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The Role of Neuropsychological Profiles in Adult ADHD and Ischemic Stroke Implementation of a Protocol for Acute Management of Hyperglycemia in Adult Patients Following an Ischemic Stroke Cardiovascular Disability Plasticity Mechanisms Underlying Motor Recovery After Developmental and Adult Ischemic Stroke in Rats Transthoracic Echocardiography in Adult Patients with Ischemic Stroke Ischemic stroke in adults with congenital heart disease - a population-based cohort study Ischemic Stroke in Adults with Congenital Heart Disease Ischemic Stroke as a Health Concern in Young Adults Differential Adult and Neonatal Response to Cerebral Ischemia-hypoxia Acute Post-Ischemic Seizures are Associated with Increased Mortality and Brain Damage Following Hypoxia-Ischemia in Adult Mice Psychological Determinants of Stroke Outcome in Mice Neurohospitalist Medicine An Adult and a Neonatal Rat Model of Cerebral Ischemia Long-term Outcome After Arterial Ischemic Stroke in Children and Young Adults Ischemic Stroke Universe of Florida Patients with Ischemic Brain Attack The Lived Experience of Young Adults, 20-40 Years Old, 6-12 Months Post Ischemic Stroke Idiopathic Ischemic Necrosis of the Femoral Head in Adults Advances in the Treatment of Ischemic Stroke Early Identification of Dysphagia Post Pediatric Ischemic Stroke Novel Strategies in Ischemic Heart Disease ABCC-JNIH Adult Health Study Hiroshima, 1958-59 Outcomes in Young Adults with Ischemic Stroke in Ontario Cortical Neurogensis in Adult Brains After Focal Cerebral Ischemia Brain Hypoxia-Ischemia Research Progress Acute Stroke Nursing Acute Ischemic Stroke Cerebral Small Vessel Disease Cerebral Ischemia in Young Adults Inflammation Combined with Ischemia Produces Myelin Injury and Plaque-like Aggregates of Myelin, Amyloid-[beta] and A[beta]PP in Adult Rat Brain The Effects of Remote Limb Ischemic Conditioning on Functional Motor Outcome of the Paretic Limb After Ischemic Stroke Effects of TRPM2 Inhibition in Neuroprotection Following Neonatal Hypoxic-Ischemic Brain Injury Fetal Hippocampal Transplants Into the Ischemic Hippocampi of the Adult Mongolian Gerbil as a Means of Ameliorating Functional Deficits

Due to Global Cerebral Ischemia Effects of Early Neonatal Infection N Adult Cerebrovascular Health Regeneration in the Adult Brain After Focal Cerebral Ischemia SHABU ABUSE AND ISCHEMIC STROKE IN AN ASIAN ADULT Regenerative Therapies in Ischemic Stroke Recovery Insights Into Ischemia-induced Dendritic and Vascular Plasticity Through In Vivo Imaging Ischemic Stroke Vitamin D and the Neuroimmune Network in Ischemic Stroke

The first edition of this book will provide a comprehensive overview of ischemic heart disease, including epidemiology, risk factors, pathogenesis, clinical presentation, diagnostic tests, differential diagnosis, treatment, complications and prognosis. Also discussed are current treatment options, protocols and diagnostic procedures, as well as the latest advances in the field. The book will serve as a cutting-edge point of reference for the basic or clinical researcher, and any clinician involved in the diagnosis and management of ischemic heart disease. This book is essentially designed to fill the vital gap existing between these practices, to provide a textbook that is substantial and readable, compact and reasonably comprehensive, and to provide an excellent blend of "basics to bedside and beyond" in the field of ischemic heart disease. The book also covers the future novel treatment strategies, focusing on the basic scientific and clinical aspects of the diagnosis and management of ischemic heart disease. Stroke is the fifth leading cause of death in the United States and is a leading cause of adult disability and discharge from hospitals to chronic care facilities. Despite the frequency and morbidity of stroke, there is a relative paucity of “ stroke experts, ” such as vascular neurologists and neurocritical care physicians, to care for these patients. Clinical research in the diagnosis and treatment of stroke has grown exponentially over the past two decades resulting in a great deal of new clinical information for attending physicians to absorb. Grounded in cutting-edge and evidence-based strategies, Ischemic Stroke closes the gap in stroke care by providing a cogent and intuitive guide for all physicians caring for stroke patients. Key topics explored cover all elements of stroke care, including examinations of: emergent evaluation of the suspected stroke patient, clinical signs and symptoms of stroke, mechanisms of ischemic stroke, neuroimaging, cardiac-based evaluation, thrombolytic therapy, endovascular therapy, critical care management,

rehabilitation, cardiac arrhythmias, and structural heart disease. The initial goal of our studies was to develop a model of ischemia generating reproducible white matter injury within the adult rodent brain, with the aim of evaluating evidence of a causal relationship between white matter hyperintensity and ischemic comorbidities implied in human clinical scenarios. The bacterial endotoxin LPS, ischemia produced via either middle cerebral artery occlusion or common carotid artery ligation, andhypoxia (LPS/I/H) were each required conditions for the generation of white matter injury. The first evidence of injury was blood brain barrier damage at 24-48 hours with histologically detected accumulation of myelin basic protein around vessels and concomitant increases in degraded myelin basi protein detected by Western blotting. This was followed by the observation of foci of myelin sheath injury and associated axonopathy throughout the cortex and hippocampus which continued to evolve over several months, accompanied by a prolonged inflammatory state in the brain characterized by elevations in IL-1 and granzyme B. At 8-12 weeks, myelin basic protein and myelin associated glycoprotein-positive aggregates appeared in the brain, and unexpectedly co-localized with deposits of APP and AB 1-40/1-42 resembling the amyloid plaques found in Alzheimer's disease. In the first study, our LPS/I/H model and the resulting white matter injury and Alzheimer's-like pathology are detailed. The second study provides an in-depth examination of the blood-brain barrier injury following LPS/I/H and how myelin proteins distribute within the neurovascular unit. Cortical Neurogensis in Adult Brains After Focal Cerebral Ischemia. This book illustrates the importance and significance of regenerative medicine in stroke recovery. It discusses stem-cell-based treatment strategies and offers mechanistic insights into their role in neurological recovery. It also examines the challenges and advances in using adult stem cells for enhanced therapeutic efficacy. Further, it presents the strategies as well as the strengths and weaknesses of various delivery methods to administer stem cells in ischemic stroke. It examines the role of non-coding RNA in our understanding the stroke pathogenesis, their regulatory role in ischemic stroke and potential as biomarkers and therapeutic targets. Lastly, it explores exosomes in the treatment of stroke, and the underlying mechanism of their action as therapeutic vectors for stroke. Given its scope, it is an excellent resource for neurologists, neuroscientists and researchers involved in regenerative

therapy for stroke. Background: The overall stroke incidence among young adults is increasing. Our objective was to provide contemporary report on stroke severity, risk factors, complications and outcomes of young adults with ischemic stroke (IS) in Ontario. Methods: We collect information on demographics, risk factors, stroke severity, care/management and on clinical outcomes from the Ontario Stroke Registry (OSR) on adults (18-50yrs old) presenting with an acute stroke at participating facilities (2003-2013). Using t-tests for continuous and Chi-square tests for categorical variables we performed comparisons analysis across gender and two age categories (18-39vs.>40-50). The time-to-event outcomes (TIA, stroke, MI, composite vascular outcomes and death at 30-days and 1-year) were analyzed using Cox proportional hazards.Results: We identified 2,247 patients with IS. Stroke severity was equal in all groups (63%minor and 13.1%severe). Young man had significantly (p In recent years research on ischemic stroke has developed powerful therapeutic tools. The novel frontiers of stem cells therapy and of hypothermia have been explored, and novel brain repair mechanisms have been discovered. Limits to intravenous thrombolysis have been advanced and powerful endovascular tools have been put at the clinicians' disposal. Surgical decompression in malignant stroke has significantly improved the prognosis of this often fatal condition. This book includes contributions from scientists active in this innovative research. Stroke physicians, students, nurses and technicians will hopefully use it as a tool of continuing medical education to update their knowledge in this rapidly changing field. Stroke is the most common cause of severe long-term adult disability in the US. Stroke survivors are often left with severe motor impairments of the arm and hand on one side, termed hemiparesis. Motor rehabilitative training (RT) can induce structural plasticity to improve functional outcome, but RT alone is usually insufficient to restore normal function. Thus, there is a critical need for treatments that can enhance the effect of rehabilitation training. Remote limb ischemic conditioning (RLIC), which consists of short repeated bouts of ischemia to a limb using a blood pressure cuff, is a non-invasive intervention known to be neuroprotective in the setting of stroke. Recently, however, RLIC was also shown in several small studies to enhance motor learning in healthy humans and thus may have the potential to enhance the adaptive plasticity induced by RT and improve

functional outcome. However, this effect isn't always replicable and may be dependent on age and comorbidities. The central hypothesis of these dissertation studies is that RLIC done in conjunction with RT, can improve the functional motor outcome of the paretic limb after stroke compared to RT alone. All studies utilized the Endothelin-1 (ET-1) cortical ischemic lesion model in rats. RT was modeled using the single pellet retrieval task. RLIC was modeled by inflating a blood-pressure cuff on the hindlimb of the rat to intermittently restrict blood flow. The first study investigated RLIC+RT during the subacute phase of stroke in middle-aged rats. No beneficial effect of RLIC was found. The second study investigated RLIC+RT during the chronic phase of stroke in middle-aged rats, and no beneficial effect of RLIC was found. The third study investigated RLIC+RT during the subacute phase of stroke in adult rats and similarly found no beneficial effect of RLIC. Together these results indicate that RLIC is unlikely to enhance the effects of RT after stroke during the subacute or chronic phases. This lack of effect seen in our studies is a significant finding given the interest of the field in RLIC after the initial positive results in healthy, intact adults Abstract: Activation of the HPA-axis is among the first responses to cerebral ischemia, and glucocorticoid exposure prior to and following stroke is a critical determinant of ischemic outcome. Perinatal environment may be an important determinant of HPA-axis efficiency, influencing the onset of psychopathology and age-related disease. The present work examined the effects of brief (BMS) and extended (EMS) maternal separation, and prenatal stress (PNS), on stroke recovery in adult mice. Despite improved HPA-axis responsivity to acute stressors, BMS male mice had significantly more ischemia-induced neuronal death and increased functional deficits following MCAO in adulthood. The increase in corticosterone-induced neuronal death, and increased inflammation, suggest that BMS mice are more sensitized to the detrimental effects of elevated corticosterone during ischemia. Female BMS mice exhibited similar exacerbation of stroke outcomes to the males when exposed to the MCAO model of focal ischemia, but not the CA/CPR model of global ischemia. EMS and PNS had minimal consequences on focal ischemic outcome in male and female mice. Though additional experiments are needed to determine mechanisms, these studies clearly demonstrate that neonatal environment can drastically affect adult sensitivity to ischemic injury by altering the HPA-axis and inflammatory

responses. Psychobiological determinants of stroke outcome are not limited, however, to manipulations of the early environment. Affiliative social interaction via pair-housing was determined to decrease infarct size, functional deficits, and inflammation in comparison to socially isolated mice. In addition, pair-housing reduced the negative consequences of BMS, with pair-housed BMS males exhibiting decreased infarct and functional deficits compared to socially isolated BMS males. In contrast to affiliative social behaviors, post-stroke depression impairs functional recovery and increases mortality following stroke. The final experiments investigated glucocorticoid- and interleukin-1-activity in post-stroke anhedonia. The data suggested that anhedonia is a valid index of post-stroke depressive-like behavior in mice, and can be effectively attenuated with IL-1ra. Together, the experiments suggest that psychobiological processes that alter glucocorticoid exposure significantly influence neuronal survival and functional outcome following ischemia in adult mice. Indeed, perinatal determinants of HPA axis development, social modulation of stress responsivity, and post-stroke depression are important mediators of peri-ischemic glucocorticoid- and inflammatory cytokine exposure, significantly influencing physiological and behavioral outcomes. Post-stroke seizures are associated with worsened outcome following stroke, but the underlying pathophysiology is poorly understood. Here I combined behavioral, electrophysiological and histological assessments to examine acute seizures in adult mice following hypoxia-ischemia (HI). C57BL/6 mice aged 4-9 months received a permanent occlusion of the right common carotid artery and were then exposed to systemic hypoxia (8% O2, ~30 minutes). The HI episode resulted in decreases in cerebral blood flow, suppression of EEG activities and extensive brain injury in the hemisphere ipsilateral to the carotid artery occlusion. Generalized motor seizures were observed within 72 hours following HI. These seizures occurred nearly exclusively in animals with the extensive ipsilateral brain injury, but their generation was not associated with EEG discharges in bilateral hippocampal and cortical areas. Animals exhibiting these seizures had a high rate of mortality. Post-HI treatments with diazepam and phenytoin suppressed these motor seizures and improved post-HI animal survival. Based on these data, I conclude that these seizures are a consequence of HI brain injury, contribute to mortality following HI, and

that they may originate from deep subcortical structures. This updated second edition of Acute Ischemic Stroke: Imaging and Intervention provides a comprehensive account of the state of the art in the diagnosis and treatment of acute ischemic stroke. The basic format of the first edition has been retained, with sections on fundamentals such as pathophysiology and causes, imaging techniques and interventions. However, each chapter has been revised to reflect the important recent progress in advanced neuroimaging and the use of interventional tools. In addition, a new chapter is included on the classification instruments for ischemic stroke and their use in predicting outcomes and therapeutic triage. All of the authors are internationally recognized experts and members of the interdisciplinary stroke team at the Massachusetts General Hospital and Harvard Medical School. The text is supported by numerous informative illustrations, and ease of reference is ensured through the inclusion of suitable tables. This book will serve as a unique source of up-to-date information for neurologists, emergency physicians, radiologists and other health care providers who care for the patient with acute ischemic stroke. Up-to-date discussion of the etiology, diagnosis, treatment, and prevention of this common cause of stroke and cognitive impairment. Over the past decade, the hospitalist model has become a dominant system for the delivery of inpatient care. Forces such as national mandates to improve safety and quality, and intense pressure to safely reduce length of hospital stays, are now exerting pressure on neurologists. To meet these challenges, a new neurohospitalist model is emerging. This is the first authoritative text to detail the advances and strategies for treating neurologic disease in a hospital setting. It includes chapters on specific acute neurologic diseases including stroke, epilepsy, neuromuscular disease and traumatic brain injury and also addresses common reasons for neurologic consultation in the hospital including encephalopathy, electrolyte disturbances and neurologic complications of pregnancy. Ethical and structural issues commonly encountered in neurologic inpatients are also addressed. This will be a key resource for any clinician or trainee caring for neurologic patients in the hospital including practising neurologists, internists and trainees across multiple subspecialities. Brain hypoxia-ischemia is a disorder characterised by a reduction in oxygen supply (hypoxia) combined with reduced blood flow (ischemia) to the brain. This condition may result from a localised

obstruction of a cerebral artery or from systemic hypoperfusion. Prolonged hypoxia-ischemia is associated with ischemic attack, transient; brain infarction; brain oedema; coma; and other conditions. This book presents the latest research in this field from around the world. Neonatal hypoxic-ischemic (HI) brain injury is a major cause of acute mortality and chronic neurological morbidity in infants and children. Studies indicate that transient receptor potential melastatin 2 or TRPM2 (a non-selective cation channel with high permeability to calcium that can be activated by intracellular adenosine diphosphoribose [ADPR] and H2O2) can mediate neuronal death following acute ischemic insults in adult mice as well as HI brain injury in neonatal mice. My study tested the effect of a newly described TRPM2 channel inhibitor AG490 by using a H2O2-induced neuronal cell death in vitro model and mouse HI brain injury in vivo model. I found that the inhibition of TRPM2 channels by AG490 demonstrates a neuroprotective effect both in vitro and in vivo. The neuroprotective effect of AG490 following post-injury treatment suggests the potential clinical implications of this drug, including the possible prevention of HI related neurological complications such as hypoxic-ischemic encephalopathy. Stroke is the third leading cause of death in industrialized countries and the number one cause of disability in adults. Ischemic strokes constitute approximately 80% of all strokes, and until recently, no effective therapies had been available. The successful tissue plasminogen activator (tPA) trials open a new era of stroke management with a proven therapeutic now available to patients if they arrive at the hospital within hours of the onset of symptoms. Recent advances in our understanding of the molecular mechanisms of ischemic brain injury and vascular pathology have opened up new avenues for the development of novel therapeutic strategies. The premise that substantial reduction of brain damage after ischemic stroke by therapeutic intervention has now been confirmed in animal stroke models, sets a realistic goal for future treatment of stroke patients. This book covers topics ranging from fundamental molecular mechanisms of ischemic brain injury to the most current views on the clinical stroke trials and academic and industrial efforts to develop new drugs. The authors are leading authorities in their own fields in basic and clinical stroke research. As a timely and comprehensive volume, it will be of immense value to basic and clinical investigators, neurologists and neurosurgeons who manage stroke

patients, neuroscientists interested in various aspects of brain injury, and researchers in the pharmaceutical industry who are engaged in new drug development. The literature concerning cerebral circulation, its control, its relation to the metabolism of the brain and its disorders, including cerebral ischemia, is very large and reflects an interest which goes back to Greek and Arabian medicine and which has greatly intensified, particularly over the past decades. Data of the literature remain, however, too sparse to propose a pragmatic approach to cerebral ischemia when affecting people at young age. The first purpose of this monograph has been therefore to bring together from such a literature a coherent account of how present concepts and approaches have developed. In this, the authors have been guided by what seems to be the important lines of investigation. Secondly, the evidence from studies which form the basis of the present understanding has been reviewed critically and, where possible, an attempt has been made to pose present controversies in terms which would be helpful in everyday clinical practice. Stroke is a medical emergency that requires immediate medical attention. With active and efficient nursing management in the initial hours after stroke onset and throughout subsequent care, effective recovery and rehabilitation is increased. Acute Stroke Nursing provides an evidence-based, practical text facilitating the provision of optimal stroke care during the primary prevention, acute and continuing care phases. This timely and comprehensive text is structured to follow the acute stroke pathway experienced by patients. It explores the causes, symptoms and effects of stroke, and provides guidance on issues such as nutrition, continence, positioning, mobility and carer support. The text also considers rehabilitation, discharge planning, palliative care and the role of the nurse within the multi-professional team. Acute Stroke Nursing is the definitive reference on acute stroke for all nurses and healthcare professionals wishing to extend their knowledge of stroke nursing. Evidence-based and practical in style, with case studies and practice examples throughout Edited and authored by recognised stroke nursing experts, clinicians and leaders in the field of nursing practice, research and education The first text to explore stroke management from UK and international perspectives, and with a nursing focus Neurological disorders caused by acute disruption of cerebral blood flow are a major clinical concern in both the adult and newborn. Loss of oxygen utilization

despite adequate blood flow marks brain tissue fated for death in stroke, but the reason for this is not clear. In this study, we used a cerebral ischemia-hypoxia model in mice to determine the effects of hypoxia on partially ischemic tissue. We found this engaged simultaneous cell survival and death signaling, as well as autophagy, a process by which the cell digests its contents to stay alive during an energy crisis. This was associated with oxidative stress, extensive clotting, and impaired reperfusion after ischemia-hypoxia. This perfusion impairment was prevented in fibrinogen deficient mice, who also exhibited less injury. These results indicate the loss of tissue oxygen utilization in stroke may lead to clotting in the vasculature, and that this must be resolved for effective recovery. In contrast, in neonatal rat hypoxia-ischemia, a model of hypoxic-ischemic encephalopathy, perfusion impairment was restored by 4 hours recovery, and the mechanism for this is also unclear. We found this restoration was associated with an induction of the clot busters tissue-type (tPA) and urokinase-type (uPA) plasminogen activators. tPA deficient mice had greater fibrin deposition and mortality during hypoxia than wildtype mice, confirming a protective role. However, the induction of tPA, which can also be potentially damaging, was sustained throughout the future lesion in rats, and associated with blood-brain-barrier damage, white matter degradation, neuronal axon loss, and apoptosis. Inhibition of the downstream plasmin with alpha-2 antiplasmin offered significant protection, and the injection of additional tPA made the injury worse. These results indicate that inhibition of deleterious plasminogen activator / plasmin effects may aid in treatment of hypoxic-ischemic encephalopathy. All together, this study shows a differential response between the neonate and adult brain to hypoxia in a partially ischemic environment, the consequences of which may provide prime targets for clinical intervention. Abstract: Transient focal ischemia occurs when a portion of the brain is cut off from oxygen. Damage occurs in two phases: the initial phase, during which cell death by necrosis occurs, and the secondary phase, during which cell death by apoptosis and inflammation occurs. Exposure to lipopolysaccharide (LPS), a component of the Gram-negative bacteria, initiates an innate immune response resulting in inflammation. Specifically, LPS activates cytokines which then induce cyclooxygenase-2 (COX-2) to synthesize PGE2, a prostaglandin that causes inflammation. COX-2 also contributes to the activation of the glutamate

receptors during the initial stages of ischemic damage. Early LPS exposure up-regulates the basal levels of COX-2 in adulthood. To determine the role of COX-2 in stroke Escherichia coli LPS was administered to fourteen-day-old male and female neonatal mice and transient focal ischemia was later induced in adulthood. The control group was given an equal amount of pyrogen-free sterile saline solution. It was hypothesized that after induction of experimental stroke mice administered LPS would show greater stroke damage, both initially and secondarily, compared to their vehicle counterparts. To assess initial damage 24 h post-surgery mice were perfused and level of edema was determined. To assess secondary damage 72 h post-surgery infarct was measured. Behavioral deficits occurring at both 24 h and 72 h post-surgery were determined by preferential paw use and level of anxiety. Females neonatally treated with LPS had a significant decrease in infarct size 72 h post-surgery compared to their vehicle counterparts. These results are indicative of LPS acting as a neuroprotective agent against ischemic stroke. The 72 h survival females, however, showed a significant decrease in contralateral paw use compared to the vehicle suggesting that the LPS had a negative effect on behavioral outcome despite its neuroprotective capability. There was no significant effect of LPS on any of the other experimental groups. The study was reproduced with a doubled dose of LPS and no significant effect was seen across the board. This suggests that the amount of LPS administered is a critical factor. An effect only on females indicates a gender bias. Ischemic stroke, an interruption of cerebral blood flow leading to death or permanent loss of function, is responsible for significant mortality and disability, particularly in the older population. Transient ischemic attack (TIA), in which there is no tissue infarction and symptoms rapidly resolve, is associated with an elevated risk of subsequent stroke. Investigation of cardiac risk factors and possible sources of emboli of stroke/TIA is undertaken to identify modifiable risk factors, and reduce the risk of subsequent stroke. Transthoracic echocardiography (TTE) involves ultrasound visualization of the heart through the chest wall. Transoesophogeal echocardiography (TOE) involves introducing the probe into the patient's esophagus, which allows better resolution of most of the cardiac structures due to proximity (with the exception of the left ventricle), but is more invasive and less well tolerated, more time-consuming and costly, and potentially less readily

available. Both procedures are considered to have low diagnostic yields in identifying cardiac sources of emboli in patients with no other identifiable reason for stroke, but they do enable intervention in patients with positive findings. This report examines the clinical and cost effectiveness of using TTE with clinical evidence to identify cardiac sources of emboli in adult patients with ischemic stroke, alone and compared with TOE. The Social Security Administration (SSA) uses a screening tool called the Listing of Impairments to identify claimants who are so severely impaired that they cannot work at all and thus immediately qualify for benefits. In this report, the IOM makes several recommendations for improving SSA's capacity to determine disability benefits more quickly and efficiently using the Listings. BACKGROUND:Methamphetamine abuse is an increasingly recognized risk factor for stroke, causing up to 6% of ischemic strokes in young people. u201cShabuu201d, the purest form, is widespread in Southeast Asian communities. Shabu is frequently assumed by inhalation and it is tipically associated with ischemic stroke. We are reporting a case of ischemic stroke in a Shabu abuser. METHODS:A 55-year-old Asian active Shabu abuser male presented with a 24-hours history of headache and left limbs weakness. The patient suffered from hypertension, diabetes and dyslipidemia. Neurologic evaluation revealed left facial palsy, left hemiparesis with sensory loss. Brain Magnetic Resonance Imaging (MRI) showed recent ischemic lesions located in the frontal right deep white matter and corpus callosum. Multiple focal vessel wall thickening and narrowings affecting cervical and intracranial vessels and subocclusive stenosis of the right anterior cerebral artery were disclosed by MR Angiography (MRA) and confirmed by cerebral angiography. To rule out central nervous system (CNS) or systemic vasculitis, autoimmune screening, lumbar puncture and total body positron emission tomography (PET) were performed with negative results. Intravenous metilprednisolone was administered without clinical and radiological improvements. CONCLUSIONS: Amphetamine-related cerebral vasculopathy (ARCV) is associated to higher risk of strokes. Ischemic strokes may result from accelerated atherosclerosis, often associated with cardiovascular risk factors, rather than necrotizing arteritis. Even if radiological findings are suggestive for CNS vasculitis, there is no evidence of inflammation or necrosis in histological samples. In support of this hypothesis, our patient did not recover after steroids

treatment. Accelerated atherosclerosis represents the most likely pathogenic mechanism underlying ARCV. Pediatric stroke has received increasing attention over the last few decades. Dysphagia is commonly reported following acute stroke in adults, leading to negative health complications such as pneumonia and mortality; however, dysphagia is not a well understood outcome post-stroke in pediatrics. This dissertation sought to explore early identification of dysphagia, with a focus on pediatric stroke, and was comprised of four studies: 1) a meta-analysis of high-level evidence on the benefit of dysphagia screening in adult stroke, 2) a systematic review of available literature on pediatric stroke and dysphagia; 3) a retrospective chart review to identify frequencies, co-occurrence and associations of dysphagia, motor speech and language impairment in pediatric stroke; and 4) a prospective survey of dysphagia experts to determine opinion of relevance and feasibility of assessment items to move forward in the development of a dysphagia screening tool. This dissertation confirmed the benefit of dysphagia screening in the adult stroke population. Further, this work identified dysphagia as a relatively common impairment post-stroke in the acute phase in pediatrics, similar to adults, and especially highlighted the need for standardized assessment protocols in the younger population. Lastly, this dissertation identified a list of assessment items to move forward in the development of a pediatric dysphagia screening tool post-stroke. Future work is indicated and discussed to further advance this under-researched area of dysphagia and pediatric stroke. ABSTRACT: Stroke is the third leading cause of death worldwide, and Vitamin D deficiency is associated with increased stroke risk, severity, and mortality. Vitamin D potently regulates the immune system and is associated with chronic inflammatory diseases such as diabetes and hypertension. The post-stroke inflammatory response plays a critical role in ischemia-induced CNS pathogenesis and neuroimmune interactions in the CNS and periphery regulate the nature, extent, and duration of inflammation. Interplay between systemic and centrally mediated signals and processes can dramatically influence the phenotype and duration of the immune response. In both the brain and the periphery, Vitamin D interacts with the events mediating CNS injury after cerebral ischemia primarily through immunomodulation and regulation of other endocrine factors such as IGF-1 that play an important role in stroke outcome. The experiments outlined here aim to determine

the unique contribution of Vitamin D deficiency to stroke and possible therapeutic interventions. Sensory motor behavioral deficits, ischemic cell death, and resolution of CNS injury is exacerbated by Vitamin D deficiency in adult rats subject to middle cerebral artery occlusion, while manipulation of peripheral CD4+ T cell subpopulations improves these parameters as compared with VDD animals injected with vehicle alone. However, supplemental Vitamin D treatments do not ameliorate the negative effects of Vitamin D deficiency. Under each condition, changes in inflammatory cytokines were also observed post-ischemia and the phenotype and secretions of recruited immune cells, especially CD4+ T cells, plays an important role in the pathogenesis of the post-stroke inflammatory response. Cytokine expression in VDD suppresses development of neuroprotective Tregs. Together, these results suggest that peripheral immune status significantly influences stroke severity and that acute manipulation of the peripheral immune system post-stroke can prevent or ameliorate inflammation-associated neurodegeneration. Vitamin D deficiency may exacerbate stroke outcome by altering the CD4+ Treg:Th17 ratio and associated cytokine/chemokine expression and diminishing protective endocrine function, resulting in neurotoxic consequences. Our findings indicate that Vitamin D deficiency promotes an immunocompromised, pro-inflammatory state in rats subject to experimental stroke surgery. Stroke remains a leading cause of long-term disability in adults, and impairments in the upper extremities are particularly common. Many post-stroke remodeling events are activity dependent and can be influenced by post-ischemic behavioral experience through similar mechanisms as experience-dependent plasticity. The overarching goal of these dissertation studies was to understand how behavioral experience, in the form of rehabilitative training (RT), after ischemia impacts neuronal and vascular structural remodeling. This was tested using a mouse model of ischemia-induced upper-limb impairments in adult transgenic mice containing yellow or green fluorescent protein (YFP/GFP) in a subset of layer V cortical pyramidal neurons. First, I examined the impact of manual skill learning on dendritic spine dynamics in vivo in the trained motor cortex (MC) of intact mice (Chapter 2). We found that spine formation was significantly enhanced after 3 days of training, which was followed by an equal and opposite increase in spine elimination by day 6 and then a return to baseline levels for the remainder

of the training duration. New spines formed on day 3 were preferentially stabilized and were correlated with performance gains. Next, I tested whether a variation of the photothrombotic stroke model that confines laser illumination to individual arteries on the cortical surface, could better reproduce aspects of the vascular penumbra, such that it would be better suited for examining how structural remodeling events are influenced by the penumbra. We monitored post-ischemic cerebral blood flow (CBF) at 6, 48, and 120 h following MC infarcts and found that artery-targeted photothrombosis created a wider, more graded penumbra. In addition, it instigated vascular structural remodeling, and caused impairments in skilled-reaching performance in mice. Lastly, I examined the impact of RT on spine dynamics and recovery of skilled reaching performance following photothrombotic infarcts to MC. We found that ischemia instigated widespread increases in spine turnover that persisted for up to 5 weeks. RT increased the stabilization of new spines formed in weeks 2 and 3 after ischemia, which was correlated with improvements in skilled reaching, indicating that new spine maintenance could represent a structural mechanism for the recovery of reaching performance.

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- Cerebral Small Vessel Disease
- Cerebral Ischemia In Young Adults
- Inflammation Combined With Ischemia Produces Myelin Injury And Plaque like Aggregates Of Myelin Amyloid beta And AbetaPP In Adult Rat Brain
- The Effects Of Remote Limb Ischemic Conditioning On Functional Motor Outcome Of The Paretic Limb After Ischemic Stroke
- Effects Of TRPM2 Inhibition In Neuroprotection Following Neonatal Hypoxic Ischemic Brain Injury
- Fetal Hippocampal Transplants Into The Ischemic Hippocampi Of The Adult Mongolian Gerbil As A Means Of Ameliorating Functional Deficits Due To Global Cerebral Ischemia
- Effects Of Early Neonatal Infection N Adult Cerebrovascular Health
- Regeneration In The Adult Brain After Focal Cerebral Ischemia
- SHABU ABUSE AND ISCHEMIC STROKE IN AN ASIAN ADULT
- Regenerative Therapies In Ischemic Stroke Recovery
- Insights Into Ischemia induced Dendritic And Vascular Plasticity Through In Vivo Imaging
- Ischemic Stroke
- Vitamin D And The Neuroimmune Network In Ischemic Stroke