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EEG and SPECT Changes in Acute Ischemic Stroke

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Abstract

Acute ischemic stroke is one of the prominent roots of mortality and morbidity all over the world. Core ischemic regions, penumbral regions and extra penumbral regions occur proximal or distal to arterial occlusion where the margins of ischemia are hyperemic with either one, minimal or no parenchymal damage. Electroencephalography (EEG) and single photon emission computed tomography (SPECT) remains the investigative practices that let economical, noninvasive learning of physiological and pathological actions in the human brain in acute ischemic stroke. Mutually these procedures may detect different patterns resonant of severity, prognosis, and secondary injury allied to acute ischemic stroke. Also, these readings can be intensely linked to cerebral metabolism which is sensitive to ischemia. This review summarizes the EEG and SPECT changes and their limitations in monitoring patients with acute ischemic stroke patients.

Keywords: Acute ischemic stroke; Electroencephalography; Single photon emission computed tomography; Cerebral blood flow

Introduction

Acute cerebral ischemia is one of the leading causes of mortality and morbidity with age-adjusted incidence rate accounting for around 200 cases per 100,000 population/years [1]. Ischemic stroke may manifest in the form of thrombotic stroke, embolic stroke, systemic hypoperfusion or venous thrombosis [2]. If hemodynamic instability is severe and prolonged, it may produce focal as well as diffuse cerebral changes where the effect of ischemia whether reversible or irreversible, is dependent on the degree and duration of blood flow. In stroke, paradoxical increment of cerebral blood flow (CBF) can be witnessed at the involved site and is referred to as luxury perfusion [3]. Core ischemic regions (blood flow<15%), penumbral regions (blood flow<40%) and extrapenumbral cortical regions (blood flow rate>40%) can result both proximal and distal to arterial occlusion. This approximate flow based definition of core and of penumbra as well as extrapenumbral region is generally agreed upon but still ample argument exists [4]. Ischemic penumbra generally arises in the periphery of the brain when blood flow is significantly reduced to cause hypoxia, but not severe enough to cause irreversible failure of energy metabolism and cellular necrosis [5].

The margins of ischemia are hyperemic, either there is minimal or no parenchyma damage. This can be identified prior by EEG and SPECT than any other structural imaging modalities and thus with timely supervision and management may help reverse the condition. Currently there are many means available for clinicians to detect an ischemic stroke. However, EEG and SPECT are among the excellent options especially for hospitals that cannot afford expensive instruments as both these investigative techniques are economical and noninvasive functional studies of the brain that are sturdily linked to cerebral metabolism and ultimately lead to cerebral ischemia [6,7].

Hossmann summarized the results from different studies in different species, and expanded the threshold concept of pathophysiological changes in cerebral ischemia. The inhibition in protein synthesis begins to deteriorate of the CBF below 55ml/100g/min. When CBF drops to below 35ml/100g/min, there is an increased glucose utilization and lactate accumulation. As CBF further declines below 26ml/100g/min, the resulting acidosis leads to decline in phosphocreatine and adenosine triphosphate levels. At CBF around 23 ml/100g/min, neurological dysfunction and suppression of electrical activity in the brain and evoked potentials appear. With further deterioration of CBF to 5-18ml/100g/min, irreversible hemiparesis and infarction occurs with terminal depolarization, potassium efflux and calcium influx [8].

EEG in Cerebral ischemia

The EEG is the recording of cerebral electrical activity along scalp due to the firing of neurons within the cerebral cortex. The potentials recorded by EEG are cumulated excitatory and inhibitory postsynaptic potentials in neuronal dendrites, usually in most superficial regions of cerebral cortex. Also, deeper structures like the thalamus and brainstem create potentials of low amplitude reflecting the functional status of these brain structures. Electrographic activity contains spectrum of frequencies, recording physiological as well as pathological changes in cerebral function with delta (<3.5Hz), theta (4-7Hz), alpha (8-13Hz) and beta (>14Hz). Cerebral function represented on EEG show slower frequencies (especially delta and theta), produced by the thalamus and by cells in layers II-VI of cortex while faster frequencies (especially alpha) from cells in layers III and V of cortex [9].

For stroke patients, EEG presents repeatable changes as CBF falls off from normal to low [10]. Abnormal EEG changes in cerebral ischemia can be categorized into four types depending on the failure of cerebral blood flow. At CBF 25-35 ml/100g/min, EEG may show decrease in amplitude of faster frequencies. As CBF shrinks to 18-25 ml/100g/min, slowing of EEG changes to theta frequencies may be visualized. Marked suppression of frequencies may appear with a further drop of CBF in 12-18 ml/100g/min (Figure 1). Finally suppression of all frequencies is seen to drop of CBF in <8-10 ml/100g/min (Table 1) [11,12].

EEG is budgeted, noninvasive and convenient technique to

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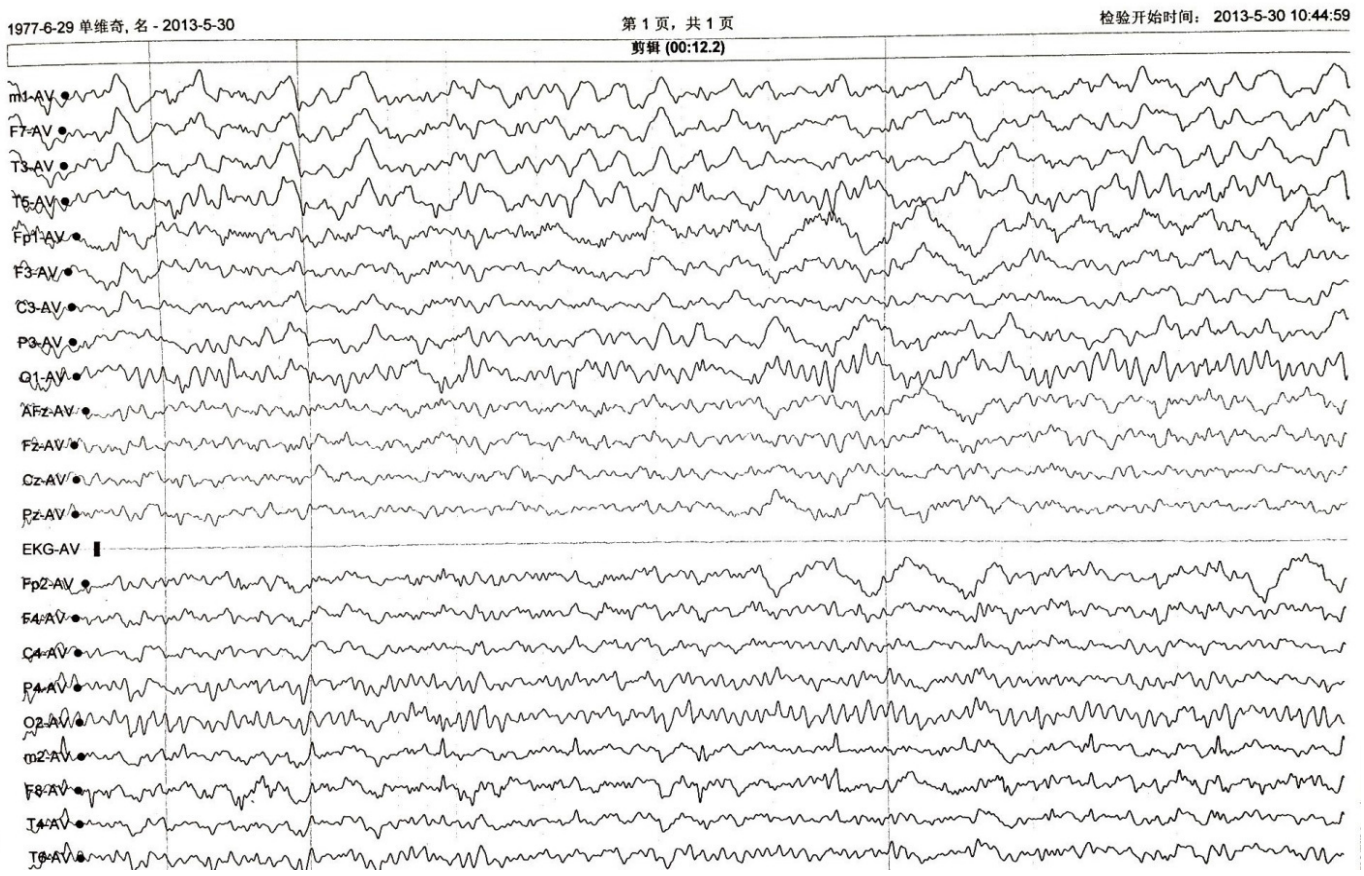

Figure 1: EEG in a 63 years old male patient with acute ischemic stroke shows low to middle amplitude slow waves visualized in the left hemisphere and are more obvious in M1, F7, T3, T5 leads.

Grade	CBF (ml/100g/min)	EEG changes
0	35-70	Normal
I	25-35	Loss in faster beta frequencies
II	18-25	Slowing of background to 5-7 Hz (theta)
III	12-18	Slowing to 1-4 Hz (delta)
IV	<8-10	Suppression of all frequencies with neuronal death (Cortical suppression)

Table 1: Morphologic and frequency changes in EEG correlating with reductions in cerebral blood flow (CBF) (modified from Composite data from Sharbrough et al. 1973, Ingvar et al. 1976, Astrup et al. 1981, Nagata et al. 1989, Jordan and Stringer 1991, Anita L Schneider et al. 2005).

assess the cerebral activity with reasonable spatial and high temporal resolution strongly linked to cerebral metabolism, sensitive to ischemia and finest existing method for detecting epileptic activity [13]. The EEG is the utmost delicate diagnostic tool for detecting acute cerebral ischemia and correlates well with its location and degrees. Intraoperative EEG monitoring and animal model readings have shown that EEG changes occur within 5 minutes of acute cerebral ischemia, better than current imaging techniques and clinical examination if a patient is sleeping, sedated, paralyzed or has altered level of consciousness [11]. EEG provides functional state of the brain as synchrony of electrophysiological events occur in close proximity to the electrodes before computed tomography (CT) or magnetic resonance imaging (MRI) can detect [6]. EEG may be able to detect patterns suggestive of severity, prognosis, and secondary injury related to acute ischemic stroke [13]. Electrodes placed directly on the surface or in the depth of the brain increases the spatial resolution exterminating distorting features of electrical conductance [14]. EEG may be helpful in determining prognosis of spontaneous neurological improvement, early neurological deterioration and death in the acute setting of presumed ischemic origin [15].

Numerous limitations have to be considered when evaluating EEG signals. Both raw EEG and quantitative EEG (qEEG) are sensitive to states ranging from stress, alertness to rest, hypnosis and sleep. In addition, different variables including biochemical, metabolic, circulatory, hormonal, neuroelectrical and behavioral function have variable effects on the EEG patterns [16]. Murri et al. found EEG to be highly sensitive for fronto-central, temporal and parieto-occipital cortical-subcortical infarctions than for lesions in basal ganglia and internal capsule [17]. When EEG does not correlate well, it tends to localize too laterally or miss deep lesions signifying that it is not uniformly sensitive or accurate than others. They may supplement with other imaging modalities in the diagnosis and prognosis of ischemia [18]. Involvement of small areas of tissue of the brain is associated with greater attenuation of activity while bustle arising from cortex

within the walls or depths of sulci may not be recorded in the EEG [19]. Jordon KG conveyed that cortical infarctions<3 cm may not be able to produce EEG abnormalities. Medial occipital lesions may produce bilateral EEG changes and not focal changes [20]. Florence et al. found that EEG could not distinguish a CBF decrease due to hemodynamic disturbances or embolism [21].

Continuous EEG (cEEG) is being increasingly practiced in neurological ICU and carotid surgery to monitor functional status of the brain including non-convulsive seizures, outcome prediction, and sedation level. It is also used to detect ischemia and secondary brain insult [12,20]. cEEG monitoring may be constructive in stroke patients as it may detect changes in brain function in a possible reversible state, allowing for early intervention [22].

qEEG as compared to conventional EEG has established an improved detection and localization of pathophysiology of brain ischemia [23]. Like the raw EEG, qEEG is proficient of demonstrating changes in blood flow and metabolism in the early stage of cerebral ischemia. Clinically, qEEG correlates more with severity, radiographic findings, and response to treatment in stroke patients. qEEG may be more sensitive to subtle changes and some parameters may even detect improvement earlier than in the clinical exam. qEEG may also provide a method of determining short-term and long-term prognosis and may improve the predictive value of acute ischemic stroke [13].

Regional attenuation without delta (RAWOD) is a distinctive pattern seen in acute ischemic stroke characterized by marked suppression of all frequencies including delta activity in the ischemic hemisphere suggestive of massive and irreversible hemispheric infarction with increased risk for malignant cerebral edema [11].

Periodic Lateralized Epileptiform Discharges (PLEDs) are EEG abnormalities that signify acute brain dysfunction or unilateral brain lesion, usually destructive in nature, usually recorded in the area adjacent to cerebral infarction [24]. PLEDs have mostly been related to cerebral infarctions [25]. PLEDs, usually associated with obtunded patients, focal seizures and focal neurological signs tend to be transient and resolve spontaneously within 2-3 weeks. The discharges tend to decrease in amplitude, repetition rate and then discharges cease in PLEDs [24].

SPECT in Acute Ischemic Stroke

Brain SPECT imaging is a functional neuroimaging technique in the nuclear medicine study that employs isotopes bound to neurospecific pharmaceuticals to assess regional cerebral blood flow (rCBF) and indirectly metabolic activity. SPECT uses low energy, photon emitting lipophilic radiotracers that easily cross the blood–brain barrier and ideally are completely extracted during the first pass through the cerebral circulation [26]. The major blood flow agents used in brain SPECT imaging are technetium-99m hexamethylpropylene amine oxime (Tc-99m HMPAO), Tc-99m ethyl cysteinate dimer (Tc-99m ECD), Xenon-133 and N-isopropyl 1-123 p-iodoamphetamine [27]. The SPECT using [99mTc] HMPAO) is also a well established imaging technique in acute cerebral ischemia that might be useful to predict the risk of hemorrhagic transformation, severe edema, and spontaneous reperfusion after acute cerebral ischemia [28].

Development of newer techniques in vivo using SPECT and Positron Emission Tomography (PET) measures the cerebral metabolic rate of glucose (CMRglc), oxygen (CMRO2), and rCBF in human subjects [29]. Several studies have cited the usefulness of SPECT in the diagnosis of acute ischemic stroke patients, displaying that the perfusion abnormality observed on SPECT images correlates with extension of injury, its severity, and immediate outcome in acute stroke patients as shown in Figure 2. Toshihiro Ueda et al. demonstrated that observed SPECT patterns of brain perfusion (normal, high, mixed, low, and absent) correlating with the severity of stroke, size of lesion and immediate outcome [30]. It is used to measure CBF and brain patency in patients with stroke, tightly coupled with brain metabolism in several neurological disorders. Abnormal patterns of blood flow are identified either as areas of hypoactivity (focal or diffuse) or hyperactivity (hyperemia or luxury perfusion). It is a sensitive indicator of perfusion; it supplements the anatomic information from CT and MRI in evaluating cerebrovascular disease [31]. SPECT has clinical value in the diagnosis, therapeutic management, and follow-up of patients with acute ischemic stroke. This helps to determine patients at increased risk for stroke, patients most likely to benefit from intervention and can even determine the degree of tissue at risk for hemorrhage and severe edema [32,33]. Fibrinolytic therapy must be carried out immediately after the onset of clinical symptoms, when no structural technique can reveal the extent and severity of acute ischemia. In such condition, brain perfusion SPECT patterns predict the outcome of stroke patients and thus help in the selection of candidates for fibrinolytic therapy. Sequential SPECT images can display changes in regional cerebral perfusion over time, thus might help with follow-up management. SPECT is a favorable investigative technique to monitor follow-up of functional defects in anatomically preserved cerebral areas [34]. The SPECT camera has been merged to x-ray CT in the same device to provide an inherent imaging modality able to depict morphological as well as functional changes in one imaging session. Brain SPECT is considered superior to anatomic imaging modalities such as CT or MRI in detecting acute ischemic stroke in the first few hours following the events. Immediately after acute stroke, a focal or regional area of hypoperfusion or no perfusion will be seen which is larger than the lesion that is later seen on CT or MRI. The high sensitivity of SPECT is counterbalanced by poor specificity in detecting functional impairment [7].

SPECT fails to detect anatomical lesions. The hemorrhage and infarction cannot be unequivocally distinguished by SPECT. Therefore, SPECT should be used in amalgamation with other imaging modalities such as non-contrast CT and diffuse weighted imaging MRI [35]. The results of the study must be correlated with those from an anatomic technique such as CT or MR imaging. The data in SPECT are of low resolution compared with those obtained with CT and MR technologies, so that subtle areas of low flow, such as in the white matter, can be missed. SPECT is usually only semi-quantitative technique, providing ratios of CBF failing to reflect a true relationship between stable, absolute entities as CBF to the control may vary, depending on a variety of states and abnormalities [36].

Computed tomography perfusion (CTP) is a relatively new imaging modality that permits rapid qualitative and quantitative estimation of cerebral perfusion by generating maps of CBF, cerebral blood volume (CBV), and mean transit time (MTT) [37]. CTP allows early detection of cerebral ischemia and yields valuable information about the extension of perfusion disturbances [38]. CTP precisely allows for a quantitative assessment of rCBF and is available for the routine clinical practice. CTP would greatly contribute to improving managements of patients with cerebrovascular diseases and helps to differentiate reversible from irreversible ischemia [39]. CTP maps are relatively low in resolution, with slower data acquisition; hence, may miss small infarcts. It has difficulty in identifying areas of acute infarction adjacent to areas of chronic ischemic infarct [36,40].

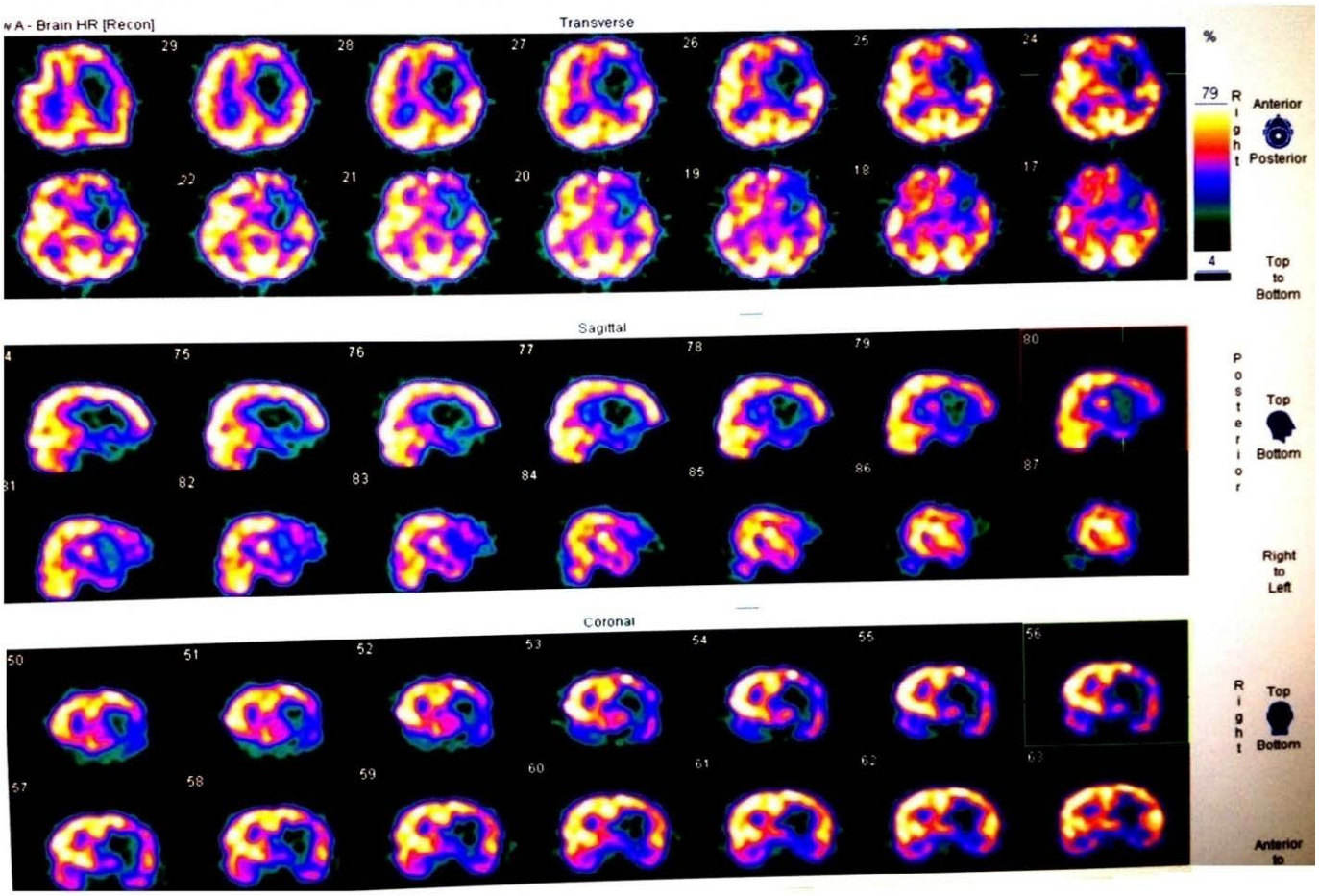

Figure 2: SPECT in a 63 years old male patient with acute ischemic stroke shows severe ischemia and infarction seen in part of left temporal lobe, left frontal lobe, left parietal lobe, left insular part, left basal ganglia.

Stable Xenon-enhanced computed tomography (XeCT), which uses inert gas to measure CBF in various brain regions, is an alternative to SPECT [35]. XeCT provides reproducible quantitative information coupled with anatomic CT imaging and also provides accurate estimations of cerebral blood flow, but it also reflects regional alterations in flow [40]. XeCT can be used to determine local cerebral blood flow in a small area, allowing the evaluation of hemodynamic states including acute stroke, vascular disease with occlusion, carotid occlusion testing, vasospasm, arteriovenous malformations, and management of head trauma [35]. This technique is useful for identifying patients at risk for ischemic compromise. XeCT is useful when assessing the patient with an acute neurological change those being considered for thrombolytic therapy and for patients with carotid artery stenosis to evaluate cerebrovascular reserve [41].

Summary

EEG and SPECT are the functional studies of the brain that are extensively available and thus can be used in emergency examinations. Identification of acute ischemic stroke in the primary stage is the utmost significant prerequisite for prevention of further impairment to the brain. This can be through EEG and SPECT, earlier than any other structural imaging studies. EEG and SPECT have confines in anatomical localization of lesions. These can supplement to imaging modalities like contrast enhanced CT and MRI in diagnosis and prognosis. EEG and SPECT also help in assessing severity and secondary injury which is related to acute ischemic stroke.

References

1. Soler EP, Ruiz VC (2010) Epidemiology and risk factors of cerebral ischemia and ischemic heart diseases: similarities and differences. Curr Cardiol Rev 6: 138-149.
2. Deb P, Sharma S, Hassan KM (2010) Pathophysiologic mechanisms of acute ischemic stroke: An overview with emphasis on therapeutic significance beyond thrombolysis. Pathophysiology 17: 197-218.
3. Cho IH, Hayashida K, Kume N, Shimotsu Y, Miyashita K (1998) Visualization of pressure-dependent luxury perfusion in a patient with subacute cerebral infarction. Ann Nucl Med 12: 217-220.
4. Lipton P (1999) Ischemic cell death in brain neurons. Physiol Rev 79: 1431-1568.
5. Paciaroni M, Caso V, Agnelli G (2009) The concept of ischemic penumbra in acute stroke and therapeutic opportunities. Eur Neurol 61: 321-330.
6. [No authors listed] (1991) Assessment: positron emission tomography. Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology 41: 163-167.
7. Camargo EE (2001) Brain SPECT in neurology and psychiatry. J Nucl Med 42: 611-623.
8. Peter Frykholm (2002) Cerebral ischemia studied with PET and microdialysis. Comprehensive Summaries Uppsala Dissertations 91: 5319-5328.
9. Rijsdijk M, Leijten FSS, Slooter AJC (2008) Continuous EEG monitoring in the Intensive Care Unit. Neth J Crit Care 12: 157-162.

10. Wang J, Chen L, Wu B, Hu X, Wang X, et al. (2010) Correlation study of cerebral blood flow and EEG feature based on CO2 stimulation. Proc Intl Soc Mag Reson Med 18: 1.
11. Schneider AL, Jordan KG (2005) Regional attenuation without delta (RAWOD): a distinctive EEG pattern that can aid in the diagnosis and management of severe acute ischemic stroke. Am J Electroneurodiagnostic Technol 45: 102-117.
12. Diedler J, Sykora M, Bast T, Poli S, Veltkamp R, et al. (2009) Quantitative EEG correlates of low cerebral perfusion in severe stroke. Neurocrit Care 11: 210-216.
13. Foreman B, Claassen J (2012) Quantitative EEG for the detection of brain ischemia. Crit Care 16: 216.
14. Mazziotta JC (1994) Mapping human brain activity in vivo. West J Med 161: 273-278.
15. Sheorajpanday RV, Nagels G, Weeren AJ, De Surgeloose D, De Deyn PP (2010) Additional value of quantitative EEG in acute anterior circulation syndrome of presumed ischemic origin. Clin Neurophysiol 121: 1719-1725.
16. Teplan M (2002) Fundamentals of EEG measurement. Measurement Science review 2: 1-11.
17. Murri L, Gori S, Massetani R, Bonanni E, Marcella F, et al. (1998) Evaluation of acute ischemic stroke using quantitative EEG: a comparison with conventional EEG and CT scan. Neurophysiol Clin 28: 249-257.
18. Nuwer MR, Arnadóttir G, Martin NA, Ahn SS, Carlson LG (1994 Apr) Comparison of quantitative electroencephalography, computed tomography, and behavioral evaluations to localize impairment in patients with stroke and transient ischemic attacks. J Neuroimaging 4: 82-84.
19. Niedermeyer E, Lopes da Silva F (2004). Electroencephalography: Basic Principles, Clinical Applications, and Related Fields. Lippincot Williams & Wilkins.
20. Jordan KG (2004) Emergency EEG and continuous EEG monitoring in acute ischemic stroke. J Clin Neurophysiol 21: 341-352.
21. Florence G, Guerit JM, Gueguen B (2004) Electroencephalography (EEG) and somatosensory evoked potentials (SEP) to prevent cerebral ischaemia in the operating room. Neurophysiol Clin 34: 17-32.
22. de Vos CC, van Maarseveen SM, Brouwers PJ, van Putten MJ (2008) Continuous EEG monitoring during thrombolysis in acute hemispheric stroke patients using the brain symmetry index. J Clin Neurophysiol 25: 77-82.
23. Finnigan SP, Rose SE, Walsh M, Griffin M, Janke AL, et al. (2004) Correlation of quantitative EEG in acute ischemic stroke with 30-day NIHSS score: comparison with diffusion and perfusion MRI. Stroke 35: 899-903.
24. Andraus ME, Andraus CF, Alves-Leon SV (2012) Periodic EEG patterns: importance of their recognition and clinical significance. Arq Neuropsiquiatr 70: 145-151.
25. YF Dan, ABS Pan, SH Lim (2004). Periodic lateralized epileptiform discharges: Aetiology and association with EEG seizures. Neurology Asia 9: 107-108.
26. Markus HS (2004) Cerebral perfusion and stroke. J Neurol Neurosurg Psychiatry 75: 353-361.
27. Mountz JM (2007) Nuclear medicine in the rehabilitative treatment evaluation in stroke recovery. Role of diaschisis resolution and cerebral reorganization. Eura Medicophys 43: 221-239.
28. Martín A, Macé E, Boisgard R, Montaldo G, Thézé B, et al. (2012) Imaging of perfusion, angiogenesis, and tissue elasticity after stroke. J Cereb Blood Flow Metab 32: 1496-1507.
29. Eliassen JC, Boespflug EL, Lamy M, Allendorfer J, Chu WJ, et al. (2008) Brain-mapping techniques for evaluating poststroke recovery and rehabilitation: a review. Top Stroke Rehabil 15: 427-450.
30. Toshihiro Ueda, William TC Yuh, JR Sonnad (2001) Value of Perfusion Imaging in the Management of Acute Ischemic Stroke Appl Radiol 30: 9.
31. [No authors listed] (1996) Assessment of brain SPECT. Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology 46: 278-285.
32. Thrall JH, Ziessman HA, O'Malley JP (2006) Central Nervous System. Nuclear medicine: The Requisites. 3rd edition, Mosby 432-434.
33. Umemura A, Suzuka T, Yamada K (2000) Quantitative measurement of cerebral blood flow by (99m)Tc-HMPAO SPECT in acute ischaemic stroke: usefulness in determining therapeutic options. J Neurol Neurosurg Psychiatry 69: 472-478.
34. Catafau AM (2001) Brain SPECT in clinical practice. Part I: perfusion. J Nucl Med 42: 259-271.
35. The Internet Stroke center (2013) SPECT and Xenon Contrast CT.
36. Latchaw RE (2004) Cerebral perfusion imaging in acute stroke. J Vasc Interv Radiol 15: S29-46.
37. Hoeffner EG, Case I, Jain R, Gujar SK, Shah GV, et al. (2004) Cerebral perfusion CT: technique and clinical applications. Radiology 231: 632-644.
38. Koenig M, Klotz E, Luka B, Venderink DJ, Spittler JF, et al. (1998) Perfusion CT of the brain: diagnostic approach for early detection of ischemic stroke. Radiology 209: 85-93.
39. Wintermark M, Maeder P, Thiran JP, Schnyder P, Meuli R (2001) Quantitative assessment of regional cerebral blood flows by perfusion CT studies at low injection rates: a critical review of the underlying theoretical models. Eur Radiol 11: 1220-1230.
40. Lui YW, Tang ER, Allmendinger AM, Spektor V (2010) Evaluation of CT perfusion in the setting of cerebral ischemia: patterns and pitfalls. AJNR Am J Neuroradiol 31: 1552-1563.
41. Massaro LM (1998) Xenon-enhanced CT: clinical applications. J Cardiovasc Nurs 13: 45-56.

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