# EXTRACRANIAL INTRACRANIAL ANASTOMOSES FOR CEREBRAL BLOOD FLOW AUGMENTATION

**Essay**

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# ABSTRACT

Cerebrovascular occlusive disease is one of the important causes of cerebral ischemic injury. We have attempted through our essay to define the physiologic and pathophysiologic mechanisms involved in ischemia due to occlusive cerebrovascular disease.

We have also attempted to define the clinical manifestations and investigative techniques involved in investigating the patients and selecting the best surgical candidates.

We have also defined various surgical procedures involved in augmentation of cerebral blood flow in such patients as well as the surgical technique, pre and postoperative managements and possible complications.

**Key Words:**

Cerebral Blood Flow, Hemodynamic Ischemia, Pathology of Ischemic, Cerebrovascular Disease, Clinical Syndromes of Brain Ischemia, Moyamoya Disease, Imaging of Ischemic Cerebrovascular Disease

***This work is dedicated***

***To my Father, the soul of my Mother***

***and to my Family***

***Special dedication to N***

# LIST OF ABBREVIATIONS

| | |
|---|---|
| ACA | : Anterior cerebral artery |
| AchA | : Anterior choroidal artery |
| CBF | : Cerebral blood flow |
| CBV | : Cerebral blood volume |
| CNS | : Central nervous system |
| CPP | : Cerebral perfusion pressure |
| CSF | : Cerebrospinal fluid |
| CT | : Computerized tomography |
| CTA | : Computerized tomography angiography |
| CVR | : Cerebrovascular resistance |
| CW Doppler | : Continuous wave Doppler |
| 3D | : 3 Dimensional |
| DSA | : Digital subtraction angiography |
| ICA | : Internal carotid artery |
| MCA | : Middle cerebral artery |
| MRA | : Magnetic resonance angiography |
| MRI | : Magnetic resonance imaging |
| OA | : Occipital artery |
| OEF | : Oxygen extraction fraction |
| PC | : Phase contrast |
| PCA | : Posterior cerebral artery |
| PET | : Positron emission tomography |
| PICA | : Posterior inferior cerebellar artery |
| RAG | : Radial artery graft |
| rCBF | : Regional cerebral blood flow |
| RIND | : Reversible ischemic neurological deficit |
| SCA | : Superior cerebellar artery |
| SPECT | : Single photon emission from computerized tomography |
| SSS | : Superior sagittal sinus |
| STA | : Superficial temporal artery |
| Tc | : Technetium |
| TCD | : Transcranial Doppler |
| TIA | : Transient ischemic attack |
| TOF | : Time of flight |
| Xe | : Xenon |

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# INTRODUCTION AND AIM OF THE WORK

The convergence of microneurosurgical techniques and more refined understanding of the pathophysiology of cerebral ischemic disease set the stage for the EC-IC bypass. Fisher had quite astutely stated in 1951, "it is even conceivable that some day vascular surgery will find a way to bypass the occluded portion of the artery" **(Amin-Hanjani et al., 2005)**.

Cerebral revascularization has developed from the culmination of several technologies, from the early beginnings of vascular surgical techniques in animals, to the development of the microscope, the bipolar coagulation, and suitable suture material. Major questions concerning the indications and benefits of cerebral revascularization are being addressed and interest in further advancing this field to make it safer for patients remains strong. Important research into perioperative blood flow assessment will play an important role in determining the success of cerebral revascularization. It is hoped that with better understanding of the pathophysiology of cerebral ischemia and better patient selection, cerebral revascularization will remain an important tool in contemporary neurosurgery **(Chmayssani et al., 2007)**.

The **aim** of this work to describe the procedural steps in the selection of possible candidates for revascularization surgery and we will describe details the various diagnostic technique used in the perisurgical evaluation of such candidates specially importance will be given to the description of the different surgical techniques available for the treatment of various bypass surgeries outlining the most modern and up to date research relevant to this topic outcome of bypass surgery based on the most recent studies in such field.

# CEREBRAL BLOOD FLOW

Our understanding of the relationship between cerebral circulation and cerebral function has evolved through refinements in the delineation of cerebral anatomy and physiology.

Ischemic injury may be a final common pathway in many types of cerebral insults. During cerebrovascular procedures, ischemic injury may result from an unanticipated complication of planned permanent or temporary vessel occlusion. An understanding of the physiologic controls of normal cerebral blood flow and the pathophysiology of ischemic injury is necessary for planning effective strategies to minimize the consequences of cerebral ischemia **(Rosenblum et al., 1990).**

**Normal Cerebrovascular Control:**

The brain is unique in that it is supplied by four major arteries that join in an equalizing manifold, the circle of Willis. The carotid arteries each supply approximately 40 percent of the total perfusion requirements of the brain.

The traditional view of the cerebral circulation saw the arterial supply as being functionally and morphologically separated into two distinct categories: the extracerebral vessels, including the major

arteries at the base of the brain and the pial vessels, and the intracerebral vessels, or the penetrating arteries.

Four major, interdependent mechanisms are involved in the control of cerebral blood flow: metabolic coupling: neural control, involving both extrinsic and intrinsic neural pathways; $Pco_3$; and autoregulation. Although this division may be somewhat artificial and these control mechanisms probably operate in concert, it is useful to consider each separately **(Bhatti et al., 1992).**

**Metabolic control:**

Local cerebral blood flow (CBF) is regionally heterogenous. The varied pattern of CBF is neither random nor related to the anatomic organization of the cerebral vasculature or to known differences in the innervation patterns of the cerebral vessels. Neuronal activity is the principal energy-consuming process in the brain. Local cerebral blood flow adjusts to the level of energy generation; therefore, it is the activity in the neuronal circuits that is the major determinant of variations and regional patterns of cerebral blood flow.

Normally there is exquisite coupling between the regional cerebral metabolic demand for oxygen and glucose generated by local neuronal activity and the volume of blood flowing through that tissue.

Alterations in the concentrations of local metabolites may lead to changes in regional CBF. Several chemical species capable of altering local vascular tone generated during periods of enhanced neuronal or glial activity have been considered as mediators of the coupling between How and metabolism.

These include extracellular pH, $Pco_2$, adenosine, glycolytic intermediates, and extracellular potassium (indirectly, through its role in neuronal and smooth muscle cell membrane function, even though it is not directly involved in energy metabolism) **(Brayden et al., 1986; Selman et al., 1994)**.

**Neurogenic control:**

It is important to consider not only the extrinsic nerve supply from the cranial ganglia to the cerebral arteries, and veins, but also the role of intracerebral neurons serving the intracerebral vasculature. A dense plexus of nerve fibers in the walls of cerebral vessels, forming a "minibrain" or regulator) center, has been documented. Given this arrangement, neurons could form the coupling mechanism between metabolism and flow.

Most of the neuron fibers investing the cerebral vasculature are sympathetic. They appear to function by reducing CBF under conditions where it has been increased by metabolic demand, and they

may raise the threshold for the breakthrough of autoregulation that occurs with arterial hypertension.

With the advent of both autoradiographic methods for the determination of regional CBF and positron emission tomography (PET) studies of cerebral circulation and metabolism in humans, as well as an explosion of techniques and knowledge in biochemical neuroanatomy, a growing body of evidence supports the concept that the brain can regulate its own blood flow through intrinsic neural networks **(Edvinsson et al., 1993).**

**Carbon Dioxide ($CO_2$):**

It has been well established that alterations in $Paco_2$ result in marked vasodilation. There is an exponential relationship between $Paco_2$ and CBF within a $Paco_2$ range of 25 to 60 mmHg, with a CBF change of approximately 4 percent per millimeter of mercury.

**Autoregulation:**

Autoregulation is defined as the physiologic maintenance of a constant flow over a moderate range of perfusion pressures.

CBF can be described by the relationship between cerebral perfusion pressure (CPP) and cerebrovascular resistance (CVR):

Although myogenic, neurogenic, and metabolic mechanisms have been postulated, the precise control of the autoregulatory response remains unknown.

A growing body of evidence suggests that endothelium-dependent mechanisms function as the primary mediating factor of vascular tone, and they are now considered as a facet of the myogenic hypothesis of autoregulation. The endothelium acts as a transducer of hemodynamic forces that lead to the release of vasoactive substances.

In the brain, autoregulation is manifest as the lack of major fluctuation in CBF despite changes in mean arterial blood pressure between 60 and 150 mmHg.

A knowledge of the cerebrovascular status of the patient with respect to hypertension may also be a consideration in the planning of temporary vessel occlusion during cerebrovascular surgery **(Iadecola et al., 1992).**

**Cerebral Blood Flow and Ischemic Thresholds:**

Although the brain represents only 2 percent of the total body weight, it receives 18 percent of the cardiac output and uses 20 percent of the oxygen supply. Its high metabolic demand and lack of appreciable energy reserves render the central nervous system uniquely susceptible to alterations of blood supply. The use of PET