AJNR

Cranial Sonography of the Infant Treated with Extracorporeal Membrane Oxygenation

Richard A. Bowerman, Joseph B. Zwischenberger, Alice F. Andrews and Robert H. Bartlett

AJNR Am J Neuroradiol 1985, 6 (3) 377-382
http://www.ajnr.org/content/6/3/377

Cranial Sonography of the Infant Treated with Extracorporeal Membrane Oxygenation

Richard A. Bowerman[1]
Joseph B. Zwischenberger[2]
Alice F. Andrews[3]
Robert H. Bartlett[2]

Neonates with severe but reversible pulmonary disease may require therapy beyond conventional ventilatory care. Extracorporeal membrane oxygenation (ECMO) serves as a temporary artificial lung for such infants. Since anticoagulation with systemic heparin is required in the extracorporeal circuit, antecedent hemorrhage may be exacerbated or new hemorrhage precipitated in ECMO patients. While the "usual" periventricular/intraventricular hemorrhage seen in a premature infant may develop, contrasting hemorrhages of unusual extent, uncommon location, or demonstrating unique alterations in internal sonographic character may be precipitated, presumably due to the requisite anticoagulation. Representative examples of such variations are presented along with guidelines for the use of cranial sonography in selecting and monitoring ECMO patients.

Neonates with respiratory failure and a predicted 80%–90% risk of mortality despite optimal medical and/or surgical therapy are considered candidates for extracorporeal membrane oxygenation (ECMO) at our institution. As ECMO serves only as a temporary artificial lung, such respiratory failure must be potentially reversible. Neonates are candidates to be placed on ECMO when, despite maximal therapy, one or more of five criteria are met: (1) acute respiratory deterioration, (2) greater than an 80% mortality risk by the neonatal pulmonary insufficiency index [1], (3) persistent air leak from barotrauma, (4) failed treatment of persistent fetal circulation, and (5) congenital diaphragmatic hernia unresponsive to ventilator management. Contraindications to initiation of ECMO are twofold: (1) the presence of intraventricular or intracerebral hemorrhage and (2) a nonreversible medical condition incompatible with a "quality-life" potential.

Premature ECMO candidates with respiratory failure are at increased risk for developing intracranial hemorrhage. With the systemic anticoagulation necessary in the ECMO circuit, the potential for significant intracranial hemorrhage increases. An antecedent intracranial hemorrhage may extend, or a new intracranial hemorrhage may be precipitated. Both are serious considerations in ECMO therapy and require close cooperation between the sonographer and the surgical/neonatal team. Portable real-time sonography of the neonatal brain is an accurate and established procedure for the identification of intracranial hemorrhage and its sequelae [2–7]. It serves as an integral component of the pre-ECMO protocol for patient selection as well as the follow-up of ECMO patients.

Our experience with cranial sonography in a large population of ECMO patients is presented. The combination of prematurity, respiratory failure, and systemic anticoagulation in many of these patients resulted in hemorrhages of unusual extent, location, and internal sonographic character. All sonographers currently involved with scanning such neonates, or those at an institution considering an ECMO program, should be aware of these sonographic features.

Materials and Methods

Twenty-eight neonates were placed on ECMO at the University of Michigan Medical Center

This article appears in the May/June 1985 issue of *AJNR* and the July 1985 issue of *AJR*.

Received May 30, 1984; accepted after revision September 11, 1984.

Presented at the annual meeting of the American Roentgen Ray Society, Las Vegas, April 1984.

[1] Department of Radiology, Ultrasound Division, Box 013, University of Michigan Medical Center, Ann Arbor, MI 48109-0010. Address reprint requests to R. A. Bowerman.

[2] Department of Surgery, University of Michigan Medical Center, Ann Arbor, MI 48109-0010.

[3] Department of Pediatrics, Division of Neonatology, Wayne County General Hospital, Wayne, MI 48104.

***AJNR* 6:377–382, May/June 1985**
0195–6108/85/0603–0377

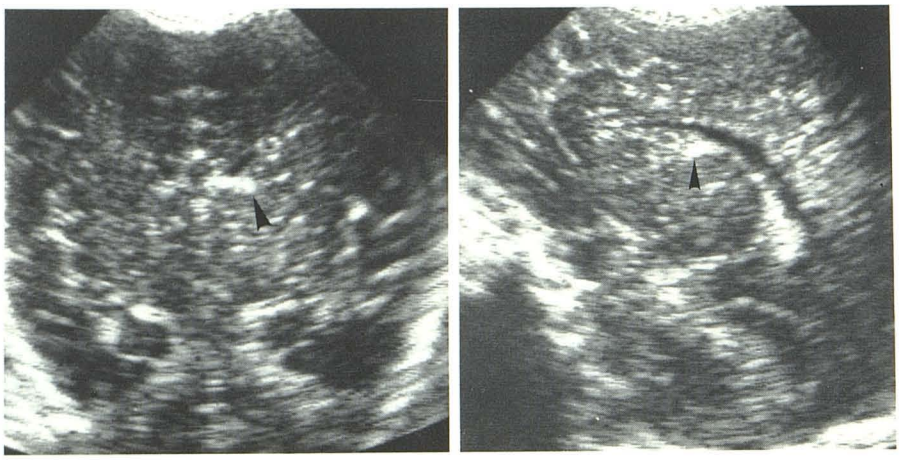

Fig. 1.—**A**, Midcoronal sonogram. New left-germinal-matrix hemorrhage (*arrowhead*) after 1 day on ECMO. **B**, Left parasagittal sonogram confirms small grade 1 hemorrhage in thalamocaudate notch (*arrowhead*).

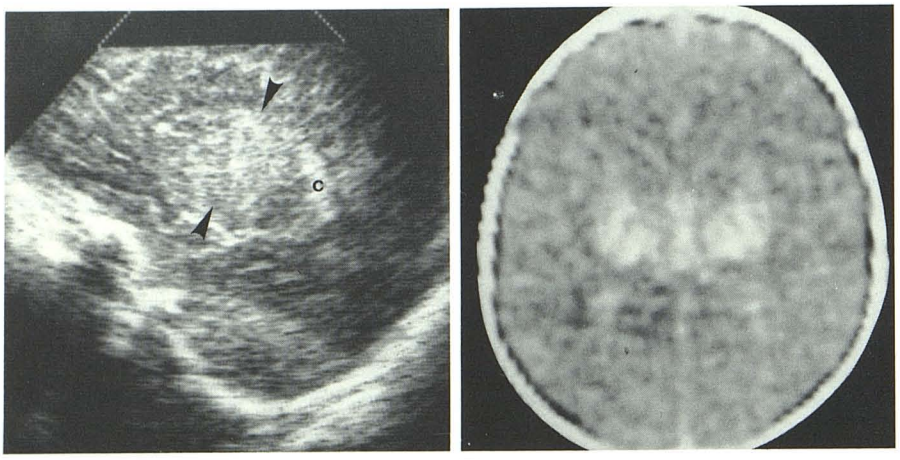

Fig. 2.—**A**, Right parasagittal sonogram after 1 day on ECMO. Compressed lateral ventricle consistent with cerebral edema. Increased echogenicity within anterior and mid thalamus (*arrowheads*) consistent with acute hemorrhage. C = choroid plexus in trigone. **B**, CT scan. Hemorrhage within thalami and basal ganglia, confirming sonographic findings.

between March 1981 and March 1984. Twenty were male and eight were female. Gestational ages were 27–42 weeks, and birth weights were 1000–4200 g. Cranial sonograms were obtained before, during, and after ECMO in all cases. Eight of the 28 infants developed an intracranial hemorrhage after placement on ECMO.

Results

Three patients developed intracranial hemorrhage and survived. Two infants of 33 and 36 weeks gestational age, each weighing more than 2000 g, developed only a grade I hemorrhage (fig. 1) and survived with normal neurologic development. One postterm infant with severe perinatal asphyxia developed hemorrhage within the thalami and corpora striata (fig. 2) and survived with marked developmental delay.

More extensive intracranial hemorrhages occurred in five infants of lower gestational age and birth weight. Grade III hemorrhages (figs. 3 and 4) were identified in three patients with gestational ages of 30, 30, and 27 weeks and birth weights of 1.2, 2.1, and 1.0 kg, respectively. The largest infant was grossly hydropic secondary to Rh isoimmunization, with an estimated adjusted weight of 1600 g. The degree of intraventricular hemorrhage in each case was pronounced, with marked ventricular distension secondary to either the direct volume effect of intraventricular blood or the acute obstruction of cerebrospinal fluid circulation. One infant developed a large intraventricular hemorrhage of echogenicity somewhat less than that usually associated with acute hemorrhage, shortly after being placed on ECMO (fig. 3). A second neonate showed rapid progression from an initial intraventricular hemorrhage of moderate size to a considerably larger hemorrhage. A third neonate demonstrated an unusual sonographic appearance of intraventricular hemorrhage (fig. 4). One component of mid- to high-level echogenicity was closely adherent to the choroid plexus throughout the lateral ventricle. A second hypoechoic and presumably unclotted component surrounded the echogenic clot (fig. 4C). This dichotomy presumably was from partial coagulation of intraventricular blood secondary to systemic heparinization. With a reduction in heparin concentration to minimal therapeutic levels, the peripheral component rapidly progressed to a more echogenic

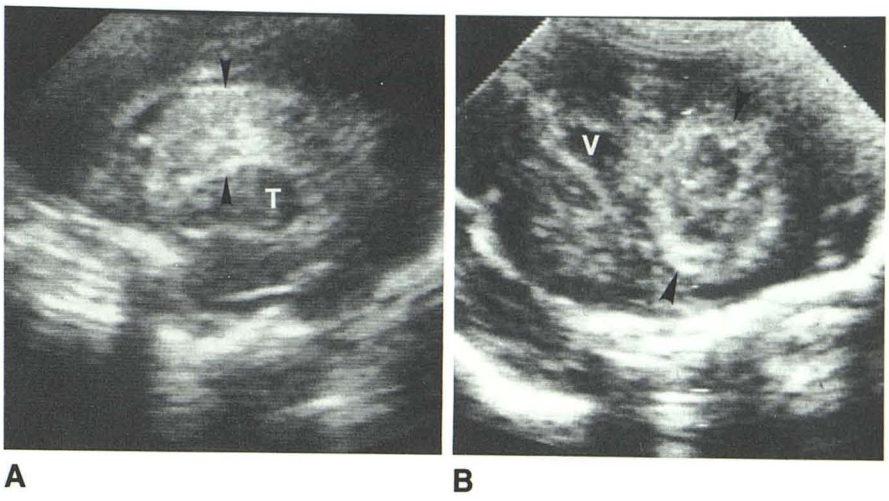

Fig. 3.—**A**, Left parasagittal sonogram 1 day after ECMO cannulation. Marked distension of left lateral ventricle by moderately echogenic hemorrhage (*arrowheads*). T = thalamus. **B**, Anterior coronal sonogram. Marked distension of left lateral ventricle with hemorrhage of mixed, but predominantly low, echogenicity (*arrowheads*). Mild distension of right lateral ventricle (V).

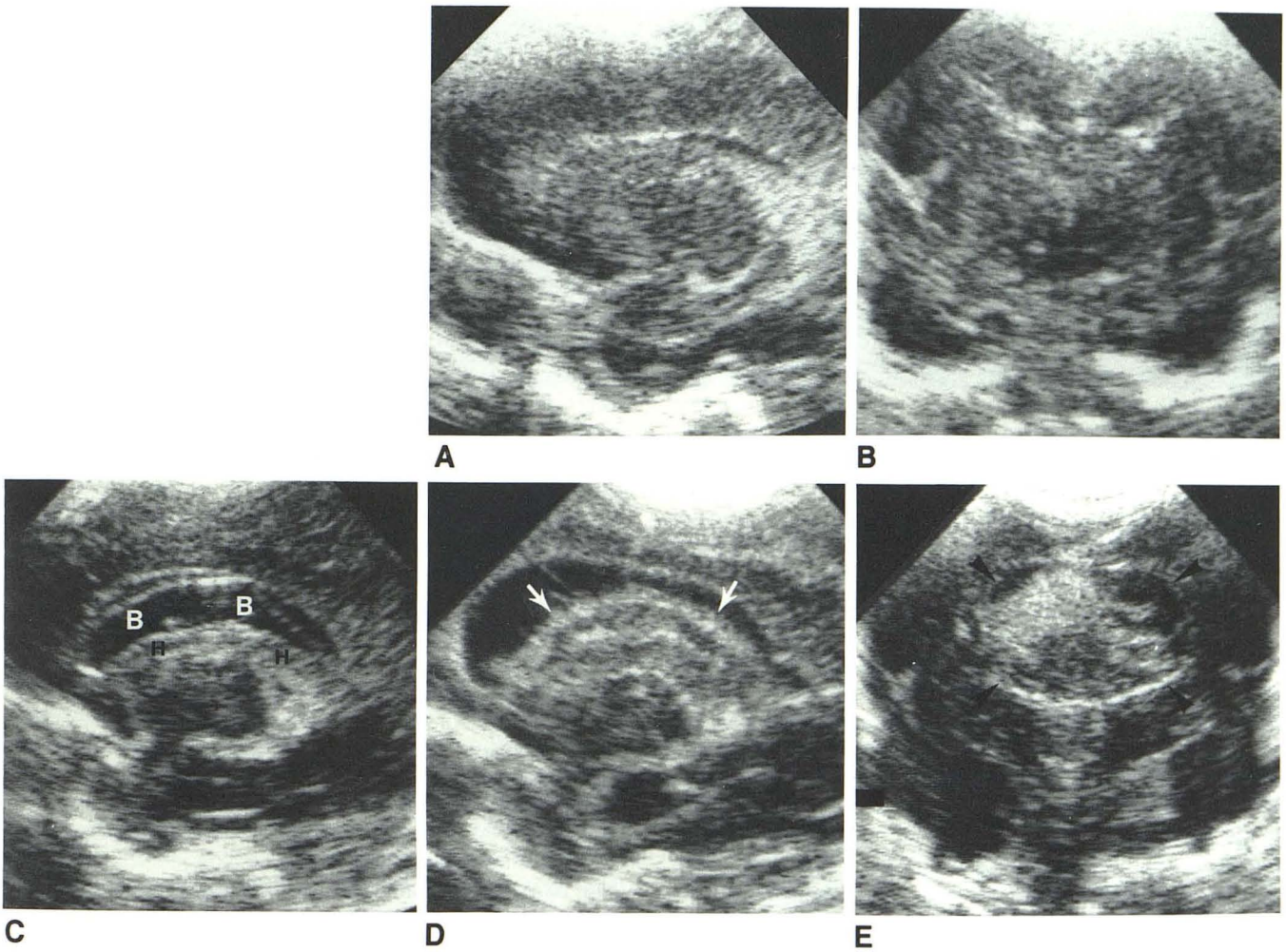

Fig. 4.—**A**, Left parasagittal (**A**) and midcoronal (**B**) sonograms before initiation of ECMO. Attenuated lateral ventricles with no intracranial hemorrhage. **C**, Left parasagittal sonogram ½ hr after placement on ECMO. Ventriculomegaly due to distension by intraventricular hemorrhage. Midlevel echogenic clot closely adherent to choroid plexus in floor of lateral ventricle (H). Second, hypoechoic, unclotted component of intraventricular hemorrhage (B) bordered by echogenic margin within confines of ventricle peripheral to clotted hemorrhage. **D**, Left parasagittal sonogram 5 hr after initiation of ECMO. Progression of hydrocephalus. After reduction in systemic heparin concentration, much of previously liquid blood has now clotted, with layered appearance of midlevel echogenic hemorrhage adherent to floor of lateral ventricle (*arrows*). **E**, Midcoronal sonogram 16 hr after initiation of ECMO. Further gross distension of lateral ventricles (*arrowheads*), with distortion of midline structures and considerable intraventricular clot of mixed echogenicity. Heterogeneity of hemorrhage was presumed secondary to variable levels of heparinization during evolution of this rapidly progressing intraventricular hemorrhage.

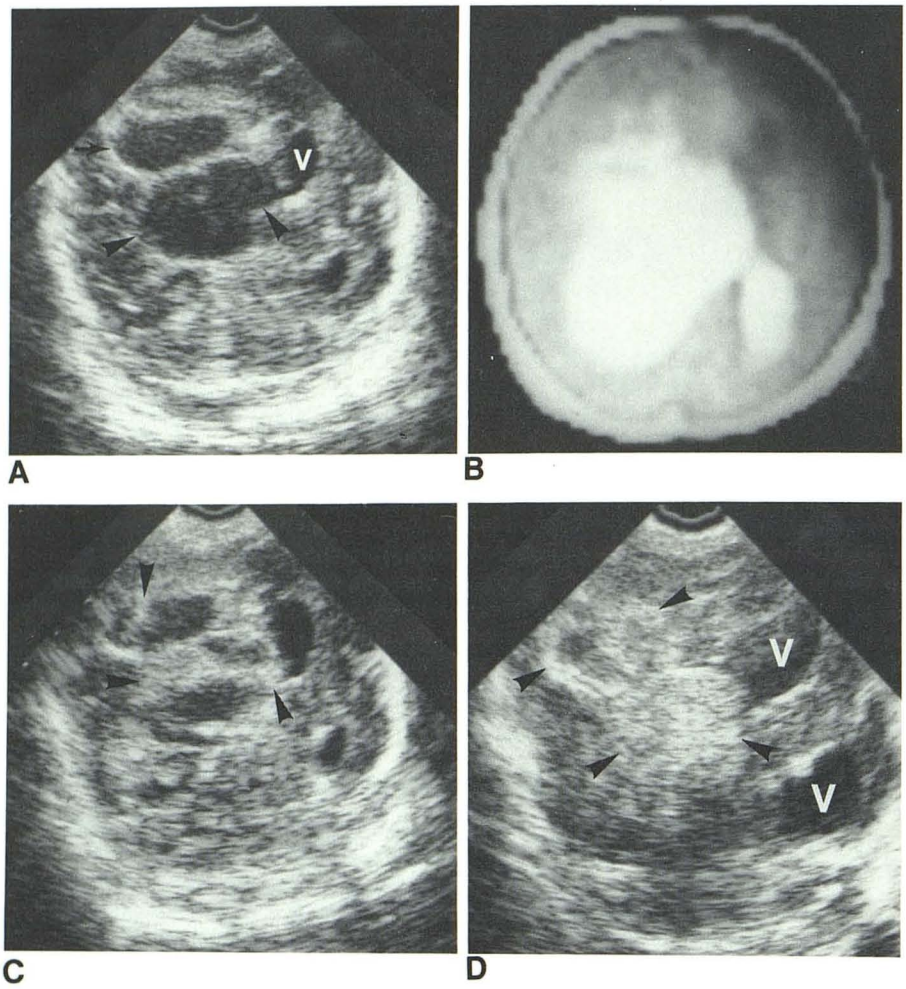

Fig. 5.—**A**, Midcoronal sonogram 3 days after ECMO cannulation. Large hypoechoic areas in right parietal lobe (*arrow*) and right lateral ventricle (*arrowheads*). Latter extends across midline to abut mildly dilated left lateral ventricle (V). **B**, CT scan confirms abnormal hypoechoic masses delineated sonographically to represent extensive, predominately right-sided intraventricular hemorrhage, with midline shift and intracerebral extension. **C**, Midcoronal sonogram 1 day after initial appearance of hemorrhage and after reduction of heparin concentration. Parts of previously hypoechoic hemorrhage are now echogenic, consistent with partial interval clotting (*arrowheads*). **D**, Midcoronal sonogram 3 days after appearance of hemorrhage documents further clotting of intraventricular and intracerebral hemorrhage, which is now predominately echogenic (*arrowheads*). Midline shift persists, and left hydrocephalus has progressed (V).

pattern (fig. 4D), although further significant extension of hemorrhage was also documented (fig. 4E).

A severe grade IV intracranial hemorrhage developed in an infant of 1.4 kg and 32 weeks gestational age. The initial sonographic findings were atypical for acute hemorrhage, with large hypoechoic areas containing low-level internal echoes filling the right lateral ventricle and extending into the right cerebrum, with a pronounced mass effect (fig. 5A). As this was an early case in our series and clinical concern for an intracerebral abscess was raised, a computed tomographic (CT) scan was obtained confirming the presence of gross intracranial blood (fig. 5B). Again, with anticoagulation reduced to a minimal therapeutic level, serial sonograms showed increasing echogenicity of the intracranial hemorrhage as clotting ensued (figs. 5C and 5D).

One infant of 1.6 kg and 32 weeks gestational age developed an unusual focal subarachnoid hemorrhage between the cerebral peduncles and the medial aspect of the left temporal lobe. While less echogenic than acute hemorrhage in the nonheparinized patient, the associated mass effect on the left temporal horn made it readily apparent (fig. 6).

Discussion

ECMO therapy for severe respiratory disease is a highly sophisticated treatment available at few medical centers [8–12]. Considerable expertise is necessary to establish an ECMO team skillful in the successful management of such neonates. As success is demonstrated in selected infants, however, undoubtedly more centers will endeavor to offer such a therapeutic option. As this occurs, it will be important that sonographers recognize the sonographic features of intracranial hemorrhage in anticoagulated infants that are markedly at variance with those characteristics usually associated with intracranial hemorrhage [7].

As part of the ECMO team, sonographers must cooperate closely with their surgery and neonatology colleagues to provide frequent and timely cranial sonography where necessary. Our criteria for the use of sonography in evaluating ECMO candidates and patients are dependent on patient weight. When the appropriate clinical and physiologic criteria have been met for potential placement on ECMO, cranial sonography is performed. For infants under 2.0 kg, this must

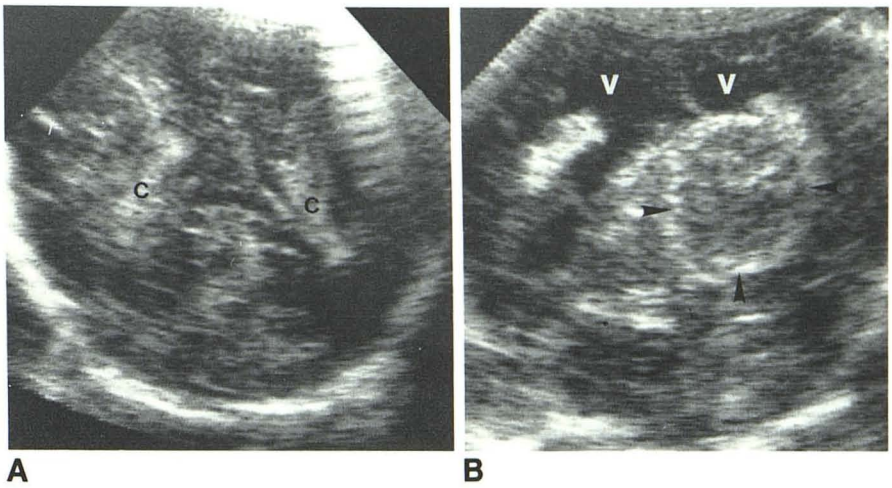

Fig. 6.—**A**, Posterior coronal sonogram 1 day after placement on ECMO documents normal position and symmetry of choroid plexus (C) within atria of lateral ventricles. **B**, 4 days after initiation of ECMO. Hypoechoic retrothalamic mass displaces choroid plexus and left temporal lobe laterally (*arrowheads*). Moderate ventriculomegaly (V) bilaterally.

occur within 2 hr of cannulation, while infants greater than 2.0 kg require a study within 12 hr of bypass. Unequivocal sonographic evidence of a grade II, III, or IV hemorrhage precludes placement on ECMO. All infants must be studied after cannulation and subsequently with any sign of clinical deterioration. The smaller neonates are studied daily during ECMO, while clinically stable infants over 2 kg need only be scanned before discharge. ECMO may be administered safely for 1 week, with an increasing complication rate beyond that time.

The steps of the cranial sonographic examination are well described [13, 14], and require no modification in the ECMO patient. The bypass cannulas enter the neck, with no impediment to scanning through the anterior fontanelle. The types of intracranial hemorrhage most frequently documented sonographically in the non-ECMO patient may be classified according to Papile et al. [15]. In the premature infant, such hemorrhage usually originates in the germinal matrix at the level of the head of the caudate nucleus, with extension possible intraventricularly and/or intracerebrally [16]. Acute hemorrhage is uniformly echogenic in nature. Its subsequent sonographic characteristics of diminishing internal echo texture, fragmentation, and resorption have been well defined [7].

The use of systemic heparinization in the premature infant on ECMO appears to precipitate hemorrhage in unusual locations (figs. 2 and 6) and of atypical sonographic character (figs. 4 and 5). The potential for rapid progression (fig. 4) and the marked extent (figs. 3–5) of such hemorrhage also appears secondary to systemic anticoagulation. Most striking is the hypoechoic or anechoic appearance of acute hemorrhage in some patients (figs. 4 and 5). This is probably a direct result of systemic anticoagulation, as a reduction in the heparin concentration to minimal therapeutic levels has resulted in subsequent clotting of these hemorrhages with a progression to a "normal" echogenic appearance. Subarachnoid hemorrhage is not reliably imaged sonographically [17]. However, a large focal accumulation of subarachnoid blood with an associated mass effect was readily imaged in one patient (fig. 6). One premature ECMO patient with an intraventricular hemorrhage had no germinal matrix bleed at autopsy (fig. 3). The choroid plexus, more often the origin of intraventricular hemorrhage in the term infant [16], was postulated as the source.

No neonate in our series survived once an intraventricular or intracerebral hemorrhage was noted. In each case these infants were of 32 weeks gestational age or less and 1600 g or less birth weight (corrected weight for the hydropic patient was 1600 g). The larger and more mature infants placed on ECMO generally did well, however. Twenty of our 28 neonates had no intracranial hemorrhage, while a presumably insignificant grade I hemorrhage was seen in two.

It is important that sonographers recognize these unusual features of intracranial hemorrhage to insure an accurate intracranial examination in the ECMO patient. Particularly, the potential for unclotted hemorrhage must be recognized to differentiate it from other fluid collections or even ventriculomegaly. As more health-care centers start ECMO therapy programs, these sonographic considerations will gain even further significance.

REFERENCES

1. Wetmore N, McEwen D, O'Conner M, Bartlett RH. Defining indications for artificial organ support in respiratory failure. *Trans Am Soc Artif Intern Organs* **1979**;25:459–461
2. Babcock DS, Han BK. The accuracy of high resolution, real-time ultrasonography of the head in infancy. *Radiology* **1981**; 139:665–676
3. Sauerbrei EE, Digney M, Harrison PB, Cooperberg PL. Ultrasonic evaluation of neonatal intracranial hemorrhage and its complications. *Radiology* **1981**;139:677–685
4. Rumack CM, Johnson ML. Role of computed tomography and ultrasound in neonatal brain imaging. *J Comput Assist Tomogr* **1983**;7:17–29
5. Johnson ML, Rumack CM, Mannes EJ, Appareti KE. Detection of neonatal intracranial hemorrhage utilizing real-time and static ultrasound. *JCU* **1981**;9:427–433
6. Grant EG, Borts FT, Schellinger D, McCullough DC, Sivasubra-

manian KN, Smith Y. Real-time ultrasonography of neonatal intraventricular hemorrhage and comparison with computed tomography. *Radiology* **1981**;139:687–691

7. Bowerman RA, Donn SM, Silver TM, Jaffe MH. Review. Natural history of nenoatal periventricular/intraventricular hemorrhage and its complications: sonographic observations. *AJNR* **1984**;5:527–538, *AJR* **1984**;143:1041–1052
8. Bartlett RH, Andrews AF, Toomasian JM, Haiduc NJ, Gazzaniga AB. Extracorporeal membrane oxygenation (ECMO) for newborn respiratory failure: 45 cases. *Surgery* **1982**;92:425–433
9. Bartlett RH, Gazzaniga AB. Extracorporeal circulation for cardiopulmonary circulation. *Curr Probl Surg* **1978**;15(5): 1–96
10. Hardesty RL, Griffith BP, Debski RF. Extracorporeal membrane oxygenation: successful treatment of persistent fetal circulation following repair of congenital diaphragmatic hernia. *J Thorac Cardiovasc Surg* **1981**;81:556–563
11. Toomasian JM, Haiduc NJ, Wetmore NE, Gazzaniga AB, Bartlett RH. Refinements in prolonged extracorporeal membrane oxygenation in children and neonates. *J Extracorporeal Technology* **1979**;11:109–118
12. Kirkpatrick BV, Krummel TM, Mueller DG, Ormazabel MA, Greenfield LJ, Salzberg AM. Use of extracorporeal membrane oxygenation for respiratory failure in term infants. *Pediatrics* **1983**;72:872–876
13. Shuman WP, Rogers JV, Mack LA, Alvord EC Jr, Christie DP. Real-time sonographic sector scanning of the neonatal cranium: technique and normal anatomy. *AJNR* **1981**;2:349–356, *AJR* **1981**;137:821–828
14. Grant EG, Schellinger D, Borts FT, et al. Real-time sonography of the neonatal and infant head. *AJNR* **1980**;1:487–492, *AJR* **1981**;136:265–270
15. Papile L, Burstein J, Burstein R, Koffler H. Incidence and evolution of subependymal and intraventricular hemorrhage: a study of infants with birth weights less than 1500 gms. *J Pediatr* **1978**;92:529–534
16. Volpe JJ. Current concepts in neonatal medicine. Neonatal intraventricular hemorrhage. *N Engl J Med* **1981**;304:886–891
17. Mack LA, Wright K, Hirsch JH, et al. Intracranial hemorrhage in premature infants: accuracy of sonographic evaluation. *AJR* **1981**;137:245–250