present with infantile onset psychomotor regression, hyperacusis, macular cherry red spot, central hypotonia and blindness. As the disease progresses megalencephaly and generalized seizures are seen. Sandhoff's disease in addition exhibits hepatosplenomegaly and cardiomyopathy.

Source: R. Dubey et al, A child with global neuroregression with refractory epilepsy, International Journal of Epilepsy 1(2014) 92–93.

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