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A COMPUTERIZED MODEL OF THE CIRCLE OF WILLIS

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The circle of Willis (CW) is the most important anastomotic system of the cerebral arterial circulation.

CW is a heptagon with three anterior sides (the proximal portions of the two anterior cerebral arteries — ACA, and the anterior communicating artery — ACoA), two lateral sides (posterior communicating arteries — PCoA) and two posterior sides (the proximal portions of the two posterior cerebral arteries — PCA). It should be underlined that the middle cerebral arteries (MCA), the most important ones in supplying the brain, are oriented in the extension of the axis of the internal carotid arteries (ICA), and they are not integral parts of CW. Normally it is considered that the anterior and lateral components of CW, as well as MCA are tributary to the carotid system, whereas PCA belong to the vertebrobasilar system (1—5).

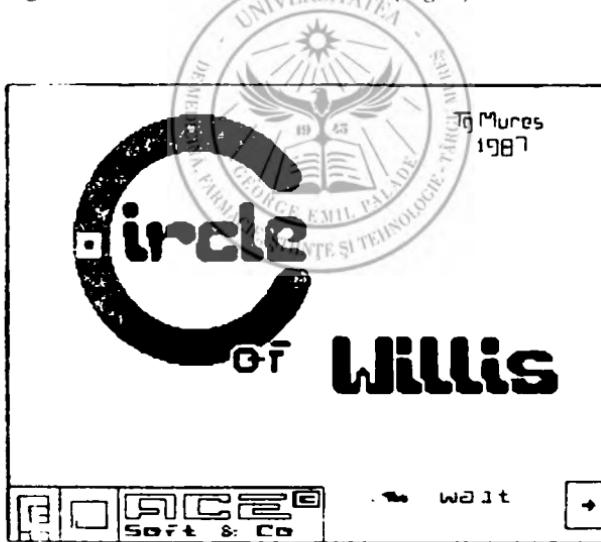
Regarded as a unitary and indivisible structure, CW functions well when it is perfectly constituted ("ideal" CW type with diameters of its components over 1 mm). Under these conditions CW succeeds in homogenizing and equilibrating the perfusion pressures of the three blood sources (the two ICA and basilar artery — BA), thus ensuring the redistribution of blood in the cerebral arteries. The functionality of CW is considered potential, becoming real either in normal conditions (rotation and extension of the head, when one of the vertebral arteries can be compressed), or in pathological conditions, when there occurs a total or partial interruption of the pre-CW cerebral blood sources (4.6—14).

The functionality of CW becomes unforeseeable with regard to its anatomical varieties especially when one or more component segments are missing or their diameter is under 1 mm. Detailed anatomical studies have shown that CW percentage with "ideal" structure in the series of unselected cases ranges between 25—52% (1.2.15—19). This percentage is lower in the series of cases with cerebrovascular lesions (20—23).

The presented data suggested us to create a computerized model of CW capable to simulate any modifications in its anatomical structure, as well as any variation of pre- and intra-CW blood flow (BF), and which can offer finally the possibilities of redistributing blood towards the brain. Special literature reported some electric, hydrodynamic, mathematic or electronic models of CW, which however, have limited capacities of simulating pre- intra- and post-CW circulation (24—31).

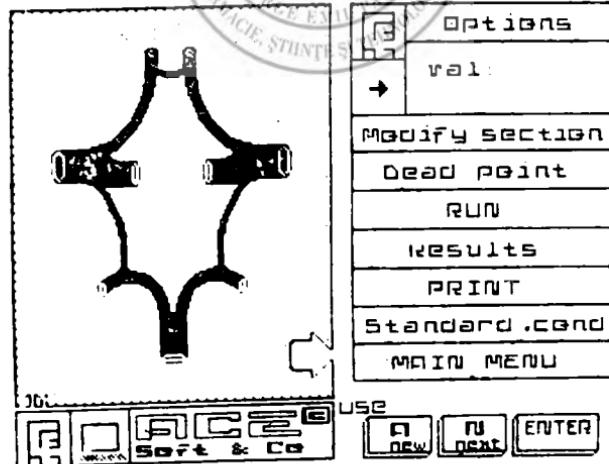
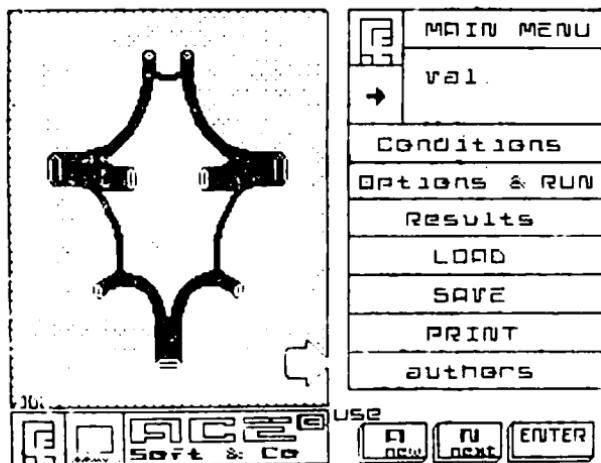
The model proposed by us can be running on a personal microcomputer, it is continuously improvable, makes use of the graphic possibilities of the computer, it is interactive, and it can be easily understood and utilized. The achieved program uses electric analogy of laminar flow for incompressible viscous fluids. This analogy is possibly due to the linear dependence of BF upon the difference of pressure. Obviously, the intensity of current corresponds to the fluid flow, the difference of potential to the difference of pressure, the electric resistance to the viscosity, the conservation theorem of the electric charge to the equation of continuity etc. An electric analogue was constructed to the circle, and for this a set of equations were deduced by using currents (flows) as variable, and as known values of CW geometric parameters (resistances) and potentials at certain points (arterial blood pressures accesible for measurements) were made use of. As the initial data can be modified at will, there is a possibility of simulating a great anatomic and hemodynamic variety practically applicable in determining post-CW BF under the conditions of stenosis of pre-CW arterial occlusions, or CW anomalies. The calculated parameters can be introduced in card-index or printed.

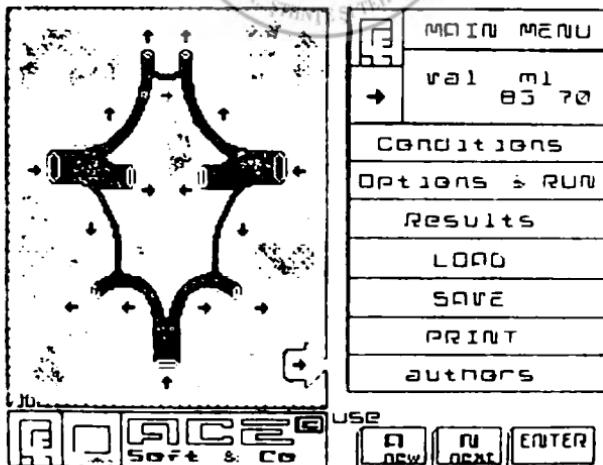
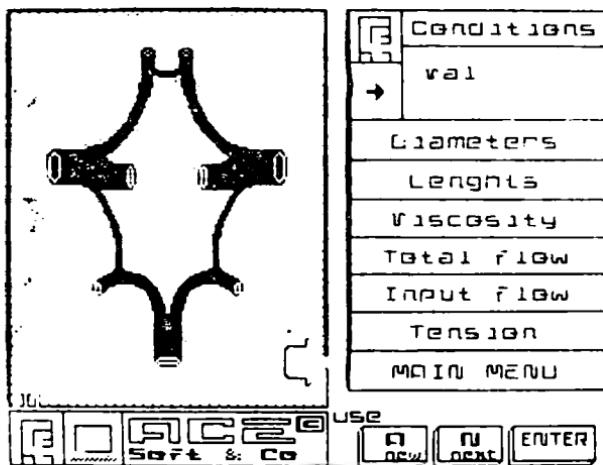
After running, the title of the program appears by itself exposing the first image on the screen of the monitor (Fig. 1).



Then the CW scheme follows, and the main menu comprises: conditions, options & run, results, load, save, print, authors (Fig. 2).

"Conditions" include the following parameters: diameters, lengths, viscosity, total flow, input flow, tension (blood pressure). After making the desired modifications, it is possible to enter again into the main menu (Fig. 3).





On passing to "options & run" the following sections are shown: modify section, dead point, run, results, print, standard conditions and again the main menu (Fig. 4).

The standard conditions of the program were established by taking into consideration the anatomical and physiological data from special literature and by one of us (I.P.), gathered for 20 years by studying in details the cerebral blood supply in over 2000 brains.

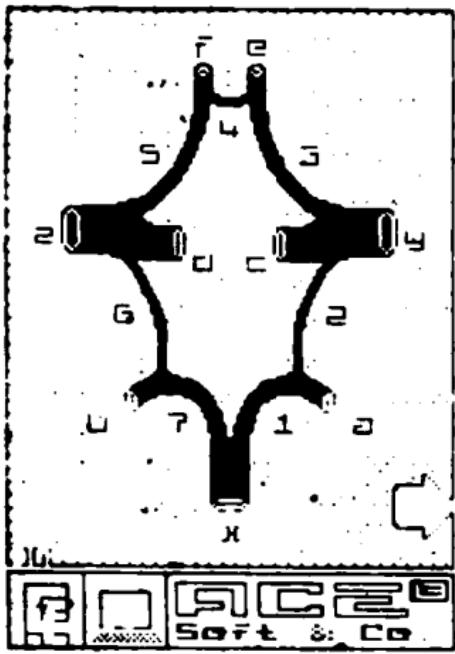
Thus, it was admitted that the lumen diameters of PCA and ACA are 3 mm, those of MCA 4 mm, and of ACoA and PCoA 1.5 mm. The lengths of CW components were considered to have the following dimensions: PCA 10 mm, ACA and PCoA 15 mm each, and ACoA 2 mm (1—4, 17—19, 32—38). The viscosity of the blood for 50% hematocrite was taken as equal to 0.04 poises (39,40). Total BF through the two ICA and through BA was evaluated up to 760 ml/minute for a brain of 1400 gr with mean cerebral BF of 54.3 ml/100 gr/minute (39, 41—48). Considering the volume of the brain supplied by the two arterial systems, it was admitted that through both ICA 630 ml/minute blood enters the brain (82.9% of total cerebral BF), and through BA 130 ml/minute blood (17.1%). On leaving CW, by making use of volume of brain supplied by each cerebral artery, and the data obtained by transcranial Doppler ultrasound method, the standard data of cerebral BF show the following values: 136 ml/minute for PCA (17.9%), 456.6 ml/minute for MCA (60.1%) and 167.4 ml/minute for ACA (22.0%). These values refer to the whole brain, and they are the same for both hemispheres (39, 49—53).

When the control „run“ is used, the program solves the set of equations. Then, the control „results“ shows the input, internal and output values and directions of BF. Finally, the control „print“ exposes a synoptic table with the following values: total flow, tension, viscosity, dead points and modified sections (Fig. 5; Fig. 6; Fig. 7).

In our future papers we shall present the results obtained by our model in certain particular situations of the cerebral circulation.

References

1. Alpers B.J., Berry R.G., Paddison R.M.: Arch. Neurol. Psychiat. (1959), 81, 409; 2. Riggs H. E., Rupp C.: Arch. Neurol. (1963), 8, 24; 3. Baptista A. G.: Acta Neurol. Scand. (1964), 40, 398; 4. Lazorthes G., Gouazé A.: C.R. Ass. Anat. (1968), 140, 1; 5. Lazorthes G., Gouazé A., Santini J.J., Lazorthes Y., Laffont J.: Neurochirurgie (1971), 17, 361; 6. Lowe R. D.: Lancet (1962), 1, 395; 7. Braun J.P., Baumgartner J., Woringer E., Rust F.: Neurochirurgie (1966), 12, 741; 8. Hawkins T. D.: Clin. Radiol. (1966), 17, 203; 9. Rovira M.: Neurochirurgie (1966), 12, 733; 10. Lehrer H. Z.: Brain (1968), 91, 339; 11. Zülch K. J.: Patterns of the collateral circulation of the cerebral arteries, in: Zülch K. J. — Cerebral circulation and stroke., Springer-Verlag, Berlin (1971), 106; 12. Nornes H.: Acta Neurochir. (1973), 28, 165; 13. Levy L. L., Wallace J. D., Stolwijk J. A. J., Poindexter E. R.: Stroke (1976), 7, 147; 14. Fukuyama H., Akiguchi I.,



	input	
↑	130	
↓	115	
→	515	internal
←	65	
↑	80	
↓	70	
→	80	
←	70	
↑	65	
↓	60	
→	68	Output
←	68	
↑	220	
↓	220	
→	200	
←	200	
↑	85	
↓	70	
→	70	

- FLOWS table



Circulation

Total flow (ml) 760

Tension (mmHg) 120/80

Viscosity (Pa) 0.6

Dead points

2 4 6

Sections model

1	+
2	+
3	+
4	+
5	+
6	+
7	+
8	+
9	+
10	+
11	+
12	+
13	+
14	+
15	+



- On conductors

- Kameyama M., Taky W., Handa H., Higa T., Tanaka S., Fujita T., Torizuka K.: *J. Neurol.* (1983), 230, 7; 15. Fisher C. M.: *Vasc. Dis.* (1965), 2, 99; 16. Baptista A. G.: *Acta Neurol. Scand.* (1966), 42, 161; 17. Lazorthes G., Gouazé A.: *C.R. Ass. Anat.* (1970), 149, 826; 18. Saeki N., Rhoton A. L.: *J. Neurosurg.* (1977), 46, 563; 19. Lazorthes G., Gouazé A., Santini J. J., Salamon G.: *Anat. Clin.* (1979), 1, 241; 20. Alpers B. J., Berry R. G.: *Arch. Neurol.* (1963), 8, 398; 21. Fazio C., Fieschi C., Agnoli A., Bugiani O., Gottlieb A.: Fréquence et rôle des anomalies du polygone de Willis et de l'artériosclérose dans l'apoplexie cérébrale. *Symp. Intern. Circ. Cérébr.*, Ed. Sandoz, Paris (1966), 225; 22. Battacharji S. K., Hutchinson E. C., McCall A. J.: *Brain* (1967), 90, 747; 23. Pascu I., Popoviciu L., Lázár L.: *Rev. Roum. Neurol., Psychiat.* (1974), 11, 151; 24. Pallie W., Samarsinghe D. D.: *Brain* (1962), 85, 569; 25. Murray K. D.: *J. Neurosurg.* (1964), 21, 26; 26. Chao J. C., Hwang N. H. C.: *J. Biomechanics* (1971), 4, 141; 27. Himwich W. A., Clark M. E.: *J. Appl. Physiol.* (1971), 31, 873; 28. Chao J. C., Hwang N. H. C.: *J. Life Sci.* (1972), 2, 81; 29. Dürös J., Nádvorník P.: *J. Neurosurg. Sci.* (1977), 21, 243; 30. Nádvorník P., Dürös J.: *Acta Neurochir.* (1979), suppl. 28, 278; 31. Hillen B., Hoogstraten H. W., Post L.: *J. Biomechanics* (1986), 19, 187; 32. Lavieile J., Choux M., Sedan R.: *Neurochirurgie* (1966), 22, 717; 33. Perlmutter D., Rhoton A. L.: *J. Neurosurg.* (1976), 45, 259; 34. Zeal A. A.. Rhoton A. L.: *J. Neurosurg.* (1978), 48, 534; 35. Kamath S.: *J. Anat.* (1981), 133, 419; 36. El Khamlichi A., Azouazi M., Bellakhdar F., Ouhcein A., Lahlaidi A.: *Neurochirurgie* (1985), 31, 287; 37. Milenkovic Z., Vučetić R., Puzic M.: *Surg. Neurol.* (1985), 24, 563; 38. Orlandini G. E., Ruggiero C., Zecchi Orlandini S., Gulisano M.: *Acta Anat.* (1985), 123, 72. 39. Lazorthes G., Gouazé A., Salamon G.: Vascularisation et circulation de l'encéphale, Masson, Paris (1978). 28, 40. Wood J. H., Kee D. B.: *Stroke* (1985), 16, 765; 41. Lassen N. A., Hoedt-Rasmussen K., Sorensen S. C., Skinhøj E., Cronqvist S., Bodfoss B., Ingvar D. H.: *Neurology* (1963), 13, 719; 42. Geraud J., Bés A., Delpla M., Marc-Vergnes J. P., Guiraud B.: *Acta Neurol. Scand.* (1965), 41, suppl. 14, 169; 43. Ingvar D. H., Cronquist S., Ekberg R., Risberg J., Hoedt-Rasmussen K.: *Acta Neurol. Scand.* (1965), 41, suppl. 14, 72; 44. Fieschi C., Agnoli A., Battistini N., Bozzao L.: *Arch. Neurol.* (1966), 15, 653; 45. Hoedt-Rasmussen K.: *Acta Neurol. Scand.* (1967), 43, suppl. 27, 1; 46. McHenry L. C., Jaffe M. E., Goldberg H. I.: *Neurology* (1969), 19, 1198; 47. Olesen J., Paulson O.B., Lassen N. A.: *Stroke* (1971), 2, 519; 48. Sveinsdóttir E., Torlof P., Risberg J., Ingvar D. H., Lassen N. A.: *Europ. Neurol.* (1972), 6, 228; 49. Aaslid R., Markwalder T., Nornes H.: *J. Neurosurg.* (1982), 57, 769; 50. Arnolds B. J., von Reutern G. M.: *Ultrasound Med. Biol.* (1986), 12, 115; 51. Bishop C. C. R., Powell S., Rutt D., Browse N. L.: *Stroke* (1968), 17, 913; 52. Padayachee T. S., Kirkham F. J., Lewis R. R., Gillard J., Hutchinson M. C. E., Gosling R. G.: *Ultrasound. Med. Biol.* (1986), 12, 5; 53. Hennerici M., Rautenberg W., Sitzer G., Schwartz A.: *Surg. Neurol.* (1987), 27, 439.