



Get Clarity On Generics

Cost-Effective CT & MRI Contrast Agents



FRESENIUS
KABI

WATCH VIDEO

AJNR

Digital intravenous cerebral angiography in neonates.

B C Lee, T M Voorhies, M E Ehrlich, E Lipper, P A Auld and R C Vannucci

AJNR Am J Neuroradiol 1984, 5 (3) 281-286

<http://www.ajnr.org/content/5/3/281>

This information is current as
of August 8, 2025.

Digital Intravenous Cerebral Angiography in Neonates

B. C. P. Lee¹
T. M. Voorhies²
M. E. Ehrlich²
E. Lipper³
P. A. M. Auld³
R. C. Vannucci²

Digital intravenous cerebral angiography was performed in 13 neonates. Injections were made either centrally into the right atrium or peripherally into a distal vein. Seven infants suffered from anoxia, one infant had clinical brain death, another had focal infarcts, and two had intracranial hemorrhage. One infant had an intracranial tumor and another had a neck tumor. Venous sinus thrombosis was seen in five of the seven anoxic infants. A total absence of intracranial arterial circulation was demonstrated in the clinically brain-dead infant. Vascularity and venous involvement by neoplasm were excellently delineated by this technique.

Abnormalities of the intracranial circulation of neonates are studied infrequently because of the difficulty and morbidity of angiography [1]. Intravenous or inhalation radioactive xenon scintillation and nonisotopic xenon computed tomographic (CT) studies are sometimes used to evaluate cerebral blood flow, but are limited by the poor spatial resolution of both techniques compared with angiography [2-5]. Digital subtraction angiography (DSA) is a relatively noninvasive method that may potentially provide detailed images of the intracranial vessels [6-8]. We present our initial experience with this technique in the evaluation of a number of neonatal disorders.

Materials and Methods

Thirteen neonates (36 weeks to full term) were studied with the Technicare DR960 attached to a GE fluoroscopic unit. Seven patients had anoxia, and one neonate was investigated because of suspected brain death. One infant had a brain tumor, one, a neck tumor. Three infants, two with CT diagnoses of hemorrhage and the other an infarct, were also studied. All the patients except the brain-dead infant were sedated with Demerol 25 mg, Phenergan 25 mg, and Thorazine 12.5 mg/kg.

Contrast material was delivered directly into an indwelling umbilical vein catheter in eight cases, into a peripheral venous catheter in three cases using a 23 gauge needle, and into the right atrium after transfemoral venous catheterization in two cases using 3 French pigtail catheters (table 1). Four ml of Conray 76 followed by a bolus of 4 ml of saline was injected by hand, through the umbilical or peripheral venous routes. Not more than a total of 4 ml/kg of contrast material was given.

Anteroposterior (AP), off-lateral, and oblique AP head and neck views were taken (fig. 1). Off-lateral head views were routinely obtained; AP views were obtained when arterial and cortical vein occlusions were suspected, and oblique AP head views when confirmation of sagittal sinus thrombosis was required. Oblique neck views were required for visualization of extracranial arterial occlusions or neck pathology.

CT scans in the axial plane were obtained in 12 cases. The initial scans were obtained within 48 hr of birth. Follow-up scans were obtained from 7 to 21 days in four cases. Most scans were obtained without intravenous contrast enhancement.

Received January 26, 1983; accepted after revision October 24, 1983.

¹ Department of Radiology, Division of Neuro-radiology, New York Hospital-Cornell Medical Center, 525 E. 68th St., New York, NY 10021. Address reprint requests to B. C. P. Lee.

² Department of Pediatric Neurology, New York Hospital-Cornell Medical Center, New York, NY 10021.

³ Department of Neonatology, New York Hospital-Cornell Medical Center, New York, NY 10021.

AJNR 5:281-286, May/June 1984
0195-6108/84/0503-0281 \$00.00
© American Roentgen Ray Society

TABLE 1: Digital Intravenous Cerebral Angiography in Neonates

Pathology: Case No. (weight in kg)	Injection Site	DSA Findings		CT Findings*
		Arterial	Venous	
Anoxia:				
1 (3.5)	RA (umb v)	Attenuated caliber of arteries; prolonged transit time	Sagittal sinus thrombosis	1 d: generalized decreased density, compressed lateral ventricles; 14 d: hemorrhage into white matter, dilated lateral ventricles
2 (2.8)	RA (umb v)	Occluded MCA, ACA; prolonged transit time	Sagittal sinus thrombosis	1 d: generalized decreased density, compressed lateral ventricles; 10 d (with contrast): hemorrhage into white matter, ventricles, straight sinus
3 (3.8)	RA (umb v)	Generalized prominence of arterioles, capill; rapid transit time	Normal	1 d: generalized decreased density, compressed lateral ventricles; 7 d: hemorrhage into white matter
4 (4.0)	(Arm v)	Patchy areas of prominence of capill	Normal	. . .
5 (3.5)	(Fem v)	Normal	Partial transverse sinus thrombosis	2 d: multiple areas of decreased density
6 (3.5)	RA (umb v)	Attenuated caliber of arteries; prolonged transit time	Partial sagittal sinus, deep vein thrombosis	21 d: multiple focal atrophy
7 (4.0)	RA (fem v)	Normal	Partial sagittal sinus thrombosis	1 d: generalized decreased density, compressed lateral ventricles
Brain death:				
8 (3.4)	RA (fem v)	Occluded internal carotid, vertebral arteries	Veins not visualized	2 d: generalized decreased density, compressed lateral ventricles
Tumor:				
9 (3.7)	(Arm v)	Normal	Filling defect in sagittal sinus	4 d: midline mass over sagittal suture, bone defect
10 (3.5)	RA (umb v)	External carotid tumor circulation	Venous puddling	3 d: multiloculated enhancing facial, neck mass
Hemorrhage:				
11 (2.5)	RA (umb v)	Normal	Normal	1 d: hemorrhage: quadrigeminal cistern, velum interpositum
12 (2.9)	RA (umb v)	Normal	Normal	2 d: hemorrhage: parietooccipital lobe interhemispheric fissure; 14 d: dilated ventricles
Infarct:				
13 (3.5)	RA (umb v)	Normal	Normal	4 d: focal frontal infarct

Note.—DSA = digital subtraction angiography; RA = right atrium; umb v = umbilical vein; fem v = femoral vein; MCA = middle cerebral artery; ACA = anterior cerebral artery; capill = capillaries; d = day(s).

* CT was performed without contrast enhancement unless stated otherwise.



Fig. 1.—Normal cerebral DSA. AP (A) and lateral (B) views show normal carotid and vertebrobasilar circulations. C, Lateral view shows normal venous drainage.

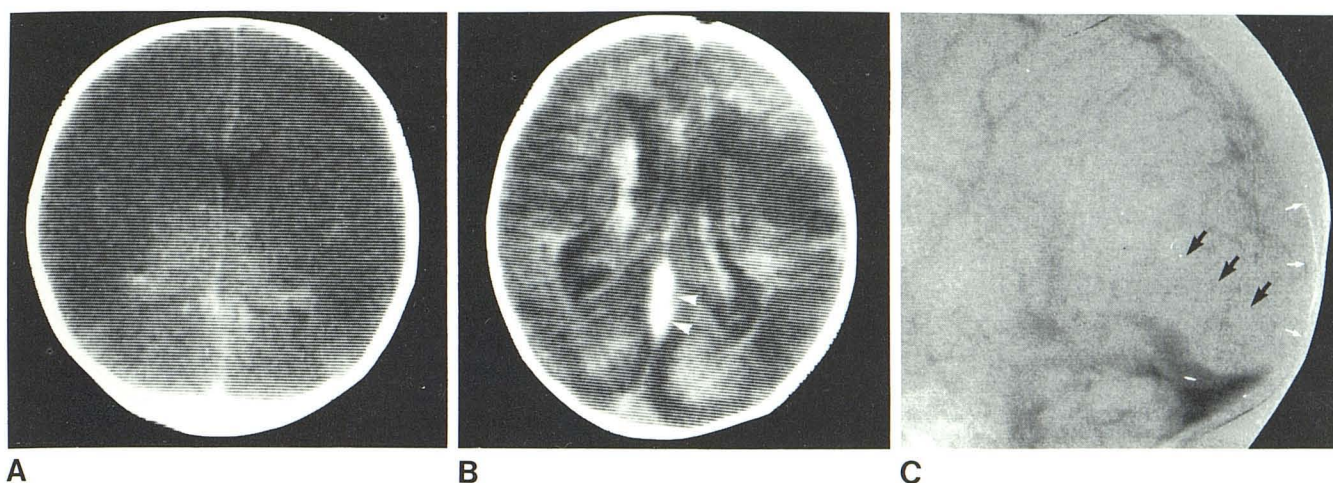


Fig. 2.—Case 1, intraventricular hemorrhage and sagittal sinus thrombosis. **A**, CT without contrast shows decreased attenuation of white matter with compression of ventricles. **B**, CT without contrast 14 days after anoxic episode shows mild intraventricular hemorrhage, parenchymal hemorrhage, enlarged

straight sinus with increased attenuation (presumably due to thrombosis), and ventricular dilatation (arrowheads). **C**, DSA, lateral view, shows occlusion of posterior part of superior sagittal sinus (white arrows), straight sinus, and deep veins (black arrows).

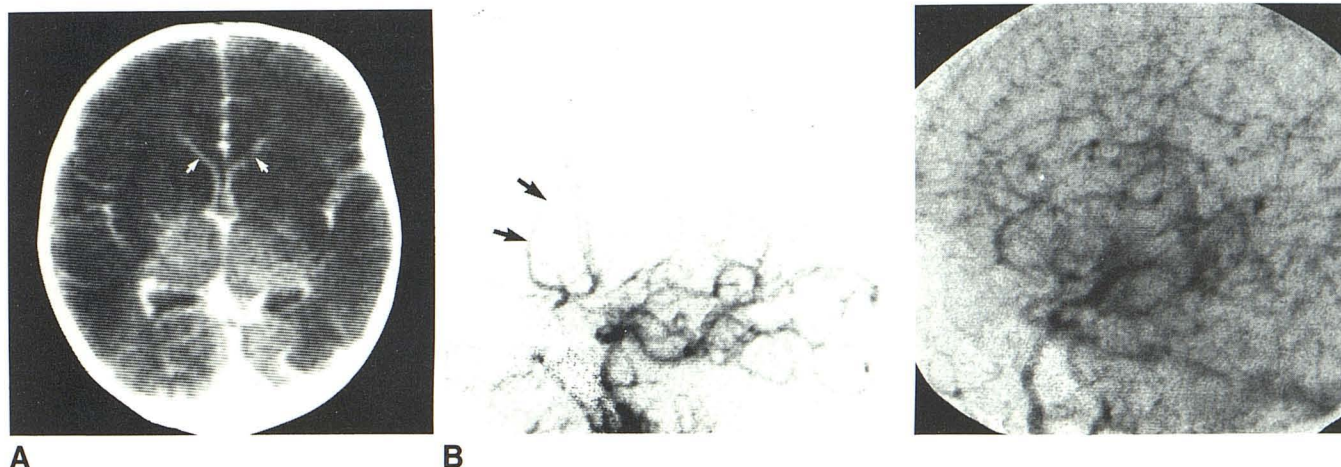


Fig. 3.—Case 2, occlusion of internal carotid artery, sagittal sinus thrombosis. **A**, CT with contrast shows prominent enhancement of paraventricular vessels (subependymal veins, arrows) and falx, suggesting collateral flow secondary to sinus thrombosis. **B**, DSA, lateral view, shows occlusion of internal carotid artery with no filling of middle cerebral arteries and filling of only proximal part of anterior cerebral arteries through circle of Willis collaterals from posterior circulation (arrows).

Fig. 4.—Case 3, nonoccluded cerebral circulation. DSA, lateral view, shows generalized increased caliber and prominence of intracranial arteries and capillaries. Circulation time was rapid.

Results

Anoxia

Noncontrast CT scans obtained within 48 hr of birth showed generalized decreased density of the white matter with ventricular compression in all cases. Repeat scans 7–14 days later revealed intracranial hemorrhages that were intraparenchymal in cases 1–3; in case 1 there was also an intraventricular hemorrhage and enlargement and increased attenuation of the straight sinus (fig. 2B). Repeat CT in case 5 at 21 days showed focal cerebral atrophy at the previously

demonstrated areas of decreased density. Intravenous contrast material was administered only in case 2 and showed prominent opacification in the paraventricular and tentorial regions (fig. 3A).

DSA showed occlusion of the middle and anterior cerebral arteries with intact posterior circulation in case 2 (fig. 3B). There was prominence of the arterioles and capillaries in cases 3 and 4 (figs. 4 and 5) and decreased caliber of the arteries in cases 1 and 6. Transit time appeared prolonged in cases 1, 2, and 6 and decreased in cases 3 and 4. Complete occlusion of the sagittal sinus was demonstrated in cases 1

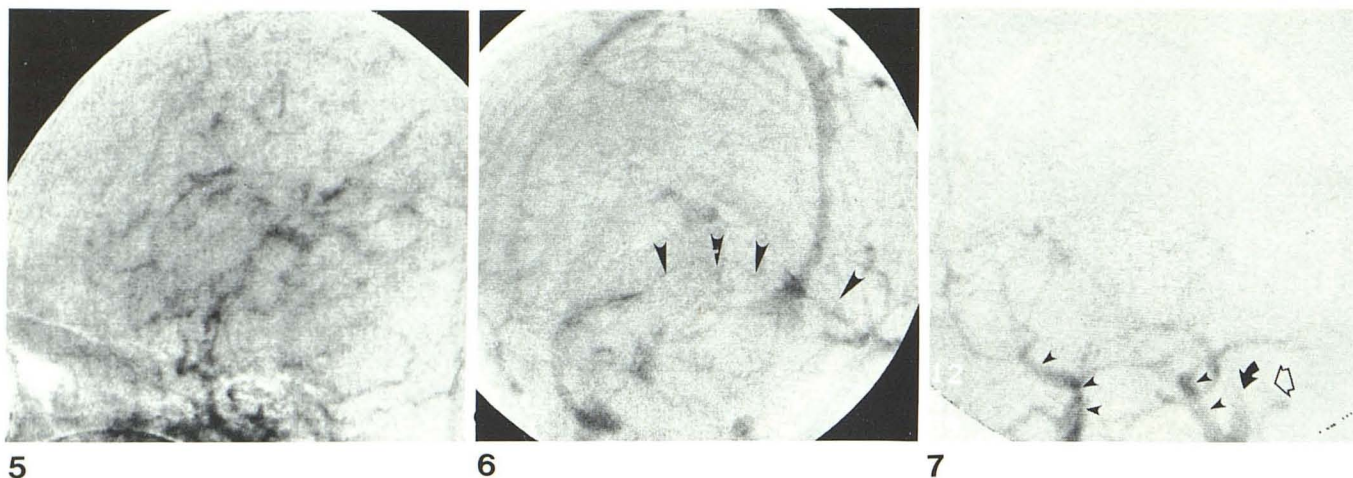


Fig. 5.—Case 4, nonoccluded cerebral circulation. DSA, lateral view, shows patchy areas of prominence of capillaries.

Fig. 6.—Case 5, anoxia. DSA, AP oblique view, shows partial occlusion of transverse sinuses (arrowheads).

Fig. 7.—Case 8, brain death. DSA, off-lateral view, shows nonfilling of intracranial circulation. Only external carotids are filled (arrowheads). Sites of occlusion of internal carotid (curved arrow) and vertebral (open arrow) arteries are shown.

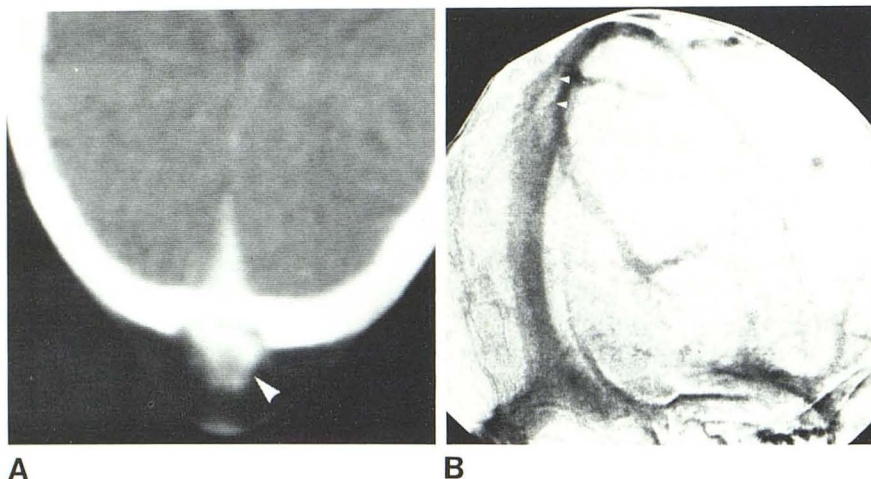


Fig. 8.—Case 9, dermoid with involvement of sagittal sinus. A, CT with contrast shows midline dermoid with extracranial extension (arrowhead). B, DSA, AP oblique view, shows filling defect within sagittal sinus (arrowheads).

and 2 (fig. 2C). Partial sagittal sinus occlusion with associated occlusion of the transverse sinus and deep veins were shown in cases 5–7 (fig. 6).

Brain Death

In the one case of clinical brain death (case 8), noncontrast CT showed generalized decreased density and compressed lateral ventricles. DSA showed complete occlusion of the internal carotid and vertebral arteries with no intracranial circulation. The external carotid circulation was intact (fig. 7).

Tumors

CT with contrast enhancement showed a midline tumor over the sagittal sinus in case 9 (fig. 8A). DSA demonstrated a filling defect of the sagittal sinus caused by this pathologi-

cally proved dermoid (fig. 8B). Another case of a teratoma of the neck (case 10) was shown to have pathologic circulation on the DSA examination.

Hemorrhage and Infarct

DSA was normal in these three cases.

Discussion

Technique

At the beginning of the study various volumes of contrast material (2–4 ml) were injected either centrally into the right atrium or peripherally into an arm vein. It was found that the route of contrast delivery had only a slight effect on the visualization of intracranial vessels: peripheral injections

through small needles (23 gauge) provided images only slightly inferior to central injections of contrast material. Four ml of Conray 76 was found to be the optimal volume. No adverse effects were encountered when the total volume of injection was limited to 4 ml/kg. Patient movement was eliminated by sedation except when respiratory movement was transmitted to the head.

The AP projection was best for visualizing the distal internal carotid artery; branches of the anterior and middle cerebral arteries; and the torcula, transverse sinuses, and cortical veins (fig. 1A). A slightly off-lateral projection was the view of choice for viewing the carotid siphons, basilar artery, deep veins, and straight sinuses. A 30° oblique AP view was best for visualizing the sagittal sinus and torcula. A 512 × 512 matrix provided the best resolution at the expense of a slower acquisition rate of 1.25 frames/sec. The resolution of 256 × 256 matrix was inadequate. The entire cranium was included in the 11.4 or 15.2 cm fluoroscopic fields.

Anoxia

The pathologic literature on perinatal asphyxia is controversial: border-zone arterial infarcts have been observed in autopsies of long-term survivors [9–11] and have also been seen in animal models when anoxia was combined with hypotension [12, 13]. Similar infarcts have not, however, been observed in infants who died acutely from asphyxia. In some autopsy studies the infarcts were arterial, in the distribution of the middle cerebral arteries [11, 14–16], while other observers have noted a high incidence of venous occlusive disease [17, 18]. Hemorrhagic infarcts, presumably secondary to venous thrombosis, are said to be more common in stillborn fetuses [14, 16–19].

The available radiologic literature has defined CT appearances only [20–22]. CT scans of six of our seven patients with anoxia showed massive edema with generalized hypodensity and slitlike ventricles in five. The extent of the edema was larger than would be seen with border-zone infarcts. DSA revealed partial or total sinus occlusion in five of the seven patients. Prominent dural structures in the contrast CT scan in one case probably was from collateral venous flow. Sequential CT scans showed massive hemorrhages in three cases, which was most likely due to venous infarcts.

Although arterial occlusions were demonstrated in one case it is not possible to know whether this occurred spontaneously or was secondary to venous occlusion and stasis. Prominence of arterioles and capillaries may reflect hyperemia secondary to disturbances of autoregulation [3, 22], and slow transit times may be the result of venous stasis and occlusions. The large number of cases with patent arteries would tend to suggest that venous occlusion is the primary result of anoxia, and arterial occlusions occur when prolonged stasis is present. Border-zone ischemia may play an additional role, but is probably not as important as the venous component.

Demonstration of the patency of the venous sinuses and arteries has great importance in the treatment of anoxic infants [8]. It would appear from our study that DSA can reliably evaluate the patency of the major venous sinuses and

deep intracranial veins. Cortical venous occlusion is more difficult to determine, as only widespread occlusion of all the hemispheric veins would be detected. Careful administration of anticoagulants and/or antiplatelet agents reduces progression of venous thrombosis and may potentially prevent arterial occlusions.

Brain Death

Currently the radiologic diagnosis of brain death is by selective arterial injections and intravenous radionuclide examinations [23, 24]. Arteriography is time-consuming and radionuclide studies lack resolution. DSA in our one clinically dead infant showed the same appearances as arteriography: absent internal carotid and vertebrobasilar circulations. The ease of DSA would make it the ideal method for confirming death.

Vascular Tumors

Masses of the head and neck are ideally evaluated by DSA. The vascular nature of the lesion and the origins of blood supply may be shown. Lesions situated close to dural sinuses may involve the sinus. DSA is the method of choice for determining the degree of invasion, as in our case with a midline dermoid, where the tumor extended into a nonoccluded superior sagittal sinus.

Intracranial Hemorrhage and Infarcts

Intraventricular hemorrhage in premature infants is usually secondary to subependymal germinal matrix bleeding. DSA probably offers little diagnostic data or help in management. Arteriovenous malformations, however, may be revealed on DSA. Although small malformations still require selective arteriography for confirmation, DSA may be useful as a screening procedure in the planning of subsequent contrast studies.

Arterial infarcts shown on CT may be evaluated by DSA, which is useful for demonstrating occlusion of primary and secondary branches of the intracranial arteries. Selective intraarterial injections are required for showing small vessel disease.

REFERENCES

1. Harwood-Nash DC, Fitz CR. *Neuroradiology in infants and children*. St. Louis: Mosby, 1976:318–340
2. Lou NC, Lassen NA, Friis-Hausen B. Impaired autoregulations of CBF in the distressed newborn infant. *J Pediatr* 1979;94: 118–121
3. Ment LR, Ehrenkrantz RA, Lange RC, et al. Alterations CBF in preterm infants with intraventricular hemorrhage. *Pediatrics* 1981;68:763–769
4. Drayer BP, Wolfson SK Jr, Reinmuth OM, et al. Xenon enhanced CT for analysis of cerebral integrity, perfusion and blood flow. *Stroke* 1980;9:123–130
5. Meyer JS, Hayman LA, Yamamoto M, Sakai F, Nakajima S. Local cerebral blood flow measured by CT after stable xenon inhalation. *AJNR* 1980;1:213–225, *AJR* 1980;135:239–251

6. Chilcote WA, Modic MT, Pavlicek WA, et al. Digital subtraction angiography of the carotid arteries: a comparative study in 100 patients. *Radiology* **1981**;139:287-295
7. Mistretta CA, Crummy AB, Strother CM. Digital angiography: a perspective. *Radiology* **1981**;139:273-276
8. Modic MT, Weinstein MA, Chilcote WA, et al. Digital subtraction angiography of the intracranial vascular system: comparative study in 55 patients. *AJNR* **1981**;2:527-534, *AJR* **1982**;138:299-306
9. Christensen E, Melchior J. Cerebral palsy—a clinical and neuropathological study. In: *Clinics in developmental medicine*, no. 25. Spastic International Medical. Lavenham, England: Lavenham, **1967**:26-110
10. Courville CB. Pathogenesis of nodular atrophy of the cerebral cortex. A common cortical change in cases of cerebral palsy. *Arch Pediatr* **1960**;77:101-129
11. Clark RM, Linell EA. Case report: prenatal occlusion of the internal carotid artery. *J Neurol Neurosurg Psychiatry* **1954**;17:295-297
12. Brierley JB, Brown AW, Excell BJ, et al. Brain damage in the rhesus monkey resulting from profound arterial hypotension: its nature, distribution and general physiological correlates. *Brain Res* **1969**;13:68-100
13. Brierley JB, Meldrum BS, Brown AW. The threshold and neuropathology of cerebral anoxic-ischemic cell change. *Arch Neurol* **1973**;29:367-374
14. Banker BQ. Cerebral vascular disease in infancy and childhood: 1. Occlusive vascular disease. *J Neuropathol Exp Neurol* **1961**;20:127-138
15. Cocker J, George WW, Yates PO. Perinatal occlusion of the middle cerebral artery. *Dev Med Child Neurol* **1965**;7:235-243
16. Malamud N. An etiologic and diagnostic study of cerebral palsy. In: McMeemy WH, ed. *Selective vulnerability of the brain*. Philadelphia: Davis, **1963**:211-225
17. MacGregor AR. *Pathology of infancy and childhood*. London: Livingston, **1960**:28-54
18. Towbin A. Central nervous system damage in the human fetus and newborn infant. *Am J Dis Child* **1970**;119:529-542
19. Towbin A. Cerebral hypoxic damage in fetus and newborn. *Arch Neurol* **1969**;20:35-43
20. Flodmark D, Becker LE, Harwood-Nash DC, et al. Correlation between computed tomography and autopsy in premature and full-term neonates that have suffered perinatal asphyxia. *Radiology* **1980**;137:93-103
21. Harabayashi S, Kitalara T, Hiokida T. Computed tomography in perinatal hypoxia and (hypoglycemic) encephalopathy with emphasis on follow-up studies. *J Comput Assist Tomogr* **1980**;4:451-456
22. Sherman DA, Fine M, Masden JC, Palacio E. Postischemic hypervascularity of infancy: a stage in the evolution of ischemic brain damage with characteristic CT scan. *Ann Neurol* **1981**;9:358-365
23. Greitz T, Gordeu E, Kolmodin G, Widen A. Aortocranial and carotid angiography in determination of brain death. *Neuroradiology* **1973**;5:13-19
24. Goodman JM, Mishkin FS, Dyker NM. Determination of brain death by isotope angiography. *JAMA* **1969**;209:1869-1872