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Cerebral CT in Fatal Courses of Resuscitated Sudden Infant Death

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Three children revived after sudden infant death syndrome were observed by cerebral computed tomography (CT). The scans were correlated with clinical and electroencephalographic findings of brain deterioration. The salient features of the CT presentations are described. Necropsies performed in two of the three cases revealed liquefactive necrosis.

Sudden infant death syndrome (SIDS) is the abrupt and inexplicable death of an apparently healthy infant. This event is said to be the most common cause of death in infants between 2 weeks and 1 year of age. Statistically it occurs in one to three or four individuals per 1,000 live births [1]. Most of these infants are discovered after death has occurred. In rare cases, sudden death occurs in the presence of persons who try to revive the infants. Sometimes resuscitation attempts result in stabilization of circulatory parameters and subsequent hospital admission. These comatose patients can be included in the definition of SIDS although complete brain death is temporarily postponed. In these circumstances the course of events and treatment after resuscitation is seldom recorded. This is a report of cerebral computed tomographic (CT) follow-up of three such cases.

Materials and Methods

Cranial CT scans were performed on a Siemens Siretom 2000 scanner in 16,500 patients in our hospital during the 4 year period ending in August 1982. Of these patients, 847 were less than 1 year old. Three of these young patients had suffered from SIDS but were temporarily revived. The CT scans were obtained to exclude injury, arteriovenous malformation (AVM), or hemorrhage as the cause of the sudden death. Clinical and electroencephalographic (EEG) findings are also reported. Necropsies were performed in two of the three cases.

Case Reports

Case 1

A male infant whose gestational age at delivery was 40 weeks weighed 3,025 g at birth. Apgar scores were 9 and 10 at 1 min and 10 min, respectively. The boy developed an inguinal hernia and was admitted for surgery in the 11th week of life, but surgery was postponed because of rhinitis. Four days after admission, cardiac

arrest occurred and breathing ceased. Resuscitation attempts led to stable circulatory parameters but no spontaneous regular breathing. CT 28 hr later (fig. 1A) showed widespread variable hypodensity without distinction between gray and white matter. The basal cist-

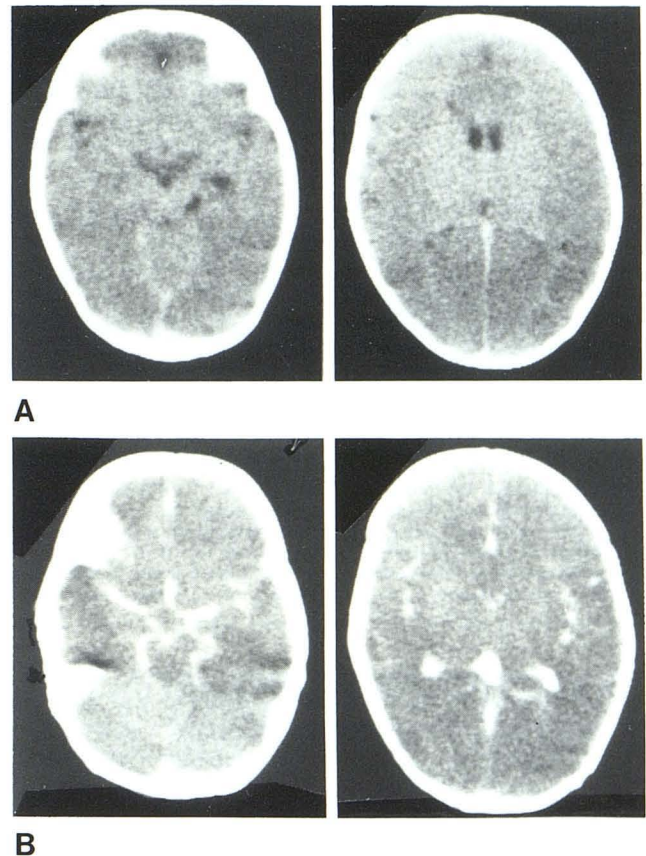


Fig. 1.—Case 1. CT scans in 3-month-old infant revived after SIDS. **A**, 28 hr postresuscitation. Widespread variable hypodensity (26 H) with lack of gray- and white-matter contrast. No mass effect. **B**, Contrast-enhanced CT 8 days postresuscitation. Parenchymal structures show increased hypodensity (13 H). Ventricular system is not visualized, but vascular structures are well delineated. No AVM is seen.

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terns and the ventricular system were within normal limits. No signs of hemorrhage were seen. EEG documented abnormal discharges of short duration and depression of amplitudes, diminishing to absence of electrical activity on the sixth day after SIDS. On the

eighth day after SIDS a second CT scan was obtained (fig. 1B) with administration of contrast medium under suspicion (by bruit) of AVM. This contrast-enhanced scan showed delineation of vascular structures but no evidence of AVM. The ventricular system was not visualized and the parenchymal structures showed homogenous hypodensity with a CT value of 13 Hounsfield units (H), a decrease from 26 H on the previous scan. The patient died on the 25th day after SIDS. Permission for necropsy was refused by the parents.

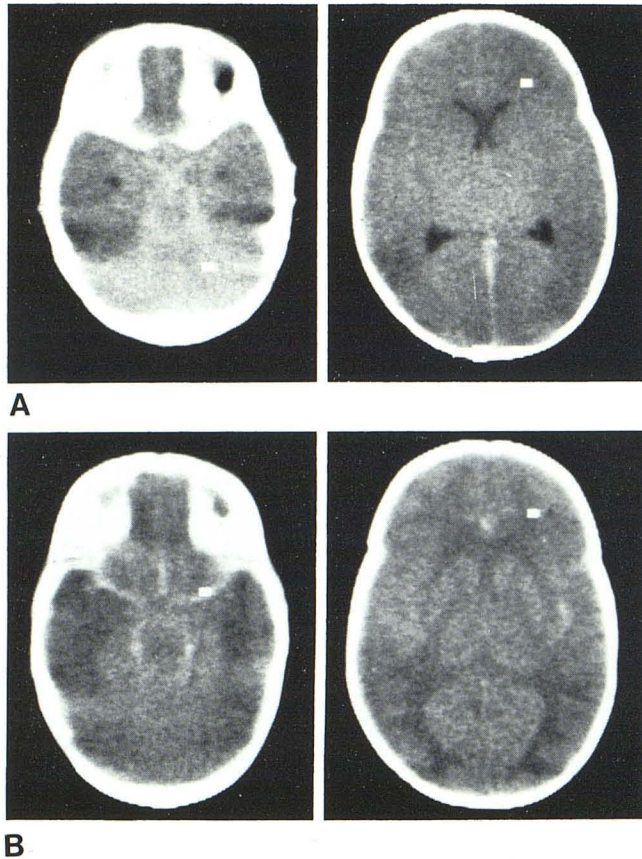


Fig. 2.—Case 2. CT scans in 3-month-old infant revived after SIDS. **A**, 3 days postresuscitation. Nearly homogeneous hypodensity (17–24 H) with lack of gray- and white-matter contrast. Ventricular system appears normal. **B**, 24 days postresuscitation. Increased general hypodensity (13 H) with severe leukomalacia. Ventricular system is no longer delineated, but vascular structures are visualized.

Case 2

A male infant was delivered normally at term. Birth weight was 3,110 g. The boy was found apparently dead in his crib at 3 months of age. Resuscitation led to stable circulatory parameters but assisted ventilation was necessary. EEG monitors showed gradual decrease in amplitudes but no absence of electrical activity. The first CT examination was performed 3 days after SIDS (fig. 2A). The scan showed nearly homogeneous hypodensity of the cerebral tissue (17–24 H) without gray- and white-matter discrimination, but the ventricular system appeared normal. A second CT scan 24 days after SIDS (fig. 2B) demonstrated increased general hypodensity (13 H) and severe leukomalacia. The ventricles could no longer be delineated, but the vascular structures were better visualized than in the earlier scan and contrast between gray and white matter was improved. There were no clinical signs of increased intracranial pressure. The patient died in a coma on the 29th day after SIDS.

Necropsy manifested gross cerebral and clear cerebellar liquefactive necrosis with dural adhesion (fig. 3A), but revealed no evidence of increased intracranial pressure. The ventricular system was collapsed. Microscopic examination (fig. 3B) documented the complete absence of ganglial cells in the cerebrum with only a few glial cells remaining. The cerebral tissue showed infiltration by inflammatory and mesenchymal cells via the leptomeninges.

Case 3

A male infant was delivered normally at term. Birth weight was 3,750 g. At 10 months of age, the boy was found unconscious with breathing arrested. Initial resuscitation was fully effective, but aspiration had occurred and hyperpyrexia developed. CT scan 10 hr after SIDS (fig. 4A) showed only zones of mild posterior periventricular hypodensity. A convulsive episode occurred 14 hr after resuscitation. Under signs of increasing intracranial pressure despite medication, SIDS recurred with circulatory and cardiac arrest 2

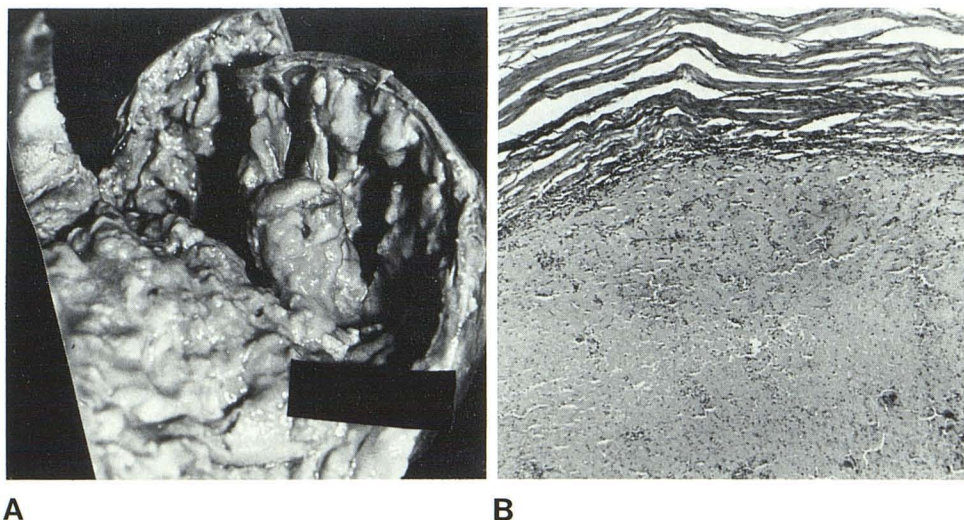
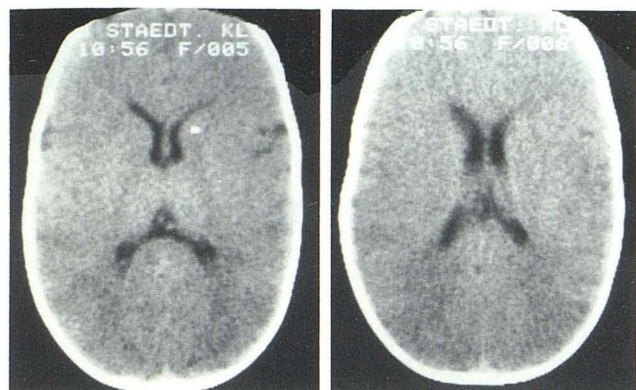


Fig. 3.—Case 2. Necropsy, 29 days postresuscitation after SIDS. **A**, Macroscopy. Gross liquefactive necrosis with dural adhesion. **B**, Microscopy of cerebrum. No ganglial cells and few glial cells. Slight infiltration by inflammatory and mesenchymal cells via leptomeninges.



A

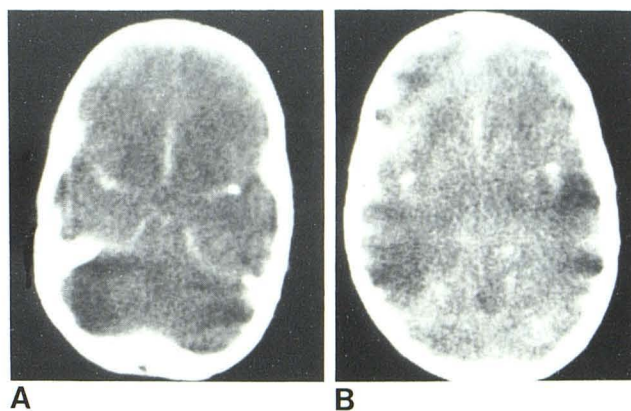
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Fig. 4.—Case 3. CT scans in 10-month-old infant revived after SIDS. A, 10 hr postresuscitation. Mild posterior periventricular hypodensity. B, 2 hr after recurrence of SIDS 2 days after initial resuscitation. Increased periventricular and cortical hypodensity with edema or incipient leukomalacia. Ventricles are not well delineated.

days after initial resuscitation. Further attempts at resuscitation stabilized only circulation. A CT scan 2 hr later (fig. 4B) showed no signs of hemorrhage but revealed severe periventricular hypodensity and edema or incipient leukomalacia. The ventricles were not well delineated but no ventricular dilatation was seen. EEG diminished gradually to absence of electrical activity the next day, and the infant died 3 days later (on the sixth day after the initial SIDS). Necropsy confirmed cerebral edema and incipient liquefactive necrosis of the cerebellum. There was no evidence of hemorrhage or AVM.

Discussion

In our three cases, as in most such cases, the factors precipitating SIDS remain unclear. We found no intracranial malformation or hemorrhages. Two of the three resuscitated infants showed neither clinical nor CT signs of increased intracranial pressure. In none of the three cases did CT findings suggest that respiratory and cardiac arrest were induced by cerebral abnormalities. On the contrary, the



A

B

Fig. 5.—CT scans in 8-year-old boy (A) and 9-year-old boy (B), comatose 16 days after succumbing to carbon monoxide. Severe general hypodensity. Ventricles not visualized, but intracranial vascular structures are well delineated. Liquefactive necrosis was found on necropsies.

brain damage appeared to have been the consequence of these events.

In contrast to cases of nonfatal perinatal asphyxia, where edema and encephalomalacia are present but limited in extent, only our case 3 exhibited such findings. But in this case dilation of the ventricles was not evident, possibly because the infant died in a toxicogenic edematous phase and the clinical course could be followed for only a short time.

In cases 1 and 2 the ventricles could not be visualized on the later CT scans. Necropsy in case 2 showed collapsed ventricles. The precise reason for this is not clear, but it seems to be the result of liquefactive necrosis secondary to a generalized anoxic lesion. The same phenomenon was found in the necropsies of two siblings who were resuscitated after succumbing to carbon monoxide and died comatose 40 and 20 days later, respectively. Cerebral CT scans in these two cases 16 days after resuscitation (fig. 5) were morphologically similar to those of the SIDS infants.

CT scans in all three cases of delayed SIDS demonstrated increasing general hypodensity of cerebral and cerebellar tissue while blood vessels and connective tissues such as the falx and tentorium retained their normal attenuation values. Despite the normal aspect of vascular structures on necropsy, we suggest that angiographic and microscopic evaluations be done in the future in such cases to establish the integrity of the cerebral circulatory system.

Only clinical and EEG findings have heretofore been reliable indicators of brain death after attempted resuscitation. Careful recording and reporting of cerebral CT findings in the preterminal course of more such cases may eventually provide enough data so that CT can be used as a diagnostic and prognostic tool in selected cases.

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