CLINICO-ETIOLOGICAL PATTERN OF NEONATAL SEIZURES

By

Hassan Ali Hassan*, Ahmed Ismaieel*, Eslam Elsherbeiny Qotb*

Pediatric department*, Faculty of Medicine, Al-Azhar University

ABSTRACT

Background: *Incidence of seizures is highest during the neonatal period, and it is considered a medical emergency that necessitate proper clinical and etiological assessment.*

Aim: *This study aimed to focus on the etiological, clinical classification of neonatal seizures and proper assessment for appropriate diagnosis.*

Patients and methods: *This prospective study was performed on 50 neonatal patients who developed neonatal seizures either prior or after admission to the NICU of Al-Hussein University Hospital and Sayed Galal University Hospital below the age of 28 days after an informed written consent from parents in the period between December 2018 and September 2019*

Results: *In this study, neonatal seizures were more common in males 27 (54%) than females 23(46%); their mean birth weight was 2748 grams. 40% of patients of the study were complaining of perinatal asphyxia evidenced by history of prolonged or instrumental labor, 11 babies (22%) with sepsis,6 babies (12%) with hypocalcemia, 6 babies (12%)with intraventricular hemorrhage, 3 babies (6%)with intracranial hemorrhage, 3 babies(6%) with kernicterus, one baby (2%) with metabolic disorder (Non ketotic hyperglycinemia) .*

Many types of seizures are represented in our study with different ratios; out of which 18 (36%) had myoclonic seizures, 14(28%) had mixed, 8 (16%) babies had subtle, 4(8%) had clonic and 5(10%) had tonic types), and only 1(2%) baby had multifocal clonic fit.

Conclusion: *This study confirms that Hypoxic ischemic encephalopathy was most important etiology of neonatal seizures and most important risk factor for poor neurological outcome followed by sepsis, intraventricular hemorrhage, intracranial hemorrhage, kernicterus and metabolic respectively and most important risk factor for poor neurological outcome and myoclonic seizures being most common clinical type of seizure observed.*

Key words: *Neonatal seizures-etiology-clinical-Neonates.*

INTRODUCTION

Neonatal Seizures (NS) are the most frequent and distinctive clinical manifestation of neurological dysfunction in the newborn. Infants with neonatal seizures are at a high risk of neonatal death or neurological impairment/epilepsy disorders in later life **(Silverstein et al., 2007)**.

Though mortality due to etiology of NS has decreased from 40% to about 20% over the last years, the prevalence of long-term neurodevelopment sequelae has largely remained unchanged at around 30%. Improper and inadequate management of seizures could be one of the major reasons behind this phenomenon **(Tekgul et al., 2010)**.

The National Neonatal Perinatal Database (NNDP; 2002-03), which collected data from 18 tertiary care units across the country, has reported an incidence of 10.3 per 1000 live-births. The incidence was found to increase with decreasing gestation and birth weight- for example, preterm infants had almost twice the incidence when compared to term neonates (20.8 vs. 8.4 per 1000 live births) while very low birth weight infants had more than 4-fold higher incidence (36.1 per 1000 live-births) (Accessed Jan 8, 2012).

The decreased seizure threshold in the newborn is due to transient overdevelopment of excitatory systems compared to inhibitory systems **(Tekgul et al., 2006)**.

Broadly speaking, neonatal seizures diagnosis can be categorized into: Etiological, Clinical diagnosis. The etiologies of neonatal seizures ordered according to their incidence are [Cerebral hypoxia-ischemia (55%), Intracranial hemorrhage (15%), Metabolic diseases (6%), Central nervous system infection (5%), Cerebral dysgenesis (5%), Neonatal epileptic syndromes (1%), Neonatal abstinence syndrome (1%), and Unknown cause (10%)] **(Tekgul et al., 2006)**.

The clinical neonatal seizures types can be classified into four groups: Subtle seizures, clonic seizures, Tonic seizures, and Myoclonic seizures **(Du Plessis et al., 2008)**.

Cranial ultra-sonography (CUS) is frequently used for the diagnosis of major cerebral lesions (MCL) during the early neonatal period **(Vollmer et al., 2006)**.

Although (CUS) is a useful and minimally invasive, a study on 42 preterm neonates suffering from

neonatal seizures was performed at Essen University Hospitals showed that (MCL) appeared in 28.5% of cases <7 days of life & in 71.5% of cases >7days of life, so several days or few weeks are required for the brain insult to become evident on (CUS) **(Horsch et al., 2006)**.

It is critical to recognize neonatal seizures, to determine their etiology, and to treat them for three major reasons. First, the seizures are usually related to significant illness, sometimes requiring specific therapy. Second, neonatal seizures may interfere with important supportive measures, such as alimentation and assisted respiration for other associated disorders frequently present in neonates. Third, experimental data give reason for concern that the seizures per se may be a cause of brain injury and mental retardation and/or cognitive delay **(Nunez, 2008)**.

Aim of the Work

The aim of this work is to focus on the etiological, clinical classification of neonatal seizures and proper assessment for appropriate diagnosis.

Ethical consideration:

- Written Parent consent for the study was obtained before the study.
- Approval of the local ethical committee in the pediatrics department, college and university were obtained before the study.
- The authors declared no potential conflict of interest with respect to the research & publication of this article.
- All the data of the patient & results of the study are confidential & the patient has the right to keep it.
- The authors received no financial support for the research & publications of the article.

PATIENTS AND MATERIALS

This prospective study was performed on 50 neonatal patients who developed neonatal seizures either prior or after admission to the NICU of Al-Hussein University Hospital and Sayed Galal University Hospital below the age of 28 days after an informed written consent from parents in the period between December 2018 and September 2019.

Inclusion Criteria:

- Any full-term>37wk or pre-term neonate manifested by more than one attack of seizure.

Exclusion Criteria:

- Any baby above 28 days of age.

• Conditions that mimic neonatal seizures such as jitteriness, Benign Neonatal Sleep Myoclonus.

Methods:

All babies were subjected to:

1. Full maternal history:

Age, acute or chronic medical problems as (diabetes mellitus, hypertension, thyroid disease, renal disease, urinary tract diseases, heart and lung disease, and blood disease), and medications.

2. Neonatal history:

Gestational age, sex, birth weight, mode of delivery, full anthropometric measurement, prenatal risk factors, assessment of seizure type, duration, age of onset & frequency, neonatal illness, oxygen supply, nutrition, and medications.

3. Family history:

Asking about consanguinity, other sibling with neonatal seizure and similar condition, history of abortion or previous death.

4. Clinical Evaluation:

A. General examination:

a. Apgar score:

b. Vital signs: Heart rate, respiratory rate, temperature and capillary perfusion time.

c- Gestational age assessment: By Ballard scoring system:

1. Physical maturity.

2. Neuromuscular-maturity.

B. Local examination:

Head and neck, limbs, skin, back, genitalia, birth injuries.

C. Systemic examination:

a. Cardiac examination: Central cyanosis, murmur, tachycardia or bradycardia.

b. Chest examination: Chest movement, cyanosis, air entry, and auscultatory finding.

c. Abdominal examination: distension, liver and spleen enlargement.

d. Neurological examination:

Standardized neurologic examination was performed for all neonates within 24h after birth. Serial neurological examinations were done later during their period of stay using Sarnat & Sarnat Staging for diagnosing Hypoxic ischemic encephalopathy including; level of consciousness, muscle tone, neonatal reflexes and presence of seizures (type &duration).

5. Laboratory investigation:

- First line investigations was done in all neonates included blood glucose, serum

electrolytes (calcium, sodium, magnesium, potassium and phosphorus), complete blood count, c-reactive protein, arterial blood gases, CSF for any evidence of infection.

- Second line investigations e.g. blood culture, serum urea/creatinine, and liver function tests, ammonia, lactate, also organic acid profile &ketones in urine, Tandem mass spectrometry were done in selected neonates as guided by history, physical examination and essential investigations suspected to have metabolic disorder.

6. Cranial ultrasound (CUS):

At least one imaging examination during the neonatal period was performed using a standardized protocol for screening of major cranial U.S lesions was performed through the anterior fontanel to obtain standard Coronal, Sagittal, Para-sagittal images using a portable sectorial ultrasound apparatus.

Statistical Methods:

Statistical analyses in this study were described in terms of: frequencies (number of cases), percentage, mean and standard deviation.

- Comparison of quantitative variables between the study groups was done using:

  1. Chi-square test: for comparing categorical data.

  2. Statistical significance: is achieved when the probability value (P-value) is less than or equal (<=0.001).

All statistical calculations were done using computer programs Microsoft Excel 2003 (Microsoft Corporation.NY.USA) and SPSS (Statistical Package for Social Science; SPSS Inc. Chicago, II, USA) version 15 for Microsoft windows.

RESULTS

Table (1): Demographic characteristics of neonates with seizures in this study

Variables		Count	Column N %
Sex	F	23	46 %
	M	27	54%
Weight	<= 2500 g	20	40.0%
	> 2500g	30	60.0%
GA	FT (37-40w)	30	60.0%
	POST (>40w)	6	12 %
	PT (<or=35w)	14	28 %
Mode of delivery	NVD	25	50%
	CS	25	50%
Consanguinity	No	39	78 %
	Yes	11	22%

This table shows that: neonatal seizures were more common in males (M: F 1:1.17) with no consanguinity (78%).

Table (2): Neurological finding of All Patients Included in the Study

Variables		No	%
Seizure type	focal clonic	4	8 %
	Multifocal clonic	1	2 %
	Mixed	14	28%
	Myoclonic	18	36 %
	Subtle	8	16 %
	Tonic	4	8 %
Seizure duration	< 3 minutes	35	70%
	>=3 minutes	15	30%
Seizure Frequency	<5 times/day	29	58 %
	>= 5 times/day	21	42%
Neonatal Reflexes	Weak	9	18 %
	Absent	16	32 %
Deep Reflexes	Hyperreflexia	8	16 %
	Hyporeflexia	35	70 %
	Normal	7	14 %
Muscle Tone	Hypertonia	18	36 %
	Hypotonia	28	56 %
	Normotonia	4	8 %
Level of consciousness	Coma	2	4 %
	Conscious	13	26 %
	Disturbed conscious level	11	22 %
	Lethargy	24	48 %

This table shows that 35 cases (70%) with hyporeflexia, with hypotonia 28 cases (56%), Duration of seizures mostly <3 minutes in 35 cases (70%) and

Frequency of seizures mostly <5 times/day in 29 cases (58 %).

Table (3): laboratory finding in studied cases

Variables		Count	%
Metabolic acidosis	Uncompensated	17	34%
	Compensated	20	40%
	Normal	13	26%
Serum Calcium	Low (<7mg/dl)	6	12%
	Normal	44	88%
Serum Magnesium	Low(<1.7mg/dl)	0	0.0%
	Normal	50	100%
Serum Sodium	High(>145mg/dl)	13	26%
	Low(<135mg/dl)	8	16%
	Normal	29	58%
Serum Creatinine	High(>1mg/dl)	16	32%
	Low(<0.3mg/dl)	5	10%
	Normal	29	58%
CRP (C-reactive protein)	Negative(<6mg/dl)	39	78%
	Positive(>6mg/dl)	11	22%

This table shows that: 6 cases (12%) with hypocalcaemia, 11 cases (22%) with positive CRP.

Table (4): Etiology of seizures in the neonates included in the study

Etiology	No. of neonate	%
Hypoxic ischemic encephalopathy	20	40%
Sepsis	10	20%
Hypocalcemia	6	12%
Intraventricular hemorrhage	6	12%
Intracranial hemorrhage	3	6%
Kernicterus	3	6%
Metabolic	1	2%
Meningitis	1	2%

This table shows: Hypoxic-ischemic encephalopathy was the commonest cause of seizures (40%) followed by sepsis (20%).

Table (5): Clinical presentation of seizures in the neonates included in the study

Clinical	No. of neonate	%

Myoclonic	18	36
Mixed	14	28
Subtle	8	16
Focal clonic	4	8
Tonic	5	10
Multifocal clonic	1	2

This table shows: myoclonic seizures being the most common clinical type of seizure (36%) followed by mixed type (28%).

Table (6): Relation between seizure types of studied cases & their outcome

Outcome / Seizure Type	Death		Improved		P- value
	Count.	%	Count.	%	
Myoclonic	10	55.5%	8	44.5%	0.02
Mixed	4	28.6%	10	71.4%	
Subtle	1	12.5%	7	87.5%	
Focal clonic	0	0.0%	4	100.0%	
Tonic	4	80%	1	20%	
Multifocal clonic	1	100.0%	0	0.0%	

This table shows that tonic and myoclonic seizures were associated with poor outcome (death in ratios 80%, 55.5% respectively) whereas most of cases with subtle, mixed types were improved (87.5%), (71.4%) respectively.

Table (7): Seizure's Etiology as regard to outcome

Outcome / Etiology	Death		improved		P-value
	Count.	%	Count.	%	
HIE	10	50.0%	10	50.0%	0.002
Hypocalcemia	0	0.0%	6	100.0%	
ICH	1	33.3%	2	66.7%	
IVH	6	100.0%	0	0.0%	
Kernicterus	0	0.0%	3	100.0%	
Meningitis	1	100.0%	0	0.0%	
Metabolic	1	100.0%	0	0.0%	
Sepsis	1	10.0%	9	90.0%	

This table shows that the highest mortality was due to intraventricular hemorrhage (100%), followed by HIE (50%) while complete recovery were (100%) in hypocalcemia and kernicterus but in sepsis recovery occurred in (90%).

Table (8): Relation between Cranial sonar findings & seizure's etiology

Etiology	Cranial ultrasound				P-value
	Normal (15)	%	Abnormal (35)	%	
Hypoxic ischemic Encephalopathy (HIE)	0	0	20	40%	0.000
Sepsis	9	18%	1	2%	
Intracranial Hemorrhage (ICH)	0	0	3	6%	
Intraventricular Hemorrhage (IVH)	0	0	6	12%	
Hypocalcemia	6	12%	0	0	
Meningitis	0	0	1	2%	
Kernicterus	0	0%	3	6%	
Metabolic disorder	0	0	1	2%	

This table shows that: all cases with HIE, ICH, IVH, meningitis, kernicterus has abnormal cranial ultrasound findings.

DISCUSSION

Seizures in the neonatal period are usually concomitants of serious neurological disease. The convulsive phenomena take certain distinctive and often subtle forms because of the status of the neuroanatomical and neurophysiologic development of the neonatal brain **(Zupanac et al., 2013)**.

Diagnosis of neonatal seizures was based on clinical observation, etiological identification and correct description of seizure type according to Volpe's classification **(Nunes et al., 2009)**.

Neonatal seizures are easy to be detected, diagnosed, and managed. Management of seizures involves identifying and treating the underlying etiology of the seizure and appropriate use of pharmacologic interventions **(Khan et al., 2010)**.

It has been widely known that the golden standard method to recognize neonatal seizures is video-EEG. However, this equipment is not always available in many neonatal units around the world **(Nagarajan et al., 2012)**.

The aim of this study is to focus on the etiological, clinical classification of neonatal

"convulsions" thus proper assessment for appropriate diagnosis and proper management.

In this study (as shown in table 1) majority of neonates with seizures were full-term (60%), while (28%) babies were preterm and (12%) babies were post-term, similar observation was seen in study by Das et al (2016) where term babies were (91.3%), pre-term were (7.8%) and post-term were (0.9%).

Regarding to the etiology of neonatal seizures;(as shown in table 2):

The most common cause of neonatal seizures encountered in our study was HIE (40%) mainly during the first 24 hours. Most of HIE events occurred in full term infants. And this agreed with **(Volpe J, 2001)** who stated that risk of seizures is similar between full term and pre term neonates.

This finding compares with Loman **(AM et al., 2014)** who also found HIE (53.9%) in their study as the earliest common cause of neonatal seizures, and similar results were documented by, **Khan et al (2008)** who found that the majority of his cases, suffered from HIE.

Also **Levene M et al (2013)** reported that HIE as etiology of seizures was seen in 45.7% of cases in the first 24 hours with a higher incidence rate in comparable with the results of our study.

Neonatal sepsis constituted an important etiology of neonatal seizures which is the second most common etiology in the current study accounting for (20%) of the cases, and this agreed with Aziz A et al (2015) with higher incidence (22%).

A study conducted by **Baral D et al (2006)** reported that out of 90 babies with neonatal seizures, 10 (11%) developed septicemia and 10 (11%) had meningitis, which is comparable with the present study in which out of 50 babies, 10 (20%) developed septicemia and 1 (2%) neonate only had meningitis. On contrary, in a study conducted by **Das et al (2016)** 24 (21%) cases had meningitis and **Rabindran et al (2015)** also reported meningitis as a cause of neonatal seizures in (17.2%)

Intra-cranial bleeding (including intracranial hemorrhage &intraventricular hemorrhage) was diagnosed as the etiology of neonatal seizures in 9 cases (18%) of our study. And this agreed with **Mizrahi (2001)** who found intra-cranial hemorrhage as a frequent cause of neonatal seizures accounting for (15-25%) of cases in his study. While **Aziz et al**

(2015), **Anand et al (2014)** & **Kumar et al (2016)** noted that the incidence of ICH was (13%), (7.4%) and (2%) respectively which lower incidence than in our study are.

In the present study, hypocalcaemia was responsible for (12%) of cases of neonatal seizures as the only metabolic disturbance etiology. Similarly, **Bushra A et al (2005)** reported that (12.5%) of the neonates had metabolic disturbances. Also in **Talebian et al (2014)** study, (8%) of cases had hypocalcemia. In contrary **Pressler RM (2005)** stated that hypocalcaemia and hypomagnesaemia as the etiology of (4-22%) of cases. Also **Sood A et al (2003)** reported metabolic disturbances in (49%) of cases with seizures, most common being hypocalcaemia followed by hypoglycemia.

Cases due to Kernicterus were mostly presented after 48 hours of age, which constituted (6%) of all cases. Similar results (2%) & (3%) were found by **Anand et al (2014)** and **Sarker SA et al (2014)** respectively, and this was compared with **Rushda (2007)** who found Kernicterus presented after 72 hours of age constituting (34.7%) in his study.

Furthermore, Metabolic disorders were responsible for (2%) of cases which is lower incidence compared to a study conducted by **Kumar et al (2016)** where 13 (11%) cases had metabolic disorders.

Regarding to the clinical presentation of neonatal seizures; (as shown in table 5).

Many types of seizures are represented in our study with different ratios; out of which 18 (36%) had myoclonic seizures, 14 (28%) had mixed, 8 (16%) babies had subtle, 4 (8%) had clonic and 5 (10%) had tonic types), and only 1 (2%) baby had multifocal clonic fit. In comparable to these results, Noreen **Faiz et al (2007)** studied 101 babies, out of which 20 (19.8%) had subtle seizures, 1 (0.9%) neonate had myoclonic, both tonic and clonic fits were there in 40 (39.6%) cases each. In another study conducted by **Das et al (2016)**, 49 (43%) cases had subtle seizures, followed by tonic in (34%), and clonic seizures were present in (16%).

The type of seizures and its prognostic value remain a challenge for the clinician; **Pisani F et al (2009)** reported that subtle seizures have a worse outcome compared with those with clonic seizures. In our study (as shown in table 6) only 16% of seizures were subtle and most neonates had

myoclonic seizures, which was comparable with this study.

The incidence of mortality (as shown in table 7) was 20 cases (40%) out of 50 cases, of which 10 cases (20%) died of hypoxic ischemic encephalopathy, 6 cases (12%) died of intra-ventricular hemorrhage. These findings agree with the study done by **Tekgul et al., (2006)** who reported that mortality due to HIE was 7%, in contrary to other studies; our results was higher in comparable with mortality in a study reported by **Sandhu Ravneet et al (2003)** 11.25% and 9% respectively. This higher rate could be due to the etiology of the seizures.

CONCLUSION

In our prospective study on infants with neonatal seizures we found that:

- GA, birth weight, gender, route of delivery didn't distinguish which infant with neonatal seizures would have a poor prognosis.
- Early control of neonatal seizures helps to improve the outcome, while prolonged time of cessation of seizures carry poor prognosis.
- Hypoxic ischemic encephalopathy was the major cause of neonatal seizures.
- The myoclonic seizures carry the worst prognosis. Also, it is the commonest seizure type in our study.

RECOMMENDATION

- Careful pre-natal follow up to pregnant women for early detection of risk factors such as maternal illness, fetal distress, maternal infection, and the antecedent events to prevent neonatal seizures.
- Mandatory availability of well experienced residents in neonatal resuscitation program (N.R.P) with appropriate equipment for successful resuscitation to prevent HIE.
- Neurological follow-up to all cases of neonatal seizures should be meticulous to reassure and early pick up the cases with neurological sequelae for rehabilitation and multidisciplinary management.

REFERENCES

1. **Anandeveena, P M C Nair (2014):** Neonatal seizures: Predictors of adverse outcome. J PediatrNeurosci. 2014 May-Aug; 9(2): 97–99.
2. **Aziz A, Gatto I, Aziz M, Rasool G. Clinical and etiological profile of neonatal seizures (2003):** a tertiary care hospital based study. Int J Res Med Sci. 2015,3:2198-2203.

3. **Bushra AM, Butt MA, Shamoon M, Tehseen Z, Fathima A, Hashmat N. (2005):** Seizures etiology in the newborn period. J Coll Physicians Surg Pak; 15(12):786-90, 2005.
4. **Chapman KE, Mizrahi EM, Clancy RR (2011):** Neonatal seizures. In: Wyllie's Treatment of Epilepsy, 2011.
5. **Dinesh Das, Sanjib Kumar Debbarma. (2016):** A study on clinico-biochemical profile of neonatal seizure. J Neurol Res. 2016; 6 (5-6):95-101.
6. **Du Plessis AJ (2008):** "neonatal seizures" in manual of neonatal care, 27(A):483-498, 2008
7. **Horsch S and Muentjes C (2006):** Pediatric Department, Essen University Hospital, Germany, 2006.
8. **Khan RL, Nunes ML, Fernando L, DaSilva G and DaCosta JC (2008):** "Predictive value of sequential electroencephalogram in neonates with seizures and its relation to neurological outcomes"; J.Child. Neurol. Vol. 23, 144, 2008.
9. **Levene M. (2013):** The clinical conundrum of neonatal seizures. Arch Dis Child 2013; 8675–77.
10. **Loman AM, Ter Horst HJ, Lambrechtsen FA, Lunsing RJ. Neonatal seizures (2014):** etiology by means of a standardized work-up. Eur J Paediatr Neurol. 2014; 18 (3): 360 - 7.
11. **McIntire DD, et al. (2005):** Birth weight in relation to morbidity and mortality among newborn infants. New England Journal of Medicine. 2005; 340:1234–8.
12. **Mills PB, Camuzeaux SS, Footitt EJ, Mills KA, Gissen P, Fisher L, Das KB, Varadkar SM (2014):** Epilepsy due to PNPO mutations: genotype, environment and treatment affect presentation and outcome.Brain disorder ;137(Pt 5):1350-60. doi: Epub 2014 Mar 18. 2014.
13. **Nagarajan L, Palumbo L, Ghosh S (2012):** Classification of clinical seminology in epileptic seizures in neonates. Eur J PediatrNeurol; 16:118-25, 2012.
14. **Nunez P, Srinivasan R: Electric fields of the brain (2009):** The Neurophysics of EEG, 2nded. New York: Oxford University Press,2009.
15. **Rabindran, HemantParakh, Ramesh JK, Prashant Reddy (2012):** Phenobarbitone for the Management of Neonatal Seizures - A Single Center Study.Int J Med Res Rev. 2015; 3(1):63-71.Rahman S (2012): Mitochondrial disease and epilepsy. Dev Med Child Neurol 54: 397-406
16. **Silverstein FS, Jensen FE.**

Neonatal seizures (2007): Ann Neurol. Aug 2007; 62(2): 112-20.

17. **Talebian, Mohammad JAHANGIR, MahinRabiee: Tekgul H, Gauvreau K, Soul JS (2006):** "the current etiologic profile and neurodevelopmental outcome of seizures in term newborn infants". Pediatrics; 117(4): 1270-1280, 2006.The Etiology and Clinical Evaluations of Neonatal Seizures,Iranian journal of child neurology 2014.
18. **Vollmer B and Roth S (2006):** "Neurodevelopmental outcome of preterm infants". Dev. Med. Child Neural, 48: 348-352; 2006.
19. **Volpe JJ (2008):** "Neonatal seizures". In: Neurology of the newborn, 6th ed., Philadelphia, Saunders, 178-214, 2008.
20. **Zupanac M.L.:** Neonatal seizures. PediatrClin N Am 51. 961-978.

الملخص العربي

تعرف التشنجات بانها النشاط الغير طبيعى بالخلايا العصبيه نتيجة النشاط الزائد للطبقه المخيه القشريه كما انها تعرف بالتغير فى وظائف المخ (السلوكيه والحركيه والمحرك اللارادى) وتعتبر نسبة حدوث التشنجات اعلى فى الاطفال حديثى الولاده عن اى فئه عمريه اخر.

وهناك اسباب عديده لحدوث التشنجات لدى هؤلاء الاطفال بانماط مختلفه وهذا يستلزم الدقه فى اختيار العلاج المناسب لوقف هذه التشنجات كما انه يوجد مسببات متعدده تكثر فى الاطفال حديثى الولاده لذا كان من الضرورى استبعاد بعض الاسباب التى من السهل علاجها مثل نقص نسبة السكر بالدم نقص نسبة الكالسيوم والماغنسيوم بالدم قبل بدء العلاج الخاص لوقف هذه التشنجات.

ان اكثر الاسباب شيوعا لتشنجات الاطفال حديثى الولاده هى الاختناق الوليدى نزيف داخلى بالمخ والتسمم الوليدى لكن الاسباب التى تنتج عن اختلال فى الايض اقل شيوعا.

وهناك انواع كثيره من التشنجات لكن الرمع العضلى هو النوع الاكثرشيوعا ثم ياتى بعد ذلك النوع المختلط يليه التشنجات الدقيقه.

يعتبر رسم المخ الكهربائى من الطرق المستقبليه فله القدره على معرفة الوليد الاكثر عرضه للاصابه الدماغيه وكذلك القدره على معرفة نشاط نوبة التشنج عند حدوثها كما ان رسم المخ الكهربائى المستمر قادر على تسجيل نشاط النوبات الغير ظاهره.