The brain is critically dependent on sufficient blood flow. Strickland's laboratory investigates how dysfunction of the circulatory system contributes to neurological conditions such as Alzheimer’s disease in humans and in mice.

Neurological disorders of the central nervous system represent profound medical problems worldwide. For example, Alzheimer's disease affects millions of people and has severe physical, psychological, and financial consequences. By studying patients and mouse models with neurological diseases, Strickland is working to elucidate the molecular mechanisms by which neural function is disrupted.

In investigating neurovascular dysfunction, the Strickland lab studies the mechanisms underlying the pathogenesis of Alzheimer's disease. Cerebrovascular defects contribute to the progression of Alzheimer's pathology, and members of the lab are using transgenic mouse models of Alzheimer's to evaluate blood-brain barrier damage and the roles that blood clot formation and degradation play in this disease. Their research has determined that the β-amyloid peptide, which is considered to be a causative factor in Alzheimer's, interacts with fibrinogen to promote irregular fibrin accumulation in the brain and increase brain inflammation. This peptide also hinders blood clot degradation, which could compromise blood flow, exacerbate inflammation, and lead to neuronal death. These findings suggest that fibrin and the mechanisms involved in its accumulation and clearance may present novel therapeutic targets for slowing the progression of Alzheimer's disease.

The Strickland lab has also recently found that β-amyloid can activate coagulation Factor XII (FXII) in the plasma of both Alzheimer's patients and mouse models. The activation of FXII initiates fibrin clotting as well as inflammatory processes, both of which are recognized pathologies in Alzheimer's disease. Promotion of FXII activation by β-amyloid could help explain the association between Alzheimer's disease and vascular diseases. This knowledge may ultimately identify new pathogenic mechanisms that could disrupt neuronal function, aiding in the discovery of novel diagnostic and therapeutic approaches.