ISSN 0362-1197, Human Physiology, 2006, Vol. 32, No. 1, pp. 37–42. © Pleiades Publishing, Inc., 2006.

Differences in Vulnerability between the Hemispheres in Early Childhood and Adulthood[1]

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Received June 23, 2005

Abstract—There are more left hemisphere damaged than right hemisphere damaged children and adults if one relies on studies of congenital hemiparesis as well as on those done on groups with radiologically demonstrated perinatal and postnatal unilateral stroke, hemispherectomy, or unilateral epilepsy in childhood or adulthood. The main pathogenetic factor seems to be a hemodynamic one, responsible for insufficient blood supply to the left hemisphere. Since adults show a difference in the same direction as children, the blood supply to the left internal carotid artery would seem to be the crucial factor. Around birth, an open ductus arteriosus may play an additional role. The hemodynamic asymmetry does not exclude an intrinsic maturational hemispheric tissue factor, making the left hemisphere more vulnerable than the right to detrimental influences around birth and during the early postnatal period. Immature white matter is especially vulnerable to asphyxia.

DOI: 10.1134/S0362119706010051

INTRODUCTION

Many studies suggest that in early childhood left hemisphere damage (LHD) predominates over right hemisphere damage (RHD). However, relatively few authors mention this difference explicitly in their reports when it occurs in their studies of left hemisphere damaged (LHD) and right hemisphere damaged (RHD) subjects. Some authors mention it for early cerebral infarcts in the vascular distribution of the middle cerebral artery [1, 2]. De Vries *et al.* [1] mention the difference in 23 infants with a lateralized infarct without, however, quantifying it. The difference is also found in periventricular hemorrhage [3], in neonatal stroke [2, 4], and in cerebral palsy [5]. Most authors seem to take the difference for granted; few have hypothesized about the cause of this phenomenon in neonates [6].

I first noted the above-mentioned difference between LHD and RHD while reviewing hemispherectomy studies of congenitally hemiparetic–epileptic children and therefore decided to do a larger metastudy on other groups, such as those with perinatally acquired unilateral damage and acquired unilateral damage in childhood or adulthood. It was found that the difference in the number of LHD and RHD subjects comes to light only in studies in which there is no selection bias between these two subgroups.

In this study it was found that the left–right asymmetry in frequency of hemispheric damage is not limited to brain damage acquired during early childhood (the pre- and perinatal time). The question that remains is what the cause of this uneven distribution might be. Is the cause intrinsic to the brain itself or do other factors, for example, vascular ones, play a role?

METHOD

Studies after 1970 were involved, but only studies whose aim was to look for qualitative differences between LHD and RHD subjects. Studies with a selection bias to one side were excluded. Most of the studies were done in order to investigate the neuropsychological status or the outcome of LHD or RHD, which means that their authors carefully divided LHD and RHD subgroups to exclude bilaterally damaged subjects. Aphasics were sometimes excluded when the aim of a study was cognition in LHD and RHD. Although not all authors mention it, this could influence the statistics. Although one can find a higher number of left brain damaged subjects, studies with a total of less than ten subjects have not been included. Some studies are not mentioned because other authors from the same research group used the same subjects in several subsequent studies, which might bias the results. In such cases, the most recent study has been used, which often entails more subjects than older studies done by the same laboratory.

Age was the key criterion in our study. Other covariables such as within-hemisphere localization, gender, and etiology other than vascular were not studied.

The difference between the numbers of LHD cases and RHD cases is expressed in a percentage, calculated as (nLHD – nRHD)/nLHD × 100. The percentage is negative in the case of a higher number of (n) RHD.

[1] The text was submitted by the author in English.

Table 1. Studies of congenital hemiplegia with perinatal insult to one hemisphere

References		(*n*) LHD	(*n*) RHD	% difference
[7]	CHP	161	109	+32
[8]	OSS	24	14	+42
[9]	CHP	19	6	+68
[10]	CHP	8	9	–12
[11]	CHP/EPI	28	15	+46
[12]	CHP	10	12	–25
[13]	CHP	10	7	+30
[14]	CHP	18	13	+28
[15]	CHP	19	6	+68
[2]	OSS	12	3	+75
[16]	OSS	60	49	+22
[17]	OSS	7	5	+28
[18]	CHP	88	61	+31
[3]	IPE	46	28	+39
[19]	CHP/EPI	34	23	+32
[20]	CHP	20	11	+45
[21]*	CHP	32	24	+25
[22]	CHP	9	5	+44
[23]	OSS	14	6	+57
[24]	CHP/EPI	23	15	+35
[25]	CHP	15	13	+15
[26]	OSS	10	6	+40
[27]	CHP	16	6	+62
[28]	CHP/OSS	26	13	+50
[5]	CHP	80	71	+11
[29]	+EPI	42	40	+5
[30]	CHP	23	20	+13
[31]	CHP/OSS	10	4	+60

Note: The studies published are of cases called CHP (congenital hemiplegic children), OSS (one-sided nonhemorrhagic stroke), IPE (intraparenchymal echodensities), and EPI (epilepsy). The table shows the number of subjects (*n*) for each damaged hemisphere (LHD, left hemispheric damage; RHD, right hemispheric damage). The right-hand column shows the percentage by which LHD is more frequent than RHD. The percentage is positive in all studies except two.
* Kolk and Talvik's [21] study only partially involves the same subjects as the study done by these authors in 2000.

RESULTS

Unilateral pre- or perinatally acquired damage or early childhood stroke. These studies mainly involve children with congenital hemiparesis. In most research done before the 1970s, the side of hemiparesis is taken to suggest contralateral hemispheric involvement. Since the mid-1970s, the side of injury has also been assessed using neuroimaging (CT or MRI). Table 1 summarizes these studies. Except for two of them, they all show more LHD than RHD cases.

Postnatal stroke, i.e., after the perinatal period, and subsequent hemiplegia is not rare in children. In these children one can also find an unequal occurrence of LHD and RHD. Table 2 summarizes the results of some studies.

Mixed etiology. A large number of studies do not meet the inclusion criteria of our survey. This is especially so when vascular etiology is mixed with other causes such as infection or tumors. The LHD–RHD difference in these patient populations is lower or may even be reversed. Table 3 gives some examples. We did not try to study the influence of etiologies other than the vascular one in detail.

Epileptic children with unilateral brain damage, usually perinatally acquired, in a minority of cases acquired in early childhood. Table 4 summarizes these studies. Most subjects have temporal lobe epilepsy (TLE) and are assessed before surgery (hemispherectomy or lobectomy) or even when no operation is done. The purpose of these studies is usually behavior, learning, cognitive status, or the prognosis of left or right brain damage or the sequelae of lobectomy. These studies therefore carefully divide left and right hemisphere cases. It is noticeable that even Rasmussen's encephalitis, i.e., unilateral epilepsy with unknown or heterogeneous etiology, occurs more frequently on the left side.

Adults with unilateral epilepsy. Giovagnoli [45] studied 112 patients with TLE. A clear left–right difference is present. Divided according to localization (frontal or temporal), the left–right difference is present in both subgroups. Divided according to mesial or lateral temporal localization, the difference is present in lateral TLE only. If the author divides the patients according to brain pathology (etiology), the differences are still present in all three subgroups when the pathology is temporomesial: hippocampal sclerosis (26 left, 15 right), low-grade glioma (17 left, 11 right), and cavernous angioma (4 left, 3 right). Manaut *et al.* [46] studied adult and child patients with unilateral epilepsy. They mention 395 subjects with left-sided discharges and 276 subjects with right-sided discharges, a difference of 30%. However, among them there were 218 children under 14 years of age (left–right hemisphere difference 23%) and the etiology is mixed and partially nonvascular. Table 5 summarizes adult subjects with unilateral epilepsy before they underwent a brain operation.

Adults with first-ever ischemic stroke. In adults with first-ever ischemic stroke, one also finds a left–right difference in hemispheric damage (Table 6). One study does not show this difference [53]; all the subjects in this study were males and the study protocol, based on an interview (personal communication), required that severe aphasics be left out. Two other studies are mentioned because of their high number of

patients [55, 56], but in these study groups there were also kinds of pathology other than ischemic stroke.

DISCUSSION

In which brain-damaged people does one find an unequal number of RHD and LHD subjects?

1. Although hemispherectomy is done for unilateral epilepsy caused by vascular as well as other causes, the number of studies concerning children who undergo right-sided hemispherectomy is significantly lower than that of studies of left-sided hemispherectomy. These data suggest a lower occurrence of right hemispheric (RH) lesions. The main cause is usually perinatal or postnatal stroke. Other causes are of a cardiac or infectious nature or arteriovenous malformation.

2. In infants there are fewer RH lesions from a vascular cause. This comes to light in the unequal number of RHD and LHD cases in studies with perinatal hemispheric damage from stroke or congenital hemiplegia, which is usually of a vascular nature [authors mentioned in Table 1].

3. In children with postnatally acquired hemiplegia caused by nonhemorraghic stroke several months to years after birth, the occurrence of LHD is also higher than that of RHD (Table 2). In children with LHD and RHD and etiologies other than vascular, the left–right difference is not significant or may even be absent (Table 3).

4. The difference is also found in adults with unilateral first-ever stroke (Table 4).

5. In children with unilateral epilepsy, usually TLE with or without hemiplegia, there are more left- than right-sided cases (Table 4). Given the pathologic nature of the lesions, it is highly probable that these epilepsies have a perinatal hypoxic-ischemic cause.

6. In adults with unilateral TLE, we find a clear asymmetry, except in lateral TLE (Table 5). It is known that the cause of TLE is often a morphological abnormality of developmental nature or is acquired early. Asphyxia is a likely cause. In this respect, this group does not differ from the children described under point 5.

What might be the cause of an unequal number of RHD and LHD subjects?

Disturbances in cerebral blood flow are a major cause of cerebral damage in the neonate, especially the preterm baby. Although brain damage is nearly always caused by hypoxia and/or ischemia, differences might be either a hemispheric vulnerability difference for hypoxia or a difference in blood supply, i.e., a vascular factor. The difference in the number of lesions on the left and on the right side, however, is not restricted to damage acquisition in early childhood.

Mullaart *et al.* [6] were the first to find an interesting explanation for perinatal cases. Perinatal hypoxic-ischemic damage is asymmetrically distributed, according to these authors, because of a hemodynamic left–right asymmetry. Using a blood flow study, they were able to show that the left hemisphere is more often damaged by hypoxic-ischemic insults because the ductus arteriosus Botalli, connecting the left pulmonary artery and the descending aorta in the fetus and closing soon after birth, steals blood from the left carotid artery,

Table 2. Studies of hemiplegia with postnatal insult to one hemisphere

References	Pathology	(*n*) LHD	(*n*) RHD	% difference
[9]	OSS Postn.	27	14	+48
[32]	OSS Postn.	14	13	+7
[33]	OSS angio	8	6	+25
[34]	OSS NI	16	5	+68

Note: This table shows postnatal stroke and acquired hemiplegia in children. The study by Banich *et al.* [9] includes one stroke at 1.5 months, while the other children studied had strokes between 7 and 114 months. Angio, results of unilateral angiographic findings, occlusion or stenosis; NI, neuroimaging. See explanations under Table 1.

Table 3. Studies of childhood hemiplegia with mixed etiology and mixed time of acquisition

References	Pathology	(*n*) LHD	(*n*) RHD	% difference
[35]	Mixed	39	36	+5
[36]	Mixed	22	26	+18
[37]	Acute	8	8	0
[38]	Mixed	6	8	–33
[29]	Mixed	28	25	+11
[39]	+EPI	10	8	+20

Note: Mixed, mixed etiology; EPI, epilepsy.

Table 4. Unilateral damage and epilepsy in children with or without hemiplegia

References	Pathology	(*n*) LHD	(*n*) RHD	% difference
[40]	TLE	13	14	–07
[41]	RAS	14	8	+43
[41]	DEV	12	11	+8
[20]	+CHP	8	6	+33
[42]	TLE/Other	13	7	+46
[43]	+CHP	10	7	+33
[44]	TLE	51	29	+43

Note: If TLE (temporal lobe epilepsy) is the case, it is mentioned in the table; "Other" is used for other types of epilepsy. Some authors explicitly mention that their epileptic children are also congenitally hemiplegic (CHP); most of the authors do not mention that the children are CHP. DEV, developmental idiopathic epilepsy; RAS, Rasmussen's encephalitis. For other explanations, see Table 1.

Table 5. Unilateral epilepsy in adults (before temporal lobectomy)

References		(*n*) LHD	(*n*) RHD	% difference
[47]*	TLE	15	11	+27
[48]	TLE	38	11	+71
[45]	MTLE mesial	47	29	+53
[45]	LTLE lateral	18	18	0
[45]	TLE total	65	47	+27
[45]	FLE	30	23	+23
[49]	MTLE	102	82	+20
[42]	TLE & EPI	13	7	+46
[50]	TLE	20	11	+45

Note: LTLE, lateral temporal lobe epilepsy; MTLE, mesial temporal lobe epilepsy; FLE, frontal lobe epilepsy; EPI, other types of epilepsy. For other explanations, see Table 1.
* No etiology and no time of insult mentioned.

Table 6. Adults with first-ever unilateral ischemic stroke

References		(*n*) LHD	(*n*) RHD	% difference
[51]		50	26	+48
[52]		194	173	+11
[53] (males only)		56	54	+3
[54]		32	15	+53
[55	68% ischemic lesions only	291	188	+36
[56]		29	16	+45
[57]	Ischemia and tumors	39	23	+41
[58]	Vascular occlusion (UIS)	33	21	+66
[59]		151	119	+21

Note: UIS, unilateral ischemic stroke. For other explanations, see Table 1.

causing hypoperfusion on the left side. A patent ductus arteriosus might be unfavorable. Earlier, Saliba *et al.* [60] showed that acute ligation of the ductus arteriosus in preterms was associated with an increase in the systemic systolic blood pressure in the anterior cerebral arteries.

Dabbs [61] stated in an earlier study that "… when a person drinks ice water, circulation of the blood will carry cooling to the head, causing a slight temperature drop on each side proportional to the blood flow on that side. Data from right and lefthanded subjects showed greater temperature drop on the nonverbal side of the head, consistent with radiological findings that the nonverbal side receives more blood." Other authors who found that the right hemisphere receives more blood are quoted by Dabbs. The left hemisphere thus receives less blood in adults, and it is also possible that this happens in children as well. This difference might then work in concert with ductal shunting, making the hemodynamic asymmetry decisive for unilateral damage in unfavorable circumstances. That a vascular cause plays a role seems probable because, in studies of postnatal stroke from causes other than vascular ones, the left–right difference is smaller (Table 3).

According to Coker *et al.* [2], the left circulation predilection suggests a differential flow in the left circulation, but anatomic studies by others did not reveal differences in angulation, size, or tortuousity between the left and the right carotid arteries. Doppler sonography by the authors did not demonstrate flow differences. The authors state that laminar flow and turbulence may be different on both sides.

Corballis and Morgan [62] argue that the left hemisphere is immature at birth, undergoes a rapid maturation process, and is therefore more vulnerable. Immature white matter is especially vulnerable to asphyxia [63]. The results of Kolk and Talvik [20] support the hypothesis that the period of vulnerability is more prolonged for the left hemisphere than for the right hemisphere.

Taylor [61], looking at the first fit in 168 cases of temporal lobe epilepsy before age 10, found left-sided lesions common in the first year and rare after two years, while right-sided lesions were equally prevalent during the first four years of life. In females the fall of the inception rate was more precipitate than in males in the first four years. The author concluded that cerebral maturation is more rapid in girls and the right (temporal) lobe. Brain tissue less active in the first year and immature (the left hemisphere) would be at greater risk for insults. For research purposes the number of left and right cases in this study were kept equal.

Another indication of maturity differences in the hemispheres at birth is the earlier formation of gyri and sulci around the sylvian fissure (temporal superior and frontal superior gyri) on the right than on the left side [65].

The difference in number of cases between LHD and RHD seems to have two pathogenetic mechanisms in children. The main factor seems to be a vascular one, which causes diminished blood supply to the left hemisphere. However, the vascular hypothesis cannot be attributed principally to ductal steal because the left–right difference is also found in childhood and adult cases with first-ever stroke. An additional, probably maturational factor, may be intrinsic to the hemispheres, favoring the right one in early childhood. Beginning white matter maturation may be the main vulnerability factor. The perinatal factors favoring the right hemisphere over the left hemisphere are probably the same in childhood and adult unilateral epilepsy.

We conclude, therefore, that blood hypoperfusion to the left carotid artery probably plays a significant role in the causation of the difference in frequency of RHD compared to LHD in cases at all ages.

REFERENCES

1. De Vries, L.S., Groenendaal, F., Eken, P., *et al*., Infarcts in the Vascular Distribution of the Middle Cerebral Artery in Preterm and Fullterm Infants, *Neuropediatr*., 1997, vol. 28, p. 88.
2. Coker, S.B., Beltran, R.S., Myers, T.F., *et al*., Neonatal Stroke: Description of Patients and Investigation into Pathogenesis, *Ped. Neurol*., 1988, vol. 4, p. 219.
3. Guzzetta, F., Shackelford, G.D., Volpe, S., *et al*., Periventricular Intraparenchymal Echodensities in the Premature Newborn: Critical Determinant of Neurologic Outcome, *Pediatrics*, 1986, vol. 78, p. 995.
4. Perlman, J.M., Rollins, N., and Evans, D., Neonatal Stroke: Clinical Characteristics and Cerebral Blood Flow Velocity Measurements, *Ped. Neurol*., 1994, vol. 11, p. 281.
5. Uvebrant, P., Hemiplegic Cerebral Palsy. Aetiology and Outcome, *Acta. Ped. Scan. Suppl*., 1988, vol. 345, p. 1.
6. Mullaart, A., Daniels, O., Hopman, J.C., *et al*., Asymmetry of the Cerebral Blood Flow: An Ultrasound Doppler Study in Preterm Newborns, *Ped. Neurol*. 1995, vol. 13, p. 319.
7. Annett, M., Laterality of Childhood Hemiplegia and the Growth of Speech and Intelligence, *Cortex I*, 1973, vol. 9, p. 4.
8. Aram, D.M., Language Sequelae of Unilateral Brain Lesions in Children, *Lang. Comm. Brain*, 1988, p. 171.
9. Banich, M.T., Cohen, D., Levine, S., Hongkeu, K., *et al*., The Effects of Developmental Factors on IQ in Hemiplegic Children, *Neuropsychol*., 1990, vol. 28, p. 35.
10. Ballantyne, A.O., Scarvie, K.M., and Trauner, D.A., Verbal and Performance IQ Patterns in Children after Perinatal Stroke, *Dev. Psychol*., 1994, vol. 10, p. 39.
11. Bates, E., Vicari, S., and Trauner, D., Neural Mediation of Language Development: Perspectives from Lesion Studies of Infants and Children, *Neurodevelopmental Disorders*, H. Tager Flusberg, Ed., Bradford Books, MIT Press, Cambridge, Massachusetts, 1999, p. 533.
12. Bouza, H., Dubowitz, L.M.S., Rutherford, M., *et al*., Late Magnetic Resonance Imaging and Clinical Findings in Neonates with Unilateral Lesions on Cranial Ultrasound, *Dev. Med. Child. Neurol*., 1994, vol. 36, p. 951.
13. Brizzolara, D., Pecini, C., Brovedani, P., *et al*., Timing and Type of Congenital Brain Lesion Determine Different Patterns of Language Lateralization in Hemiplegic Children, *Neuropsychol*., 2002, vol. 40, p. 620.
14. Carlsson, G., Uvebrant, P., Hugdahl, K., *et al*., Verbal and Non-verbal Function of Children with Right Versus Left-hemiplegic Cerebral Palsy of Pre- and Perinatal Origin, *Dev. Med. Child. Neurol*., 1994, vol. 36, p. 503.
15. Cohen, D., Levine, S., Huttenlocher, P., Banich, M.T. *et al.*, Factors Affecting Cognitive Functioning of Hemiplegic Children, *Dev. Med. Child. Neurol*., 1987, vol. 29, p. 27.
16. DeVeber, G.A., MacGregor, D., Curtis, R., *et al*., Neurologic Outcome in Survivors of Childhood Arterial Ischemic Stroke and Sinovenous Thrombosis, *J. Child. Neurol.*, 2000, vol. 15, p. 316.
17. Estan, J. and Hope, P., Unilateral Neonatal Cerebral Infarction in Full Term Infants, *Arch. Dis. Childh*., 1997, vol. 76, F88.
18. Goodman, R. and Yude, C., IQ and Its Predictors in Childhood Hemiplegia, *Dev. Med. Child. Neurol*., 1996, vol. 38, p. 881.
19. Isaacs, E., Christie, D., Vargha-Khadem, F., *et al*., Effects of Hemispheric Side of Injury, Age At Injury, and Presence of Seizure Disorder on Functional Ear and Hand Asymmetries in Hemiplegic Children, *Neuropsychol*., 1996, vol. 34, p. 127.
20. Kolk, A. and Talvik, T., Cognitive Outcome of Children with Early-Onset Hemiparesis, *J. Child. Neurol*., 2000, vol. 15, p. 581.
21. Kolk, A. and Talvik, T., Cerebral Lateralization and Cognitive Deficits after Congenital Hemiparesis, *Pediatr. Neurol*., 2002, vol. 27, p. 356.
22. Mercuri, E., Spano, M., Bruccini, G., *et al*., Visual Outcome in Children with Congenital Hemiplegia: Correlation with Mri Findings, *Neuropediatrics*, 1996, vol. 27, p. 184.
23. Mercuri, E., Rutherford, M., Cowan, F., *et al*., Early Prognostic Indicators of Outcome in Infants with Neonatal Cerebral Infarction: A Clinical, Electroencephalogram, and Magnetic Resonance Imaging Study, *Pediatrics*, 1999, vol. 103, p. 39.
24. Muter, V., Taylor, S., and Vargha-Khadem, F., A Longitudinal Study of Early Intellectual Development in Hemiplegic Children, *Neuropsychol*., 1997, vol. 35, p. 289.
25. Nass, R., Peterson, H., and Koch, D., Differential Effects of Congenital Right and Left Hemisphere Injury on Intelligence, *Brain Cogn*., 1989, vol. 9, p. 258.
26. Sran, S.K. and Baumann, R.J., Outcome of Neonatal Strokes, *Am. J. Dis. Child*., 1988, vol. 141, p. 1086.
27. Steinlin, M., Good, M., Martin, E., *et al*., Congenital Hemiplegia: Morphology of Cerebral Lesions and Pathogenetic Aspects from MRI, *Neuropediatr*., 1993, vol. 24, p. 224.
28. Trauner, D., Nass, R., and Ballantyne, A., Behavioural Profiles of Children and Adolescents after Pre- or Perinatal Unilateral Brain Damage, *Brain*, 2001, vol. 124, p. 995.
29. Vargha-Khadem, F., O'Gorman, A.M., and Watters, G.V., Aphasia and Handedness in Relation to Hemispheric Side, Age at Injury and Severity of Cerebral Lesion During Childhood, *Brain*, 1985, vol. 108, p. 677.
30. Vicari, S., Albertoni, A., Chilosi, A.M., *et al*., Plasticity and Reorganization during Language Development in Children with Early Brain Injury, *Cortex*, 2000, vol. 36, p. 31.
31. Wulfeck, B.B., Trauner, D.A., and Tallal, P.A., Neurologic, Cognitive, and Linguistic Features of Infants after Early Stroke, *Pediat. Neurol*., 1991, vol. 7, p. 266.
32. Lanska, M.J., Lanska, D.J., Horwitz, S.J., *et al.*, Presentation, Clinical Course, and Outcome of Childhood Stroke, *Pediatr. Neurol*., 1991, vol. 7, p. 333.

33. Wanifuchi, H., Kagawa, M., Takeshita, M., *et al*., Ischemic Stroke in Infancy, Childhood, and Adolescence, *Child's Nerv. syst*., 1988, vol. 4, p. 361.

34. Mancini, J., Girard, N., Chabrol, B., *et al*., Ischemic Cerbrovascular Disease in Children: Retrospective Study of 35 Patients, *J. Child. Neurol*., 1997, vol. 12, p. 193.

35. Ganesan, V., Hogan, A., Shack, N., *et al*., Outcome after Ischaemic Stroke in Childhood, *Dev. Med. Child. Neurol*., 2000, vol. 42, p. 455.

36. Riva, D. and Cazzaniga, L., Late Effects of Unilateral Brain Lesions Sustained Before and after Age One, *Neuropsychol*., 1986, vol. 24, p. 423.

37. Riva, D., Cazzaniga, L., Pantaleoni, C., *et al*., Acute Hemiplegia in Childhood. The Neuropsychological Prognosis, *J. Ped. Neuroscie*., 1986, vol. 2, p. 239.

38. Sztriha, L., Al Suhaili, A.R., Prais, V., *et al*., Regional Cerebral Blood Perfusion in Children with Hemiplegia: A SPECT Study, *Neuropediatrics*, 1996, vol. 27, p. 178.

39. Villemure, J.G. and Rasmussen, T., Functional Hemispherectomy in Children, *Neuropediatrics*, 1993, vol. 24, p. 53.

40. Camfield, P.R., Gates, R., Ronen, G., *et al*., Comparison of Cognitive Ability, Personality Profile, and School Success in Epileptic Children with Pure Right Versus Left Temporal Lobe EEG Foci, *Ann. Neurol*., 1984, vol. 15, p. 122.

41. Carson, S.C., Javedan, S.P., Freeman, J.M., *et al*., Hemispherectomy: a Hemidecortication Approach and Review of 52 Cases, *J. Neurosurg*., 1996, vol. 84, p. 903.

42. Kuehn, S.M., Keene, D.L., Richards, P.M.P., *et al*., Are There Changes in Intelligence and Memory Functioning Following Surgery for the Treatment of Refractory Epilepsy in Childhood? *Child. Nerv. Syst*., 2002, vol. 18, p. 306.

43. Lindsay, J., Ounsted, C., and Richards, P., Hemispherectomy for Childhood Epilepsy: A 36-year Study, *Dev. Med. Child. Neurol*., 1987, vol. 29, p. 592.

44. Whitman, S., Hermann, B.P., Black, R.B., *et al*., Psychopathology and Seizure Type in Children with Epilepsy, *Psychol. Med*., 1982, vol. 12, p. 843.

45. Giovagnoli, A.R., Relation of Sorting Impairment to Hippocampal Damage in Temporal Lobe Epilepsy, *Neuropsychol*., 2001, vol. 39, p. 140.

46. Manaut, E., Gomez, C.M., Vaquero, E., *et al*., Hemispheric Lateralization of Language in Epileptic Right-handed Children with Unihemispheric Discharge, *J. Child. Neurol*., 2002, vol. 17, p. 505.

47. Adolphs, R., Tranel, D., and Damasio, H., Emotion Recognition from Faces and Prosody Following Temporal Lobectomy, *Neuropsychol*., 2001, vol. 15, p. 396.

48. Briellmann, R.S., Berkovic, S.F., and Jackson, G.D., Men may Be More Vulnerable to Seizure-Associated Brain Damage, *Neurol*., 2000, vol. 55, p. 1479.

49. Janszky, J., Jokeit, H., Heinemann, D., *et al*., Epileptic Activity Influences the Speech Organization in Medial Temporal Lobe Epilepsy, *Brain*, 2003, vol. 126, p. 2043.

50. Tebartz van Elst, L., Woermann, F.G., Lemieux, L., *et al*., Affective Aggression in Patients with Temporal Lobe Epilepsy, *Brain*, 2000, vol. 123, p. 234.

51. Basso, A., Burgio, F., and Caporali, A., Acalculia, Aphasia and Spatial Disorders in Left and Right Brain-damaged Patients, *Cortex*, 2000, vol. 36, p. 265.

52. De Haan, R.J., Limburg, N., Van der Meulen, J.H.P. *et al.*, Quality of Life after Stroke. Impact of Stroke Type and Lesion Location, *Stroke*, 1995, vol. 26, p. 402.

53. Gerritsen, M., Cognitive aftermath of ischemic stroke. A Longitudinal Community-Based Study, *Thesis, Univ. of Groningen*, 2004.

54. Herrmann, M., Bartels, C., Schumacher, M., *et al*., Poststroke Depression. Is There a Pathoanatomic Correlate for Depression in the Postacute Stage of Stroke? *Stroke*, 1995, vol. 26, p. 850.

55. Kimura, D. and Watson, N., The Relation between Oral Movement Control and Speech, *Brain Lang*., 1989, vol. 37, p. 565.

56. Kimura, D., Acquisition of a Motor Skill after Left-hemisphere Damage, *Brain*, 1977, vol. 100, p. 527.

57. Mammucari, A., Caltagirone, C., Ekman, P., *et al*., Spontaneous Facial Expression of Emotions in Brain-damaged Patients, *Cortex*, 1988, vol. 24, p. 521.

58. McGlone, J., Sex Differences in the Cerebral Organization of Verbal Functies in Patients With Unilateral Brain Lesions, *Brain*, 1977, vol. 100, p. 775.

59. Pohjasvaara, T., Leppavuori, A., Siira, I., *et al*., Frequency and Clinical Determinants of Poststroke Depression, *Stroke*, 1998, vol. 29, p. 2311.

60. Saliba, E.M., Chantepie, A., Gold, F., *et al*., Intraoperative Measurements of Cerebral Haemodynamics during Ductus Arteriosus Ligation in Preterm Infants, *Eur. J. Ped*., 1991, vol. 150, p. 362.

61. Dabbs, J.M., Left-Right Differences in Cerebral Blood Flow and Cognition. *Psychophysiol*, 1980, vol. 17, p. 548.

62. Corballis, M.C. and Morgan, M., On the Biological Basis of Human Laterality: I. Evidence for a Maturational Left-Right Gradient, *Behav. Brain Sci*., 1978, vol. 2, p. 261.

63. Leviton, A. and Gilles, F.H., Acquired Perinatal Leucencephalopathy, *Ann. Neurol*., 1984, vol. 16, p. 1.

64. Taylor, D.C., Differential Rates of Cerebral Maturation Between Sexes and Between Hemispheres. Evidence From Epilepsy, *Lancet*, 1969, vol. 19, p. 140.

65. Chi, J.G., Dooling, E.C., and Gilles, F.H., Gyral Development of the Human Brain, *Ann. Neurol*., 1977, vol. 1, p. 86.