REFERENCES

- Kelling G. Ueber die Oesophagoscopie, Gastroskoppie und Caoelioskopie. Muench Med Wochenschr 1902; 49:21-4.
- Jacobaeus HC. Konnen durch die durch die Laparoskopie Indikationen zu chiruugischen Eingriffen gewonnen werden? Nord Med Ark 1914: 14:1-5.
- Goetze O. Die Rontgendiagnostik bet bei gasgefullter Bauchhohle. Eine neue methode. Muench Med Wochenschr 1918; 65:1275-9.
- Eisenburg J. Ueber eine Apparatur zur schonenden und kontrollierbaren Gasfullung der Bauchhohle fur die Laparoskopie. Klin Wochenschr 1966; 44:593-7.
- 5. Semm K. Das Pneumoperitoneum mit CO2. Visum 1967; 6:1-4.
- Scott DB. Some effects of peritoneal insufflation of carbon dioxide at laparoscopy. Anesthesia 1970; 25:590-5.

- Graff TD, Arbegast NR, Phillips OC, Harris LC, Frazier TM. Gas embolism: a comparative study of air and carbon dioxide as embolic agents in the systemic venous system. Am J Obstet Gynecol 1959; 78:259-63.
- O'Boyle CJ, deBeaux AC, Watson DI, Ackrovd R, Luffarde T, Leong JY, Williams JA, Jamieson GG. Helium vs carbon dioxide insufflation with or without saline lavage during laparoscopy. Surg Endosc 2002; 16:620-5.
- Tsereteli Z, Terry ML, Bowsers SP, Spivak H, Archer SB, Galloway KD, Hunter JG. Prospective rendomized clinical trial comparing nitrous oxide and carbon dioxide pneumoperitoneum for laparoscopic surgery. J Am Coll Surg 2002; 195:173-9.
- Alexander GD, Noe FE, Brown EM. Anesthesia for pelvic laparoscopy. Anesth Analg 1969; 48:14-8.

- Lewis DG, Ryder W, Burn N, Wheldon JT, Tacchi D. Laparoscopy – an investigation during spontaneous ventilation with halothane, Br J Anesth 1972; 44:685-9.
- Seed RF, Shakespeare TF, Muldoon MJ. Carbon dioxide homeostasis during anesthesia for laparoscopy. Anaesthesia 1970; 25:223-8.
- Magno R, Medegard A, Bengtsson R, Tronstad SE. Acid-base balance during laparoscopy. Acta Obstet Gynecol Scand 1979; 58:81-6.
- 14. Rademaker BM, Meyer DW, Bannenberg JJ, Klopper PJ, Kalkman CJ. Laparoscopy without pneumoperitoneum. Effects of abdominal wall retraction versus carbon dioxide insufflation on hemodynamics and gas exchange in pigs. Surg Endosc 1999; 9:797-801.
- Cantarow A, Trumper M. Clinical Biochemistry, 6th Ed., WB Saunder, Philadelphia, 1962, p. 776.

Tiraboshi RB, Domingos ALA, Dias-Neto JA, Paschoal RM, Travassos J, Martins ACP, Suaid HJ, Cologna AJ, Tucci Jr S. O insuflador de gás CO2 é necessário para treino de laparoscopia em animais? Acta Cir Bras [serial online] 2003 vol 18 suppl 5. Disponível em www.scielo.br/acb.

RESUMO – Objetivo – Testar a eficácia e seguraça do pneumoperitônio com ar comprimido para cirurgias videolaparoscópicas em porcos em treinamento de residência médica. Métodos – Porcos da raça Dalland de peso variável de 15 a 17kg foram submetidos a anestesia geral e respiração controlada. Eles foram divididos em 3 grupos: A – 38 animais com pneumoperitônio feito com insuflador automático de CO₂ usando este gás; B – 7 animais sujeitos ao mesmo procedimento exceto que o CO₂ foi substituído por ar comprimido; e, C – 11 animais em que o pneumoperitônio foi feito com ar comprimido diretamente da rede hospitalar. Nos 3 grupos a pressão intra-abdominal foi mantida entre 12 e 14mmHg. Os procedimentos realizados foram distribuídos proporcionalmente nos 3 grupos: nefrectomia bilateral – 20, pieloplastia desmembrada – 20 e nefrectomia parcial – 16. Antes e 2h após o pneumoperitônio foi colhido sangue arterial para gasometria em 5 porcos do grupo C. Resultados – Foram consumidos 25 torpedos de 4,5kg de CO₂ a um custo total de R\$ 3.150,00 no grupo A. A duração média da cirurgia nos grupos A, B e C foram respectivamente: 181±30min, 196±39min e 210±47min (p>0.05). Alcalose respiratória foi observada em 3/5 porcos testados do grupo C. Nenhum animal apresentou sinais de embolia gasosa ou faleceu durante o procedimento. Conclusão - O uso de ar comprimido para laparoscopias em porcos mostrou-se método seguro com redução de custos e tornou desnecessário o uso de insuflador automático.

DESCRITORES - Laparoscopia. Nefrectomia. Pieloplastia. Nefrectomia. Parcial. Pneumoperitônio. CO₂. Ar comprimido.

Correspondence address: Antonio Carlos Pereira Martins Departamento de Cirugia – HCFMRP-USP Av. Bandeirantes, 3900 – 9° Andar Ribeirão Preto, CEP: 14048-900

5-ARTIGO ORIGINAL

Study of corpus callosum in experimental hydrocephalic wistar rats¹

Luiza da Silva Lopes², Hélio Rubens Machado³, João-José Lachat²

Lopes LS, Machado HR, Lachat J-J. Study of corpus callosum in experimental hydrocephalic wistar rats. Acta Cir Bras [serial online] 2003 vol 18 suppl 5. Avaliable in www.scielo.br/acb

ABSTRACT – Purpose: Hydrocephalus causes countless cerebral damages, especially on the structures around the ventricles. Hydrocephalic children present deficiencies in the nonverbal skills more than in the verbal skills, and not always revertible with an early treatment. As the corpus callosum has an important role in the nonverbal acquisition it is possible that the injuries in this structure are responsible for the cognitive dysfunctions of these children. This present study tries to establish the alterations caused by hydrocephalus on the corpus callosum of developing Wistar rats, induced by intracisternal injection of kaolin. Methods: Seven, fourteen and twenty one days after the injection, the animals were killed, and the corpus callosum was dissected and prepared for the study of the axonal fibers. Results and Conclusion: The seven-day old rats in hydrocephalus development presented a delay in myelination in relation to the control rats. With the fourteen-day old rats in hydrocephalus development the corpus callosum showed a recovery of myelin, but with the twenty one-day old rats in hydrocephalus development the axonal fibers were damaged and reduced in number.

KEY WORDS: Corpus callosum, Hydrocephalus, Myelin, Wistar rat, Development.

^{1.} Article from the Laboratory of Neuroanatomy, Department of Surgery and Anatomy, Faculty of Medicine of Ribeirão Preto, University of São Paulo, São Paulo, Brazil.

^{2.} PhD, Department of Surgery and Anatomy, Faculty of Medicine of Ribeirão Preto, University of São Paulo, São Paulo, Brazil.

^{3.} Associate Professor, Department of Surgery and Anatomy, Faculty of Medicine of Ribeirão Preto, University of São Paulo, São Paulo, Brazil.

INTRODUCTION

Hydrocephalus is a complex disease, characterized by ventricular enlargement, secondary to the imbalance between the formation and absorption of the cerebrospinal fluid (CSF)1. The first structure that is affected by the increase of the CSF pressure and the expansion of the ventricles is the ependyma, by compressing and stretching of the structure, besides ruptures from isolated points until the nearly total destruction of the epithelium^{2,3}. In the white matter axonal degeneration is observed, as well as the loss of the myelin in the more accentuated degrees of ventricular enlargement4. The distortion of the brain leads to stretching and distortion of the brain vessels, besides reducing the density of the capillaries. As a final result, there is a reduction of the cerebral blood flow⁵. Hydrocephalus also affects the neurons and the synapse, it is not known if by direct compression or secondary suffering to the axons. The cytoarchitecture is affected in the density and neuronal size6,7,8, with alterations in the cortical lamination. The neuronal connectivity is also damaged, and the neurotransmitters have their concentrations reduced, but the degradation products of its metabolism are increased, which mirrors the exposure of the clearance of the extracellular space9.

Children suffering from hydrocephalus often present a retarding in the development of their cognitive functions, with a tendency of showing more faults in the development of nonverbal cognitive functions than in the development of speech^{10,11,12,13}. Rare studies have associated these alterations of the nonverbal cognitive functions with a degree of exposure of the corpus callosum and of periventricular white matter in the hydrocephalus. Assumptions suggest that the development of the nonverbal functions is dependent on the integrality of the white matter in both the brain hemispheres, as well as the integrality of the commissures, especially that of the corpus callosum¹⁴. There are authors who attribute the cognitive deficiencies to the lesions of the vascular elements of the cerebral cortex¹⁵. Even with the improvement of the ventricular shunts not all the neurological and structural alterations are reverted with the treatment of the hydrocephalus. Neurological residuary damage occurs on 10% to 74% of the children with hydrocephalus receiving treatment with shunts16. The recovery of the white matter with remyelination and the neuronal recovery, and the restore of the levels of the neurotransmitters only occur when the treatment with shunts is precociously established^{17,18}.

The objectives of this study were to determine the myelination progression in the corpus callosum development of the normal rats, and the alterations caused by hydrocephalus in different anatomic regions of this structure, according to the time of progression of the ventricular enlargement.

METHODS

Induction of hydrocephalus. This study was done according to the CIOMS (Council for International Organization of Medical Sciences)

ethical code for animal experimentation (WHO Chronicle 1985; 39(2):51-6) and the principles of the COBEA – Colégio Brasileiro de Experimentação Animal.

Sixteen litters of 7-day old Wistar rats were used. without sexual distinction. Each litter was constituted of the mother rat and 6 to 8 newborn rats. Each pup rat was held by an auxiliary, who held the head with one hand and the body with the other, bending the animal's neck, leaving the posterior cervical area free. By palpation the space between the posterior extremity of the foramen magnum, on the occipital bone, and the posterior arch of the first cervical vertebra was identified. With an Mise 0.3 odontologic needle, with a short bezel, the suboccipital percutaneous injection was done, and 0.4 ml of a kaolin suspension (Merckâ) dilated in distilled water of 20% was slowly injected. One rat of each litter was not injected as it was used as a control. Following this the rats were put back into their cages with their mothers. All the injected animals were observed daily to see any possible clinical alterations. Those rats that did not develop hydrocephalus after the injection were dismissed and were not computed in the number of animals of each group.

Brain perfusion and dissection of the structures. The animals of each group injected with kaolin and their respective controls of the same age, were weighed one by one, on a standard animal scale. Following anesthesia with vapors of sulphuric ether, they were submitted to a transcardiac perfusion with PBS solution (about 1ml/g of the animal's weight), after the perfusion, with the same volume, with a 2% paraformaldehyde and 1% glutaraldehyde in a 0.1M, pH 7.4 phosphate buffer. The animals were decapitated, their brains removed in block by a craniectomy of the vertex, weighed on a precision scale and immersed in the same fixative solution of formaldehyde and glutaraldehyde for more than 24 hours at 4°C. After washing with a 0.1M phosphate buffer, the brains were sectioned at the medium sagittal plane, and the corpus callosum identified and dissected, using its natural cleavage plane, removing samples of the genu, mid-body and splenium.

Preparation of samples for regional evaluation of the corpus callosum. The samples obtained from the corpus callosum were fixed in a 1% osmium tetroxide in a 0.2M phosphate buffer for 2 hours, at 4°C, washed with a 0.1M phosphate buffer and then dehydrated in an increasing series of acetone and embedded in araldite. This inclusion was done to permit that the callosum fibers could always be sectioned transversally. After the polymerization of the resin, the blocks were cut in sections of 0.5 mm of thickness and the sections stained with 1% toluidine blue.

The sections were analyzed in a light microscope and photographed on the Axiophot (Carl Zeiss) photomicrography system. The aspect, the distribution and the number of myelinated fibers and the neuroglia cells were evaluated considering the region of the corpus callosum and the time of evolution of hydrocephalus. The maturity of the corpus callosum of the normal rats and of the hydrocephalus ones, were analyzed.

Statistic Analysis. The body and brain weight data of the control rats and the ones with hydrocephalus, obtained on the day they were killed, were compared by ANOVA, with significance level of 5%.

RESULTS

Clinic alterations of hydrocephalus. Around the 2nd day after the injection a slight convexity of the skull vertex was able to be observed on the animals that developed hydrocephalus. As the days went by this convexity was more evident. Those rats that were not killed after 7 days of the kaolin injection and that belonged to the groups of 14 and 21 days of hydrocephalus evolution, also presented staring eyes on the 10th day besides skull convexity. Some rats with accentuated skull convexity, became lethargic on the 3rd week of hydrocephalus evolution, and withdrew themselves to a corner of the cage, presenting trembling of the head and hyperexcitement, jumping without coordination.

Body and brain weight of the normal rats and the hydrocephalus ones. All the animals were weighed on the day that they were killed, immediately before the anesthesia to transcardiac perfusion. The results of the weights can be seen in Table 1. The average body weight of the hydrocephalus animals killed on the 7th day after the kaolin injection (group H1) was significantly less than that of the control (group C1). Although the difference between the average weight of the hydrocephalus rats killed on the 14th day after the injection (group H2) and their controls (group C2), it is not statistically significant, but there was a tendency of the hydrocephalus animals to present less weight. Finally in the animals killed on the 21st day after the kaolin injection (group H3) we noticed that there was not a statistically significant difference, but they also presented a tendency of less weight than the control rats of the same age (group C3). The result of brain weighing presented a similar relation between the control rats and the hydrocephalus ones. When the brain weights are related to the body weights we observed that the average decreases with age, in the normal rats, or better, in the younger animals, the brains present a bigger proportion in the animal's total weight than the older animals. In the hydrocephalus rats of group H1, the brains are heavier than their controls. In the hydrocephalus rats of group H2, the brains continue to be heavier than the controls, but the difference is less than that of the rats of 1 week younger. In the rats of group H3, there is a tendency of the hydrocephalus brains being heavier than the controls, but the difference is not statistically significant.

Maturation of the brain in the control animals.

The observation of the sections of the corpus callosum stained with toluidine blue showed that the myelinated fibers of the control animals of group C1 are disperse, in increasing number from the posterior extremity to the anterior of the structure, or better, the splenium presents myelinated fibers in less number than the body, that in turn presents a smaller number of these

fibers than the genu of the corpus callosum. Neuroglia cells are present in a great number, with big nucleus and evident nucleolus. Some neuroglia cells appear withdrawn and dark (Figure 1A, B and C). In the rats of group C2, the number of myelinated fibers, are really increased,

although compacted, but the crescent gradient of the splenium for the genu maintains. The neuroglia cells are in a reduced number in relation to the 14th day controls (Figure 1 D, E and F). The controls of group C3 present myelinated fibers even more exuberant in number and

density. The crescent gradient of the number of myelinated fibers of posterior to anterior callosal portion is still identified, but the number of fibers with myelin of the splenium increased considerably, and the fibers are bigger and better defined (Figure 1 G, H and I).

TABLE 1: Averages of the body and brain weights and the brain/body rate of the normal rats with 14 (C1), 21 (C2), and 28-day old (C3), and the hydrocephalic ones, killed on the 7th (H1) 14th (H2) and 21st (H3) days after the injection.

	Experimental Groups					
	C1	C2	C3	H1	H2	Н3
Average of body	$38,67 \pm$	$54.8 \pm$	93 ±	$27\pm3,\!98$	49,79	49,79
weight ± SD	1,37	12,15	13,89		$\pm 11,443x$	11,126
Average of the	$1,271 \pm$	$1,419 \pm$	$1,551 \pm$	$1,425 \pm$	$1,499 \pm$	$2,038 \pm$
brain weight ± SD	0,029	0,118	0,034	0,239	0,127	0,536
Brain/body rate	$3,289 \pm$	$2,656 \pm$	$1,696 \pm$	$5,393 \pm$	$3,116 \pm$	$2,66 \pm$
$x100 \pm SD$	0,095	0,382	0,237	1,199	0,543	0,846

Lesions caused by hydrocephalus. The observation of the sections of the corpus callosum of the hydrocephalic rats stained with toluidine blue showed that hydrocephalus increases the density of the neuroglia cells that however show up pyknotic and dark most of the time. The hydrocephalic animals of group H1 present a smaller number of myelinated fibers of the corpus callosum when compared to their

controls. These fibers are reduced in the 3 areas of the examined corpus callosum, appearing more in the splenium as scattered groups. The splenium also shows vacuolization that appears as light areas (Figure 2 A, B and C). However the hydrocephalic rats of group H2, show a big number of myelinated fibers, more densely compacted than the controls. This compaction occurs in the more distant regions of the

ventricular cavity, that is on the body and on the splenium of the corpus callosum, the immediate areas of the lateral ventricles roof have signals of edema with areas of spacing between the fibers (Figure 2D, E and F). The animals from group H3 also show important losses of myelinated fibers, which are disperse, with small areas of compacting (Figure 2 G, H and I).

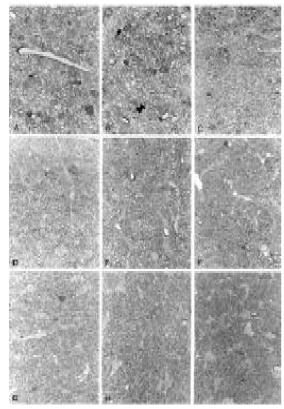


FIGURE 1: The development and maturing of the corpus callosum of the normal Wistar rat. A, B, and C: 14-day old animal (group C1); D, E and F: 21-day old animal (group C2); G. H and I: 28-day old animal (group C3). The first figure of each row is the sample of the genu of the corpus callosum, the second figure is the sample of the body of the corpus callosum and the third figure of each row is the sample of the splenium. Solid arrow in B: myelinated fiber of the corpus callosum. Open arrow in B: Neuroglia cell. Toluidine blue stain. 500 X.

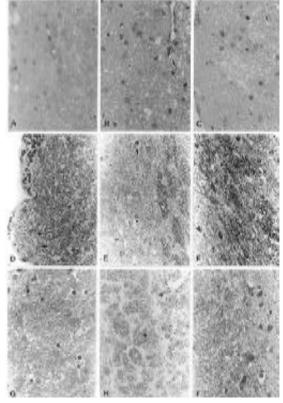


FIGURE 2. The development and myelination of the corpus callosum in hydrocephalic rats, killed on the 7th (H1) 14th (H2) and 21st (H3) days after the injection (moderate hydrocephalus). A, B and C: Group H1; D, E and F: Group H2; G, H and I Group H3. The first figure of each row is the sample of the genu of the corpus callosum, the second figure is the sample of the body of the corpus callosum and the third figure of each row is the sample of the splenium. Toluidine blue stain. 500 X.

DISCUSSION

Kaolin is an inactive chemical substance, composed of hydrated aluminum silicate, which causes an intense inflammatory response of the meninges, with an obstruction of the outflow of the cerebral spinal fluids of the 4th ventricle, without leading to brain parenchyma lesions¹⁹. One or two days after the cisternal injection of kaolin, the ventricular enlargement begins, on the lateral ventricle, then advancing afterwards. This model of experimental induction was chosen due to it being efficient, inexpensive, and it does not need surgical proceedings or cause anatomic modifications and does not even lead to other alterations unless those results that are totally of hydrocephalus^{9,20,21}.

In this study, the induced hydrocephalus by transcutaneos, intracisternal injection, of the kaolin suspension, on 7-day old post-birth rats, lead to skull convexity, identified after the first days of the injection. This shows that the cranial sutures of these animals are still obvious therefore this model of experiment can simulate hydrocephalus that develops in very young children. The skull convexity was directly proportional to the degree of ventricular enlargement. Similar findings were obtained by Kiefer et al., although they used the H-Tx rats, genetically affected by hydrocephalus³. These animals, however, are expensive and restricted to few researchers, besides not being able to guarantee that all the lesions found are entirely a result of hydrocephalus.

Besides the skull convexity, the hydrocephalic rats presented staring eyes that could correspond to "setting-sun phenomenon" of the hydrocephalic children. Similar findings were seen by Wright et al., although in kittens¹⁵. Following this these animals begin to present apathy and trembling, also in the same way as hydrocephalic patients. Trembling is referred to by several authors, so much so as in hydrocephalic rats as in hydrocephalic cats^{8,15}.

The hyperexcitability of some animals with moderate-severe ventriculomegaly could be explained by the compromising of the periaqueductal grey matter, compressed by the enlargement of the more peripheral levels of the ventricular system at this stage of hydrocephalus evolution. Nevertheless, detailed and directed studies for this structure have to be done. Nothing was found in the pertinent literature that something similar was discovered.

The 14-day old post-birth animals with 7-day hydrocephalus evolution (group H1), presented slightly less body weight than its correspondent controls. But the 21-day old hydrocephalic animals, therefore 14 days of hydrocephalus evolution (group H2), although they presented a tendency of less weight when compared with their controls, did not have a significant difference of body weight gain. This can represent an adaptation of the animals with the new condition and then return to normal feeding. The same was observed with animals 1 week older, reinforcing this idea of readaptation. Several authors obtained similar data with hydrocephalic animals with less weight than the controls at the beginning of the ventricular enlargement process, recovering on the second week of hydrocephalus progression, but no explanations were given for these findings^{22,23,24,25}

As for the brain weight in relation to the body weight, the normal animals presented a progressive decrease of the relation, or better, at this phase of development the body gains more weight than the brain, indicating that the final brain weight is a lot affected before the animal reach body maturity. In the 2-week old hydrocephalic rats (group H1), the brains are heavier than their controls. These alterations in weight can reflect of brain edema and the proper accumulation of fluid in the enlarged ventricular cavities. However, this difference is accentuated with aging, as time goes by the brain begins to represent a smaller volume of the total body weight of the rat.

In rats the myelination of the corpus callosum begins around the 13th day of birth, increasing a lot at the end of the 1st month, but this continues until the 10th month of age²⁶. The presence of a big number of neuroglia cells on the corpus callosum of the younger animals indicates a role of these cells in the axonal myelination process, as after the increase in the number of fibers with myelin as time goes by, the neuroglia cells are inclined to reduce in number.

With the development of hydrocephalus the number of neuroglia cells of the corpus callosum increase in number, mainly in the posterior areas. This can be a sign of an attempt of the myelin of the damaged areas to repair. The apparent partial recovery of the myelin was also identified by Del Bigio et al.24. Hydrocephalus although it initially makes the body and the genu to become thinner, first goes to the edema and alters the myelination of the posterior areas of the corpus callosum. In this study an initial loss of myelinated fibers in the seven days of hydrocephalus evolution (group H1) was noticed, an important increase of these fibers with the 14 days of hydrocephalus evolution (group H2) and once again a drastic reduction of myelinated fibers in the 21 days of hydrocephalus evolution (group H3). This initial increase of the fibers with myelin, that still present themselves in a smaller number than in the controls, can be an indication of an attempt of the recovery of the corpus callosum damages. However, this recovery was not maintained while the ventricular enlargement persisted. These findings were similar to those of Del Bigio et al.24. An explanation for the increase of the damages of the corpus callosum fibers during the hydrocephalus time, could be an alteration of the tissue perfusion that hydrocephalus causes in the white matter that leads to the accumulation of toxic metabolites in this matter, as is suggested by Massicotte et al.27. Another explanation for this fact could be the maintenance of the biomechanical alterations in respect to the stabilization of the ventricular enlargement, as is proposed by Shapiro

It should be considered that the histochemical methods is qualitative and for a more criteria evaluation of the myelination, it is necessary a quantitative study, with an ultrastructural study, so much as for the number of existing fibers as the thickness of the myelin border of each fiber.

The results of the present study allows us to conclude that the development of the corpus callosum of the Wistar rat, in relation to myelination, is essentially after birth following a standard of anterior-posterior sequence. Initially in hydrocephalus the myelination process tends to follow the normality standard, but as time goes by the myelinated fibers are slowly affected. Complementary studies are necessary, including morphometric and ultrastructural analysis for a better comprehension of the physiopathology of the damages produced by hydrocephalus in the corpus callosum and other structures of the brain parenchyma, close to the ventricular system.

REFERENCES

- Del Bigio MR, MCallister II JP. Hydrocephalus – Pathology. In: M Choux, C Di Rocco, AD Hockley, ML Walker. Pediatric Neurosurgery. London, Churchill Livingstone 1999; 217-36.
- Del Bigio MR, Bruni E, Fewer HD. Human Neonatal Hydrocephalus. An Electron Microscopic Study of the Periventricular Tissue. J Neurosurg 1985; 63: 56-63.
- Kiefer M, Eymann R, Von Tiling S, Müller A, Steudel W-I, Booz K-H. The ependyma in chronic hydrocephalus. Child's Nerv Syst 1998; 14: 263–70.
- Mabe H, Suzuki K, Nagai H. Cerebral blood flow after ventriculoperitoneal shunt in children with hydrocephalus. Child's Nerv Syst 1990; 6: 388-91.
- Portnoy HD, Branch C, Castro ME. The relationship of intracranial venous pressure to hydrocephalus. Child's Nerv Syst 1994; 10: 29-35.
- MCallister II JP, Maugans TA, Shah MV, Truex RC. Neuronal Effects of Experimentally Induced Hydrocephalus in Newborn Rats. J Neurosurg 1985; 63: 776-83.
- Harris NG, Jones HC, Patel S. Ventricle shunting in young H-Tx rats with inherited congenital hydrocephalus: a quantitative histological study of cortical grey matter. Child's Nerv Syst 1994; 10: 293-301.
- Del Bigio MR, Vriend JP. Monoamine neurotransmitters and amino acids in the cerebrum and striatum of immature rats with kaolin-induced hydrocephalus. Brain Res 1998; 798: 119–26.
- Shoesmith CL, Buist R, Del Bigio MR. Magnetic resonance imaging study of extracellular fluid tracer movement in brains of immature rats with hydrocephalus. Neurol Res 2000; 22:111-6.
- Fletcher JM, Bohan TP, Brandt ME, Brookshire BL, Beaver SR, Francis DJ, Davidson KC, Thompson NM, Miner ME. Cerebral white matter and cognition in hydrocephalic children. Arch Neurol 1992; 49: 818-24.
- Riva D, Milani N, Giorgi C, Pantaleoni C, Zorzi C, Devoti M. Intelligence outcome in children with shunted hydrocephalus of different etiology. Child's Nerv Syst 1994; 10: 70-3.

- Brookshire BL, Fletcher JM, Bohan TP, Landry SH, Davidson KC, Francis DJ. Verbal and nonverbal skill discrepancies in children with hydrocephalus: A Five-Year Longitudinal Follow-Up. J Pediat Psycol 1995; 20: 785–800.
- 13. Fletcher JM, Bohan TP, Brandt ME, Kramer LA, Brookshire BL, Thorstad K, Davidson KC, Francis DJ, McCauley SR, Baumgartner JE. Morphometric evaluation of the hydrocephalic brain: Relationships with cognitive development. Child's Nerv Syst 1996; 12: 192-9.
- 14. Brodal A. Anatomia neurológica com correlações clínicas. 3ª ed. São Paulo, ROCA, 1984: 338 e 679-84.
- Wright LC, MCallister II JP, Katz SD, Miller DW, Lovely TJ, Salotto AG, Wolfson BJ. Cytological and cytoarchitectural changes in the feline cerebral cortex during experimental infantile hydrocephalus. Pediatr Neurosurg 1990; 91: 139-155.
- McLone DG. Consensus: modelling of hydrocephalus. Child's Nerv Syst 1994; 10: 24–8.

- Donders J, Canady AI, Rourke BP. Psychometric intelligence after infantile hydrocephalus. A critical review and reinterpretation. Child's Nerv Syst 1990; 6: 148-54.
- Hirsch J-F. Consensus: long-term outcome in hydrocephalus. Child's Nerv Syst 1994; 10: 64–9.
- Hochwald GM. Animal models of hydrocephalus: recent developments. Proc Soc Exp Biol Med 1985; 178: 1-11.
- Hasan D, Tanghe HLJ. Distribution of cisternal blood in patients with acute hydrocephalus after subarachnoid hemorrhage. Ann Neurol 1992; 31: 374-8.
- Matsumoto T, Nagai H, Fukushima T, Mase M. Analysis of intracranial pressure pulse wave in experimental hydrocephalus. Child's Nerv Syst 1994; 10; 91–5.
- Chumas PD, Drake JM, Del Bigio MR, Silva M, Tuor UI. Anaerobic glycolysis preceding white-matter destruction in experimental neonatal hydrocephalus. J Neurosurg 1994; 80: 491-501.
- 23. Da Silva MC, Drake JM, Lemaire C, Cross A, Tuor UI. High-energy phosphate

- metabolism in a neonatal model of hydrocephalus before and after shunting. J Neurosurg 1994; 81: 544-53.
- 24. Del Bigio MR, Kanfer JN, Zhang YW. Myelination delay in the cerebral white matter of immature rats with kaolin-induced hydrocephalus is reversible. J Neuropathol Exp Neurol 1997; 56: 1053–66.
- 25. Tashiro Y, Drake JM. Reversibility of funtionally injured neurotransmitter systems with shunt placement in hydrocephalus rats: implications for intellectual impairment in hydrocephalus. J Neurosurg 1998; 88: 709-17.
- Elberger AJ. The functional role of the corpus callosum in the developing visual system: a review. Prog Neurobiol 1982; 18: 15-79.
- 27. Massicotte EM, Buist R, Del Bigio MR. Altered diffusion and perfusion in hydrocephalic rat brain: a magnetic resonance imaging analysis. J Neurosurg 2000; 92: 442-7.
- Shapiro K, Takei F, Fried A, Kohn I. Experimental feline hydrocephalus. The role of biomechanical changes in ventricular enlargement in cats. J Neurosurg 1985; 63: 82–7.

Lopes LS, Machado HR, Lachat J-J. Estudo do corpo caloso de ratos Wistar submetidos à hidrocefalia experimental. Acta Cir Bras [serial online] 2003 vol 18 suppl 5. Disponível em www.scielo.br/acb

RESUMO— Objetivo: A hidrocefalia causa inúmeros danos cerebrais, especialmente nas estruturas ao redor dos ventrículos cerebrais. As crianças com hidrocefalia apresentam déficits nas aquisições não verbais mais do que nas verbais, nem sempre revertidos com o tratamento precoce. Como o corpo caloso tem um papel essencial nas aquisições não verbais, é provável que as lesões nesta estrutura estejam envolvidas com as disfunções cognitivas dessas crianças. Este trabalho procura estabelecer as alterações causadas pela hidrocefalia, induzida pela injeção de caulim intracisternal ao corpo caloso de ratos Wistar em desenvolvimento. Métodos: Sete, 14 e 21 dias após a injeção, os animais foram sacrificados, sendo o corpo caloso dissecado e processado para estudo das fibras axonais. Resultados e Conclusões: Os ratos com 7 dias de evolução da hidrocefalia apresentaram um atraso na mielinização em relação aos controles. Em ratos com 14 dias de evolução da hidrocefalia, o corpo caloso mostrou recuperação da mielina, mas em ratos com hidrocefalia com 21 dias de evolução, as fibras apresentavam-se lesadas e reduzidas em número.

DESCRITORES: Corpo caloso. Hidrocefalia. Mielina. Rato Wistar.

Correspondence to:

Luiza da Silva Lopes Faculdade de Medicina de Ribeirão Preto/USP Departamento de Cirurgia e Anatomia Setor de Anatomia Av. Bandeirantes, 3900 14049-900 Ribeirão Preto, São Paulo, Brasil e-mail: luslopes@fmrp.usp.br

Conflict of interest: none Financial support: FAEPA

6-ARTIGO ORIGINAL

Brachial plexus variations in its formation and main branches

Valéria Paula Sassoli Fazan^{1,} André de Souza Amadeu^{2,} Adilson L. Caleffi^{2,} Omar Andrade Rodrigues Filho³

Fazan VPS, Amadeu AS, Caleffi AL, Rodrigues Filho OA. Brachial plexus variations in its formation and main branches. Acta Cir Bras [serial on line] 2003 vol 18 suppl 5. Available in www.scielo.br/acb.

ABSTRACT – **Purpose:** The brachial plexus has a complex anatomical structure since its origin in the neck throughout its course in the axillary region. It also has close relationship to important anatomic structures what makes it an easy target of a sort of variations and provides its clinical and surgical importance. The aims of the present study were to describe the brachial plexus anatomical