Brief Communication

Holter Monitoring in Patients with Transient Focal Cerebral Ischemia

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SUMMARY The value of continuous long-term ECG monitoring in patients with transient generalized neurological symptoms is well established, but its value in transient focal neurological deficits is less clear. A patient is reported who had significant dysrhythmias and transient focal cerebral symptoms which did not clear after cardiac pacing. Nineteen other patients were monitored as part of their evaluation for transient focal cerebral symptoms and were found to have essentially no cerebrally significant dysrhythmias. This report suggests cardiac dysrhythmias rarely cause such symptoms.

THE EFFECT of cardiac dysrhythmia on cerebral circulation has been well established. The importance of a careful search for hemodynamically significant dysrhythmias in patients presenting with symptoms of non-focal transient cerebral dysfunction, such as episodic dizziness and syncope, has been the subject of several recent reports, but detection of such dysrhythmias by routine resting ECG monitoring has been difficult. The development of the continuous ambulatory ECG system by Holter and his associates has provided an effective and relatively simple method for detection of these dysrhythmias.

Several authors have alluded to the role of a diminished cardiac output in various dysrhythmias as an etiologic agent in transient focal cerebral ischemia and the need for continuous ECG monitoring in these patients. Others have stressed the rarity of transient focal cerebral events in dysrhythmias associated with a significant reduction in cardiac output. A recent experience with a patient who had significant cardiac dysrhythmias and transient focal cerebral ischemic symptoms which did not abate after cardiac pacing prompted a review of Holter monitoring results in patients with such transient focal cerebral symptoms.

A 66-year-old man was admitted for evaluation of two episodes of dysesthesias in the right arm and hand associated with an expressive aphasia, lasting approximately 30 minutes. The most recent episode occurred on the morning of admission. Cardiac history was significant in that the patient had suffered an acute myocardial infarction at age 60. In addition, there was a history of frequent angina occurring approximately 1 to 2 times monthly. There was no history of syncope attacks or presyncope episodes.

General physical examination disclosed a coarse systolic murmur at the apex. Pulses were full, but bruits were audible over the left common carotid artery and both femoral arteries. The neurological examination was essentially unremarkable. A baseline 12-lead ECG disclosed sinus bradycardia and non-specific ST-T wave flattening. On 24 hour Holter monitoring, sinus bradycardia with sinus arrest was noted as well as runs of paroxysmal atrial tachycardia and multifocal premature ventricular beats with coupling. In view of these findings a permanent cardiac pacemaker was implanted.

The patient was discharged and did well for a 2 week period, but he then suffered 4 episodes of transient right-sided numbness and expressive aphasia within a 4-day period. His examination remained unchanged and the pacemaker was noted to be functioning adequately. Angiography disclosed a 90% stenosis of the proximal left internal artery as well as severe narrowing of the left vertebral artery. The patient subsequently underwent an uncomplicated left carotid endarterectomy.

Nineteen patients have been studied up to 24 hours by continuous ECG monitoring as part of their evaluation for transient focal cerebral dysfunction. Only patients who satisfied the criteria established by the Joint Commission for Stroke Resources and who were monitored within 14 days of their most recent transient neurological event were considered. Dysrhythmias were classified as to their significance by the recent classification of Jonas et al. Aside from the patient outlined above, none of the other patients had a dysrhythmia known to be significantly correlated with transient neurological symptoms as per the Jonas classification. One patient had a short duration dysrhythmia which, in itself, may not cause transient neurological symptoms, but when of greater duration, could do so. Six patients had dysrhythmias of variable significance, premature ectopic beats or premature ventricular contractions in succession. The remaining 12 patients had dysrhythmias not ordinarily related to cerebral symptoms or entirely normal recordings. These findings are seen in the table.

None of the patients had neurological symptoms recorded in his clinical diaries at the time of ECG documented disturbances of the cardiac rhythm.
The pathogenesis of transient focal cerebral ischemia is a topic which has generated much attention in the past few decades. Recently the role of platelet emboli originating from stenotic or ulcerated extracerebral cranial vasculature as having a major etiologic role in their pathogenesis has been stressed. The role of transient hypotension, "the hemodynamic crisis of Denny-Brown," in conjunction with localized cerebrovascular stenosis as an etiologic agent of these episodes has received scant investigation but significant speculation in recent years.

Denny-Brown, in a series of experiments in the late 1950's, demonstrated that in the monkey occlusion of the carotid or middle cerebral artery did not produce symptoms. Transient hemiplegia only developed with significant lowering of blood pressure. Others have commented on the selective transient or permanent vulnerability of the central nervous system in such metabolic disturbances as hyponatremia, hypoglycemia, and the non-ketotic hyperosmolar state. The implication is that a focal area of the central nervous system, already compromised by relative arterial insufficiency, may be selectively vulnerable to generalized ischemic anoxia or other metabolic disturbances.

Kendell and Marshall evaluated the effects of hexamethonium-induced hypotension on a series of 37 patients with complaints of transient focal cerebral ischemia. Only 1 patient developed a focal deficit in the appropriate vascular territory prior to the development of the signs of severe generalized cerebral ischemia. The role of hypotension in producing completed cerebral infarction was also challenged by a recent report of Torvik and Skutlerud. They found almost no correlation between the incidence of recent focal cerebral infarcts and severity of cerebral atherosclerosis in patients dying within a few days to weeks after successful resuscitation from an episode of cardiovascular collapse. These studies imply that generalized hypotensive episodes produce generalized and not focal clinical or pathologic deficits, be they transient or permanent.

The introduction of continuous long-term ECG monitoring has made it possible to correlate hemodynamically significant cardiac dysrhythmias with transient, generalized or focal cerebral ischemic episodes. Such a correlation seems well established in those patients with generalized cerebral symptoms. Correlation of these dysrhythmias with transient focal events is poor. The lack of response of the present patient's transient focal ischemic events to cardiac pacing and the finding of essentially no significant cardiac dysrhythmias in another 19 patients who were monitored supports the conclusion of Reed, Siekert et al. Based on the present data, patients who have focal transient cerebral ischemic attacks seldom appear to have significant cardiac dysrhythmias as an etiologic agent of their symptoms. Various non-cerebrally significant dysrhythmias may be found in this patient population because of age and underlying generalized atherosclerotic vascular disease with cardiac involvement.

Failure to document a frequent association between dysrhythmia and subjectively reportable symptoms of focal cerebral dysfunction during a finite period of ECG recording and clinical monitoring is not an entirely conclusive argument against a positive correlation. The great variability in the temporal occurrence of dysrhythmias, coupled with the usually widely spaced timing of the TIAs, makes correlative evaluation of the 2 phenomena difficult. Further work related to the use of patient-activated ECG monitors may well be useful in confirming the rarity of a cause and effect relationship between dysrhythmia and transient focal cerebral symptoms.

**Table: Summary of Dysrhythmias in Patients with Transient Focal Cerebral Symptoms**

<table>
<thead>
<tr>
<th>Category</th>
<th>No.</th>
<th>Percent</th>
</tr>
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<tbody>
<tr>
<td>a) Significant arrhythmias</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>b) Short duration significant arrhythmias</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>c) Normal or insignificant arrhythmias</td>
<td>18</td>
<td>90</td>
</tr>
<tr>
<td></td>
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<td>20</td>
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**Discussion**

The pathogenesis of transient focal cerebral ischemia is a topic which has generated much attention in the past few decades. Recently the role of platelet emboli originating from stenotic or ulcerated extracerebral cranial vasculature as having a major etiologic role in their pathogenesis has been stressed. The role of transient hypotension, "the hemodynamic crisis of Denny-Brown," in conjunction with localized cerebrovascular stenosis as an etiologic agent of these episodes has received scant investigation but significant speculation in recent years. Denny-Brown, in a series of experiments in the late 1950's, demonstrated that in the monkey occlusion of the carotid or middle cerebral artery did not produce symptoms. Transient hemiplegia only developed with significant lowering of blood pressure. Others have commented on the selective transient or permanent vulnerability of the central nervous system in such metabolic disturbances as hyponatremia, hypoglycemia, and the non-ketotic hyperosmolar state. The implication is that a focal area of the central nervous system, already compromised by relative arterial insufficiency, may be selectively vulnerable to generalized ischemic anoxia or other metabolic disturbances.

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**References**

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