

Why Stroke Cannot Be Treated as One Disease

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Abstract

“Stroke” describes a clinical syndrome, not a single etiology. Ischemic arterial occlusion, intracranial hemorrhage, cerebral venous thrombosis and stroke mimics require different reasoning and sometimes mutually incompatible treatments. This paper argues that any root-cause theory of stroke must be partitioned by mechanism before it can be evaluated.

The Clinical Question and Its Context

For ischemic disease the relevant etiologies include large-artery atherosclerosis, cardioembolism, small-vessel occlusion, arterial dissection, prothrombotic disorders and other determined or undetermined causes. Even within one broad category, a proximal embolus and local in-situ thrombosis imply different preventive strategies. The central distinction is between predisposition, vessel event and brain response: hypertension can increase small-vessel risk; atrial fibrillation can generate emboli; infarct size and collateral flow shape outcome.

Proposed Biological or Explanatory Model

A terrain-based explanation can contribute if it identifies a measurable upstream exposure that maps onto a recognized phenotype. For example, cholesterol-handling abnormalities are more directly relevant to atherosclerotic mechanisms than to traumatic dissection. An infection-associated cardiomyopathy may lead to cardioembolism, whereas a retinal vascular event may signal systemic risk without revealing the precise causal pathway. Mechanism-specific thinking prevents one attractive explanation from becoming universal.

Mechanism-Specific Interpretation

Classification is not just taxonomy; it predicts different interventions. Large-artery disease may motivate evaluation of local stenosis and plaque, atrial fibrillation may require anticoagulation, and hemorrhagic stroke makes some antithrombotic interventions immediately hazardous. Small-vessel pathology is strongly shaped by longstanding vascular risk, while dissection involves a lesion in the arterial wall. There are also mixed-mechanism patients: an older person can have carotid plaque and intermittent atrial fibrillation together. The clinician must determine which abnormality explains the current event and whether both require prevention. An inclusive systems theory should make this differentiation easier, not erase it.

Competing Explanations and Limits of Inference

Stroke-like deficits can result from seizures, hypoglycemia, migraine and functional neurological symptoms. Conversely, a normal initial CT may fail to visualize an early ischemic infarct; the scan's clinical role includes excluding hemorrhage and assessing reperfusion candidacy. A convincing alternate diagnosis must explain localization and time course, not simply coexist with a negative early scan.

Discriminating Observations and Evidence Thresholds

A good stress test for any proposed unified mechanism is whether it explains a cerebral venous thrombosis, deep small-vessel lacune and embolic cortical infarct equally well. If the explanation changes definitions to encompass all three, it has probably ceased to discriminate. The value of a model depends on excluding some alternatives in addition to explaining those it favors.

Implications for Interpretation and Future Inquiry

Every future ABSURD stroke paper should state the lesion's vascular territory, likely occlusion mechanism, the relevant evidence and the uncertainty. The most humane consequence of better theoretical classification is faster recognition of situations where immediate stroke evaluation remains necessary. Exploratory explanations and emergency treatment must never compete for time.

Selected Evidence and Further Reading

1. [Navigating the clinical landscape of artery of Percheron infarction: A systematic review](#)
2. [Sitosterolemia: a review and update of pathophysiology, clinical spectrum, diagnosis, and management](#)

Source lineage: Acute stroke differential and vascular investigations

Editorial note: Conceptual scientific commentary; not an original clinical trial, systematic review, peer-reviewed publication, or medical advice. Hypotheses are not established treatment effects.