Are Terms Such as Completed Stroke or RIND of Continued Usefulness?

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MANY TERMS HAVE BEEN DEVISED in an attempt to capture the state of the clinical deficit in patients with cerebrovascular disease. Transient ischemic attack (TIA), partial non-progressing stroke, reversible ischemic neurological deficit (RIND), stroke in evolution, and completed stroke are terms born decades ago. The era in which these terms were coined was quite different: arteriography was seldom performed because of the requirement for a surgical cutdown and the need for a series of hand-pulled single films; endarterectomy was an infrequent surgical procedure; CT was but a dream, and there was no surgically-created cranial vascular shunts. Furthermore, that era knew little if anything of some stroke entities clarified only later: lacunar infarctions, ulcerated plaques with their tendency to embolize, prolapsed mitral valves, spontaneous extracranial vascular dissections. There were relatively few neurologists — scarcely enough to fill a large meeting hall — beginning to examine a small fraction of the total number of patients with stroke.

The terms were well chosen for that period. They were simple and required no neurological sophistication to apply. It was hoped that one remedy or another could be effectively applied to some of these subdivisions and be curative or at least dramatically helpful. Hope springs eternal in the human breast; the search for a panacea is understandable and forgivable. The decades since have hopefully taught us that there is no single panacea for any of these subgroups. Many clinical trials of one therapy or another have been undertaken. The results of these trials, many imperfectly conceived in retrospect, have been examined and re-examined. Statisticians have "massaged the numbers" and argued endlessly in terms relatively foreign to most clinicians about the significance or lack thereof of the results. No study has provided figures which immediately overwhelm the reader with convincing proof that a cure is available. Yet the studies go on, and the patient with no deficit. Why then do we continue to review the treatment of completed stroke? After all, these terms tell us how badly off the patient is: They say nothing about the mechanism of the stroke. None of these categories approaches homogeneity: they contain fruits as divergent as grapes and watermelons. For example, TIA may be caused by the arteriopathy underlying lacunes — lypohyalinosis, platelet emboli from an ulcerated plaque, cholesterol emboli, internal carotid artery occlusion with low flow, embolization of clot from an occluded internal carotid artery, internal carotid artery stenosis with low flow, cardiac embolization, vascular spasm as is seen in migraine, or cerebral aneurysm. This list names only some of the mechanisms and is not inclusive. Is it reasonable that a single therapy would apply to this heterogenous lot? Of course the degree of clinical deficit does matter. It tells us of the risk-benefit ratio of treatment. The patient with a severe deficit ("completed stroke") has less likelihood of gaining and the same complication rate as the patient with no deficit. Why then do we continue to use these terms as if they were the essence of the problem? Why do we continue to embark on clinical trials using these terms as the sole criteria for a treatment? Why do we continue to review studies using these terms as criteria despite the fact that they have born little fruit in the past? I will attempt to argue systematically why I feel it is time to discard or at least significantly de-emphasize these terms and move on to more fruitful pastures.

The Terms Though Simple Are Often Variously Defined, And Variously Applied

As an example, let us examine usage of "completed stroke." The Classification and Outline of Cerebrovascular Diseases II reads as follows:

"completed stroke (prolonged neurological deficit) . . . This category refers to a relatively stable neurological deficit, that is, during the period of observation for categorization, little or no change in deficit is occurring. When the duration is more than three weeks, often permanent, it is commonly known as completed stroke."

Meyer et al say "completed stroke can be defined as a neurological deficit which has persisted for considerable time (in excess of months)." The Report of the Joint Committee for Stroke Resources reads:

"term, completed stroke is usually employed to signify a focal neurological disability that came on abruptly and has become stabilized . . . some authors indicate that 18 to 24 hours without progression of the lesion in the carotid system, and up to 72 hours if

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The territory of involvement is the vertebral-basilar system, would suffice to place the patient in the stabilized category. It is also noteworthy that only a gradation in severity differentiates a partial non-progressing stroke from a completed stroke."

The timing is confusing and none define how one judges severity. Some use the term “completed stroke” to mean that all the deficit that is to occur has already accrued. Others have used the term to imply that all the deficit possible in a given vascular territory is already present. Since none of us have an infallible crystal ball, the judgment that additional deficit won’t be added is often conjectural and erroneous. This is especially true if one considers new deficits weeks or months later. To determine whether the entire deficit possible in a vascular territory has already developed, one must know which vascular territory (which vessel) is involved. A minor sensory deficit in the left arm or leg could be due to infarction of the entire territory of an occluded right thalamogeniculate artery. On the other hand, if the involved vessel were the right internal carotid artery and the lesion a small right post-central gyrus infarct, obviously, there would still be considerable brain tissue at risk for further damage. The determination of “completed stroke” then becomes complex and requires clinical, CT, and arteriographic data — information not customarily available in prior studies nor generally considered in making the determination of whether the stroke is “completed.” Similarly “RIND,” “partial non-progressing stroke,” and “stroke in evolution” are not easy to define, and are often obvious only with hindsight — long after a treatment decision must be made.

Clinical Criteria Do Not Always Predict Pathology

The advent of CT scanning has led clinicians to realize that some patients with a TIA have unexpected cerebral infarction. Beal, Williams, et al. recently reported a patient with approximately 35 episodes usually consisting of paresthesiae or weakness of either arm or face. At post-mortem, there were myriads of small infarcts due to cholesterol emboli which were the pathological counterparts of these clinical episodes. Are these designations in fact a roadblock delaying further progress in our understanding of stroke treatment?

If these terms provide us no clear pathologic, prognostic, or therapeutic guidelines, why do we continue to use them? Clearly more important than tradition is an elucidation of the mechanism of the stroke or TIA. Because of the literature bias for considering therapy only in relation to TIA, RIND, completed stroke, etc., house officers and students almost invariably stop their analysis of the case with assessment of these subdivisions. Are these designations in fact a roadblock delaying further progress in our understanding of stroke treatment?

Perhaps we should emulate the classification system of the American Heart Association which describes heart disease in terms of anatomy, physiology, and functional effects of the lesions. Should we state the cause and mechanism of the ischemic lesion, its anatomy and the functional severity of the deficit. A statement as to whether the patient is clinically neurologi-
The development of the concept of transient ischemic attack (TIA) was an extremely important advance in the understanding and management of cerebrovascular disease, which has been refined over the years to the generally accepted clinical definition of a focal neurological dysfunction which resolves completely within 24 hours. While there is almost certainly a spectrum of pathophysiology underlying TIA, it is clear that in many cases TIs are caused by emboli or microemboli from atherosclerotic plaques and are the harbinger of cerebral infarction. The entity, however, is a clinical concept rather than an anatomical one and includes at least several pathological entities, often with different diagnostic, therapeutic and prognostic implications. In particular, the term TIA is currently used to describe a variety of symptomatically transient neurological disturbances of focal nature due to local ischemia caused by a variety of pathophysiological mechanisms such as hypotension, "hemodynamic crisis," microembolism, or abnormality of blood constituents. They range from potentially reversible ischemia, on the one hand, to infarction on the other.

It is the purpose of this article to demonstrate that, in some cases, the clinician cannot accurately differentiate between a TIA without infarction and an infarction with minor residua, and to suggest the hypothesis that this distinction may have clinical significance. In this article, we call attention to episodes with the temporal profile of TIA, and a demonstrable pathological substrate, transient neurologic dysfunction in the setting of cerebral infarction.

TIs are defined as episodes of focal neurological deficit of sudden onset, referable to a specific arterial territory, lasting no longer than 24 hours. As early as 1914, Hunt referred to "cerebral intermittent claudication" due to disease of the carotid artery. Nearly forty years later Denny-Brown described hemodynamic crises or "episodic insufficiency in the circle of Willis of a temporary nature." Millikan, Siekert, and their colleagues, among others have more recently elaborated and refined the concept. They have agreed that only transient neurological dysfunction, resolving within 24 hours, can be considered a "transient ischemic attack."
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