Transcranial Doppler Ultrasound in the Evaluation of Collateral Blood Flow in Patients with Internal Carotid Artery Occlusion: Correlation with Cerebral Angiography

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PURPOSE: To determine the accuracy of transcranial Doppler (TCD) ultrasound for evaluation of collateral supply through the circle of Willis in patients with internal carotid artery (ICA) occlusion. **METHODS:** The evaluation of the collateral pathways through the circle of Willis with TCD ultrasound and with cerebral angiography was compared in 40 patients (30 men, 10 women; mean age, 55 ± 9 years) in a total of 44 ICA occlusions of which 43 had a suitable ipsilateral temporal bone window for TCD examination. **RESULTS:** By TCD, a patent anterior communicating artery is indicated by a reversal blood flow in the A1-segment of the anterior cerebral artery or by a prompt fall of blood velocity in the middle cerebral artery after compression of the nonoccluded contralateral carotid artery. In 42 of 43 instances of ICA occlusion, TCD and angiography agreed in the evaluation of a present or absent anterior communicating artery collateral supply. TCD's sensitivity was 95%, its specificity 100%. A collateral supply through the basilar artery was assumed with TCD when there was: (*a*) a basilar artery blood velocity of more than 70 cm/s; (*b*) a marked increase of basilar artery blood velocity after compression of the nonoccluded carotid artery; (*c*) an evident side-to-side asymmetry of the blood velocity of the posterior cerebral arteries with high blood velocity ipsilateral to the ICA occlusion. For evaluating the collateralization via the basilar artery, TCD and angiography agreed in 37 of 40 ICA occlusions. TCD's sensitivity was 87%, its specificity 95%. **CONCLUSIONS:** TCD is a reliable tool for the evaluation of the collateral supply in patients with ICA occlusions.

Index terms: Ultrasound, Doppler; Ultrasound, comparative studies; Arteries, stenosis and occlusion; Brain, blood supply; Cerebral angiography

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The annual rate of future ipsilateral stroke in patients with internal carotid artery (ICA) occlusion is between 0% and 7.7%, and is reported mostly to be between 3% and 5% (1–15). With the exception of a few studies (1, 14, 15), follow-up reports about the natural history of patients with ICA occlusions paid little attention to the role of the intracranial collateral supply on the development of future stroke. Recently one study revealed a significantly increased incidence of future ipsilateral strokes in patients with ICA occlusions whose state of cerebrovascular autoregulation distal to ICA occlusion was severely diminished (16). Cerebrovascular autoregulation is usually well preserved in patients with ICA occlusions when collateral blood flow is supplied by a lone patent anterior communicating artery (ACoA) or a patent ACoA accompanied by a collateralization via the basilar artery. It is impaired in patients with ICA occlusion who lack an ACoA collateralization and show a collateral supply through the vertebrobasilar artery system only or through a lone supraorbital artery (17, 18) (Müller M, Kessler CH, Maravic MV, et al, "Doppler-CO_2-Test in Carotid Occlusion: A Follow-up Study," presented at the Second European Conference on Stroke, June 1992,

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Lausanne). Therefore, the evaluation of the collateral supply seems to be of prognostic value, and the methods for its evaluation should reliably answer whether collateral blood flow via the ACoA and/or via the basilar artery (BA) exists. Cerebral angiography is risky especially in patients with cerebrovascular diseases (19–22). Because transcranial Doppler (TCD) ultrasound is noninvasive, it is a promising alternative to cerebral angiography for the evaluation of the collateral supply (23–26). To determine the accuracy of TCD for evaluation of the collateral supply, we compared the angiographic and TCD findings in patients with ICA occlusions.

Patients and Methods

In 40 consecutive patients with ICA occlusions having undergone both angiography and TCD examinations, we compared the results of the TCD examination with those of the cerebral angiography. Additionally, the extracranial arteries were investigated by the continuous-wave Doppler sonography (Velocimetre D.800, Delalande, France, 4 MHz probe). Informed consent was obtained from the patients for the angiography as well as for the ultrasound examinations. Thirty of the 40 patients were men, 10

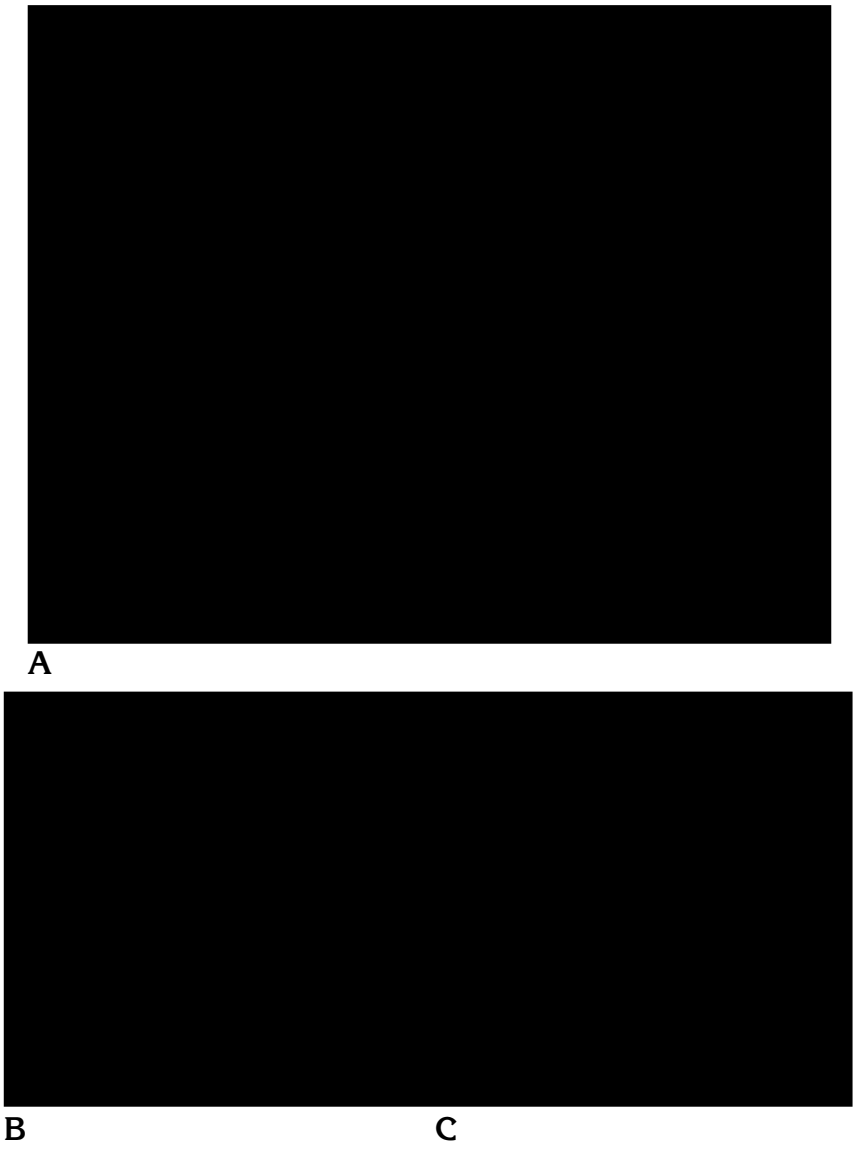


Fig 1. Transcranial Doppler (TCD) recordings of an angiographically proved collateral supply through the ACoA in a 54-year-old patient with an occlused right ICA caused by a carotid artery dissection.

A, The arrows in the right upper corners of the TCD registrations mark the blood flow direction: *arrow to the right* indicates flow towards the probe, *arrow to the left*, flow away from the probe. A high orthograde (away from the probe) mean blood velocity in the left ACA (128 cm/s, insonated from temporal left) is accompanied by a high retrograde (towards the probe) mean blood velocity in the A1 segment of the right ACA (80 cm/s, insonated from temporal right), suggesting collateral blood flow from the left ICA through the A1 segment of left ACA and ACoA, and the A1 segment of right ACA to right MCA.

B and *C,* The corresponding angiography confirms the TCD recordings. The lateral view shows the occlusion (*arrow*) of the ICA, the anteroposterior view shows the ACA and MCA perfusion from the contralateral side via the ACoA.

VA indicates vertebral artery.

women; their mean age (± standard deviation) was 55 ± 9 years (range, 34 to 72 years). Occlusion of the ICA was bilateral in 4 patients and unilateral in 36 patients, of whom 6 exhibited an additional stenosis (50% to 90%) of the contralateral ICA. The grading of the contralateral ICA stenosis followed the recommended continuous-wave Doppler criteria given by von Reutern/von Büdingen (27).

Cerebral angiography was performed by the intraarterial digital subtraction technique. The carotid arteries were visualized bilaterally in all 40 patients. The vertebrobasilar artery system was angiographically investigated in 37 of the 40 patients. Angiography did not usually include compression tests of the nonoccluded contralateral ICA.

The TCD examinations were performed with the EME TC 2-64 equipment (EME, Überlingen, Germany, 2 MHz hand-held probe). For detailed information about the physical and technical basis of TCD see previous descriptions (25, 26, 28, 29). TCD recording requires a temporal and a nuchal bone window through which ultrasound transmission is possible. The identification of the intracranial vessels of the circle of Willis is possible by the depth of the sample volume and the flow direction. To detect the middle cerebral artery (MCA) and the anterior cerebral artery (ACA) the probe must be positioned at the temporal skull above the zygomatic arch in a slightly anterior direction. The MCA is found in 50- to 55-mm depth with the blood flow directed toward the probe. The ACA is characterized by a blood flow away from the probe in 60- to 70-mm depth. Positioning the probe more posterior and inferior, the posterior cerebral artery (PCA) is identified in 55- to 70-mm depth with the flow directed towards the probe while insonating the P1 segment and away from the probe while insonating the P2 segment of the PCA. From this site of the probe the PCoA also can be insonated in 60- to 70-mm depth. By the transnuchal approach through the foramen magnum, the basilar artery was identified in 85- to 100-mm depth with the flow away from the probe.

To estimate mean blood velocities in the patients' MCA, ACA, PCA, and BA as an effect of collateralization, the mean blood velocity of the MCA, ACA, PCA, and BA were evaluated in 63 healthy subjects (33 women, 30 men; mean age ± SD, 44 ± 17; range, 17 to 80 years) without any cerebrovascular risk factors and without any central nervous system disease.

By compression of the nonoccluded carotid artery, the TCD allows the testing of a patent ACoA and PCoA (for examples see "Results"). Compression tests were performed in all patients except those with stenosis of 50% or more of the contralateral ICA or in the condition of ICA occlusion bilaterally.

The chi-square test was used for comparison of TCD and angiography. Using angiography as the reference method, the sensitivity of TCD was determined as the ratio [true positives/(true positives + false negatives)]·100%, the specificity of TCD as [true negatives/(true negatives + false positives)]·100% (29).

Results

In the control group, mean blood velocity was 56 ± 13 cm/s (range, 28 to 90 cm/s) in the MCA (n = 119), 46 ± 10 cm/s (range, 20 to 72)

Fig 2. TCD recordings of an ACoA collateral pathway in a 60-year-old patient with right-sided atherosclerotic occlusion of the ICA. Insonating from temporal right, mean blood velocity is orthograde and normal in the A2 segment (44 cm/s) of the right ACA, but mean blood velocity is retrograde and highly elevated in the A1 segment (96 cm/s) of the right ACA, indicating that the collateral blood supply via the ACoA flows through the proximal segment of the right ACA only and does not provide blood supply for a leptomeningeal anastomosis through the distal ACA.

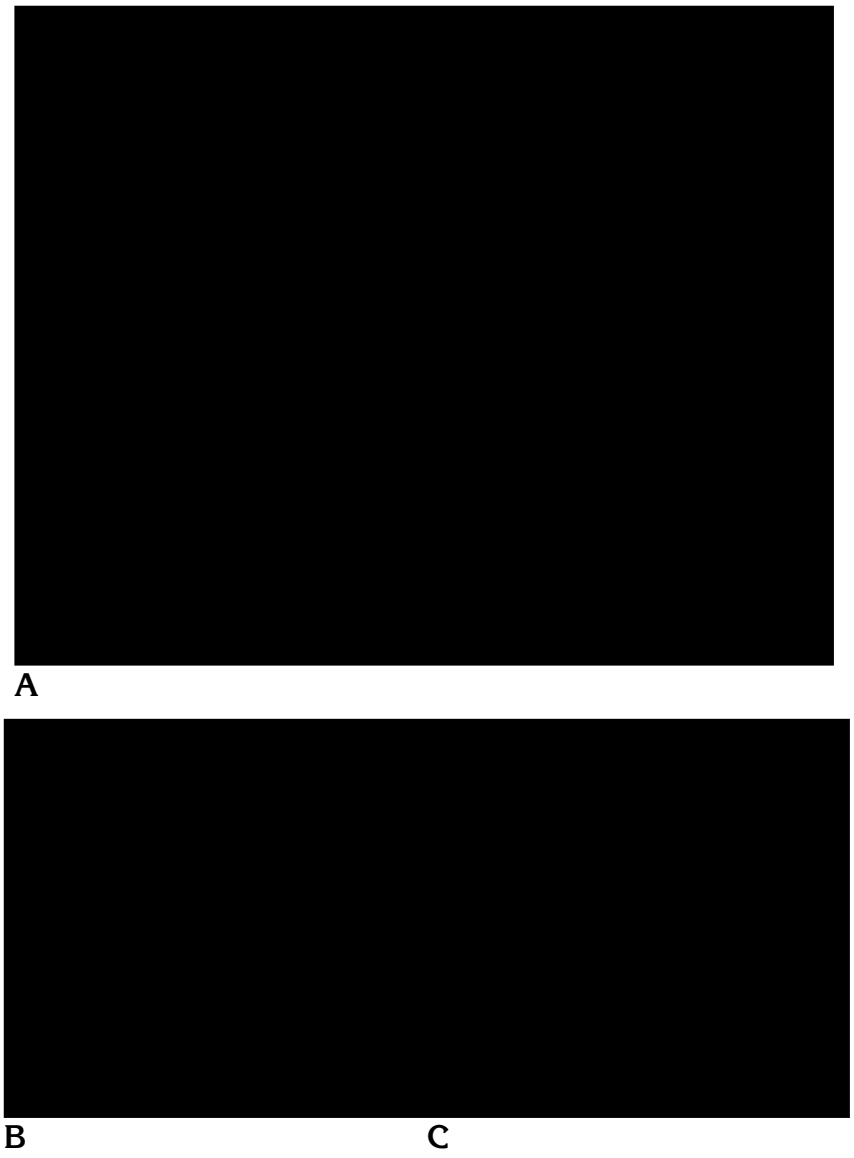


Fig 3. TCD recordings of a patent ACoA and PCoA by compression of the nonoccluded ICA in a 47-year-old patient with occlusion of the right ICA.

A, The bars indicate the duration of compression of the left carotid artery. Although the prompt drop of mean blood velocity in both ACAs and the right MCA indicate a patent ACoA, the increase of the blood velocity in the BA evidently shows a patent PCoA.

B and *C,* The corresponding angiography confirms the TCD recordings. The perfusion of the ACA and MCA is provided by the contralateral ICA via the ACoA and by the BA via the PCoA.

in the ACA (n = 109), 40 ± 6 cm/s (range, 24 to 50 cm/s) in the PCA (n = 79), and 43 ± 10 cm/s (range, 28 to 65 cm/s) in the BA (n = 43). A collateral flow was assumed if mean blood velocity exceeded the upper limit of the normal velocity range.

Because of an inadequate temporal bone window on one side of a patient with ICA occlusion bilaterally, the comparison between TCD and angiography for evaluating the collateral supply through the ACoA was possible in 43 of the 44 ICA occlusions. In Figures 1 through 3, the characteristic TCD findings are presented for evaluating the collateral supply through the ACoA. By TCD, a collateral supply through the ACoA is indicated by, first, retrograde high blood velocity in the A1 segment of the ACA ipsilateral to the ICA occlusion; second, compression of the contralateral carotid artery followed by a prompt reduction of the blood velocity in the MCA ipsilateral to the ICA occlusion. When none of these characteristic findings are demonstrable, a patent ACoA is excluded by TCD. Referring to these TCD criteria, Table 1 shows the highly significant correlation between TCD and cerebral

TABLE 1: ACoA collateral pathway: evaluation by transcranial Doppler ultrasonography (TCD) and cerebral angiography in patients with ICA occlusions (n = 43)

		Transcranial Doppler Ultrasonography	
		Absent	Patent
Angiography	Absent	12	—
	Patent	1	30
χ^2: $P < .0001$			

angiography for evaluating the collateral supply through the ACoA ($P < .0001$); sensitivity of TCD was 95%, its specificity, 100%.

Angiography of the vertebrobasilar artery system was performed in 37 of the 40 patients, allowing the angiographic evaluation of the collateral supply through the BA in a total of 40 ICA occlusions. Figures 3 through 5 represent characteristic TCD findings. Three conditions indicate a collateral supply through the BA: first, a high mean blood velocity in the BA; second, independent of BA's mean blood velocity, a markedly elevated mean blood velocity in the PCA ipsilateral to the ICA occlusion as compared with the contralateral PCA; third, after compression of the nonoccluded carotid artery, a marked increase in BA's blood velocity indicates a patent PCoA if the PCoA cannot be insonated transtemporally. If none of these conditions could be recorded by TCD, no collateral supply via the BA was assumed by TCD. Indeed, in three of the 40 ICA occlusions angiography showed small PCoAs with a marginal blood supply to the MCA ipsilateral to the ICA occlusion, whereas TCD failed to demonstrate this pathway. Comparing these TCD criteria with the corresponding angiographic findings (Table 2), the correlation was highly significant ($P < .0001$) and TCD's sensitivity was 87% and its specificity 95% for evaluating the collateral supply via the BA.

Discussion

The TCD has been recommended by the American Association of Neurology for the evaluation of the collateral pathways in the condition of an ICA occlusion (31). The great advantage of TCD is its noninvasiveness and its ability for repeated examinations without any risk to patients' health. The major disadvantage of TCD is its dependence on the temporal and nuchal bone windows, which are more frequently absent in women and older

Fig 4. TCD recordings of collateral supply via a patent ACoA and PCoA evaluated by compression of the nonoccluded left ICA in a 35-year-old patient with right-sided occlusion of the ICA eventually caused by an angiographically proved carotid artery dissection. After compression of the left ICA, as indicated by the bars, the prompt drop of the blood velocity in the right MCA reveals the patent ACoA. In both the BA (insonated transnuchally) and the PCoA (insonated transtemporally) compression of the left carotid artery leads to a further increase of the primarily elevated mean blood velocities (86 cm/s in both arteries). The mean blood velocity in the PCA (46 cm/s) is at rest and during the compression test within the normal range, not indicating a leptomeningeal collateral supply.

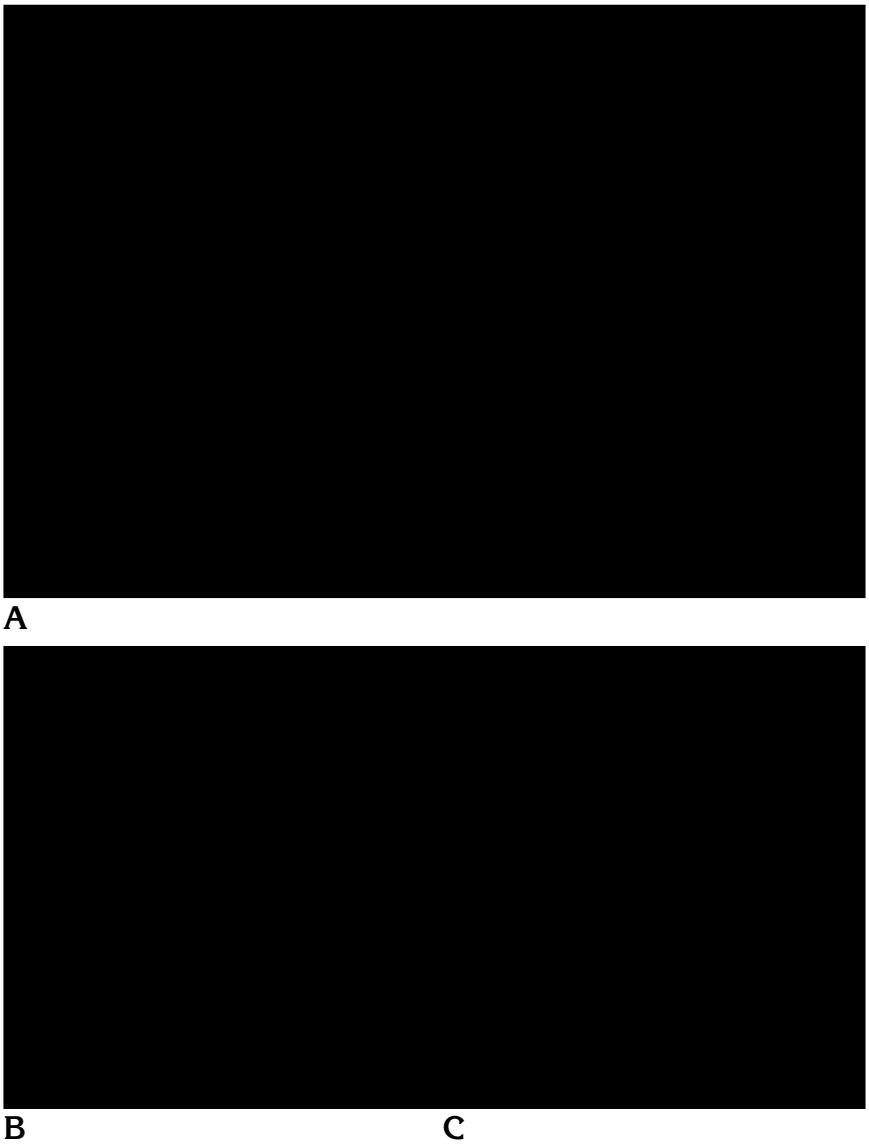


Fig 5. TCD recordings of a collateralization through the BA via leptomeningeal anastomoses of the PCA in a 50-year-old patient with an atherosclerotic occlusion of the right ICA.

A, By continuous-wave Doppler ultrasound (not shown), the left VA was strong, the right VA was classified as hypoplastic or absent. By TCD, the highly elevated mean blood velocity in the BA (98 cm/s, insonated transnuchally) is accompanied by an even higher mean blood velocity in the right PCA (P2 segment, 78 cm/s, insonated from temporal right) as compared with the normal mean blood velocity in the left PCA (P1 segment, 36 cm/s, insonated from temporal left). Compression of the left VA, as indicated by the bars, leads to a prompt and dramatic fall of blood velocity in the right PCA and MCA, indicating collateral blood flow via leptomeningeal anastomoses between right PCA and MCA.

B and *C,* The corresponding angiography shows a dominant blood supply through the left VA, whereas the right VA is hypoplastic, and the collateralization via leptomeningeal anastomosis of the right PCA.

TABLE 2: Basilar artery collateral pathway: evaluation by TCD ultrasonography and cerebral angiography in patients with ICA occlusions (n = 40)

		Transcranial Doppler Ultrasonography	
		Absent	Patent
Angiography	Absent	17	1
	Patent	3	19
χ^2: $P < .0001$			

patients than in men and younger patients, and its dependence on the experience of the examiner. An adequate temporal bone window usually will be found in about 90% of the patients, but the transnuchal approach to the BA is able to be used in only 70% of the patients (29, 32). The newly introduced color-coded transcranial duplex ultrasound can help to locate the bone windows more quickly but is also restricted principally to the limitations mentioned above. A patent ACoA can easily be shown by TCD, even without compression tests. Our results confirm previous studies that found for the TCD, as compared with angiography, a sensitivity between 84% and 94% and a specificity of 92% for the evaluation of the

collateral flow through the anterior part of the circle of Willis (25, 26).

Our results in the evaluation of the collateral flow through the BA system are comparable to the results of others (25, 26) who have found for TCD a sensitivity of 86% and a specificity of 92% for the evaluation of the collateral supply through the BA distribution, compared with angiography. The evaluation of the collateral supply through the BA distribution is more difficult and sometimes more time consuming than the evaluation of the collateral flow in the anterior part of the circle of Willis. As demonstrated in Figure 3, in some cases only the compression of the nonoccluded carotid artery will prove a patent PCoA while insonating the BA. Furthermore, leptomeningeal collateral flow may not be recognized if the PCA is not regularly insonated or its insonation is impossible because of an inadequate temporal bone window. At the least, TCD might not be able to detect marginal collateral flow through a small PCoA, although a PCoA is significantly more frequently insonated transtemporally in patients with severe occlusive carotid artery disease than in patients without occlusive carotid artery disease (24).

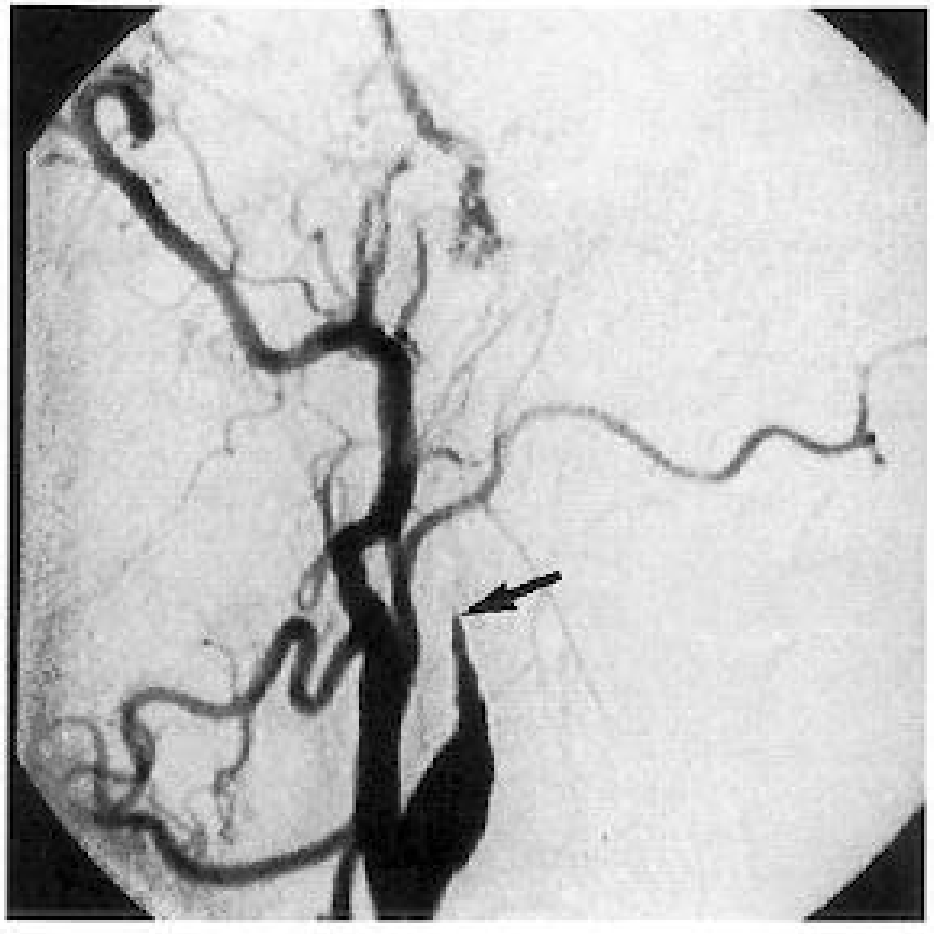
Compression tests are especially helpful in the evaluation of various types of collateral blood supply originating through the BA. But one must keep in mind the compression's risk of complications, especially the possibility of thromboembolic material from an ICA plaque.

For the evaluation of the collateral blood flow in patients with ICA occlusion, TCD is a reliable method, especially for the evaluation of the ACoA. In patients with a patent ACoA, the cerebrovascular vasomotor reactivity as a more global hemodynamic parameter is usually well preserved, whereas it is severely compromised in patients without a patent ACoA (17, 18). Depending on the experience of the TCD examiner, in the individual patient with an adequate temporal and transnuchal bone window the risk of angiography must be weighed against the potentially small benefit of angiography-proved information about the collateral supply. In patients unable to be insonated, angiography still will be needed for the evaluation of collateral blood supply.

References

1. Barnett HJM. Delayed cerebral ischemic episodes distal to occlusion of major cerebral arteries. *Neurology* 1978;28:769–774
2. Bornstein NM, Norris JW. Benign outcome of carotid occlusion. *Neurology* 1989;38:6–8
3. Cote R, Barnett HJM, Taylor DW. Internal carotid occlusion: a prospective study. *Stroke* 1983;14:898–902
4. Dyken LM, Klatte E, Kolar OJ, et al. Complete occlusion of common or internal carotid arteries. *Arch Neurol* 1974;30:343–346
5. Fields WS, Lemak NA. Joint study of extracranial arterial occlusion, X: internal carotid artery occlusion. *JAMA* 1976;235:2734–2738
6. Fritz VU, Voll ChL, Levien L. Internal carotid artery occlusion: clinical and therapeutic implications. *Stroke* 1985;16:940–944
7. Furlan AJ, Whisnant JP, Baker HL. Long-term prognosis after carotid artery occlusion. *Neurology* 1980;30:986–988
8. Grillo P, Patterson RH. Occlusion of the carotid artery: prognosis (natural history) and the possibilities of surgical revascularization. *Stroke* 1975;6:17–20
9. Hardy WG, Lindner DW, Thomas LM, et al. Anticipated clinical course in carotid artery occlusion. *Arch Neurol* 1962;6:138–150
10. Hennerici M, Hülsbömer H-B, Rautenberg W, et al. Spontaneous history of asymptomatic internal carotid occlusion. *Stroke* 1986; 17:718–722
11. Nicholls SE, Kohler TR, Bergelin RO, et al. Carotid artery occlusion: natural history. *J Vasc Surg* 1986;4:479–485
12. Sacquegna T, De Carolis P, Pazzaglia P, et al. The clinical course and prognosis of carotid artery occlusion. *J Neurol Neurosurg Psychiatry* 1982;45:1037–1039
13. The EC/IC Bypass Study Group. Failure of extracranial-intracranial arterial bypass to reduce the risk of ischemic stroke: results of an international randomised trial. *N Engl J Med* 1985;313:1191–1200
14. Bogousslavsky J, Regli F, Hungerbühler J-P, et al. Transient ischemic attacks and external carotid artery. *Stroke* 1981;12: 627–630
15. Bradac GB, Kaden B, Oppel F, et al. Occlusion of internal carotid artery: further clinical angiographic, and therapeutic considerations. *Neuroradiology* 1984;26:445–450
16. Kleiser B, Widder B. Course of carotid artery occlusions with impaired cerebrovascular reactivity. *Stroke* 1992;23:171–174
17. Ringelstein EB, Otis SM. Physiological testing of vasomotor reserve. In: Newell DW, Aaslid R, eds. *Transcranial Doppler*. New York: Raven Press, 1992:83–99
18. Weiller C, Ringelstein EB, Reiche W, et al. Clinical and hemodynamic aspects of low flow infarcts. *Stroke* 1991;22:1117–1123
19. Dion JE, Gates PC, Fox AJ, et al. Clinical events following neuroangiography: a prospective study. *Stroke* 1987;18:997–1007
20. Grzyska U, Freitag J, Zeumer H. Selective cerebral intraarterial DSA. *Neuroradiology* 1990;32:296–299
21. Mani RL, Eisenberg RL, MacDonald EJ Jr, et al. Complications of catheter cerebral angiography: analysis of 5000 procedures, I: criteria and incidence. *AJR Am J Roentgenol* 1978;131:861–865
22. Davies KN, Humphrey PR. Complications of cerebral angiography in patients with symptomatic carotid territory ischaemia screened by carotid ultrasound. *J Neurol Neurosurg Psychiatry* 1993;56: 967–972
23. Hennerici M, Rautenberg W, Schwartz A. Transcranial Doppler ultrasound for the assessment of intracranial arterial flow velocity, II. *Surg Neurol* 1987;27:523–532

24. Schneider PA, Rossman ME, Berstein EF, et al. Effect of internal carotid artery occlusion on intracranial hemodynamics: transcranial doppler evaluation and clinical correlation. *Stroke* 1988;19: 589–593
25. Lindegaard KF, Bakke SJ, Grolimund P, et al. Assessment of intracranial hemodynamics in carotid artery disease by transcranial Doppler ultrasound. *J Neurosurg* 1985;63:890–898
26. Grolimund P, Seiler RW, Aaslid R, et al. Evaluation of cerebrovascular disease by combined extracranial and transcranial doppler sonography: experience in 1,039 patients. *Stroke* 1987;18:1018–1024
27. Reutern vGM, Büdingen vHJ. *Ultraschalldiagnostik der hirnversorgenden Arterien*. Stuttgart New York: Georg Thieme, 1989: 47–209
28. Aaslid R, Markwalder TM, Nornes H. Noninvasive transcranial doppler ultrasound recording of flow velocity in basal cerebral arteries. *J Neurosurg* 1982;57:769–774
29. Fujioka KA, Douville CM. Anatomy and freehand examination techniques. In: Newell DW, Aaslid R, eds. *Transcranial Doppler*. New York: Raven Press, 1992:9–32
30. Phillips WC, Scott JA, Blasczcynski G. Statistics for diagnostic procedures. *AJR Am J Roentgenol* 1983;140:1265–1270
31. American Academy of Neurology: Assessment transcranial doppler: report of the Therapeutics and Technology Assessment Subcommittee. *Neurology* 1990;40:680–681
32. Büdingen HJ, Staudacher T. Evaluation of vertebrobasilar disease. In: Newell DW, Aaslid R, eds. *Transcranial Doppler*. New York: Raven Press, 1992:167–195



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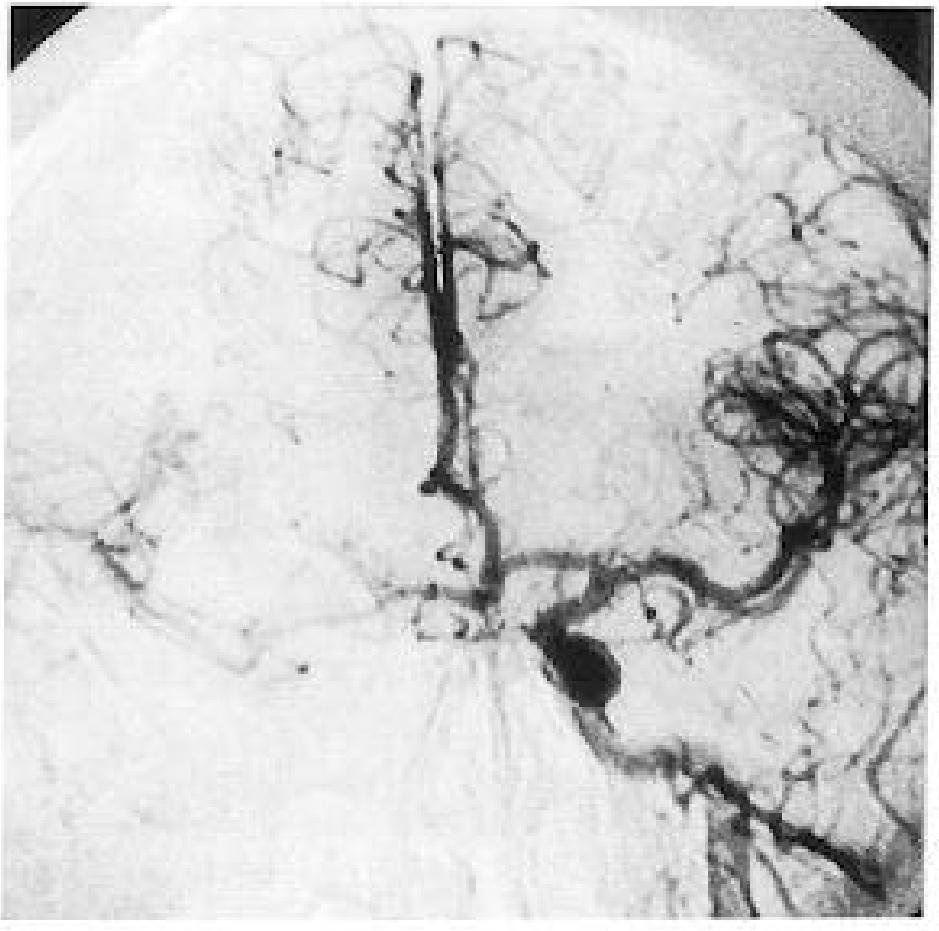

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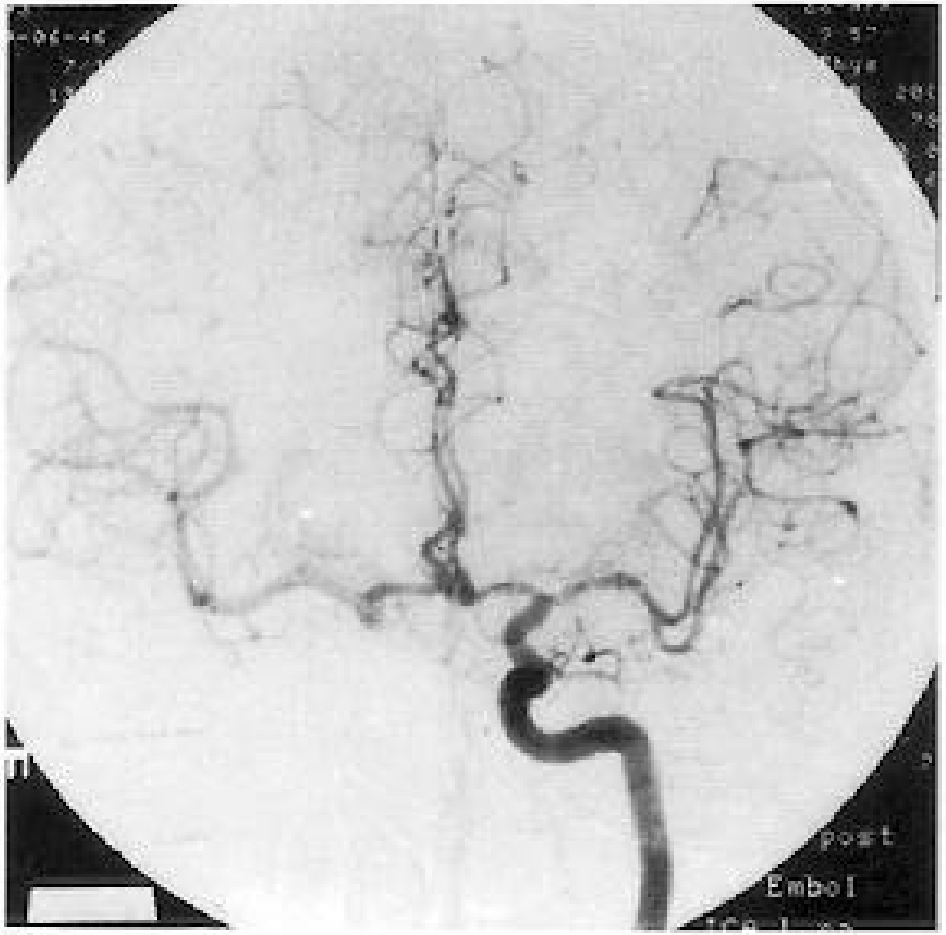

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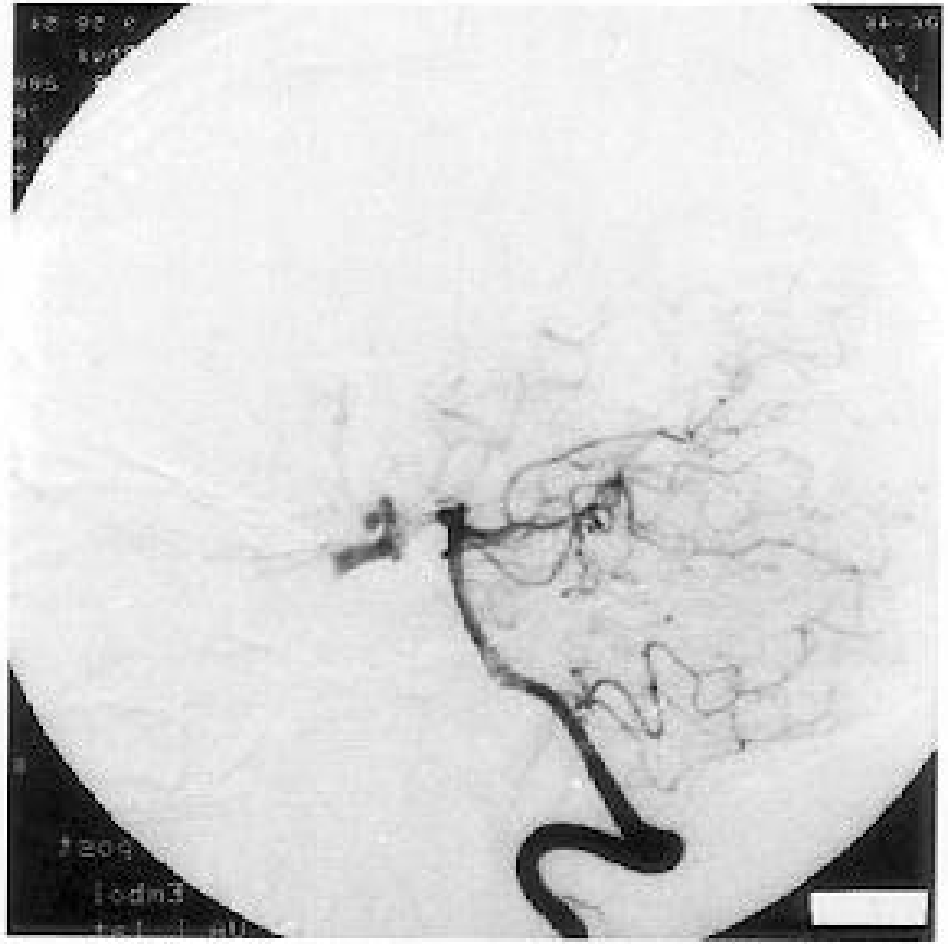

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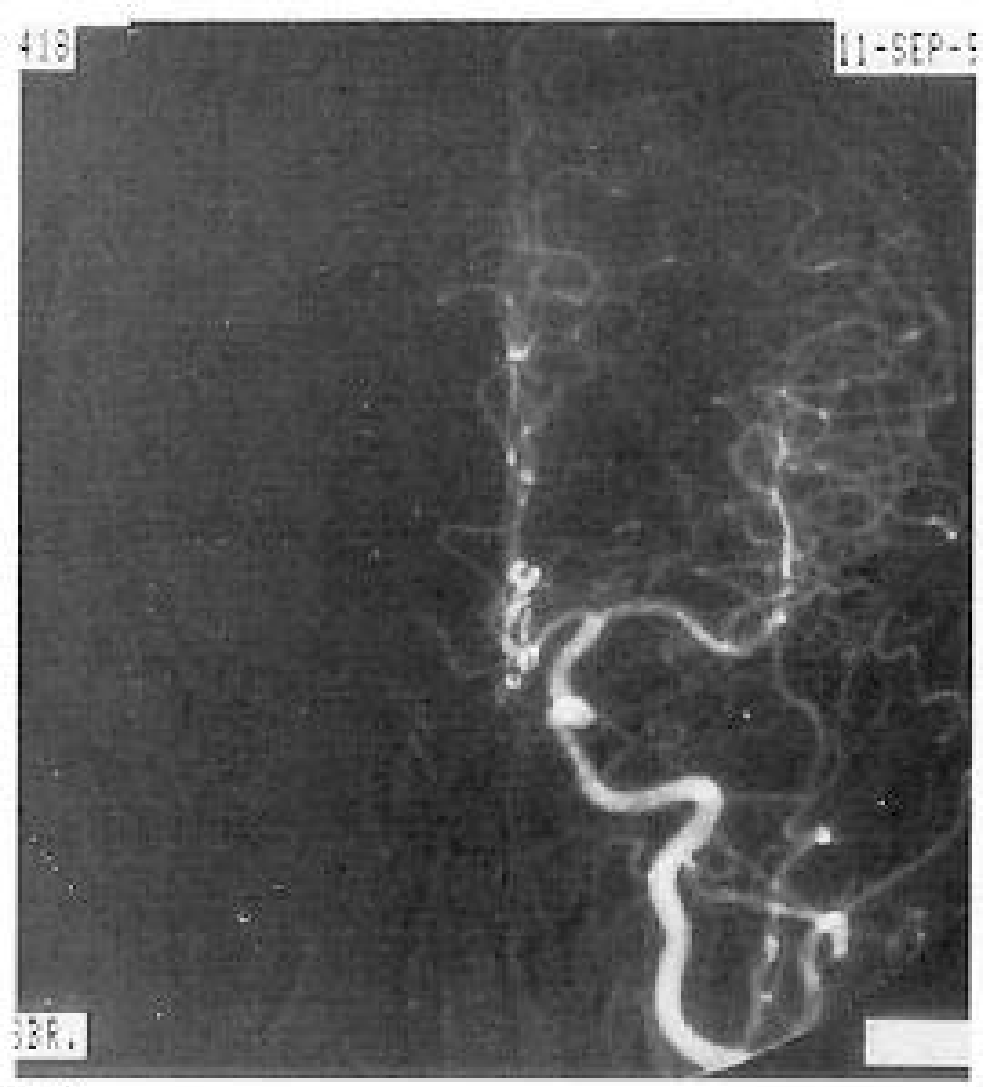

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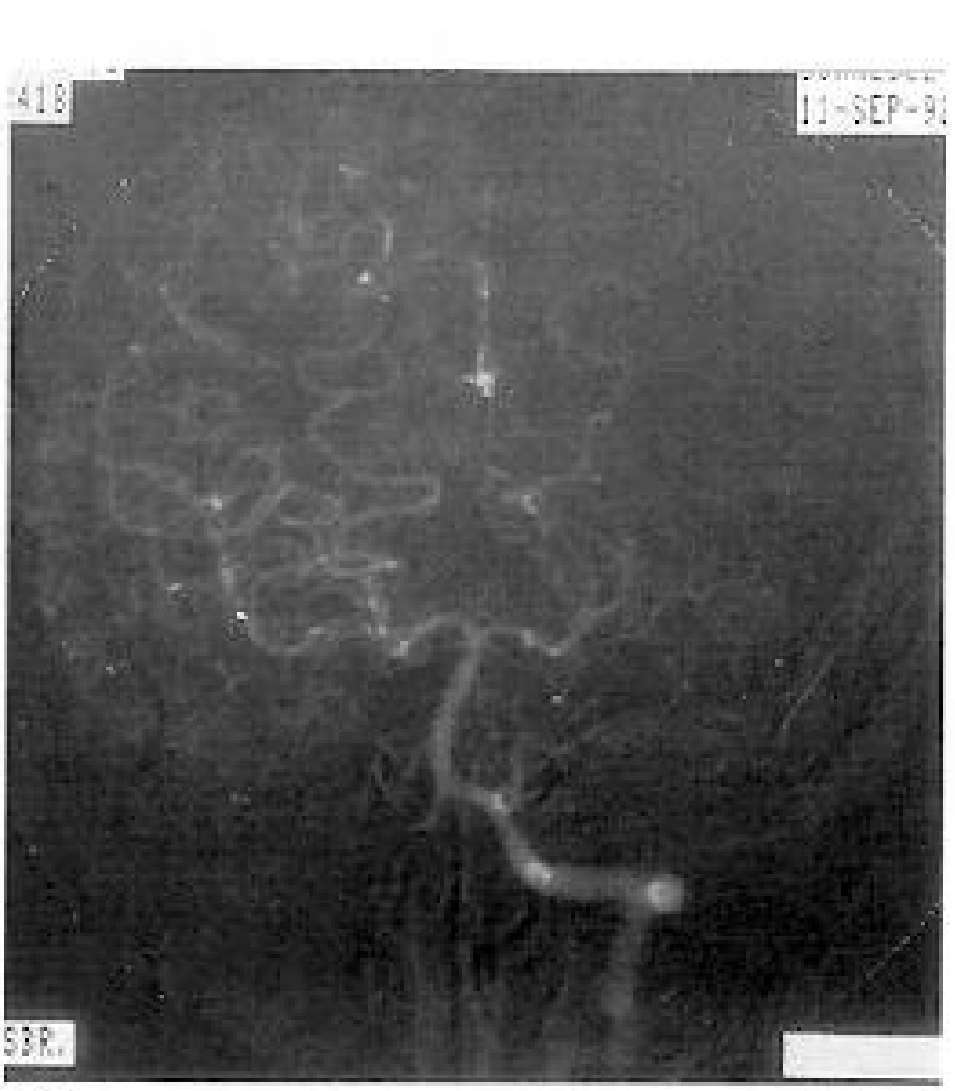

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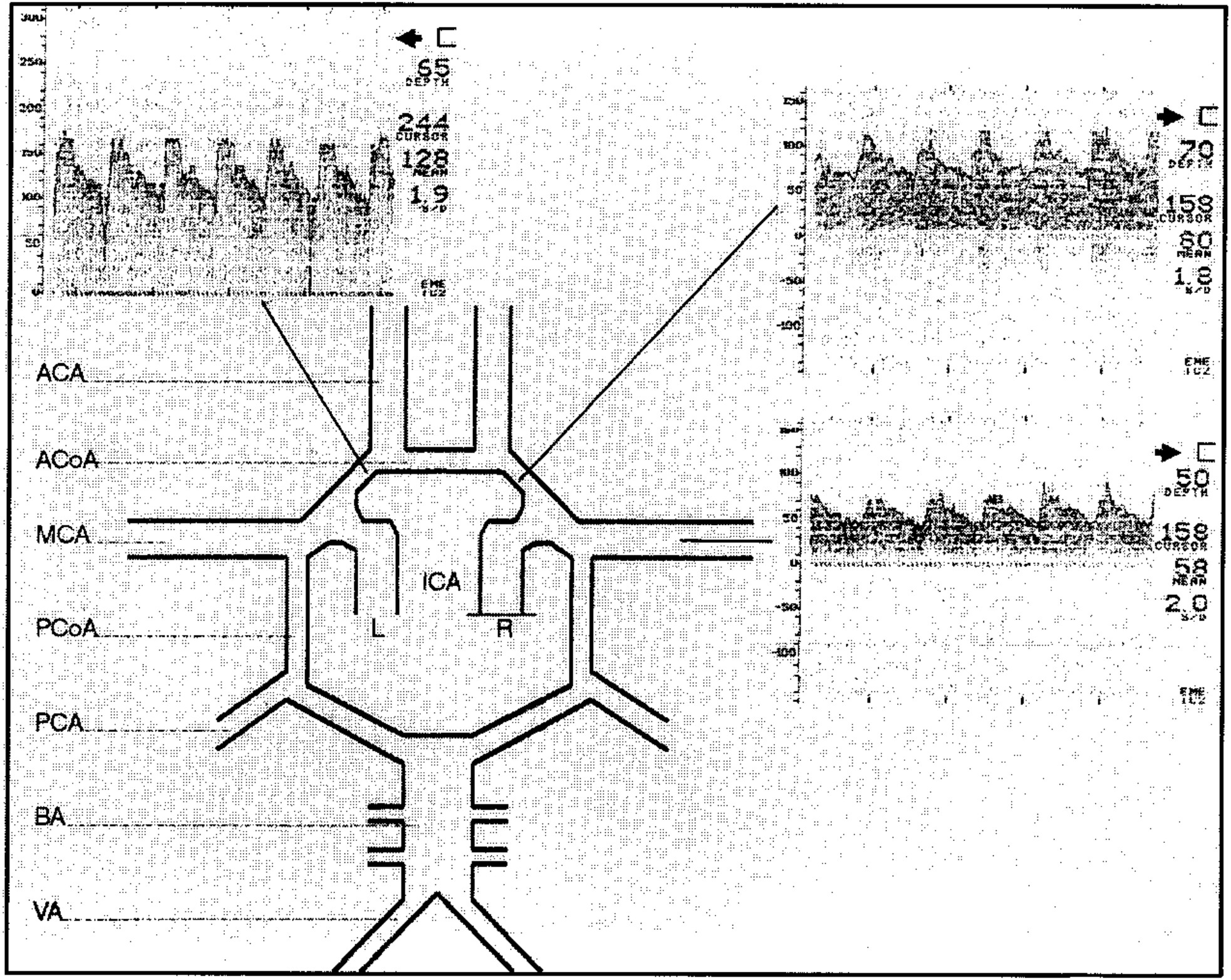
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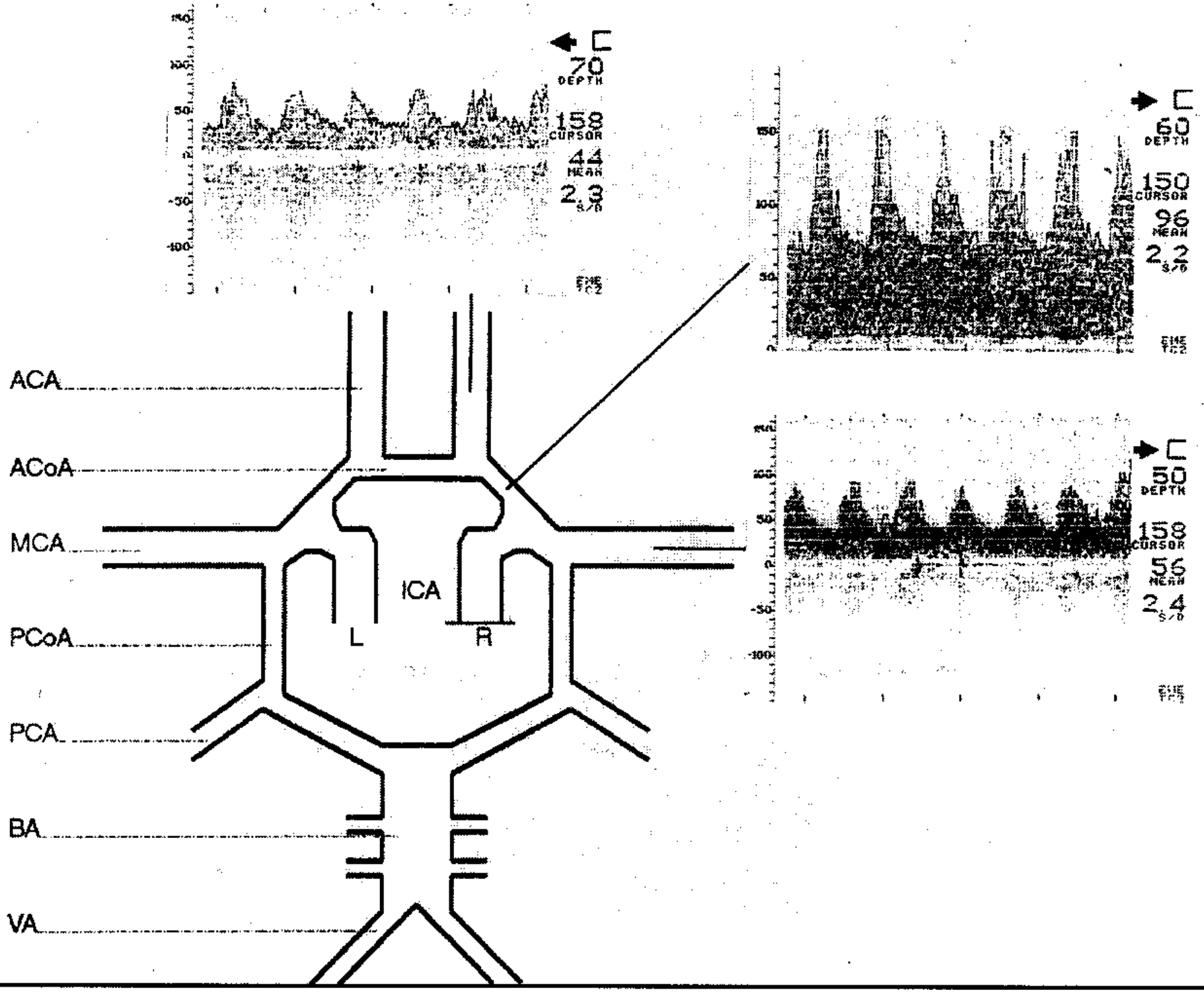
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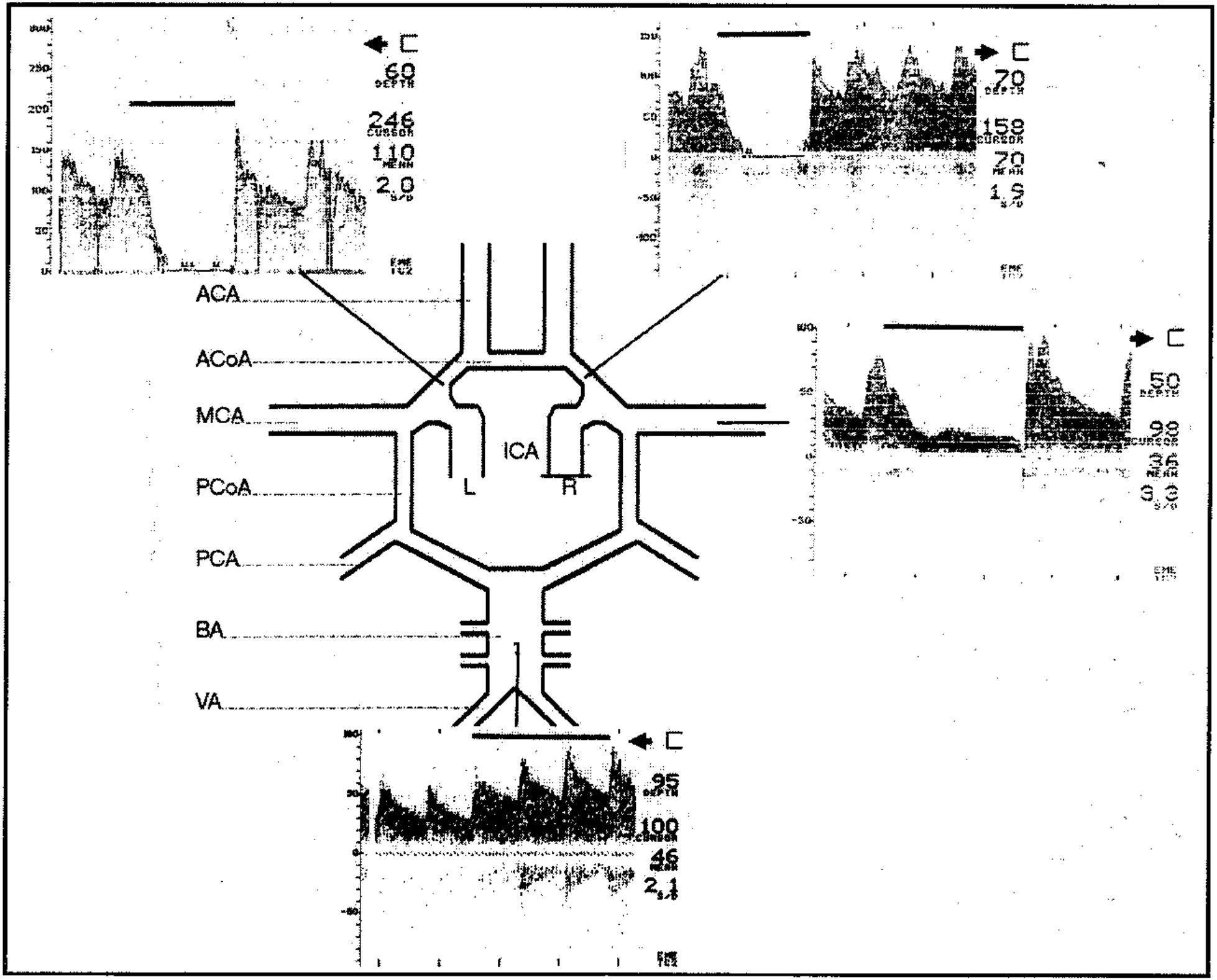

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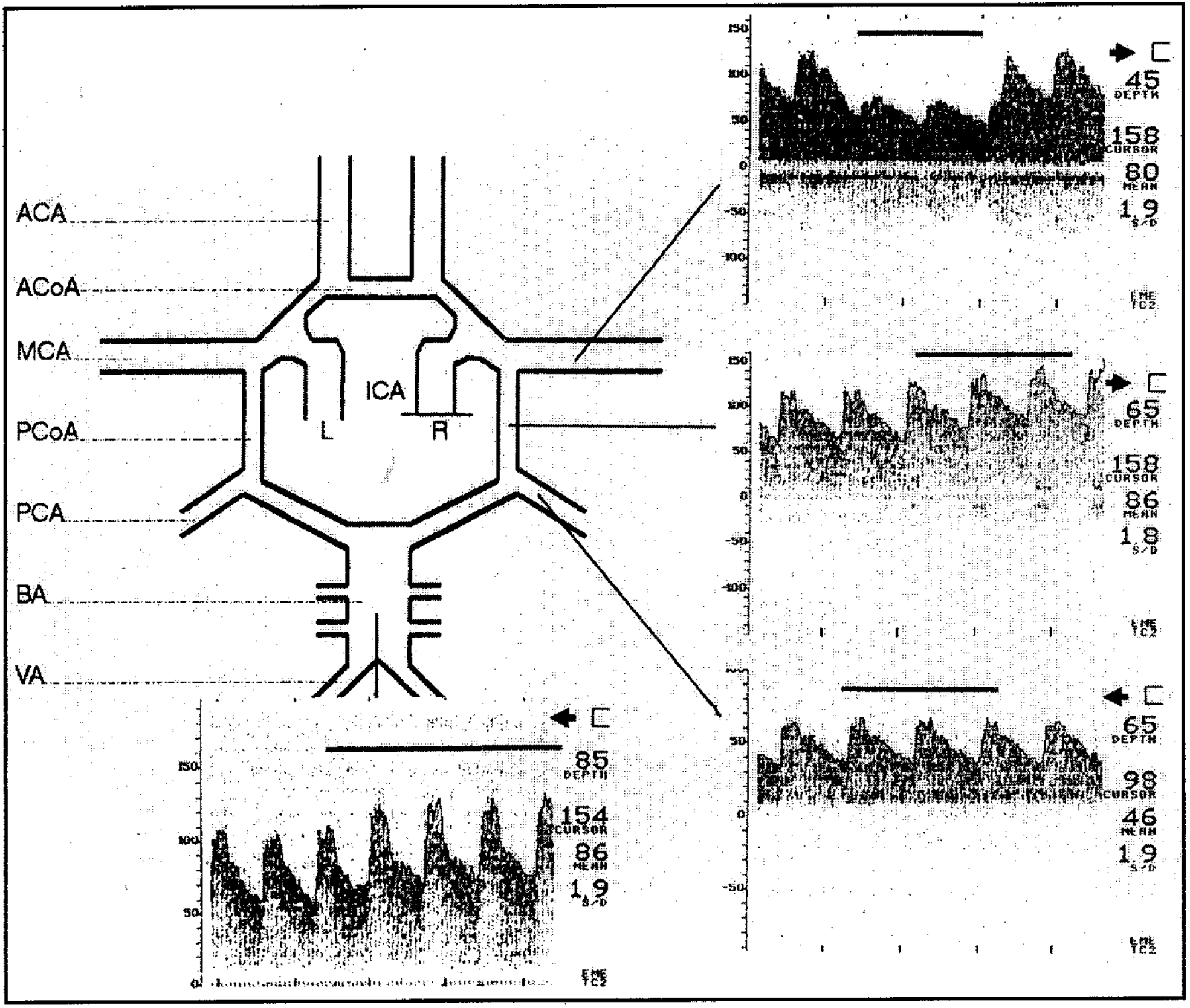
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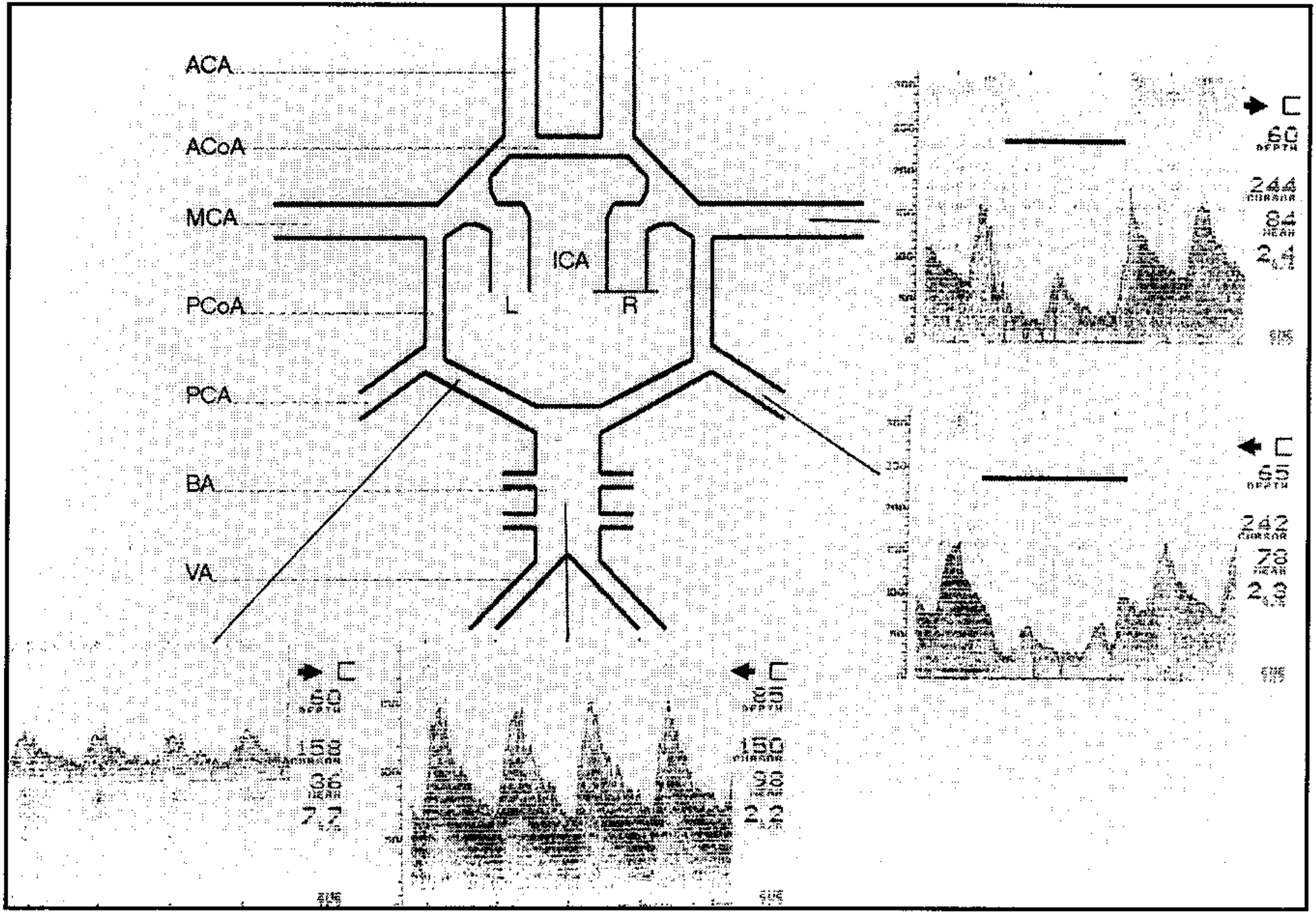

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