SUMMARY. Cardiac dysrhythmia may be responsible for signs of cerebrovascular insufficiency in some patients. Ten patients with cerebrovascular insufficiency were monitored for up to 24 hours with the Holter EKG monitor. Eight of the patients had transient ischemic attacks and two had mild deficits from a completed stroke. Eight of the ten patients had associated abnormal cerebral angiography. Holter EKG monitoring in these ten patients showed no abnormalities in four patients. The remaining six had a variety of cardiac dysrhythmias. Although there were a variety of cardiac, cerebrovascular, and neurological abnormalities in these ten patients, six of these patients had a disturbance in cardiac rhythm or conduction which could have been directly associated with or suggest an etiology for the patient's neurological event.

CORDAY et al.1, 2 and others3, 4 have emphasized that cardiac dysrhythmias may affect the cerebral circulation. As Corday et al. 2 pointed out, more than 50% of patients with cardiac dysrhythmias are not aware of a dysrhythmia and they may present to the physician with neurological manifestations or with symptoms of coronary insufficiency. Barnes2 asserted that weakness, vertigo, nervousness, insom-

nia, and even syncope may occur. Such generalized symptoms of cerebrovascular insufficiency may be more frequent than realized. Corday et al. 1 emphasized that rapid cardiac dysrhythmias are apt to induce sufficient reduction in cerebral perfusion to cause cerebral ischemia. If cerebral artery narrowing from atherosclerosis or kinking is also present, it is entirely possible that the collateral circulation to a vital area of the brain will fail because of a reduced head pressure and blood flow. Sulg et al. 5 and Shapiro et al. 6 have demonstrated in man that cardiac dysrhythmias may significantly reduce cerebral blood flow (CBF).

Recently, McAllen and Marshall7 described patients with cardiac dysrhythmia with transient ischemic attacks (TIAs). They considered that the possibility of TIAs being due to cardiac dysrhythmias seemed to be sufficiently ap-

preciated. As suggested by Corday et al., 2 the introduction of the Holter monitor10 allows one to monitor patients to detect transient dysrhythmias that may be associated with generalized or focal neurological symptoms. The purpose of this report is to describe ten patients with cerebrovascular insufficiency who had long-term Holter electrocardiographic monitoring for the detection of cardiac dysrhythmia associated with cerebrovascular symptoms.

Methods

Nine patients were admitted to the Neurology Service of the North Carolina Baptist Hospital because of neurological symptoms suggestive of cerebrovascular insufficiency. One additional patient was admitted because of two episodes of syncope. As well as complete physical, neurological, and cardiovascular examinations, aorta-cervical-cranial angiography was performed in each case. Similarly, up to 24-hour Holter EKG monitoring10 was carried out in each patient to determine whether or not cardiac dysfunction was contrib-

uting to the neurological deficit.

Case Reports

Case 1 (NCBH #54 54 07)

A 61-year-old woman had weakness and numbness of the left arm approximately four or five weeks prior to admission. Within three minutes her strength returned. On two subsequent occasions, approximately three or four weeks before admission, these same symptoms recurred. Blood pressure was 120/70 mm Hg in the right arm and 130/75 mm Hg in the left arm when the patient was sitting. Pulse was 76 and irregular with premature beats. The remainder of the physical examination was normal. On neurovascular examination there was a systolic bruit heard over the bifurcation of the right common carotid artery. Her neurological examination was normal.

Holter monitoring showed frequent premature atrial contractions with aberrant conduction throughout the tracing, particularly with activity. No sustained dysrhythmias were apparent.

Angiography showed atherosclerotic narrowing of the right internal carotid artery with stenosis of approximately 80% of the cross-section diameter. There was a small atherosclerotic plaque of the left internal carotid artery. The patient had a right carotid endarterectomy and recovered without complications. She was seen six months after discharge and was in good health and had no neurological symptoms. The diagnosis was TIAs with right internal carotid artery stenosis.

Case 2 (NCBH #45 48 51)

This 47-year-old white woman was admitted to the hospital for evaluation of a five-day history of transient numbness in her right arm. She also had some difficulty in calculations and was described as having a mild personality change. Approximately seven years ago the patient was evaluated for a mild right hemiparesis which included angiography and was reported as normal. Her right hemiparesis resolved except for a suggestion of a right cen-

tral facial weakness and she had been doing well until three weeks prior to admission. Her recent symptoms began when she awakened in the morning and noted numbness of her
right arm which lasted for about five days. She had no weakness.

Physical examination showed the blood pressure to be 170/100 mm Hg, but otherwise was normal. On neurovascular examination there were no bruits heard in her neck. Neurological examination was normal except for the suggestion of a right central facial weakness. The patient was hesitant in making calculations but was correct in her answers. She had normal strength and muscle tone without evident weakness in her right arm or leg. Sensory examination to touch and position sense was intact but pinprick in her right forearm felt different as compared to the left. She described this as being more tingling than a pinprick. Deep tendon reflexes were slightly increased on the right side without any pathological reflexes.

Holter monitoring showed normal sinus rhythm with occasional episodes of slower coronary sinus or nodal rhythm at a rate of 80 per minute. No symptoms were reported with the dysrhythmia recording. No other sustained or prolonged dysrhythmias were recorded.

Aortic study angiography performed in February, 1968, showed bilateral carotid artery disease with stenosis of the left carotid and irregularity of the right carotid. On the left side the common carotid in the neck up to the area of the bifurcation appeared normal in caliber and size. At the bifurcation, there was some irregularity in the lumen and distal to the bifurcation of the artery it tapered down and showed a smooth symmetrical narrowing to the base of the skull where the artery entered the carotid canal. At the point where the artery turned medially there was an area of stenosis with only approximately 15% to 20% of the lumen remaining. Distal to this side of the artery intracranially its branches appeared normal. The right carotid artery distal to the bifurcation showed diffuse involvement of a beaded-type irregularity of the luminal caliber without any significant compromise of the lumen. This is the appearance that is seen in fibromuscular hyperplasia of the arteries.

The patient did well during a three-day hospitalization. She was discharged with a mild right hemiparesis and was to take aspirin, 10 grains twice daily. The diagnosis was cerebral infarction in the distribution of the right middle cerebral artery due to fibromuscular hyperplasia with a mild right hemiparesis, and stenosis of the left carotid artery.

Case 3 (NCBH #55 52 08)

A 61-year-old man was admitted to the hospital with symptoms occurring two and one-half months prior to admission. While driving his car, he noted the sudden onset of vertigo and a slight change in hearing. This feeling passed in a few seconds. During the next two weeks he had a constant feeling of unsteadiness while walking but no longer had vertigo. There was no incoordination of the extremities, nausea, or vomiting. The unsteadiness gradually improved, but one morning upon awakening he had another vertiginous episode. Shortly thereafter he noted his speech was slurred. He was taken to a local hospital within five hours and both the vertigo and speech difficulty disappeared. There was no associated weakness of the extremities. Ten years ago while hospitalized for a myocardial infarction, he had a stroke. The only residual effect was a decreased sensation on the left side of his body and face. There had been no changes in his symptoms during the last ten years until the present illness. His medications prior to this admission were Digoxin, 0.25 mg daily; Quinidine, five grains b.i.d.; Coumadin, 5 mg once a day; and Probanthine, one tablet in the evening. Since his myocardial infarction he had had a cardiac dysrhythmia.

Physical examination showed the blood pressure to be 145/70 mm Hg in his right arm and 130/70 mm Hg in his left arm when sitting. Pulse was 88 and regular. The remainder of his physical examination was normal except for a low-grade aortic diastolic soft blowing murmur. On neurovascular examination there were no bruits heard over his neck, head, abdominal aorta, or femoral arteries. Neurological examination showed decreased pinprick on the left side of the body and face. Position sense and vibration were normal. Cortical sensory testing showed no abnormality. Deep tendon reflexes showed a 3+ left biceps jerk and a 3+ knee jerk on the left with a 2+ on the right side. No abnormal reflexes were noted. His gait was normal.

Holter monitoring showed infrequent premature ventricular contractions primarily unifocal, but occasionally from a separate foci with a different coupling interval. No sustained dysrhythmias were noted.

Cerebral angiography showed a smooth atheromatous plaque on the posterior wall of the left internal carotid artery at its origin. No significant stenosis was noted. In the left cerebral hemisphere there was an abnormal blush in the area of the angular gyrus with a diameter of approximately 3 cm, and an early filling vein was noted to drain this area. The right common carotid artery system was normal. Both vertebral arteries were visualized and were approximately the same size and no stenosis at the origin of these vessels was noted.

There were no complications or further recurrence of symptoms. On the third day of admission, Coumadin was discontinued. When discharged, he was instructed to continue taking his previous medications. The diagnosis was mild cerebral infarction in the distribution of the right middle cerebral artery.

Case 4 (NCBH #55 40 23)

This 53-year-old woman had had hypertension for the past ten years. Approximately three or four months before admission she first noted a useless feeling of her left upper extremity. She then noted definite weakness which interfered with her housework as well as feelings of numbness and paresthesias described as a tingling sensation and a sensation of coldness. The weakness improved considerably in the ensuing few weeks but she still had a sensation of numbness in the extremity. Approximately two months before admission she had episodes of diplopia which have recurred frequently since then and lasted for periods of approximately five minutes. There were no episodes of transient blindness or blurring of vision. Approximately five weeks prior to admission, the patient had one episode of marked weakness over the entire extent of the left side of her body associated with numbness. She was quite unsteady in her walking at this time. This episode was short-lived. Generally she had not noticed any weakness of her left extremity to the extent of her left arm weakness. There were no episodes of loss of
The liver was palpable at the costal margin. Blood pressure and posteriorly with minimal increased expiratory times. Inspiratory rales in the upper lobes of the lungs bilaterally weakness of the left arm lasting for about 12 hours. He was weakness of the left side of his face. His symptoms cleared up the following day. Approximately eight months before admission, he had a similar attack of numbness and weakness of the left arm lasting for about 12 hours. He was placed on anticoagulant medication but apparently rarely took the medication.

His general physical examination was normal except for inspiratory rales in the upper lobes of the lungs bilaterally and posteriorly with minimal increased expiratory times. The liver was palpable at the costal margin. Blood pressure was 130/70 mm Hg. Neurovascular examination was normal, as was the neurological examination except for questionable left facial weakness and a slightly decreased sensation to pinprick and light touch on the left side of the face.

Holter monitoring showed no EKG abnormalities. Normal sinus rhythm was present throughout the monitoring. Cerebral angiography was normal. During hospitalization the patient did not have further TIAs and he was discharged home to be followed by his referring physician. When seen two years later the patient had had no further neurological symptoms but had had progression of his emphysema. The diagnosis was TIAs.

Case 5 (NCBH #40 31 72)

A 68-year-old white man had been in good health until approximately one year before admission when he had sudden onset of numbness and weakness of the left arm. This episode lasted all day. The next day he had some numbness remaining. Two days later another episode occurred. When seen in the Emergency Room of this hospital, he had weakness of the left side of his face. His symptoms cleared up the following day. Approximately eight months before admission, he had a similar attack of numbness and weakness of the left arm lasting for about 12 hours. He was placed on anticoagulant medication but apparently rarely took the medication.

His general physical examination was normal except for inspiratory rales in the upper lobes of the lungs bilaterally and posteriorly with minimal increased expiratory times. The liver was palpable at the costal margin. Blood pressure was 130/70 mm Hg. Neurovascular examination was normal, as was the neurological examination except for questionable left facial weakness and a slightly decreased sensation to pinprick and light touch on the left side of the face.

Holter monitoring showed no EKG abnormalities. Normal sinus rhythm was present throughout the monitoring. Cerebral angiography was normal. During hospitalization the patient did not have further TIAs and he was discharged home to be followed by his referring physician. When seen two years later the patient had had no further neurological symptoms but had had progression of his emphysema. The diagnosis was TIAs.

Case 6 (NCBH #19 05 29)

A 55-year-old white man had had hypercholesterolemia for a number of years with cholesterol blood levels being in the range of 450 to 500 mg%. Two days before admission he noted that his eyes could not focus and his head felt like it was going to explode. This attack lasted for 10 to 15 minutes. He said that although he could not focus his eyes he was able to see. He could see his wife and pictures on the wall but could not focus on anything very clearly. He had no dizziness or vertigo. He did have tingling in both arms but was able to move all four extremities. The day prior to admission he was examined in the Emergency Room of this hospital and found to have a pulse of 60 and a blood pressure of 130/60 mm Hg. During the episode of blurred vision he had no other neurological symptoms nor did he have chest pain. In 1970 he had been admitted to this hospital for a myocardial infarction and since then his pulse had been in the range of 50 to 52 beats per minute.

In 1966, at the age of 44, the patient was admitted to the hospital because of dizzy spells and chest pain of three to four years' duration. The pain was aching in character and sometimes would last several days but improved with exercise. For approximately three to four years he had had episodes of lightheadedness associated with inability to focus his eyes. He had three such episodes which lasted five minutes each. He had never lost consciousness and could maintain normal motor activity during the episodes. On examination at that time there was a Grade 3/6 high-pitched right subclavian bruit and posterior neck bruit. There was a Grade 2-3/6 high-pitched short systolic bruit over the right common carotid artery below the bifurcation. The remainder of the pulses were equal. An aortic arch angiogram was performed at this time and there was a localized stenosis of the origin of the right subclavian artery with a steal phenomenon to the right vertebral artery. In May, 1966, a right subclavian endarterectomy with application of a dacron patch was performed. The patient did well until 1970 when he had a myocardial infarction. In 1973 he was seen by an ophthalmologist with a history of approximately three months' duration of 15 attacks with the onset of visual difficulty, lasting from 15 to 20 seconds. He had no perioral numbness, but had occasional scotoma particularly in the right visual field which was described as a yellow spot that appeared more often in his right field that left. He was diagnosed at this time as having vertebrobasilar TIAs.

On physical examination on this admission, his bloo
pressure was 130/70 mm Hg. The remainder of the physical examination was normal. Neurovascular examination showed Grade 2/6 carotid bruits bilaterally. Neurological examination was normal.

Holler monitoring showed a sinus rhythm but the rate varied from 45 to 85. The monitor strip suggested Wolff-Parkinson-White syndrome with a slow rate of 45 at rest and increasing to 70 to 80 beats per minute with walking. No paroxysmal tachyarrhythmias were noted.

The patient had no further neurological symptoms during his eight-day hospital stay. The diagnosis was TIAs possibly of the temporal lobe with carotid stenosis, and post-subclavian steal correction.

Case 7 (NCBH #55 89 20)

This 61-year-old white man had had attacks of dizziness and ataxia of five weeks’ duration. The patient described these as episodes of dizziness, ataxia, blurred vision, weakness, and perioral numbness with dysarthria which lasted for 20 to 30 minutes with complete resolution between attacks. The weakness involved all the extremities, but at times the right arm was more involved than the left. He had no syncope or diplopia. There were no other neurological symptoms.

Physical examination showed his blood pressure to be 150/90 mm Hg. His general physical including the cardiovascular system was normal. The neurovascular and neurological examinations were normal. Holler monitoring revealed a sinus arrhythmia with rates varying from 65 to 120. No dysrhythmias were noted.

Cerebral angiography showed 90% stenosis in the midportion of the basilar artery with marked slowing of flow and delayed emptying of the cephalad portion of the basilar above the stenosis. The left anterior-inferior cerebellar artery was markedly enlarged, suggesting that it served as a collateral vessel. There were marked irregularity of the more caudal portion of the basilar and some irregularity of the left vertebral. The right vertebral artery was not filled. Injection of both carotids in the neck demonstrated marked irregularity at the origins of the internal carotid arteries with slight dilatation and shaggy posterior irregular margination. The intracranial distribution of the carotid vasculature was normal. There was marked atherosclerotic disease at the origin of both internal carotid arteries and the carotid arteries in the siphon. The intracranial distribution of both carotid arteries was normal.

During hospitalization the patient had a few more transient episodes of perioral numbness with slight confusion lasting a few minutes. After such episodes the neurological examination showed the patient to be somewhat confused and incoherent for about 30 seconds but there was no hyperreflexia on the right side. The diagnosis was TIAs with vertebrobasilar insufficiency due to stenosis of the basilar artery.

Case 8 (NCBH #22 20 54)

This 61-year-old white man had had two episodes of syncope in the past one and one-half months before admission. The first one occurred while he was lying in bed. He stated that there was a twilight zone and he passed out for an unknown period of time. This was followed by a period of confusion which lasted for 5 to 10 minutes. There were no seizure, chewing of his tongue, incontinence, muscle contractions, or blurred vision. The second episode occurred several days prior to admission while he was driving his car. He said he was looking straight ahead when he had an episode similar to the one previously mentioned and had a minor automobile accident. Three or four weeks prior to admission he noted numbness of his right leg which lasted a few minutes and was relieved by walking around. He had had hypertension for the last 20 years for which he took one Diuril tablet a day. He also had had headaches followed by dizziness of 30 minutes’ duration. He had had no true vertigo. There were no other neurological symptoms.

On physical examination his blood pressure was 150/80 mm Hg. The remainder of the general physical examination was normal including examination of the cardiovascular system. The neurovascular and neurological examinations were normal except for a right Horner’s syndrome.

Holler monitoring showed normal sinus rhythm throughout. The rate ranged from 70 to 105 beats per minute.

On cerebral angiography of the right common carotid artery, some atherosclerotic plaques were noted at the origin of the internal carotid artery. There was no significant stenosis. The left common carotid artery showed a large broad-based plaque in the common carotid artery on the medial wall. A small lateral plaque was noted at the origin of the left internal carotid artery. AP and lateral views of the head showed no abnormality in the left or right cerebral hemisphere. Subclavian injection with an AP and magnification lateral views of the posterior fossa showed no abnormalities.

During hospitalization the patient did not have further TIAs. He was placed on aspirin and Diuril 250 mg daily. The diagnosis was TIAs of unknown etiology.

Case 9 (NCBH #09 24 48)

This 67-year-old white man had a history of dizziness and occasional loss of consciousness beginning in 1970. In 1971 he was seen because of dizzy spells which occurred approximately every other day at various times lasting from 15 to 30 minutes. These were characterized by dizziness during which the patient would have to lie down and this was not influenced by posture, particularly sitting up. He also noted some loss of the left visual field and marked weakness of his legs. Sometimes he was so weak that when he had a severe attack he was unable to arise. Following this, he had a severe headache and then his symptoms gradually subsided. Between spells he had some slurring of speech which lasted for several hours. He also described diplopia when looking laterally. He denied facial numbness but had some numbness in all four extremities at night in bed. He had no specific weakness of the extremities other than during the attacks. He had never actually lost consciousness with the dizzy spells. During the past year the patient’s mental status declined in that his ability to carry on a conversation and remember pertinent details were affected. This diminished to the point that he had to give up his job as a minister. His speech was quite slow and at times he was said to have been inconsistent on silly things around the house.
On physical examination the blood pressure was 130/70 mm Hg. The pulse varied from 45 to 70 with a run of bigeminy of up to 85 per minute. There were no specific abnormalities on general physical examination. The cardiovascular examination was normal except for a mild left ventricular thrust with a Grade 2/6 high frequency systolic murmur at the apex. Neurovascular examination was normal. The neurological examination was normal except for the patient's speech, which was slow but fluent. He frequently did not answer questions rather than give the improper answer. He knew the Presidents but skipped every other one. He was unable to subtract seven from 100, but knew seven times seven. He did not know what is east or west of North Carolina. He thought a peach was a vegetable. He was oriented in all three spheres. He identified objects and figures and imitated properly.

Holter monitoring showed a sinus or junctional rhythm of about 40 to 48 per minute. Ventricular rate was increased by premature ventricular contraction to 75 per minute at intervals.

On cerebral angiography the right innominate injection showed a plaque at the origin of the internal carotid artery. The remainder of the internal carotid artery appeared normal in the neck and intraorally. The vertebral artery on the right was also normal. The intracerebral distribution of the anterior middle and posterior cerebral arteries appeared normal except there was a paucity of vessels in the normal course of the ascending frontoparietal groups of vessels. Injection of the left carotid artery revealed a similar finding on the left. The carotid artery on the left appeared to be normal and the anterior and posterior cerebral arteries were otherwise normal. Injection of the left subclavian artery showed the vertebral artery to be normal. The posterior circulation was also normal. The deep venous structures were midline and normal.

Because of the patient's symptoms and cardiac abnormalities, a permanent transvenous pacemaker was inserted. Following this the pacemaker functioned well but the patient was bothered by recurrent nausea and vomiting. This was attributed to various cardiac medications. The patient also continued to have occasional episodes of severe angina pain radiating to the left arm. After several days the patient's cardiac dysrhythmia stabilized and the pacemaker only fired rarely. Two months following discharge the patient remained in good condition taking Digoxin, 0.2 mg daily. The diagnosis was atherosclerotic heart disease with angina pectoris and "sick-sinus node" syndrome, and TIAs secondary to cardiac dysrhythmia.

Case 10 (NCBH #53 81 41)

This 66-year-old man had had two episodes of unconsciousness. The first occurred when he was leaning over a box and suddenly felt as if he were going to black out. Immediately after that he did lose consciousness and fell to the floor sustaining a minor injury to his head. The patient estimated that the duration of his unconsciousness was about one minute. He had no neurological sequelae from this episode. The second occurred approximately three days later when he was reaching to put a box on the shelf at his market. On this occasion he again fell sustaining a four-inch laceration on his right posterior scalp. The patient had had no transient weakness, numbness, or loss of vision. There was no previous history of syncope or convulsions or vertiginous episodes. There were no other neurological symptoms.

On physical examination the blood pressure was 134/58 mm Hg. His pulse was 60 with occasional premature ventricular beats. General physical examination was normal. On neurovascular examination there was a Grade 3-4/6 left systolic carotid bruit. The right femoral pulse was absent and the right carotid pulse diminished. His neurological examination was normal.

Holter monitoring showed frequent premature ventricular beats unifocal with bigeminy at times. Junctional beats occurred prematurely. The basic rhythm was 45 to 70 per minute with resting and sleeping rates of 45 to 50 per minute.

Aortic study revealed a high-grade type of stenosis of the origin of the left internal carotid artery with approximately 40% stenosis of the right internal carotid artery at its origin. There was moderate stenosis at the origin of the right vertebral artery.

Because of the stenosis of the left internal carotid artery, carotid endarterectomy was performed. Two days following this a thrombectomy and an endarterectomy of the left internal carotid artery were again repeated due to occlusion of the left internal carotid artery from a fresh thrombosis. When the patient was seen four months following surgery, he was doing well. He had had no new attacks since discharge from the hospital and was working up to five or six hours a day. He had nothing to suggest transient cerebrovascular insufficiency and was not aware of any cardiac irregularity. The diagnosis was TIAs due to sinus bradycardia and stenosis of the left internal carotid artery.

Results

Ten patients with cerebrovascular insufficiency were monitored for up to 24 hours with the Holter EKG monitor. As well as neurological and neurovascular examinations, each patient had cerebral angiography. Eight of the patients had TIAs or reversible ischemic neurological deficits. Two patients (Cases 2 and 3) had mild residual deficits from a completed stroke. Of the eight patients with transient or reversible cerebrovascular insufficiency, four had clinical symptoms in the distribution of the internal carotid artery; the remaining were in the basilar-vertebral system. Four of the ten patients had carotid or supraclavicular bruits. Hypertension was present in four patients.

Holter EKG monitoring gave the following results: there were no abnormalities in four cases (Cases 2, 5, 7, and 8). The following abnormalities were present in the remaining patients: Case 1 — frequent premature atrial contraction with aberrant conduction throughout the tracing; Case 3 8 infrequent premature ventricular contractions primarily unifocal but at times from different foci; Case 4 — a constant intraventricular conduction abnormality; Case 6 — the monitor strip suggested Wolff-Parkinson-White syndrome with a slow rate of 45 beats per minute at rest and increasing to 70 to 80 beats per minute while walking; Case 9 — questionable sinus or junctional rhythm of about 40 to 48 beats per minute; rate increased by premature ventricular contraction to 75 beats per minute; Case 10 — frequent
premature ventricular beats which were unifocal with bigeminy at times. Although there were no persistent cardiac dysrhythmias throughout the time of monitoring, there were frequent conduction defects of bradycardia in six of the ten patients. In addition, Case 2 had a changing pacemaker. Eight of the ten patients had associated abnormal cerebral angiography, the most frequent finding being that of a carotid stenosis. Only one patient (Case 9) required a cardiac pacemaker. Cases 1, 6 and 7 had carotid endarterectomies. Although there were a variety of cardiac, cerebrovascular, and neurological abnormalities in these ten patients, six of them had disturbance in cardiac rhythm or conduction which could have been directly associated with or suggest an etiology for the patient’s neurological event.

Discussion

It is postulated that in the series of patients with cerebrovascular disease by history and on cerebral angiography the cardiac abnormality played a role in these symptoms in at least 60% of the cases, namely, Cases 1, 3, 4, 6, 9, and 10. The remaining, except for two patients, had angiographical abnormalities but did not have abnormal monitoring.

Walter et al.5 studied 39 patients with symptoms of cerebral ischemia. Twenty-eight had symptoms of diffuse cerebrovascular insufficiency, namely, dizziness, giddiness, or syncope, while 11 had TIAEs. They carried out continuous ten-hour tape-recorded EKG monitoring of these patients. No patient had had a significant dysrhythmia that was considered to be responsible for their symptoms previously diagnosed by clinical observation or standard EKG. Walter et al.5 found that 10 of the 39 patients monitored had transient cardiac dysrhythmia or conduction abnormalities that probably precipitated their neurological symptoms. Eight of the positive results were in patients with diffuse cerebrovascular symptoms, namely, episodic lightheadedness, dizziness, or syncope. Two with cardiac abnormalities were in the group of 11 TIAEs patients. In our report all of our positive results were in patients with TIAs. They carried out continuous twenty-four-hour tape-recorded EKG monitoring of these patients. Seven of their positive cases had supraventricular tachycardia with a heart rate greater than 150 beats per minute. Three patients had bradycardia with a heart rate less than 40 beats per minute. Two of these patients had sinus bradycardia while the other had atrial fibrillation with a slow ventricular rate.

In another study, McAllen and Marshall7 examined 16 patients who required the insertion of a permanent cardiac pacemaker to control episodes of dysrythmia producing TIAE. Eight of these cases had been examined previously without the cause of the TIAs being detected. They point out that in six of their cases no EKG was initially performed. Of the 16 patients referred for the insertion of a permanent cardiac pacemaker, eight initially had the dysrhythmia as the cause of their TIAs. Another group of 13 patients seen for similar recurrent episodes were treated by drugs and again six of these underlying dysrythmias had not been previously recognized. McAllen and Marshall7 point out that such symptoms as vertigo, blurred vision, flashing lights, diplopia, syncope, or seizures may be unrecognized signs of TIA due to cardiac dysrhythmias. They further emphasize that commonly, as their present cases show, an EKG is not recorded, or, if recorded, the significance of the abnormality is not appreciated. They further emphasize that difficulty in diagnosis is increased in some patients when several months or years elapse between episodes of dysrhythmia producing symptoms. It is for this reason they suggest not only a baseline EKG but prolonged cardiac monitoring in patients with evanescent or transient neurological symptoms.

Reed et al.6 reported on the rarity of TIAs in patients who have episodes of cardiac dysrhythmia of the type associated with decreased cardiac output. They separated 290 patients requiring cardiac pacemakers into three groups: (1) those without neurological symptoms, (2) those with generalized nonfocal neurological symptoms, and (3) those with focal neurological symptoms. They found only four patients (1.4%) in the latter group who had focal TIAs, but in group 2 there were 235 (81%) with symptoms of syncope, near-faint or “gray out,” dizziness or lightheadedness, or generalized seizures. This group is similar to the group of 28 of 39 patients of Walter et al.5 who had syncope, dizziness, or giddiness. Although Reed et al.6 found only four of 290 patients, Walter et al.5 found 11 of 39 with “classic transient ischemic attacks.” This is also similar to the high incidence of 16 patients reported by McAllen and Marshall7 who needed cardiac pacemakers for “transient cerebral ischemic attacks.” Hence, one cannot agree with the low incidence (1.4%) of Reed et al.6 One must consider both focal and generalized neurological symptoms when considering the incidence of cardiac abnormalities in patients with transient cerebrovascular insufficiency or reversible ischemic neurological deficits.

In our study we have emphasized the importance of long-term EKG monitoring along with cerebral angiography in patients with transient cerebrovascular insufficiency. This allows us to determine not only the nature and site of the cerebrovascular lesion, but also the importance of any contributing cardiac factors. Our report does not approach the subject from a cardiac standpoint as did Reed et al.6 but from the neurological standpoint as did Walter et al.5 and McAllen and Marshall.7 These conflicting reports point out the need for further studies of prolonged cardiac monitoring in patients with cerebrovascular ischemia.

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