## Clinical Features of Ischemia in Cerebral Arterial Border Zones After Periods of Reduced Cerebral Blood Flow

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The clinical syndrome of borderzone ischemia is described in eight patients. Cerebral damage in four occurred during or after cardiac surgery and the other four patients had each experienced a period of severe hypotension. Cerebral blindness and visual disorientation are the most regular features and are produced by ischemic lesions in the parieto-occipital region, the common border-zone territory between all three arterial territories. Some patients show bibrachial sensorimotor impairment and a disturbance of volitional saccadic eye movement caused by more anteriorly placed ischemia in the anterior-middle artery border zone. Some show a temporary dyslexia/dysgraphia and memory defect related to bilateral parietotemporal lesions in the middle-posterior border zone.

(Arch Neurol 1987;44:934-940)

**B**eevor<sup>1</sup> made the first systematic study of the cerebral arterial territories by injecting colored dyes at equal pressure into the major arteries. He noted a sharp demarcation between each territory, and in the pial vessels connecting the peripheral branches of the main arteries, there were null points of equal pressure where no flow occurred (Fig 1). The border zone between anterior and middle territories is a sickle-shaped line on the lateral surface of the hemi-

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Fig 1.—A through H, Distribution of three major cerebral arterial territories of cerebral hemispheres in horizontal brain stices, A (highest) through H (lowest). Left hemisphere anterior cerebral territory, vertical hatching; middle cerebral territory, diagonal hatching; posterior cerebral territory, dotted. In right hemisphere, border zones are marked in heavy lines (after Beevor<sup>1</sup>).

Accepted for publication Jan 9, 1987.

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Fig 2.—Computed tomographic scan of patient 1 to show hypodense ischemic region (hatched).

Fig 3.-Computed tomographic scan, patient 2.

sphere that traverses the middle frontal gyrus, the upper part of the precentral and postcentral gyri, and the parietal lobule. In the parieto-occipital region, there is a point where all three territories meet. The posteriormiddle border zone passes close to the occipital pole and along the inferolateral border of the temporal lobe.

Although localized infarctions in these regions had been described previously,<sup>2,3</sup> Zulch<sup>4</sup> first suggested that selective damage occurred where perfusion pressure was lowest (die letzten Wiesen). He described border zones, not only in the hemispheres, but also in basal ganglia, cerebellum, and spinal cord. Romanul and Abramowicz<sup>5</sup> reported border-zone infarcton after occlusion of the internal carotid artery, and showed that the patency of the extracranial arteries and the configuration of the circle of Willis were crucial factors in determining the distribution of brain damage.

In the brains of patients who died shortly after cardiac surgery, Adams et al<sup>6</sup> identified selective border-zone infarction as a common pattern of injury and ascribed it to one or more episodes of abrupt hypotension; the patients usually had intact carotid arteries.

Brierley and Excell<sup>7</sup> reproduced some of these changes in the experimental primate subjected to repeated periods of hemorrhagic hypotension. Cortical ischemia was maximal in the parieto-occipital regions, but was also present in the basal ganglia. Most descriptions of the clinical features of border-zone infarction describe severe, diffuse, and permanent changes<sup>8-10</sup>; we describe a group of patients who sustained milder focal brain damage following hypotension or cardiac surgery, as an illustration of some characteristic features, and to indicate the favorable outcome in some cases.

## **REPORT OF CASES**

CASE 1.—A 51-year-old right-handed woman developed angina and syncopal attacks; an aortic valve replacement was recommended. The operation was uneventful and a prosthesis was inserted. Aortic occlusion time was 46 minutes and total bypass time, 63 minutes. Sinus rhythm was restored after a brief period of atrial fibrillation.

Twelve hours after surgery, her cardiac state was satisfactory, but she was drowsy, irritable, and restless. She responded to commands, but did not move her left arm or leg. She appeared to be blind, but pupil reactions to light were normal. Two days later her vision was unchanged; she spoke fluently, but made a number of dysphasic errors; comprehension of speech was also impaired. She was able to move the left leg to command, but the left arm was still weak; both plantar responses were extensor.

Two months later her vision was examined again. She could now see shapes and colors in the upper visual fields. Her left hand remained weak, but there had been some return of power in the shoulder and elbow. Her left leg had recovered completely. She was discharged home.

She was reexamined six years later. Vision had improved; she could identify a 2-mm object in the upper part of the field in each eye at 1 m. She could recognize objects, shapes, and colors in the upper field, but had no perception of light in the lower fields. She lived at home, but remained visually handicapped, requiring assistance in finding her way around.

Although she was able to see, visually directed pointing was grossly inaccurate in both upper fields. Fixation and pursuit eye movements were also jerky and inaccurate, but voluntary saccades appeared normal. Opticokinetic nystagmus was absent and she failed to blink to a visual threat. When confronted with a group of objects, she showed restriction of visual attention and could see only one object at a time. She was unable to count a group of six coins until allowed to touch them. There was a mild left facial weakness and slight spasticity of the left arm with marked residual hand and forearm weakness with cortical sensory loss in the hand. Her left leg was normal, except for the plantar response, which remained extensor.

Psychometric testing now showed no evidence of dysphasia. There was a mild visual memory defect and a marked impairment of form perception, although object identification by silhouette appeared normal, allowing for her visual acuity. Naming and matching of colors was preserved.

A computed tomographic (CT) scan showed bilateral occipital and parietooccipital lesions, which were more extensive on the right side where there was extension forward into the anterior-middle cerebral border zone (Fig 2).

Extensive asymmetrical borderzone infarction in this patient produced severe visual impairment of both acuity and visual orientation



Fig 4.—Computed tomographic scan, patient 3.



Fig 5.—Computed tomographic scan, patient 4.

with disturbance of smooth pursuit eye movement. Visual symptoms have persisted with only slight improvement for six years. By contrast, there was good recovery of the left hemiparesis and dysphasia. There was no documented hypotension in the perioperative period, but a single brief episode of atrial fibrillation was noted.

CASE 2.—This 35-year-old right-handed man was admitted to the hospital complaining of increasing angina. He had an 11-year history of hypertension and chronic renal failure. Four years previously, he had undergone bilateral nephrectomies, and the next year he had received a cadaveric renal transplant that had functioned well.

He was grossly overweight but normotensive. All peripheral pulses, including the carotids, were palpable. His vision was normal.

Coronary angiography showed extensive disease and he underwent triple-bypass grafting. The operation was uneventful, total bypass time was 59 minutes, and aortic cross-clamp time was 35 minutes. The patient was cooled to 32°C. No perioperative hypotension was noted.

Recovery from anesthesia was delayed; a single short episode of ventricular fibrillation responded to cardioversion. He remained drowsy, and on the second day, he had a left hemiplegia and cortical blindness. He required positive-pressure ventilation. He improved slowly, and by the sixth postoperative week he was alert without dysphasia. He was able to see and recognize small objects but was unable to fix, localize, or follow. There was severe visual disorientation; his lower visual fields were particularly affected. He remained moderately weak down his left side with cortical sensory loss. A CT scan showed bilateral occipital and posterior parietal infarcts (Fig 3). A few days later, he suffered a fatal myocardial infarction.

This patient sustained cerebral infarction during or after cardiac surgery, which was probably related to an episode of cardiac dysrhythmia in the postoperative period. Physical signs included cortical blindness progressing to visual disorientation, mild left hemiparesis, and sensory loss. There were bilateral parieto-occipital abnormalities on CT scanning.

CASE 3.—This 44-year-old gardener was admitted with angina pectoris refractory to medical treatment. There was a history of treated hypertension and myocardial infarction. He smoked 20 cigarettes daily.

On examination, his blood pressure was 130/90 mm Hg. Coronary angiography demonstrated double-vessel disease with good left ventricular function. Aortocoronary bypass grafting was recommended. The operation was uneventful; total bypass time was 84 minutes, with an aortic crossclamp time of 36 minutes. The patient was cooled to 32°C. There was no documented hypotension or cardiac dysrhythmia. Recovery from anesthesia was not delayed.

Weakness of upper limbs was noted two days later. At the time, he was alert and his visual fields were normal to confrontation. There were no features of visual disorientation or optic ataxia. Voluntary conjugate gaze was impaired to both sides. There was marked distal weakness of both arms, which was more marked on the right. There was also some weakness of right ankle dorsiflexion. All tendon reflexes were brisk; plantar responses were flexor; sensation in the arms was normal. A CT scan showed a low-density lesion in the center of the right parietal lobe (Fig 4).

Recovery was slow and two months after his operation, the right upper limb was still weak and spastic, and his right hand was clumsy, with impaired rapid finger movements. The left upper limb had virtually recovered. Venous digital imaging of his neck vessels and a second CT scan were both normal. After five months, his right hand remained weak, although it was still improving. He was unable to hold a screwdriver or a cup of tea. There was a feeling of a band around his left forearm, although there was no sensory loss.

Following successful coronary artery bypass grafting, this patient developed bibrachial weakness more marked distally and more pronounced on the right side. There was no cortical sensory loss. Recovery was slow and incomplete on the right side. A transient parietal abnormality was present on CT scanning.

CASE 4.—This 52-year-old right-handed man developed mild angina following a myocardial infarction. He subsequently developed intermittent claudication and worsening angina; he continued to smoke heavily. His blood pressure was 110/70 mm Hg. Six years later, coronary angiography showed severe disease and he underwent triple-artery-bypass grafting. The total bypass time was 85 minutes and the total ischemic time was 36 minutes. There were no perioperative complications and no documented episodes of hypotension.

On recovery from the anesthetic he was confused, agitated, and completely blind. On examination, he could not perceive light, but the pupils were symmetrical and reacted normally to light; the fundi and



Fig 6.—Computed tomographic scan, patient 6.



Fig 7.-Computed tomographic scan, patient 8.

cranial nerves were normal. He was also noted to be moving his arms poorly. By the following day, he was less confused and had regained some vision. He was able to read large print with both eyes and field testing showed a left homonymous lower quadrantanopia. Voluntary eye movements were normal to command but smooth pursuit movements were jerky and irregular in all directions, particularly in the vertical plane. There was no visual inattention and he showed severe optic ataxia when attempting to reach for objects. He made errors in all three planes of space, most notably in the sagittal plane. He did not react to visual menace, but blinking was normal when he was menaced with his own hand. He had difficulty fixating on objects introduced into the periphery of his seeing field and in transferring his gaze rapidly from one object to another. The tone was increased in the arms; there was minimal weakness of elbow movements but finger movements and hand grip were profoundly weak, worse on the left side. The reflexes were abnormally brisk on the left side. There was no weakness of the legs, and the reflexes were symmetrical; however, both plantar responses were extensor. There was no sensory impairment. The gait was ataxic.

Over the course of a month, he gradually recovered normal visual acuity although the field defect remained unaltered. Although visual disorientation persisted, the power of the hands improved to virtually normal. He was able to read and write without difficulty. A CT scan (Fig 5) showed bilateral low-density lesions in the parieto-occipital and posterior frontal regions. On review at six months, the lower inferior quadrantanopia persisted and he continued to be severely disabled by visuomotor ataxia. The tone and power of the limbs were normal and the reflexes were brisk but symmetrical, although both plantar responses were extensor. Sensation was normal.

Ischemic brain damage occurred during coronary artery surgery, although no hypotensive episodes were documented. Cortical blindness was followed by visual disorientation and left lower quadrantanopia, which have persisted. Bilateral hand and forearm weakness of the cortical type was present for a few weeks but recovered completely.

CASE 5.—This 30-year-old civil servant was admitted to the hospital unconscious following an assault. He had been stabbed in the right side of his neck and had bled profusely. On admission, his blood pressure was unrecordable. He was ventilated and required a tracheostomy. After transfusion with 6 U of blood, the neck wound was explored and a large posterior pharyngeal laceration was sutured. The right carotid artery was seen and noted to have escaped injury.

On recovery from anesthesia, he developed myoclonic jerking of the left side of the face, the left arm, and right leg. He initially had a dense, right homonymous hemianopia, and pursuit eye movements were full but jerky. There was weakness of both hands and the right leg, with bilateral extensor plantar responses. A CT scan and technetium brain scan were both normal. Digital venous imaging failed to show the left vertebral artery. The patient improved, and after three days the right hemianopia had become less marked; he was able to see colors, movements, and vague shapes on the right side. It was noted that visual localization was impaired in both lower visual fields, as was shape discrimination. Voluntary eye movements were normal. Limb weakness was unchanged. Reading was slow and he made occasional dysphasic errors.

At the time of his discharge, three weeks after the assault, he had improved further. Goldmann perimetry showed central defects in both visual fields, and localization was still defective in both lower visual fields. Pursuit eye movements remained broken up. Weakness of both hands persisted, with astereognosis bilaterally and impaired joint-position sense. His reading and writing remained slow but accurate. When seen three months later, he had improved only a little. His visual fields were unaltered, although eye movements were now normal. Both hands were weak and slightly wasted, with impaired pro-prioception on the left side. Plantar responses remained extensor. Electromyography of the left hand showed no evidence of denervation. A left temporal abnormality persisted on a repeated electroencephalogram.

Bilateral cerebral hemisphere lesions developed after a period of hemorrhagic hypotension. Neurologic signs suggested damage to hand areas of the sensorimotor cortex on both sides, and to a lesser extent to parietooccipital cortex.

CASE 6.—A 50-year-old right-handed postman with a five-year history of diabetes and increasing shortness of breath awoke normally, but suddenly collapsed while eating breakfast. The cause of his collapse was later shown to be a massive pulmonary embolus. He rapidly became unconscious with intense dyspnea, cyano-

Clinical Features of Border-Zone Lesions				
Patient No./ Age, y/Sex	Causative Event	Clinical Feature (Acute Stage)	Site of Lesion on Computed Tomographic Scan	Outcome
1/51/F	Cardiac surgery	Cortical blindness; visual disorientation; dysphasia; left hemiparesis	Bilateral occipitoparietal	Persistent visual disorientation; memory defect
2/35/M	Cardiac surgery	Cortical blindness; visual disorientation; left hemiparesis	Bilateral occipitoparietal	Died
3/44/M	Cardiac surgery	Bibrachial weakness	Right parietal lesion	Improved; persisting clumsiness of right hand
4/52/M	Cardiac surgery	Cortical blindness; left quadrantanopia; visual disorientation; bilateral hand weakness	Bilateral occipitoparietal and frontal lesions	Persistent visual disorientation
5/30/M	Hemorrhage; hypotension	Right homonymous hemianopia; bibrachial paresis		Hand weakness persists
6/50/M	Pulmonary embolism; hypotension	Cortical blindness; saccadic gaze paresis; dysphasia; dyslexia	Bilateral occipitotemporal lesion	Persistent amnesia
7/70/M	Abdominał surgery; pulmonary embolism	Saccadic gaze palsy; cortical hand paresis		Persisting hand weakness; died after 18 mo
8/65/M	Pneumonectomy; post- operative hypotension	Bilateral hemianopia; visual disorientation; saccadic gaze palsy	Bilateral occipital, occipitoparietal, and frontal	Right hemianopia; visual disorientation, left side

sis, and pallor. His limbs were rigid.

On admission to the hospital 40 minutes later, he was still unconscious and unresponsive to pain. Respirations were periodic. His eyes deviated to the left. The pupils were reactive and his limbs showed generalized increase in tone. Blood pressure on admission was 90/60 mm Hg.

Within two hours, he had recovered consciousness and was irritable and aggressive, moving all limbs freely. He appeared to be blind. Spontaneous speech was limited to his wife's name. Brain-stem functions were intact and the pupils reacted normally to light. Increased tone in the limbs with hyperreflexia and extensor plantar responses were noted.

He improved gradually and five days later was alert, cooperative, fully oriented, and responding correctly to simple commands but not to two-stage commands. His speech contained many dysphasic errors and he tended to perseverate. He could perceive finger movements in all parts of the field but could not follow or fix on a moving object. There was no optic ataxia or visual inattention. Random eye movements were normal but voluntary visual saccades in all directions were hypometric and poorly sustained. The limbs showed slight left-sided weakness with hyperreflexia. Both plantar responses remained extensor. There were no grasp reflexes. Two weeks after admission, psychologic testing showed some naming difficulties and a marked dyslexia and dysgraphia. Perceptual and visuospatial tests were well performed, but both verbal and visual short-term memory was very poor.

Over the following three years, he has remained well, apart from continuing dyspnea. He has a mild residual left-sided weakness and makes occasional dysphasic errors. Comprehension of language is intact. He makes only minor reading and spelling errors. He complains of difficulty in judgment of distance when driving, but no features of visual disorientation or optic ataxia can be detected. Tests of short-term verbal memory remain impaired, but visual memory is now within the normal range.

It is probable that this patient sustained an episode of severely reduced cardiac output at the time of the pulmonary embolism. There was cerebral irritation with temporary loss of speech, cortical blindness, and limb spasticity. These features improved, leaving a state of dysphasia, dyslexia, and dysgraphia and impairment of verbal and nonverbal memory. A CT scan (Fig 6) showed bilateral symmetrical posterior temporal lesions.

CASE 7.—A 70-year-old man with mild hypertension (blood pressure, 170/100 mm Hg) developed deepening jaundice and was found to have carcinoma of the pancreas. A partial pancreatectomy was performed. Seven days after the operation, he collapsed with acute dyspnea. His blood pressure was 110/80 mm Hg, with atrial fibrillation of 100 beats per minute. He was drowsy, but did not lose consciousness. He was thought to have had a pulmonary embolus.

The following day, he was alert but noted to be not moving his arms. Examination showed hypometric and unsustained conjugate ocular movements in all directions, both volitional and on smooth pursuit. Oculocephalic eye movements were normal. There was no blinking to a visual threat and optokinetic nystagmus was absent. His visual acuity was normal and visual fields were full. He had a profound flaccid weakness of both arms with preserved reflexes. Joint-position sense was absent in the fingers of both hands. The legs showed no weakness. The left plantar response was extensor.

Two days later, volitional eye movements had improved, but smooth pursuit remained grossly abnormal. He was unable to fixate accurately on an object in his field of vision. Over the next month, he slowly improved. Voluntary power returned to the shoulders and elbows, but wrist extension and flexion and intrinsic hand muscles still remained weak on both sides. Slight wasting was noticed in the intrinsic hand muscles; however, electromyography showed no signs of denervation. Voluntary saccadic eye movements were improved, although still unsustained. Smooth pursuit movements were broken up and inaccurate. His visual fields remained full and visual judgment of relative size and distance was normal.

Three months later, bilateral hand weakness was still present and both arms showed slight spasticity. There were increased tendon reflexes in the arms with pronounced finger jerks. Joint position sense and two-point discrimination were absent in the fingers.

He returned home, but remained disabled by hand weakness and died of disseminated carcinomatosis 18 months later.

Following a period of relative hypotension probably caused by pulmonary embolus, this patient developed a bilateral supranuclear gaze palsy affecting both voluntary and pursuit eye movements. This was accompanied by bibrachial weakness, which improved, leaving him with profound hand weakness of cortical type. No visual or perceptual defects were found.

CASE 8.—A 65-year-old man underwent right pneumonectomy for carcinoma of the lower lobe. There was no previous history of cerebrovascular disease, but his right eye had been blind for many years from retinal detachment. The day following operation, he had a hypotensive episode when systolic blood pressure fell to 65 mm Hg. After this, it was noted that he moved his left arm less than the right and his head and eyes were turned to the right side. Examination some hours later showed a blood pressure of 100/70 mm Hg. He was fully conscious and replied to simple questions. He was unable to move his eyes voluntarily in any direction or to follow a moving object. Visual acuity was reduced to hand movements in his left eye. His limbs showed normal power, but there was impaired position sense in both hands.

By the next day he had improved. Visual fields on confrontation showed a right homonymous hemianopia with additional loss of the lower quadrant on the left side. In the remaining field, he could recognize and name small objects and identify colors. He had very poor localization in his remaining field. Voluntary saccades were absent in all directions except downward. He was unable to follow a moving stimulus. Oculocephalic reflexes were normal. He had slight right facial weakness. Examination of the limbs showed slight weakness of the right arm and leg and increased reflexes. Both plantar responses were extensor.

Two weeks later, he showed further improvement. The complete right hemianopia persisted with visual disorientation in the remaining left field. Voluntary saccades were hypometric and ill sustained. Pursuit movements were jerky. Judgment of distance was defective in the left field, although object recognition and appreciation of shapes and colors were normal. Reaction to visual threat was absent. He was discharged to another hospital after six weeks and no further follow-up data are available. A CT scan performed three weeks after operation showed extensive infarction involving the occipital lobe on the left side and with further infarction in the left posterior frontal region. The right side showed a parieto-occipital infarct (Fig 7).

An episode of postoperative hypotension was followed by an incomplete bilateral homonymous hemianopia with visual disorientation in the remaining field. There was a transient supranuclear palsy of voluntary eye movements and cortical sensory loss in both hands.

The right homonymous hemianopia persisted.

## COMMENT

Many of the earlier reports of brain damage occurring during cardiac surgery describe either embolic infarction or severe diffuse ischemic damage to both hemispheres caused by a period of reduced cerebral blood flow.<sup>6</sup> Many of the patients failed to recover consciousness after operation, and survived for a short time in a vegetative state with bilateral spasticity and no response to visual or auditory stimuli. Various patterns of ischemic infarction were described, the most affected regions being the posterior portion of the hemispheres and in the border-zone territories.

There have been a few reports of patients surviving after cardiac sur-



Fig 8.—Composite computed tomographic scan (superimposition of Figs 2 through 7) to show tomography of border-zone lesions in all cases.

gery or after hypotension from other causes who have shown features of brain damage localized to the parietal and occipital lobes.<sup>11</sup> Instances of considerable recovery from states of partial or complete cerebral blindness have been recorded, especially in children.<sup>12,13</sup>

Gilman<sup>14</sup> examined a series of 35 patients after cardiac surgery and found evidence of brain damage in 12. He deduced that unilateral defects were usually embolic, but that bilateral defects were caused by episodes of systemic hypotension and identified the parietal lobes as sites of special vulnerability with dyslexia and memory loss the most prominent clinical features.

Other large surveys of neurologic damage after cardiac surgery describe states of confusion, visual disorientation, or reversible delirium. Abnormal signs found most frequently were extensor plantar responses, primitive reflexes, and visual-field defects.<sup>15-18</sup>

We describe here a group of patients who show a border-zone pattern of ischemia on CT scan and who have a characteristic symptomatology (Table). In four patients, the defect appeared after cardiac surgery, in two it appeared after documented hypotension, and in two it appeared after pulmonary embolism. The clinical features confirm previous reports of selective vulnerability in the parieto-occipital regions. In addition, however, the patients showed signs of more extensive lesions involving the frontoparietal and parietotemporal regions, all situated in border-zone territories (Fig 8).

The syndrome of bilateral lesions of the parieto-occipital region is well known from earlier studies of missile wounds.<sup>19</sup> It consists of a bilateral lower altitudinal visual-field defect, a difficulty in visual judgment of size, distance, and movement, and a disorder of smooth oculomotor pursuit. There is also a restriction of visual attention and an absence of blink reaction to visual threat. A prominent sign is difficulty with movements performed under visual guidance (optic ataxia).<sup>8</sup>

Four of our patients show in addition to visual disturbance an unusual type of weakness and sensory loss involving both the upper limbs.<sup>10,17</sup> Initially involving the whole limb, the weakness may later be confined to the hands and forearms. Spasticity is not marked, but the tendon reflexes are enhanced and brisk finger jerks may be elicited. Superficial sensation is retained, but there is cortical sensory loss in the fingers. Patients find such actions as writing, handling cutlery, and dressing very difficult. There may be a difference in degree between the two sides. There is no involvement of the face and speech is normal. Extensor-plantar responses may be found but in other respects the legs are

normal. The bibrachial paresis can be attributed to lesions of the motor and sensory cortex on each side affecting the regions concerned with movements of the upper limbs and hands. This correlates well with the site of the middle-anterior cerebral border zone that traverses the precentral and postcentral gyri and the parietal lobule.<sup>20</sup>

An additional disturbance of volitional eye movements was a feature of the acute stage of the illness in three patients. Although alert and cooperative and clearly understanding the commands, patients were unable voluntarily to perform saccadic eye movements in any direction, using head thrusts in an attempt to do so. Oculocephalic movements were normal.

This abnormality was transient in all patients and although voluntary saccades recovered in a few days, they remained hypometric and impersistent for a longer period. These findings presumably indicate reversible damage to both frontal eye fields.<sup>21</sup>

The third aspect of border-zone symptomatology consists of an initial period of cortical blindness that rapidly improves, but leaves a marked dyslexia, dysgraphia, and dyscalculia and a memory defect both for verbal

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9. Geschwind N, Quadfasel FA, Segarra J: Isolation of the speech area. *Neuropsychologia* 1968;6:327-340. and nonverbal material.<sup>14,15</sup> The anatomic substrate for such a defect appears to be bilateral parietotemporal lesions in the position of the border zone between middle cerebral and posterior cerebral territories.

A feature of all the patients reported on here is the degree to which the initially severe neurologic deficit recovered. Six of eight patients had markedly impaired vision at one time, yet only one was left with a permanent defect. Three had a severe residual sensorimotor defect in both hands; three retain some elements of visual disorientation shown by a persisting disturbance of judgment of distance; and two still have some memory defect.

The degree of recovery indicates that the threshold for cerebral infarction may not have been reached and that the border-zone territories have sustained only a reversible ischemia. Computed tomographic scanning may be normal, but isotope scanning, which may detect regions of ischemic edema and vascular leakage, may prove to be more helpful in diagnosis in the early stages.<sup>22</sup>

The mechanism of cerebral damage during cardiac surgery has not been established. Global reduction in cere-

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Border-zone infarction is a particular hazard of patients with carotid occlusion; because of the increased resistance of collateral pathways the brain is perfused at a reduced pressure.<sup>25</sup> In such cases, the infarction is usually unilateral and affects only the anterior carotid border zone.

Although serious lasting brain damage is rare after cardiac surgery, the numbers of patients at risk increase yearly. More complete records of circulatory events,<sup>23</sup> better monitoring of brain function during the perioperative period, and more systematic examination of patients before and after surgery may contribute to a fuller understanding and eventually to the prevention of border-zone ischemia.

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