CITATION: Hasan v. Trillium Health Centre Mississauga, 2022 ONSC 3988
COURT FILE NO.: CV-13-5376
DATE: 20220707

ONTARIO

SUPERIOR COURT OF JUSTICE

BETWEEN:	)	
	)	
SYED JADWAL HASAN, SHAZIA HASAN, SYEDA KISA ZEHRA, SYED ZAIN JAFFARY, SYEDA ARUBA ZEHRA	)	Duncan Embury and Daniela Pacheco, for the Plaintiffs
Plaintiffs	)	
- and -	)	
TRILLIUM HEALTH CENTRE MISSSSAUGA DR. VERITY JANE JOHN, DR. ALAN CAMPBELL, DR. CHIKKAHANUMAIAII DEVARAJ, JOHN DOE and JANE DOE	)	Barbara Walker Renshaw, for the Defendant Trillium Health Care Jennifer Hunter and Nadia Marotta, for the defendant Dr. Campbell
Defendants	)	
	)	**HEARD:** January 11, 12, 13, 14, 18, 19, 20, 21, 24, 25, 26, 27, 28, 31, February 1,3,4,7,14,15,16, 2022

REASONS FOR JUDGMENT

TZIMAS J.

INTRODUCTION

[1] In the early hours of December 3, 2011, Mr. Syed Hasan felt dizzy, nauseous, and began to vomit. He felt unsteady and needed to hold somebody's hand for fear of falling. He attended Milton District Hospital and, after being assessed, was discharged with the diagnosis "probable peripheral vertigo". He returned home and went to bed.

[2] Later that morning, he continued to feel unwell and went to see his family doctor. She examined him, gave him a referral note with the express request: "Please rule out organic cause (brain lesion or stroke)", and directed him to Trillium Health Partners – Mississauga Hospital ("Trillium"), a designated Regional Stroke Centre.

[3] Dr. Campbell was the emergency physician at Trillium who assessed Mr. Hasan. He did not see the note from the family doctor. He purported to take a history from Mr. Hasan, examined him, ordered medications for the vertigo and nausea and some diagnostic tests. Ultimately, his diagnosis was "Dizzy – Bell's Palsy – Peripheral Vertigo". Dr. Campbell discharged Mr. Hasan with a prescription for his dizziness and instructed him to follow-up with his family doctor in 3-4 days. He also told him to return to the Emergency Department ("ED") if his condition worsened.

[4] In the early hours of December 4, Mr. Hasan's condition deteriorated significantly. He woke up around 3 a.m. to discover that he could not get out of bed. An ambulance returned him to the ED at Trillium. Dr. Campbell examined him again and ordered diagnostic tests. By noon, Mr. Hasan deteriorated to such a point that he had to be intubated and admitted to the intensive care unit ("ICU"). Mr. Hasan had suffered a stroke with devastating and severe long-term disabilities.

[5] Mr. Hasan commenced a negligence action against Dr. Campbell. He alleges that Dr. Campbell's care fell below the standard of care for an emergency physician. As a result of that breach, Mr. Hasan further alleges that Dr. Campbell caused him to suffer devastating long-term disabilities. He contends that his stroke was the result of a basilar artery occlusion. *But for* Dr. Campbell's breach, he would have been assessed in a timely manner, treated successfully, the blockage to the basilar artery would have been cleared, and Mr. Hasan would have had a full recovery.

[6] Dr. Campbell disagrees. In his view, as an experienced and conscientious physician, he came to a reasonable working diagnosis that met the standard of care. On causation, the stroke was the result of an intracranial arterial dissection that would have been impossible to treat.

[7] For the following reasons, I conclude on a balance of probabilities that Dr. Campbell breached the standard of care for an emergency physician. I also

conclude on a balance of probabilities that Mr. Hasan suffered an embolic stroke, and that but for Dr. Campbell's breach of the standard of care, Mr. Hasan would have been assessed and received treatment in a timely manner, which in turn would have resulted in a successful treatment and recovery. Finally, I dismiss Dr. Campbell's claim for a discount in damages on account of the possibility that Mr. Hasan would not have obtained a full recovery, even if he received treatment in a timely manner.

BACKGROUND

a. Agreed Facts

[8] The parties filed a very detailed agreed statement of facts. Highlights of those facts follow.

[9] On the evening of Friday, December 2, 2011, Mr. Hasan attended mosque with his family where he engaged in a religious ritual involving tapping on the front of the chest, near the shoulders. After, he drove himself home, ate a sandwich, and went to bed. Mr. Hasan had no pain or other symptoms.

[10] In the early morning hours of Saturday, December 3, 2011, Mr. Hasan awoke feeling dizzy, nauseous, and began to vomit. He felt so bad that he went to the ED at Milton District Hospital and detailed his symptoms. His presenting complaint was documented to be: "Dizzy, Nausea." He was assessed by a nurse

at 3:35 a.m. At triage, Mr. Hasan was assigned a CTAS score of 3 – Urgent. Shortly thereafter, Mr. Hasan was assessed by Dr. Hafiz Umer. He found that Mr. Hasan was looking well and that his vital signs were stable. Dr. Umer ultimately diagnosed Mr. Hasan with "probable peripheral vertigo". Mr. Hasan received Gravol and Zofran and was also administered Serc. Mr. Hasan was discharged at 5:20 a.m. and went directly home and to bed. At the time of his discharge, he was noted to be "stable per wheelchair".

[11] Later that morning, Mr. Hasan continued to feel unwell and went to his family physician, Dr. Odette Wahba. She examined Mr. Hasan and instructed him to immediately attend the ED at Trillium. She provided him with a referral note ("Referral Form") addressed to "Trillium ER" that read: "Please rule out organic cause (brain lesion or stroke)."

[12] Due to his dizziness, Mr. Hasan was unable to walk to Trillium by himself. He was registered in the ED at 1:49 p.m. and Dr. Wahba's note was provided to the triage nursing staff; he was assigned a CTAS score of 3 – Urgent. Mr. Hasan was assessed by two nurses, once at 1:32 p.m. and again at 3:00 p.m. They documented Dr. Wahba's Referral form and that Mr. Hasan had been in the hospital "last night for same." They noted that Mr. Hasan went outside at night, developed pain in the left occipital area, and then started feeling dizzy with headache, some visual difficulties, loss of hearing, and "right side droop."

[13] Mr. Hasan was assessed by the Defendant emergency medicine physician, Dr. Alan Campbell, at 3:05 p.m. Dr. Campbell took a history, documented various symptoms and noted that the onset of these symptoms was "last night." The symptoms documented by Dr. Campbell were: hearing a lot of noises in his head, can hear but has to be talked to loud, "lots of birds in my head noise," right facial weakness which began at 0100 hours, slight left neck pain, nausea, vomiting x 6 last night, multiple episodes of vertigo, and a slight sore throat. Dr. Campbell further documented that Mr. Hasan's speech was normal and there was no chest pain, no trauma or head injury, arms feel normal power, no fever, no extremity paresthesia no palpitations, no allergies, not on any medications, and no past or present comorbid conditions. As part of the history, Dr. Campbell documented that Mr. Hasan had gone to the ED in Milton where he was given an injection of medication and diagnosed as having vertigo. He did not document anything about a note from Dr. Wahba.

[14] Dr. Campbell conducted a physical examination and observed that Mr. Hasan was alert but had total right facial weakness. Dr. Campbell documented Mr. Hasan's condition and ordered intravenous Stemetil 5 mg to treat Mr. Hasan's vertigo and nausea, oral Prednisone 50 mg for his total right facial palsy, and one litre of intravenous normal saline to replace fluid loss from vomiting.

[15] Dr. Campbell had stroke on his differential diagnosis for Mr. Hasan, which is a potentially life-threatening condition. He ordered bloodwork – specifically, routine chemistry and CBC (complete blood count); the bloodwork was non-diagnostic. Because of the vertigo and the Bell's palsy, he also ordered a CT scan of the head to rule out a cerebral vascular accident ("CVA") or a mass. Mr. Hasan underwent a CT scan of the head without intravenous contrast. The radiologist report stated that there was no acute intracranial process, no evidence of acute intracranial hemorrhage, infarct or mass lesion, and no abnormal intracranial fluid collections.

[16] Dr. Campbell reassessed Mr. Hasan after medications were administered and the bloodwork and CT scan were completed. Dr. Campbell documented that the CT scan showed no acute abnormality and that Mr. Hasan's dizziness had improved. His diagnosis was "Dizzy – Bell's Palsy – Peripheral Vertigo". He did not speak to or consult with a neurologist regarding Mr. Hasan's condition and he did not order a CT angiogram. He discharged Mr. Hasan at 5:26 p.m. with a prescription for Stemetil and Prednisone. Dr. Campbell instructed Mr. Hasan to follow up with his family physician in 3-4 days and return to the ED if he had any problems or if his condition worsened.

[17] At around 3:00 a.m. on Sunday, December 4, 2011, Mr. Hasan woke up to go to the bathroom, only to discover that he could not get out of bed. Eventually,

his wife called the Halton Emergency Medical Services ("EMS") who assessed Mr Hasan, found that his symptoms were now more pronounced, and returned him to Trillium. Mr. Hasan was again assessed by a triage nurse at Trillium at 8:21 a.m. who assigned him a CTAS score of 2 – Emergent.

[18] Dr. Campbell assessed Mr. Hasan at 9:15 a.m. He reviewed the radiologist's report from Mr. Hasan's CT scan of the prior day, and noted that the interpretation by the radiologist was negative for any abnormality. Dr. Campbell obtained a history, conduced a physical examination, and documented Mr. Hasan's symptoms and his observations. He then ordered a CT scan of the head, with and without contrast. His note indicated that the CT scan was to rule out a CVA (stroke) and his differential diagnosis included stroke, a potentially life-threatening condition. The radiologist concluded that there was no acute intracranial process; no acute intracranial hemorrhage, infarct, or mass lesion; no abnormal intracranial fluid collections; no abnormal enhancement; and no acute or subacute infarct. Dr. Campbell was then informed that the CT scan had been completed.

[19] On reassessment, Dr. Campbell documented the worsening of symptoms, but he did not order a CT angiogram or an MRI. He then handed over Mr. Hasan's care to the internist and his involvement with Mr. Hasan ended. Mr. Hasan deteriorated and was admitted to Trillium's ICU.

[20] On Wednesday December 8, 2011, five days following Mr. Hasan's initial symptoms, Mr. Hasan underwent an MRI of the brain. This study was reported as showing an acute infarct with restricted diffusion involving the right aspect of the pons as well as left paramedian aspect and involving the bilateral paramedian medulla. It was also reported to show small acute infarcts in the inferior right cerebellar hemisphere and anterior-inferior left cerebellar hemisphere, fetal origin of the bilateral posterior cerebral arteries and a basilar artery that had a small caliber. The MRI report also noted the loss of normal flow void in the proximal basilar artery and distal vertebral arteries. The radiologist suspected a thrombosis and proposed further evaluation with CT angiography of the neck and circle of Willis arteries.

[21] On the same date, Mr. Hasan also underwent a CT angiogram of the head and neck. Observing the imaging, the radiologist opined: "Occluded distal vertebral arteries and proximal basilar trunk. The changes are highly suggestive of left vertebral artery dissection and distal thromboembolism."

[22] Mr. Hasan was then diagnosed with a brain stem stroke and placed on IV heparin (anticoagulation) protocol infusion. This stroke caused him to suffer permanent and disabling injuries, requiring assistance from another person for almost all activities of basic living, including his own bodily needs, and placing him at a score of 4 on the modified Rankin Scale.

b. Treatments Available at Trillium

[23] In December 2011, there was one interventional neuroradiologist who worked at Trillium. That doctor continues to hold privileges at Trillium today. The available treatments at Trillium in December 2011 included intra-arterial tPA, or intra-arterial thrombolysis and endovascular thrombectomy ("EVT"). Fourteen EVT procedures were conducted at Trillium from 2008 to 2011. Currently, on average, EVT is used in more than 14 cases each month.

c. Medical Definitions and Diagram

[24] The parties provided the court with a "Joint Medical Glossary" which was essential to engaging with and understanding the evidence before the court. The parties also relied on various demonstrative aids to orient the court on the relevant parts of the brain and to visualize various concepts. Schedule "A" to this judgment contains the Joint Medical Glossary and one of the diagram that both parties used.

POSITION OF THE PARTIES

a. Mr. Hasan

[25] Mr. Hasan submitted that the devastating and severe long-term disabilities he suffered in December 2011 resulted from Dr. Campbell's failure to properly

diagnose him, even though he attended Trillium with a note from his family doctor that specifically requested: "Please rule out organic cause (brain lesion or stroke)".

[26] According to Mr. Hasan, Dr. Campbell's principal failure occurred on December 3, 2011, when instead of ruling out an organic cause for his symptoms, as requested by Dr. Wahba, Dr. Campbell discharged him with a diagnosis of Bell's Palsy and peripheral vertigo. This failure continued on December 4, 2011, when Mr. Hasan returned to the ED in a deteriorating condition, exhibited the signs and symptoms of a progressive basilar artery occlusion ("BAO"), but did not receive the attention that a life-threatening condition would demand.

[27] The basilar artery is an artery that supplies blood to the vital structures of the brainstem and surrounding areas. An occlusion is a blockage. A BAO means that blood flow is disrupted. If left untreated, BAO carries a high rate of death or severe long-term disabilities. Its treatment essentially involves the removal of the blockage and the restoration of the blood flow ("recanalization") to the vital brain tissue as soon as possible. Without recanalization, the areas affected by the disruption in the blood flow will die, resulting in infarct or stroke in those areas.

[28] Dr. Campbell's failure to appreciate that Mr. Hasan's symptoms were the result of a life-threatening medical emergency meant that Mr. Hasan's BAO was not treated. He suffered devastating injuries and catastrophic deficits that have left him requiring assistance with almost all his activities of daily living.

[29] Mr. Hasan submits that had Dr. Campbell either ordered a CT angiogram or consulted a neurologist on December 3, then, on a balance of probabilities, he would have been properly assessed, received treatment, and the recanalization would have been successful.

b. Dr. Campbell

[30] Dr. Campbell denied that his care of Mr. Hasan in the ED at Trillium on December 3 and 4, 2011, fell below the standard of care. In his view, he conducted a thorough examination, ordered appropriate tests, exercised his clinical judgment, and came to a reasonable working diagnosis of a peripheral cause for Mr. Hasan's vertigo and facial palsy.

[31] Dr. Campbell also disagreed with the cause for Mr. Hasan's stroke. He rejected Mr. Hasan's theory of a BAO and submitted instead that Mr. Hasan's injury was caused by an occlusive vertebral arterial dissection at the V3/V4 level; that is, a tearing in the inner lining of the artery. Its occurrence at the V3/V4 level made it impossible to treat.

OVERVIEW OF THE EVIDENCE

[32] The court heard from Mr. Hasan. In his testimony, he amplified and elaborated on his experience just before he fell ill, his attendances at Milton Hospital, at his family doctor's office, his two attendances at Trillium, his steady

deterioration, and the extraordinary deficits that resulted from his stroke. The court also heard from his friends who took him to the hospital and from Dr. Wahba.

[33] The court then heard evidence from expert witnesses for both parties. Drs. Edwin Brankston, David Gladstone, David Mikulis, and Gordon Cheung testified for Mr. Hasan. Drs. Arun Sayal, Frank Silver, and Timo Krings testified on behalf of Dr. Campbell. What follows is a brief overview of each expert's background, the parameters of their qualification before the court, and highlights of their very extensive evidence. My assessment of each witness' evidence is contained within my analysis of each legal question. The experts are listed in the order which they appeared before the court.

a. Dr. David Gladstone

[34] Dr. Gladstone is a full-time neurologist and Associate Professor in the Department of Medicine at the University of Toronto. He practices in the Division of Neurology, Brain Science Program and Regional Stroke Centre at Sunnybrook Health Sciences in Toronto. He is also a Scientist at Sunnybrook Research Institute. He obtained his medical degree from the University of Toronto in 1996 and completed his residency training in neurology at the University of Toronto in 2003. He completed a PhD in clinical stroke recovery via the Royal College Clinician-Scientist Training Program and Institute of Medical Sciences at the University of Toronto with a CIHR Heart and Stroke Foundation Fellowship Award.

[35] Dr. Gladstone's 43-page curriculum vitae reveals that he has received multiple competitive awards, participated in multiple substantial research projects in various capacities. He has supervised students at the bachelor, masters, doctoral, and post-doctoral levels, enjoys memberships with various high profile stroke associations. He presents regularly on stroke-relates subjects, and has authored or co-authored over 150 peer-reviewed publications spanning the spectrum of stroke care.

[36] Since 2004, Dr. Gladstone has specialized in the diagnosis and management of ischemic stroke, intracerebral hemorrhage, transient ischemic attack, carotid artery disease, atrial fibrillation, antiplatelet and anticoagulant therapies, acute stroke thrombolysis ("tPA"), and patient selection for endovascular thrombectomy. In the past 15 years on the hospital full-time staff, he has provided stroke and general neurology consultation to Sunnybrook's ED, ICU, and inpatient medical and surgical wards. As part of the hospital's 24/7 Code Stroke Team, he provides regular on-call consultation to the ED for acute stroke emergencies, particularly for tPA treatment and evaluation of patients for endovascular stroke therapy. His activities also include the review and interpretation of imaging studies, participation in weekly hospital-based Stroke Imaging Rounds, the use and maintenance of certification in stroke assessment rating scales, directs the Sunnybrook Regional Stroke Prevention Clinic, regularly teaches students, residents, and fellows on wards and in clinic, and provides

regular emergency stroke and tPA/EVT consultations remotely to 28 hospitals within the Telestroke program of the Ontario Telemedicine Network.

[37] Dr. Gladstone was qualified as an expert in the medical field of stroke neurology, including the examination, diagnosis, treatment, and outcome measurement of strokes, and to provide opinion evidence in the following areas:

- The standard teaching to Emergency Room doctors regarding the assessment, testing, and diagnosis of stroke;
- The components of a proper neurological examination to test for stroke;
- Basilar artery occlusion, including the symptoms, assessment, diagnosis and treatments available in 2011;
- The cause and mechanism of stroke in Mr. Hasan's case;
- The appropriate diagnostic and treatment pathways for Mr. Hasan's condition;
- The likely course of treatment in the event that a stroke specialist was consulted on December 3, 2011;
- The likely course of treatment in the event that a stroke specialist was consulted on the morning of December 4, 2011;
- Mr. Hasan's likely outcome in the event that treatment was initiated;
- The relevant medical literature and studies on basilar artery occlusion and their relevance to predicting outcome;
- The clinical predictors of recovery in Mr. Hasan's case and their relevance to outcome measurement; and
- Whether earlier intervention was warranted and whether it would have changed Mr. Hasan's outcome, and, if so, how.

[38] Dr. Gladstone testified extensively over the course of three and a half days. He concluded that Dr. Hasan's stroke was the result of an occlusion of his basilar artery. But for the failure to diagnose the evolving stroke, Mr. Hasan would have received emergency treatment to limit the severity of the stroke. Had he received the correct diagnosis in a timely fashion, it is more likely than not that Mr. Hasan would have been treated either with tPA, intra-arterial tPA, EVT, or a combination of therapies. Combining Mr. Hasan's clinical facts and imaging with the statistics of the natural history and treatment outcomes of patients with BAO from published medical literature, Dr. Gladstone also concluded that it was more likely than not that Mr. Hasan would have had a good outcome from treatment.

b. Dr. David J. Mikulis

[39] Dr. Mikulis is a Full Professor and Director of the Functional Neuroimaging Research Lab in the Joint Department of Medical Imaging at the University Health Network and the University of Toronto. He is a practising staff Neuroradiologist at Toronto Western Hospital and the University Health Network with fellowship training in Neuroradiology at the Massachusetts General Hospital in Boston, Massachusetts. He holds a certificate in Neuroradiology awarded by the American Board of Radiology. He has been practicing in Neuroradiology exclusively since 1987 and has been involved in the interpretation of CT and MRI studies of the human central nervous system since 1989.

[40] Dr. Mikulis obtained his medical degree from Tufts University School of Medicine in 1981. His postdoctoral training was principally in radiology. His academic appointments have included Harvard Medical School, the University of Toronto, and related institutes. His hospital appointments from 1985 to the present have included Massachusetts General Hospital and Toronto Western Hospital. Highlights from his 84-page resume include the following: He has been recognized with over 20 honours and awards of excellence. He sits on a several major committees related to neuroradiology, brain and behaviour, and stroke. His editorial activities since 1987 have been connected to highly reputable and recognized medical journals, including the American Journal of Neuroradiology, The Canadian Journal of Neurological Sciences, J. Rheumatology, Neurosurgery, Radiology Brain, and Stroke. He is a member of several professional societies related to Neuroradiology.

[41] In addition to his very extensive teaching profile, he is a regular instructor and speaker at continuing medical education events and courses. He has supervised several Masters, PhD, and Post-Doc candidates and he has sat on several thesis committees. Dr. Mikulis lists in his resume over 300 scholarly publications, six chapters in textbooks, his scholarly works, publications, 10 publications in non-refereed publications, and 35 presentations. Finally, he holds several international visiting professorships.

[42] Dr. Mikulis was qualified as an expert in neuroradiology and vascular wall imaging, and to provide opinion evidence on the following:

- The findings and interpretation of the CT scan of Mr. Hasan's brain taken December 3 and 4, 2011;
- The findings and interpretation of the CT-angiogram and the MRI of Mr. Hasan's brain taken December 8, 2011;
- The findings and interpretation of the CT-angiogram of Mr. Hasan, taken May 25, 2012;
- The anatomy of Mr. Hasan's neuro-vasculature; and
- The mechanism of Mr. Hasan's stroke and how that relates to the causation issues that arise in this case.

[43] Dr. Mikulis testified over approximately three days. In his opinion, a dissection in Mr. Hasan's left vertebral artery at the C5/C6 level was the source for Mr. Hasan's stroke. Describing it as the "smoking gun", he explained that because of that dissection, a thrombus developed at the site. It then embolized to the distal left vertebral artery and filled the proximal basilar artery. Alternatively, a clot propagated from the distal vertebral artery into the proximal basilar artery. Once the left vertebral artery and the basilar artery occluded, infarcts developed in the medulla, inferior pons, and both inferior cerebellar structures.

c. Dr. Edwin Brankston

[44] Dr. Brankston was qualified as an expert in emergency medicine to provide opinion and comment on the standard of care issues. Dr. Brankston completed a

Bachelor of Science with Honours in Biology from York University in 1973. He obtained a medical degree from Queen's University in 1976. He obtained certifications in Family Medicine and Emergency Medicine from the College of Family Physicians in 1987, and then renewed it in 2010 and November 2021. In 1982 he started a residency in neurosurgery for one year at the Ottawa Civic Hospital, which involved six months in the ICU, 3 months in vascular surgery, and 3 months in general surgery. For various reasons, he continued as a family doctor and an emergency room physician at Oshawa General, later renamed Lakeridge Health Centre. From 1977 until 2012, and other various periods, Dr. Brankston worked in the emergency room on a part-time basis. From 2009 until 2018 he also did courtesy emergency room shifts at the Haliburton Highlands Health Service.

[45] Over the years, Dr. Brankston held various leadership and director roles at Lakeridge, including as Chief Hospitalist from 2001-2005. He also sat on various internal and external committees relating to various medical and physicians' issues connected with emergency medicine, pain control, record-keeping, pharmacy and therapeutics, patient care standards, and geriatric issues. He has also been an instructor for various family medicine courses, and he has been an Assistant Professor of Family Medicine at Queen's University since 2007.

[46] Dr. Brankston explained that over the course of 30 years he undertook medical-legal opinions and chart audits for the College of Physicians and

Surgeons, involving standards of physician care, opinions for the Canadian Medical Protective Association (defence opinions), clinical expert opinions for plaintiffs, and independent medical assessments for Clarica Insurance Company. He has been qualified by the Superior Court of Justice several times.

[47] Dr. Brankston testified over the better part of a day. In his opinion, Dr. Campbell fell below the standard of care on December 3, because he failed to consider the totality of Mr. Hasan's neurological symptoms, which suggested that the location of Mr. Hasan's pathological process was in the brain stem. Following the CT scan, Dr. Campbell should have ordered a CT angiogram of Mr. Hasan's head and neck or, alternatively, he ought to have consulted the neurologist on call. On either of these inquiries, Dr. Campbell would have discovered a central life-threatening cause for Mr. Hasan's symptoms.

d. Dr. Gordon Cheung

[48] Dr. Cheung is a duly licensed radiologist in the Province of Ontario and certified by the Royal College of Physicians and Surgeons (1988) and the American Board of Radiology (1987). He obtained his medical degree from the University of Toronto in 1983 and then undertook postdoctoral training at the University of Western Ontario, the Monmouth Medical Center, in Long Branch New Jersey, and was a staff member at the Yale University School of Medicine, New Haven, Connecticut, from 1990-2003. Dr. Cheung was a staff neurologist, at the

Interventional Neuroradiology Sunnybrook Health Sciences Centre. After 2003, he became a consultant for various other hospitals and founded various private radiology and imaging clinics. Dr. Cheung's present positions include: MRI Consultant, KMH Cardiology Centres Inc., Consultant in the Department of Medical Imaging, Division of Neuroradiology and Interventional Neuroradiology with Sunnybrook Health Sciences Centre, Neuroradiologist, Joseph Brant Memorial Hospital, Malton Medical Radiology and Ultrasound and Partner with Epic Capital Management, Healthcare Fund.

[49] His resume highlights 11 awards, numerous professional affiliations, and activities connected to neuroradiology. Among his accomplishments, he notes the creation of the first MRI fellowship training program in 1993 and the pioneering of carotid angioplasty. He also lists 39 publications between 1990 and 2008, 28 presentations at scientific meetings from 1989 to 2000, and 80 presentations and lectures by invitation from 1986 until 2017.

[50] He has been qualified as a medical-legal expert on several occasions. He estimated that his opinions have been equally for plaintiffs and defendants. Dr. Cheung estimated that he reviewed more than 700 MRI images of the brain, if he counted all studies, head, neck, and spine, at Sunnybrook alone. He would read up to 14,000 MRIs a year in various radiology clinics, with half of them relating to brain, neck, and spine, and the other half relating to the general body. Of the head,

neck, and spine imaging, 20-30% would be CT scans. Fewer than that would be CT angiograms. Dr. Cheung went on to explain that at Sunnybrook he would review "several a day of CT angiograms".

[51] Dr. Cheung was qualified as an expert in neuroradiology and interventional neuroradiology to provide opinion evidence as to the following:

- The findings and interpretation of the CT scan of Mr. Hasan's brain taken December 3 and 4, 2011;
- The findings and interpretation of the MRI and the CT angiogram of Mr. Hasan's brain taken December 8, 2011;
- The findings and interpretation of the MRI of Mr. Hasan's brain taken February 11, 2012;
- The anatomy of Mr. Hasan's neuro-vasculature;
- Endovascular treatments for stroke, including thrombectomy and stenting, and how such treatments relate to Mr. Hasan's case; and
- The mechanism of Mr. Hasan's stroke and how that relates to the causation issues that arise in this case.

[52] Dr. Cheung testified over almost three days. He concluded that Mr. Hasan's stroke originated in a tear to his artery at the C5/C6 level causing a clot to form. It then broke off and embolized into the brain and resulted in the occlusion of the proximal basilar artery, which in turn resulted in Mr. Hasan's stroke. Dr. Cheung opined that Mr. Hasan could have been treated successfully with tPA, intravenous tPA and / or EVT, using various devices, like the MERCI or other retrievable stents.

e. Dr. Arun Sayal

[53] Dr. Sayal is a duly qualified medical practitioner licensed to practice medicine in Ontario since 1991. He obtained his medical degree from the University of Toronto in 1990 and completed a residency in Family Medicine in 1992, followed by a fellowship in Emergency Medicine, which he completed in 1993. Since then, he has practiced Emergency Medicine at North York General Hospital, which is a community teaching hospital in Toronto, Ontario. He is an Associate Professor in the Department of Family and Community Medicine at the University of Toronto and has received over a dozen teaching awards from hospitals and universities, nationally and internationally. From 2007 to 2013, Dr. Sayal served as the co-ordinator of North York General's Emergency Medicine Update and his responsibilities included setting the teaching agenda for over 600 emergency physicians who attend the course annually from across the country.

[54] For over 20 years, Dr. Sayal has been certified Heart and Stroke Foundation Medical Director for their Advanced Cardiac Life Support course. In that capacity, he has directed and taught over 80 courses. In 2008, he created a full-day emergency course and taught it across Canada over 300 times, primarily at community hospitals. Since 2015, he has been a Peer Assessor for the College of Physicians and Surgeons of Ontario where his role has been to review the care of and identify improvements for selected Ontario emergency physicians.

[55] Dr. Sayal was qualified as an expert in emergency medicine to give opinion evidence on the standard of care that applied to Dr. Campbell as an emergency physician. He was also qualified to opine on whether Dr. Campbell met the standard of care in his treatment of Mr. Hasan on December 3 and 4 of 2011.

[56] Dr. Sayal testified for just under a day. He concluded that Mr. Hasan presented with a very atypical and uncommon disease. He said that a vertebral diagnosis was very uncommon and that Dr. Campbell's assessment of Mr. Hasan was reasonable on both December 3 and 4 and met the standard of care.

f. Dr. Frank Silver

[57] Dr. Silver is a duly qualified medical practitioner in the province of Ontario and a fellow of the Royal College of Physicians and Surgeons of Canada with certification in neurology since 1983. He is a stroke neurologist at the Toronto Western Hospital, in the Division of Neurology at the University Health Network. He was the Director of the University Health Network Stroke Program and Medical Director of the Toronto West Stroke Network from 2000 until 2019. He is a Professor of Medicine at the University of Toronto in the Division of Neurology. His area of expertise is in cerebrovascular disease. He was a research leader for the Canadian Stroke Network and played a leadership role in designing and implementing the Ontario Stroke Strategy that shaped Ontario's current,

integrated, evidence-based stroke system of care. He is the founder and medical director of the Ontario Telestroke Program.

[58] Dr. Silver's areas of research include clinical studies in stroke management and health services research. He is an Affiliated Scientist at the Institute for Clinical Evaluative Sciences and was the co-Principal Investigator of the Ontario Stroke Registry that provides data for the evaluation of stroke care in Ontario. His 47-page resume highlights numerous "Distinctions and Research Awards" as well as several Teaching and Education Awards, the several courses he has taught, and his numerous administrative positions at the University of Toronto and elsewhere. He also has more than 40 peer-reviewed and non-peer reviewed research grants, over 130 co-publications in peer-reviewed publications, several non-peer reviewed publications, international, national, and provincial presentations and lectures. He has supervised undergraduate and postgraduate MD students.

[59] Dr. Silver was qualified as an expert in the field of stroke neurology, including the examination, diagnosis, and treatment of strokes; in particular, to give opinion evidence in the following areas:

- The interpretation of Mr. Hasan's radiological imaging, including the CT head scans of December 3rd and 4th, 2011, the MRI and CT angiogram of December 8th, 2011, and the MRI of February 11, 2012;
- The relevant medical literature and studies with respect to dissection, vascular territories and clinical correlation, and treatment and outcomes for basilar artery occlusion and stroke;

- The cause and mechanism of stroke in Mr. Hasan's case; and
- Whether an earlier diagnosis of Mr. Hasan's stroke could have allowed the use of treatments that, more likely than not, would have resulted in a better outcome.

[60] Dr. Silver testified for almost three days. In his opinion, Mr. Hasan's stroke was caused by a dissection in the left wall of his left vertebral artery that started at the third segment of the vertebral artery, just below the base of the skull at C2-3 and extended up and into the basilar artery and then back down a little bit into the right vertebral artery. This resulted in multiple branch occlusions and a stepwise progressive stroke with increasing deficits from multiple infarcts. He also concluded that the injury to Mr. Hasan was such that only the most conservative approach to treatment would have been appropriate. Any intervention with coagulants, tPA or EVT, would have been too risky to pursue.

g. Dr. Timo Krings

[61] Dr. Krings studied medicine in Aachen, Germany, and at Harvard Medical School in Boston. He completed a residency in Neuroradiology in Aachen and then a fellowship in neurointervention in France. He then joined the Neuroradiology division of the University of Toronto as a diagnostic and interventional Neuroradiologist in 2008. He is currently appointed to Radiology and Neurosurgery at the University Health Network and as an interventional Neuroradiologist at the Hospital for Sick Children. He is a Full Professor of Radiology and Surgery, the

Chief of Radiology at the Toronto Western Hospital, and the Head of the Division of Diagnostic and Interventional Neuroradiology at the University Health Network, Mount Sinai, and Women's College Hospitals. He holds the David Braley and Nancy Gordon Chair in interventional Neuroradiology at the University of Toronto.

[62] Dr. Krings focused his research efforts on imaging and treatment of neurovascular diseases. He has published more than 350 peer reviewed articles, and approximately 30 book chapters and four books on spinal, pediatric, and interventional neuroradiology and neurovascular anatomy. His current research focuses on the prediction of brain bleeding from brain vascular malformations using a multidisciplinary approach, development of novel methods to treat these life-changing events and estimating the relative effectiveness of treatments. To support these activities, he leads a team of neuro-imagers, neurointerventionalists, computational scientists, geneticist biologists, and epidemiologists within the Division of Neuroradiology. For all his various activities, Dr. Krings has received distinguished grants and awards.

[63] Dr. Krings was qualified as an expert in the fields of radiology, neuroradiology, and interventional neuroradiology; in particular, to give opinion evidence in the following areas:

- The interpretation of Mr. Hasan's radiological imaging;

- The relevant medical literature and studies with respect to dissection, endovascular therapy, endovascular therapy for acute stroke, and radiological imaging related to dissection, endovascular therapy, and acute stroke;
- The cause and mechanism of stroke in Mr. Hasan's case; and
- The feasibility of endovascular therapy for treatment of Mr. Hasan's stroke.

[64] Dr. Krings testified over three days. In his opinion, the most likely cause for Mr. Hasan's strokes was a perforator ischemia, caused by a dissection that started extracranially at the atlantal loop, meaning the C1-C2 level, and extended intradurally and into the basilar artery in a spiralling manner that "knocked out" selected perforators. He concluded that Mr. Hasan's injury was such that no intervention would have prevented the damage that he suffered.

ISSUES FOR DETERMINATION

[65] The parties agree that the issues to be decided are the following:

STANDARD OF CARE: Did Dr. Campbell fall below the standard of care for an emergency physician and if so, how?

CAUSATION: This issue involves the consideration of the following three questions:

a. What was the cause of Mr. Hasan's stroke?

b. What would have occurred to Mr. Hasan, "but for" the breach by Dr. Campbell in the standard of care?

c. Based on what would have occurred, is it more likely than not that Mr. Hasan would have had a good outcome?

DISCOUNT: If liability is established, should damages be reduced to reflect Mr. Hasan's "but for" the incident position and, if so, by what percentage.

[66] The court heard evidence from Mr. Hasan, his friends who assisted him, his family doctor, and the aforementioned experts.

[67] It is well recognized that in a medical malpractice case, the court requires expert opinions to determine questions relating to the standard of care and causation: see *Chan v. Tang*, 2012 ONSC 2050, at para. 124, and *Bafaro v. Dowd*, 2010 ONCA 188, at para. 31. As noted in *Tahir* v. *Mitoff*, 2019 ONSC 7298, at paras. 46 and 47, the outcome in such a case will often turn on the opinion(s) that the court accepts or rejects.

[68] As I consider each of the issues and the various expert opinions, I will be making specific findings and expressing my views and preferences. My conclusions in my assessment of the experts is guided by the following legal principles.

[69] Rules 4.1 and 53.03 of the *Rules of Civil Procedure*, R.R.O. 1990, Reg. 194, and section 52 of the *Evidence Act,* R.S.O. 1990, c. E.23, govern the general principles of expert evidence. An expert must provide fair, objective, and non-partisan assistance to the court. An expert's independence and impartiality go to both admissibility and, once admitted, the weight of their evidence.

[70] When weighing conflicting expert evidence, the Court should have regard to the following factors:

> a) the relevance of the training, experience and specialty of the witness to the medical issue before the court;
>
> b) any reason for the witness to be less than impartial; and
>
> c) whether that testimony appears credible and persuasive compared and contrasted with the other expert testimony at the trial.

See *Latin (Litigation Guardian of) v. Hospital for Sick Children,* [2007] O.J. No. 13 (S.C.), at para. 146.

[71] Most recently, Trimble J. in *Sit v. Trillium Health Centre,* 2020 ONSC 2458, at paras. 121-124, organized the various cases that have provided guidance on how to evaluate the evidence of competing experts and accept one opinion over another. The three-step process involves i. a consideration of one's qualifications and impartiality; ii. an assessment of the evidentiary basis for the opinion; and iii. an examination of the whole opinion. When it comes to weighing competing expert opinions, the trier of fact may accept or reject any part, or all the evidence to determine the weight to be given to the expert evidence. However, the court is not entitled to pick and choose among various aspects or portions of the expert's opinion on a given issue to fashion a new opinion. "Generally, the court does not have the expertise to determine whether the expert is right or wrong in their

opinion. When the court does this, it would, in effect, assume the role of the expert", *Sit,* at para. 125.

[72] The credibility and reliability of a particular witness is not the proper subject of expert opinion. Such evidence risks usurping the court's fact-finding role: See *Parliament v. Conley*, 2021 ONCA 261, at para. 44.

[73] Although an expert may rely on factual assumptions to formulate their opinion, the value of the opinion and the weight it may be afforded will depend on the validity of those assumptions. Experts are expected to synthesize information from their work and experience and "marry it with evidence adduced through exhibits, admissions and the testimony of other witnesses in the proceedings and express an opinion about a factual inference that should be drawn from the accumulated materials": see *R* v. *C.(M).*, 2014 ONCA 611, at paras. 69-70.

[74] To be clear, where the expert merely relies on facts that are not established by otherwise admissible evidence as the basis for his or her opinion, that opinion will not be evidence: see *Marchand (Litigation Guardian of)* v. *Public General Hospital Society of Chatham* (2000), 51 O.R. (3d) 97 (C.A.). In the consideration of that opinion, the trier of fact must first decide if the facts on which the opinion is based exist. Only then may the trier of fact proceed to consider the weight to be given to the opinion. Where an expert mingles admissible and

inadmissible evidence, the weight to be attributed to that opinion will be directly related to the amount and quality of admissible evidence on which the expert relies: see *Marchand*, at paras. 60-61.

[75] Finally, the fact that an expert has an impressive resume, has written extensively in an area, and holds an important position at a large hospital, in and of itself, will not mean that their expert opinion will necessarily be accepted. Where there are conflicting expert opinions, the court must weigh each and assess the weight to be given to the evidence: see *Crawford (Litigation Guardian of) v. Penney* (2003), 14 C.C.L.T. (3d) 60 (Ont. S.C.), aff'd (2004), 26 C.C.L.T. (3d) 246 (Ont. C.A.).

STANDARD OF CARE

Did Dr. Campbell fall below the standard of care for an emergency physician and if so, how?

[76] In my review of the evidence, and having regard for the applicable legal principles, I conclude that Dr. Campbell failed to meet the standard of care for an emergency physician. Before I go any further, I recognize that such a determination comes at the conclusion of Dr. Campbell's very long career as an emergency physician. I would like all parties to appreciate that I did not arrive at this conclusion lightly. While Dr. Brankston gave evidence on Mr. Hasan's behalf on the standard of care for an emergency physician, my findings and conclusions

are based principally on Dr. Sayal's various admissions during his cross-examination, as well as some of Dr. Campbell's own testimony.

a. Applicable Legal Principles

[77] The seminal statement of law on the applicable standard of care for physicians was provided in *Crits* v. *Sylvester et al.* (1956), 1 D.L.R. (2d) 502 (Ont. C.A.), at p. 508:

> Every medical practitioner must bring to his task a reasonable degree of skill and knowledge and must exercise the degree of care and skill which could reasonably be expected of a normal prudent practitioner of the same experience and standing, and if he holds himself out as a specialist, a higher degree of skill is required of him than of one who does not profess to be so qualified by specialist training and ability.
>
> I do not believe that the standard of care required of a *medical* practitioner has been more clearly or succinctly stated than by Lord Hewart C.J. in *Rex* v. *Bateman* (1925), 41 T.K.R. 557 at 559: "If a person holds himself out as possessing special skill and knowledge and he is consulted, as a possessing such skill and knowledge, by or on behalf of a patient, he owes a duty to the patient to use due caution in undertaking the treatment. If he accepts the responsibility and undertakes the treatment and the patient submits to his direction and treatment accordingly, he owes a duty to the patient to use diligence, care, knowledge, skill, and action in administering the treatment... The law requires a fair and reasonable standard of care and competence.

[78] This requirement raises the following question: Did Dr. Campbell bring to his task a reasonable degree of skill and knowledge, and did he exercise that degree of care and skill which could reasonably be expected of a normal, prudent practitioner of the same experience and standing? The answer does not engage a consideration of whether a specific act or omission constituted fault. The standard of care should not be measured by the result; "professionals have an obligation of

means, not an obligation of result": see *St-Jean c. Mercier*, 2002 SCC 15, [2002] 1 S.C.R. 491, at para. 53. Rather, the test is whether the defendant physician, Dr. Campbell, behaved similarly to a reasonably prudent and diligent fellow professional in the same circumstances: see *St.-Jean*, at para. 53, and *Hillis* v. *Meineri,* 2017 ONSC 2845, at para. 64.

[79] An erroneous diagnosis, on its own, does not determine the physician's liability. However, if the physician does not avail himself of the scientific means and facilities open to them to collect the best factual data upon which to arrive at his diagnosis, or if they do not accurately obtain the patient's history, they have acted negligently: see *Wade v. Sisters of Saint Joseph of the Diocese of London*, [1978] O.J. No. 413 (H.C.), at para. 22; *Boyd v. Eddington*, 2014 ONSC 1130, at para. 11, citing Ellen J. Picard & Gerald Robertson, *Legal Liability of Doctors and Hospitals in Canada,* 5th ed. (Thomson Reuters, 2017).

[80] Furthermore, information upon which a judgment or decision is reached must be as complete as is reasonably available and possible in the circumstances, including tests or consultations that should have been carried out but were not: see *Crawford,* at paras. 229 and 261(o). Clinical judgment is not guesswork based on limited facts. It must be exercised only after the medical practitioner has informed themselves of all the relevant facts, subject to any time constraints

imposed by the circumstances in each case: see *MacGregor* v. *Potts*, [2009] O.J. No. 3581 (S.C), at para. 128, aff'd in 2012 ONCA 226.

[81] A central component in a standard of care analysis concerns the process of arriving at a differential diagnosis. Courts have described this process as a universally accepted standard in the medical profession that seeks to identify possible medical perils in a patient and then proceeds to eliminate them by order of severity. Treatable and more serious conditions should rank ahead of other less conditions when considering a differential diagnosis. A key feature of differential diagnosis is eliminating the most serious possibility first, rather than the most probable. If doctors were to diagnose based on probability alone, then rare and severe ailments would regularly be ignored in favour of common, non-life-threatening alternatives. When faced with symptoms that point to two or more diseases, the universally acceptable system to use is a differential diagnosis that accounts for severity: see *Adair Estate* v. *Hamilton Health Sciences Corp.*, [2005] O.J. No. 2180 (S.C.); *Campbell v. Roberts*, 2014 ONSC 5922, at para. 14; and *Boyd v. Edington*, 2014 ONSC 1130, at para. 12.

[82] Defence counsel cautioned that differential diagnosis is a medical, not legal concept, and that any findings related to Dr. Campbell's differential diagnosis would have to be based on the expert evidence before the court. In light of the concern raised, I note that Drs. Brankston, Gladstone and Sayal defined the

process of arriving at a differential diagnosis in very detailed terms. Effectively, all three experts described the process of arriving at a differential diagnosis as something that was dynamic, and subject to reassessment and re-evaluation as the information they collected enabled them to rule-in or out probable causes. Although all three also spoke about probabilities, they agreed that it would be wrong to exclude something from the differential diagnosis from the outset simply because it is rare, unlikely, or improbable. They also agreed that a physician will consider all the possibilities, and then consider the history of the patient, their physical examination, and any investigations of that patient, to conclude, as a matter of clinical judgment, that a particular diagnosis is or is not the most likely explanation for what is happening to the patient before them. I conclude that Dr. Campbell's process of arriving at a differential diagnosis is one of the facts to consider to make my findings of fact. Based on those findings, I will answer the legal question of whether Dr. Campbell fell below the requisite standard of care.

[83] Finally, a determination of whether a physician met the standard of care should also not rely on the benefit of hindsight. It would be an error of law to employ an outcome-based approach to determine whether defendant physicians met the requisite standard of care. To apply an overly critical and retrospective judgment when assessing the exercise of a physician's clinical judgment would be unfair: see *Lapointe v. Hopital Le Gardeur*, [1992] 1 S.C.R. 351, at p. 362-363; *St-Jean*, at para. 53; *Bafaro v. Dowd,* [2008] O.J. No. 3474 (S.C.), aff'd. 2010 ONCA 188.

b. Evidence on Standard of Care

[84] The Court heard evidence on the standard of care, principally from Drs. Brankston and Sayal. Drs. Gladstone and Silver made passing references to standard of care in the context of neurology examinations; they were not qualified to speak to the standard of care for an Emergency Room physician.

i. Dr. Brankston

[85] Dr. Brankston testified that Dr. Campbell fell below the standard of care because he failed to consider the totality of Mr. Hasan's neurological symptoms that suggested that the location of his pathology was in the brainstem or posterior fossa of the brain. Following CT scan, Dr. Campbell failed to order additional imaging such as a CT angiogram of Mr. Hasan's head and neck. Alternatively, Dr. Campbell should have consulted the neurologist on call.

[86] Dr. Brankston explained that Dr. Campbell's clinical judgment was compromised by his failure to obtain a thorough patient history and a general and focused physical examination. Together, these deficiencies compromised his ability to formulate an accurate differential diagnosis and to exercise his clinical judgment. He said specifically, that "to exercise reasonable judgment, the emergency room physician needs to obtain a thorough physical examination that is directed toward the patient's symptoms or presentation. And then come up with a list of differential diagnoses to explain the patient's clinical presentation".

[87] As a result of the flawed history, Dr. Campbell did not learn that Mr. Hasan had seen his family doctor, Dr. Wahba, who assessed him as having an ataxic gait just prior to attending at Trillium on December 3, 2011. He also did not learn that Dr. Wahba thought that Mr. Hasan was having a stroke and, therefore, referred him specifically to Trillium because it was a Regional Stroke Centre. Finally, Dr. Campbell did not know that Mr. Hasan had difficulty ambulating or that he did not walk into the emergency room but required the assistance of a wheelchair, even though that information was also contained in the nurse's triage notes.

[88] Regarding Dr. Wahba's referral note and her recorded findings, Dr. Brankston agreed that the emergency physician would still have had to take their own history from the patient and then conduct their own physical assessment. However, he noted that a referral note from a family physician could provide helpful information. In this instance, Dr. Wahba recorded right facial weakness and vertigo, but she also recorded ataxia, a symptom that, as will be explained below, was especially crucial to arriving at a working diagnosis. In other words, the note explained the reason for Mr. Hasan's attendance at Emergency. Although Dr. Brankston could not explain why Dr. Campbell did not see the referral note, given its reference in the nurse's triage notes, in Dr. Brankston's view, Dr. Campbell should have been aware of the note and should have asked about.

[89] Dr. Campbell's incomplete history of Mr. Hasan fed directly into Dr. Campbell's incomplete physical examination, and specifically his failure to conduct a gait assessment for Mr. Hasan. Having failed to appreciate that Mr. Hasan presented with ataxia at the family doctor's office or that he had been unable to walk on his own when he arrived at the Trillium ED on December 3, 2011, Dr. Campbell did not conduct a complete and focused neurological examination. Stated simply, he did not determine whether Mr. Hasan's difficulty walking was the result of feeling dizzy or because of a central cause in the brain stem. Dr. Campbell admitted not conducting a gait assessment, agreed it was an oversight, and went further to agree that had he found Mr. Hasan to have an ataxic gait, he would have consulted the neurologist on call.

[90] Dr. Brankston was examined and cross-examined extensively on the meaning of ataxia. He was asked to differentiate between "unsteady walking", "ataxia", and "true ataxia". He explained that ataxia is a physical finding that could only be deduced with a basic gait assessment. That would involve asking a patient to walk back and forth to observe if the patient walked in a normal fashion, and then asking the patient to walk toe-to-toe to review the patient's tandem gait. He also said that a patient with an ataxic gait would have a broad-based gait that was unsteady and uncoordinated. He also explained that true ataxia occurs in lesions of the cerebellum and/or brainstem because they are in the posterior fossa.

[91] Dr. Brankston disagreed with the suggestion, put to him in cross-examination, that "true ataxia may be difficult to discern from the unsteadiness that occurs when a patient with significant vertigo tries to walk". He said that the only way to determine whether a patient presented with true ataxia or whether there is a gait abnormality such as ataxia, would be to conduct a gait assessment, something that Dr. Campbell did not do. This meant that Dr. Campbell's differential diagnosis was compromised and impacted his decision to discharge Mr. Hasan without ruling out stroke as a cause for his symptoms. It also impacted his assessment of vertigo and the right facial palsy as two separate peripheral causes for Mr. Hasan's symptoms, instead of seeing them as a central cause.

[92] To put his evidence in greater perspective, Dr. Brankston explained that when a patient presents with an acute onset of vertigo with or without nausea and vomiting, and with no other neurological symptoms or abnormal findings, it would be reasonable to diagnose the patient with a peripheral lesion or peripheral abnormality of the 8th cranial nerve. Similarly, where somebody experiences an acute onset of complete facial weakness one side of the face, referred to as a Bell's phenomenon, it would be reasonable to diagnose that patient with a peripheral lesion or peripheral abnormality of the 7th cranial nerve. However, when a patient presents with the contemporaneous onset of acute symptoms that are connected to two separate cranial nerves, that points to a central cause for the symptoms, meaning that there may be a lesion in the brainstem or posterior fossa,

resulting in a life-threatening situation that requires immediate attention. That assessment would be even more pronounced with the additional assessment of an ataxic gait.

[93] Relating this evidence to Mr. Hasan's situation, Dr. Brankston said that symptoms in both the 7th and 8th cranial would put the location of the lesion solidly within the patient's brainstem or posterior fossa area. The triangulation of Mr. Hasan's vertigo (8th cranial nerve), his right facial paralysis (7th cranial nerve), with his ataxia, served to reinforce Dr. Brankston's conclusion that Mr. Hasan was experiencing a lesion in his brainstem that was life-threatening and that required immediate attention.

[94] In Dr. Brankston's view, Dr. Campbell misdiagnosed Mr. Hasan's pathological situation, not because he was mistaken in the exercise of an honest and intelligent exercise of clinical judgment, but because he took an incomplete history and conducted an incomplete physical examination.

[95] Dr. Brankston explained that following the results of the CT scan on December 3, 2011, he would have wanted to see the immediate ordering of a CT angiogram, which would have clarified the exact etiology for Mr. Hasan's symptoms and led to a proper diagnosis. He explained that Mr. Hasan should not have been discharged but should have been kept at the hospital for observation. Then, a CT angiogram of his neck blood vessels and intracranial blood vessels

should have been taken, or at the very least, he should have been seen by the neurologist on call.

[96] When challenged on whether it would be reasonable for an emergency physician to defer to the opinion of a neurologist on whether a CT angiogram of the head and neck were warranted, Dr. Brankston said: "Certainly that's an alternative that is open to the emergency physician. If the emergency physician is not certain, or insecure with ordering a CT angio, then by all means defer to the opinion of, in a case like this, to the neurologist on call. But certainly, it should be one or the other, not neither." Without either that imaging, or a neurological consult, Dr. Campbell neither ruled-in nor ruled-out a potentially life-threatening diagnosis; he discharged Mr. Hasan on an incomplete assessment.

[97] In Dr. Branston's view, when Mr. Hasan returned to Trillium's Emergency the following day, with much more pronounced symptoms, the evidence suggesting that his pathological process was situated in the brainstem and/or posterior fossa was even more persuasive, and required immediate attention. The process should have been sped along, but it was not. Although Dr. Brankston thought that Dr. Campbell did not err in repeating the CT scan, he should have had talked to the radiologist, obtained a CT angiogram of Mr. Hasan's neck and head, and sought the immediate advice of a neurologist. By the time the neurologist was

called, Mr. Hasan was intubated. This made it very difficult for the neurologist to obtain a thorough history or conduct a neurological assessment.

ii. Dr. Sayal

[98] Dr. Sayal testified on behalf of Dr. Campbell. He explained that he tried to "put my feet in the shoes of the physician, think of the case as it presented, and see if the care met the expected standard" to arrive at his opinion. He identified eight elements to consider in the assessment of a doctor's conduct. He said that to provide care means to do an assessment that looks at: "the history, the physical exam, the diagnostic impression that that reveals, then any investigations that may be necessary, the course in the ED of reassessments that may be indicated, and then a diagnosis, a management plan and a disposition". He explained that each of these "pieces" gets assessed for an overall impression of the care that was provided and to try to compare that with the expected standard for a physician with similar training and practising in a similar situation.

[99] Dr. Sayal described the responsibilities of an emergency physician as trying to arrive at a diagnosis as to what might be going on with a patient and offering them an appropriate treatment. He indicated that sometimes the diagnoses are life-threatening and in such circumstances such a concern is "paramount in our thinking of diagnostic considerations".

[100] Dr. Sayal explained that in a patient's assessment "essentially 80 percent of the diagnoses are actually made on history, so understanding who the patient is that is experiencing these symptoms, understanding what it is that they are going through, helps us focus somewhat, though not exclusively, but it helps to narrow our focus as to what we think is going on". He explained that the physical exam serves to confirm what the history of the patient may be suggesting. In the absence of symptoms, it could point the emergency physician to a different direction. Investigations were described as supplementary to the first two steps, dictated by one's clinical judgment and the level of concern for what might be going on with a patient.

[101] Dr. Sayal also spoke of emergency medicine in terms of probabilities. He said that one needs to be comfortable with probabilities to work in emergency medicine. He acknowledged that ruling-in or ruling-out a diagnosis was a common phrase. However, he discourages residents from using those terms because ruling out "is a very very difficult thing to absolutely say something doesn't exist, because we can never be sure of anything in emergency medicine. So, it is a very difficult thing to absolutely rule out a diagnosis". Instead, what emergency doctors ought to be doing is managing probabilities.

[102] Dr. Sayal testified that he considered Dr. Campbell's assessment of Mr. Hasan to be very detailed and supported his conclusion that the symptoms

reflected a peripheral cause. In Dr. Sayal's view, the absence of weakness in the arms or legs, double-vision, difficulties swallowing, difficulties forming words, and vertical nystagmus, any one of which could tip the assessment in favour of a central cause for Mr. Hasan's symptoms, further supported Dr. Campbell's overall assessment that Mr. Hasan was not suffering from any life-threatening condition.

[103] Dr. Sayal downplayed the significance of Dr. Wahba's referral note, and the concerns raised there. He considered Mr. Hasan's initial visit to Milton Hospital far more significant and concluded that the ordering of a CT scan as an appropriate test to undertake. He did not believe that with a peripheral diagnosis it was necessary for Dr. Campbel to order a CT angiogram or consult a neurologist. In short, Dr. Sayal concluded that "in this case, a 40-year-old gentleman with no risk factors, no trauma, no reason to actually have a stroke of any kind, or central cause, then presents with what very much looks like a peripheral cause of his symptoms". He said that Mr. Hasan's vertigo and Bell's Palsy were highly suggesting of a peripheral cause, and that "in the absence of anything else that sort of changes that, the probabilities are exceedingly high that this is a peripheral cause of his symptoms". He also added that in 2011 CT angiograms were rarely ordered by emergency physicians. Although, they could be ordered by a neurologist.

[104] Dr. Sayal acknowledged that Dr. Campbell did not assess Mr. Hasan's ataxia and gait. However, he downplayed that concern and shifted the focus to his own understanding that Mr. Hasan was able to get to the hospital "somehow" and that he was able to leave "somehow", leading him to conclude that it was safe for Mr. Hasan to manage his symptoms at home and, therefore, it was reasonable to discharge him. Dr. Sayal acknowledged that if Mr. Hasan were unable to get up or if he were unable to walk, even with a peripheral cause, he would not be discharged but admitted to the medicine service.

[105] Ultimately, Dr. Sayal said that Mr. Hasan presented with a rare and atypical stroke that "actually fooled the stroke neurologist", who was still unable to figure things out. The implication of that response was that if a stroke neurologist could be fooled, then Dr. Campbell could not be faulted for failing to appreciate what was really going in with Mr. Hasan's developing symptoms. That, in Dr. Sayal's view strengthened his assessment.

[106] In cross-examination, Dr. Sayal conceded after some extensive back and forth that Dr. Campbell's history-taking was deficient in several ways. He agreed that a gait assessment could help differentiate between a peripheral and a central cause for vertigo; he agreed that such an assessment would have been very helpful and that it would not have been very difficult to ask Mr. Hasan to stand up

or to take a few steps. He also agreed that a proper history or relevant tests were important to arrive at a conclusion as to what was going on with Mr. Hasan.

[107] Dr. Sayal agreed that the occurrence of both a Bell's Palsy (7th cranial nerve lesion) and a vertigo (8th cranial nerve lesion), showing up together as two separate peripheral causes, was not a common presentation. However, he also agreed that they were linked in the brainstem. He qualified his response by suggesting that this was not something he would have known. Although Dr. Sayal ran through several of Dr. Campbell's negative findings to conclude that Mr. Hasan's symptoms pointed to a peripheral cause, he conceded that many of those indicators could also support a central cause. Ultimately, he agreed that the listing of symptoms did not assist in determining the existence of embolic strokes.

[108] On the concept of "ruling out", and whether Dr. Campbell ought to have ruled-out stroke before he discharged Mr. Hasan, Dr. Sayal retreated to concerns with the meaning of ruling-out a diagnosis. He agreed that Dr. Campbell did not rule out stroke definitively. He surmised that Dr. Campbell must have thought the likelihood of it to be very low since he discharged Mr. Hasan to his home.

[109] Turning to the morning of December 4, Dr. Sayal said that as an Emergency Room physician who saw a patient with Mr. Hasan's constellation of symptoms he would say "there is something going on and I am not sure where it is. It wouldn't be easy for me to localize where this is". He eventually agreed that

in the face of a potentially life-threatening situation, and if it were clear that that the problem was in the brainstem, it would be necessary to act as soon as possible. He also agreed that if he knew that the diagnosis was central vertigo, and the CT scan came back negative, he would seek an expert consultation with a neurologist.

c. Assessment of Expert Opinions

[110] Both experts were professional and exceptionally knowledgeable in their areas of expertise. Very significantly, they both agreed on the components to the standard of care analysis for an emergency physician. They disagreed over whether Dr. Campbell met those components. The defence urged me to reject Dr. Brankston's opinion as lacking in impartiality and containing an unconscious bias. The plaintiff challenged Dr. Sayal's impartiality and highlighted numerous reasons to question his reliability.

[111] In my review of the respective opinions, I found Dr. Brankston to be forthright, fair, and impartial. He agreed with reasonable propositions and was neither combative nor evasive. He did not approach his assessment with the benefit of hindsight and he readily acknowledged the risks that such an approach would pose to the integrity of his opinion. The fact that his professional trajectory was different from Dr. Sayal's was no reason to either reject or give less weight to his opinion, as suggested by the defence.

[112] I find it especially significant to note that when it came to applying the components of the applicable standard of care to Dr. Campbell, Dr. Brankston demonstrated a thorough understanding of Mr. Hasan's history and his symptoms, and he approached his analysis in a systematic and accessible manner, without any evidence of any unconscious bias. His explanation that the constellation of Mr. Hasan's symptoms, those being lesions in the 7th and 9th cranial nerves, combined with ataxia, led him to find a central cause for Mr. Hasan that was potentially life-threatening, was very convincing. In this regard, I found it significant that his assessment lined up with the guidance provided in *Rosen's Emergency Medicine,* (7th edition), ("*Rosen's*"). His opinion, that Mr. Hasan should not have been discharged but that Dr. Campbell should have ordered a CT angiogram, or alternatively consulted a neurologist immediately following the negative results of the CT scan was also entirely consistent with *Rosen's*.

[113] I expressly note that *Rosen's* was brought up by the defendant and was accepted by Dr. Campbell and other expert physicians who testified, as an authoritative text and as a reference guide. It was marked as Exhibit 38 in this trial. In Chapter 12, "Dizziness and Vertigo", *Rosen's* notes that a patient who presents with vertigo and other cranial nerve abnormalities that point to a central cause must be investigated by a thorough neurologic exam. Such an exam ought to include a gait assessment with CT or MRI imaging, especially when a patient gives a history suggesting ataxia. *Rosen's* also notes that if a CT or MRI excludes hemorrhage as

the source for a patient's symptoms there ought to be an immediate neurologic consultation, emergency angiography, and possibly anticoagulation. This specific direction was put to Dr. Brankston by the defence; he noted that save the reference to anticoagulation, it mirrored his opinion and he adopted it as part of his evidence.

[114] Against such corroboration, I see no reason to conclude that Dr. Brankston arrived at his opinion with either the benefit of hindsight or due to any influence from his experience writing other expert opinions for other plaintiffs, as suggested by the defence. To the contrary, his opinion reflected an expert who was knowledgeable, whose evidence lined up with the guidance contained in an authoritative reference guide, and who offered his opinion in accordance with an expert's duty to the court in a reliable manner.

[115] In contrast to Dr. Brankston, Dr. Sayal's approach to his evidence, his response to various questions in cross-examination, and the gradual but steady unravelling of his opinion left me with very serious concerns over his reliability as an impartial expert and his understanding of his obligations to the court. Although he and Dr. Brankston agreed on the requirements of the standard of care for an emergency physician, when it came to applying them to Dr. Campbell's conduct, Dr. Sayal eventually conceded that like Dr. Campbell, he actually did not have all the information to support his assessment, he misapprehended the chronology of

Mr. Hasan's history, and he speculated about why Dr. Campbell discharged Mr. Hasan on December 3.

[116] For example, even with his recognition that 80 percent of a diagnosis is made on the basis of one's history, Dr. Sayal misapprehended Mr. Hasan's history and his actual condition when he arrived at Trillium. He thought Mr. Hasan visited Milton Hospital two days prior to his attendance at Trillium, when in fact the two visits were approximately 12 hours apart. He also failed to appreciate that Mr. Hasan's ability to walk on December 3 was already compromised. He conceded that he did not actually know that Mr. Hasan required the assistance of his friends and a wheelchair to attend Trillium. When confronted with that discrepancy in his opinion, it was particularly astonishing that he would seek to discount this significant piece of information by saying that Mr. Hasan *must have walked in* "somehow", when ataxia, and Mr. Hasan's compromised gait was a front and centre concern for Dr. Wahba.

[117] The deficiencies in Dr. Sayal's own information were compounded by his resort to certain logical fallacies, designed to minimize Dr. Campbell's failings. As Dr. Sayal was pressed to explain the reasoning underlying his opinion, it became evident that he misapprehended his duty to the court and sought instead to persuade the court to give Dr. Campbell the benefit of the doubt. Thus, although Dr. Sayal agreed that Dr. Campbell:

i. did not record anything about Dr. Wahba's note;

ii. was unaware of the note's contents;

iii. did not make inquiries about it even though it was referenced in the nurse's triage notes;

iv. failed to conduct a gait assessment;

v. neither ruled-in or out a stroke; and

vi. did not seek input from a neurologist following the results of the CT-scan,

he dismissed each of those missteps as insignificant, and in some instances, in his explanations, he was less than forthright.

[118] For example, sidestepping the content of Dr. Wahba's note, when it came to his consideration of the implications that note, Dr. Sayal expressed a pronounced disdain for the opinions or notes from family physicians. He said that referral notes from family physicians were often inaccurate and were not nearly as significant as a patient going to an emergency room. "Ataxia" is also something that family physicians get wrong. But the practices of family physicians generally was not what was on trial. When Dr. Sayal was pressed to differentiate between his general comments about family physicians and Dr. Wahba's specific concerns, he eventually revealed that in his own information gathering, he could not recall seeing Dr. Wahba's note.

[119] Rather than be up front about not having reviewed or considered Dr. Wahba's note at all, Dr. Sayal sought to whitewash family physician notes

generally. In doing so, he created the misleading impression that he was aware of the note, when that was not the case. He also implied that family physician notes are not worth very much and therefore, Dr. Campbell's failure to see the note or seek it out, was not serious. It was only during his cross-examination that Dr. Sayal admitted that he had not actually seen Dr. Wahba's note. This realization raised two concerns about his testimony. Did he criticize family doctors generally to conceal his own failure to consider Dr. Wahba's note, to minimize Dr. Campbell's oversight, or a bit of both? On any explanation, his less than forthright engagement with this subject put his objectivity and reliability into question.

[120] The more troubling example of Dr. Sayal's deliberate attempt to minimize Dr. Campbell's missteps was captured in the way he sought to cast a doubt over the exercise of "ruling-in" or "ruling-out" in a differential diagnosis. Even though all the other experts talked about "ruling-in" or "ruling out" in their practices without any reservation, Dr. Sayal made a great fuss over them. When confronted with the observation that these were common concepts used by doctors, Dr. Sayal sought to clarify that what he meant to say was that one could not "rule out" *"absolutely"* or *"definitively"*. Except that nobody suggested that the standard of care required an emergency physician to rule-out a life-threatening situation with *"absolute"* or *"definitive"* certainty. Having regard for the admission that Dr. Campbell had neither ruled-in or ruled-out stroke prior to discharging Mr. Hasan, the only explanation for Dr. Sayal's pronounced resistance to those concepts was his hope to dilute the

significance of Dr. Campbell's inadequate assessment of Mr. Hasan before discharging him.

[121] The tell-tale of Dr. Sayal's overall approach surfaced when Dr. Sayal shifted away from the consequences of "ruling-in" or "ruling-out" to the observation that in a potentially life-threatening situation, "the probability needs to be low enough that we don't think there is reason to go further or investigate further". Applying to Dr. Campbell, Dr. Sayal suggested that Dr. Campbell's concern for stroke *must have been low enough* that he was comfortable discharging Mr. Hasan to his home. Knowing that Dr. Campbell had *not* ruled-out stroke before Mr. Hasan's discharge, Dr. Sayal replaced the concept of "ruling-out" with a "low enough concern" and then speculated on Dr. Campbell's thinking without any evidence to support such a conclusion.

[122] Before I leave this particular concern, I hasten to add that even if I were to accept Dr. Sayal's speculation that Dr. Campbell's concern would have been "low enough" to permit Mr. Hasan's discharge, that logic was flawed and incompatible with what would have been Dr. Campbell's original "high enough" concern to order a CT scan for the purposes of ruling out stroke. On Dr. Campbell's own evidence, he started down the path of ruling out stroke. He did not complete that path; he neither ruled stroke in or out. Dr. Sayal had no explanation on how or

why Dr. Campbell's original "high enough" concern became "low enough" to justify his discontinued assessment.

[123] The reality is that rather than offer a credible and reliable explanation for Dr. Campbell's omissions, Dr. Sayal's attempt to reformulate the requirement to "rule-out" with the "low enough concern", and the resulting weaknesses to that opinion, only underscored the significance of Dr. Campbell's omission. In his engagement with this particular subject, Dr. Sayal dealt a serious blow to his own credibility and reliability.

[124] Against these concerns, the defence's suggestion that the court prefer Dr. Sayal's opinion over Dr. Brankston's, opinion because of Dr. Sayal's superior academic credentials was rather rich. Having regard for the guidance offered in *Sit.,* at paras. 121-124, where Dr. Sayal agreed with Dr. Brankston, I accepted his evidence. Where Dr. Sayal either disagreed with Dr. Brankston or offered an alternative explanation, I preferred Dr. Brankston's opinion.

d. Findings on Standard of Care

[125] Having assessed the strengths and weaknesses of the noted experts, I turn to my findings on standard of care. On the totality of the evidence before me, and given the very substantial agreement between Drs. Sayal and Brankston on the critical components of a standard of care analysis for an emergency physician,

I conclude that Dr. Campbell fell below the standard of care of an emergency physician.

[126] To arrive at that conclusion, I begin by adopting the evidence by *both* experts that the standard of care for an emergency physician requires satisfaction of the following:

i. The standard of care for an emergency physician requires a very thorough history of a patient and a complete physical examination. To exercise clinical judgment, it is important to first have all the facts.

ii. It is critical to rule out a central cause of vertigo as an urgent and first priority, because central causes of vertigo can be life threatening, acute emergencies.

iii. Multiple cranial nerve abnormalities suggest a central cause of vertigo.

iv. A gait assessment is part of a neurological assessment and is as simple as asking a patient to walk a few steps. Although differentiating between general unsteadiness and ataxia requires careful attention to detail, an ataxic gait, if it exists, suggests that a patient is experiencing a central cause of vertigo.

v. If the diagnosis is central vertigo and there is a negative CT scan, the emergency room physician should consult with a neurologist.

[127] Applied to Dr. Campbell, Drs. Brankston and Sayal also agreed that:

i. Dr. Campbell did not have all the facts regarding Mr. Hasan's medical condition. He did not know that Mr. Hasan was seen by his family doctor prior to attending at Trillium, who recorded ataxia among his symptoms and who specifically sent him to Trillium because it was a Regional Stroke Centre because she was worried that he was having a stroke.

ii. Dr. Campbell, also did not know that Mr. Hasan had difficulty walking prior to his attendance at Trillium and required the support of his friends and a wheelchair.

iii. Dr. Brankston confirmed that, consistent with the principles of a differential diagnosis, it is supremely important to rule-in or rule-out the most life-threatening condition on a differential diagnosis. With the caveat that Dr. Sayal did not like to use the term "ruling-out" and preferred to speak in terms of probabilities, he agreed that anything life-threatening or a potentially life-threatening diagnosis is "certainly paramount in our thinking of diagnostic considerations".

iv. The standard of care in Mr. Hasan's circumstances required Dr. Campbell to conduct a gait assessment, which Dr. Campbell did not do.

[128] Building on these admissions, I find that Dr. Campbell's flawed history intake and his compromised physical examination cascaded into a sequence of cumulative incremental failures that collectively, caused him to fall below the required standard of care. He missed Dr. Wahba's note, he did not record stroke as a central cause, he did not complete his diagnostic assessment, and he did not seek the advice of a neurologist either on December 3 or first thing on December 4, prior to Mr. Hasan's collapse later that morning. These errors are based on the following findings.

[129] First, relying on Dr. Sayal's assessment that 80% of diagnoses are typically made on one's understanding of a patient's history, that are then confirmed by the physical examination, had Dr. Campbell inquired about the missing referral note, he would have obtained a better understanding of Mr. Hasan's history, and specifically, he would have learned of Dr. Wahba's concern that Mr. Hasan was ataxic and had difficulties walking. That information would have oriented his clinical judgment towards the need to undertake a complete

neurological assessment, including a gait assessment. I conclude on a balance of probabilities, these inquiries would have resulted in the inclusion of stroke as a central cause in Mr. Hasan's differential diagnosis.

[130] The incomplete history-taking is only part of the equation in Dr. Campbell's flawed standard of care. Even if Dr. Campbell were given the benefit of the doubt over Dr. Wahba's note, his explanation on why he did not undertake a gait assessment was unconvincing. He suggested that when people are dizzy, they may not be able to stand and there may be a risk that they may fall. But to begin with, there was no evidence that Mr. Hasan was asked to sit-up and refused to do so because of his dizziness. Secondly, the information before Dr. Campbell was that he got himself to the hospital without falling and without getting hurt. Common sense suggests that there were no pronounced indicators that Mr. Hasan would be unable to attempt the assessment. If there were a genuine concern that Mr. Hasan might fall, I would expect that either Dr. Campbell or somebody could be standing next to him to prevent him from falling. In any event, faced with the request to walk, it would then have been up to Mr. Hasan to demonstrate his limitations. Finally, and most significantly, I rely on Dr. Sayal's express admission that in Mr. Hasan's circumstances, Dr. Campbell should have undertaken a gait assessment.

[131] Before I review the additional failures in Dr. Campbell's standard of care, I pause to reject the defence's suggestion that the court engage with the differences between "ataxia" and "true ataxia" to import some doubt over Dr. Wahba's assessment of ataxia and to use that uncertainty to find in Dr. Campbell's favour. With respect, whether Dr. Wahba was correct in her assessment is beside the point. Her alert ought to have prompted Dr. Campbell to verify Mr. Hassan's gait. Separate and apart from the admission in the Agreed Statement of Facts that Dr. Wahba found Mr. Hassan's gait ataxic, this issue would not have arisen if Dr. Campbell had conducted the assessment. The absence of that assessment resulted in a gap in the evidence that was Dr. Campbell's own making. In the absence of any evidence that Dr. Wahba was mistaken or that her assessment was not borne out by the evidence, it would be wrong to rely on that gap to give Dr. Campbell the benefit of the doubt: see *Ghiassi (Litigation Gaurdian of) v. Singh*, 2018 ONCA 764, at paras. 25, 29; and *Goodwin v. Olupona,* 2013 ONCA 259, at paras. 72-74.

[132] My rejection of the defence's submission is further bolstered by the recognition that Dr. Silver relied on Mr. Hasan being ataxic to explain the cause and specifically the underlying mechanism to the gradual occlusion of Mr. Hassan's basilar artery. In the face of that evidence, I found it rather misleading for the defence to put Dr. Wahba's assessment into question to diminish the

significance of Dr. Campbell's failure to assess Mr. Hasan's gait, when Dr. Silver relied on that information to support his defence opinion.

[133] Turning to the additional failures, I accept the plaintiff's submission that Dr. Campbell should have included stroke as a central cause for Mr. Hasan's vertigo in his differential diagnosis. I find support for that conclusion in Dr. Brankston's explanation that cranial nerve abnormalities in both the 7th and the 8th cranial nerves would heighten the concern that the vertigo had a central cause. I prefer this evidence to Dr. Sayal, who seemed tentative over the specific subject and went as far as to suggest that this may not have been something he would have known.

[134] More significantly, the best support for this finding lies in Dr. Campbell's own evidence, which demonstrated his own robust understanding that both the 7th and 8th cranial nerves have nuclei in the brain stem, and the connection between vertigo and the 8th cranial nerve and facial palsy and the 7th cranial nerve. He agreed that if both those cranial nerves were affected, it would be entirely consistent with something happening in the brain stem or posterior fossa and, therefore, amount to a central cause. Given that understanding, Dr. Campbell offered no satisfactory explanation as to why he did not arrive at that conclusion and take the appropriate steps to respond to Mr. Hasan's situation. At the very least, being at a Regional Stroke Centre, Dr. Campbell had easy access to a

neurologist and could have consulted them to address any uncertainties. Where such a consultation may have caught the failure to conduct a gait assessment in a timely manner, Dr. Campbell's compromised clinical judgment made such an inquiry that much more remote.

[135] Following on the cascading set of errors, the most perplexing part of Dr. Campbell's assessment rested in the way he handled the results of Mr. Hasan's CT scan and the steps he did not take thereafter. Even with all the preceding missteps, Dr. Campbell's decision to include stroke as one of his concerns and his request for a CT scan to rule it out suggests that, at least for a short period of time, he had a concern. This would be consistent with Dr. Brankston's evidence that even if a physician did not actually document a differential diagnosis one could extrapolate the thinking from the management steps taken, be they diagnostic or therapeutic.

[136] The unfortunate reality is that Dr. Campbell started on that inquiry but did not conclude it. He agreed that he knew that a CT scan could rule-in a hemorrhagic stroke, but it could not rule-out an embolic stroke. Having set out to explore the possibility of a stroke, I have tremendous difficulty with Dr. Campbell's explanation that "ruling-out" meant little more than collecting information. For somebody as experienced and articulate as Dr. Campbell, this explanation made no sense, especially when compared to his answers on the same issue in his examination

for discovery where he simply agreed that he was seeking to rule-out stroke. As I reviewed and evaluated Dr. Campbell's testimony, I wondered whether he modified his own explanation of "ruling-out" stroke to better align himself with Dr. Sayal's views about the concept of "ruling-out". However, even on that theory, Dr. Campbell had no credible explanation for discharging Mr. Hasan midstream in the investigation and without any input from a neurologist. Nor was there any explanation on why, at the very least, Mr. Hasan would not have been kept in hospital for further observation.

[137] Dr. Sayal speculated that Dr. Campbell must have concluded that the probability of a stroke was low enough to discharge Mr. Hasan to his home. But as I already noted above, there was no evidence for such a conclusion. Further, this thought process would have been contrary to Dr. Campbell's express recognition for the need to rule out a central cause of vertigo as an urgent and first-priority. He offered no explanation on why he failed to apply that principle in his assessment of Mr. Hasan. Dr. Sayal agreed that if the diagnosis were central vertigo and the CT scan were negative, the emergency room physician should consult with a neurologist. Dr. Brankston explained that the emergency physician could consult a neurologist or order a CT angiogram but that it was not an option to do neither. Dr. Campbell did neither.

[138] The experts for both parties engaged with the questions on whether an emergency physician would have ordered a CT angiogram or sought the input of a neurologist in 2011. Dr. Sayal explained that, in 2011, it would have been rare for an emergency physician to request a CT angiogram but a consultation with neurologist or an internist at his hospital would have been the way to go. Dr. Brankston thought that both options would have been available. Dr. Sayal did not work at a Regional Stroke Centre; Dr. Brankston did. Clearly, their respective views were shaped by the respective experiences. The crucial take away is that Dr. Campbell should have continued his investigations by either consulting a neurologist or proceeding with a CT angiogram. On either scenario, the investigations would have confirmed an ataxic patient having a stroke, with the prospect of turning one's mind to understanding the cause and the potential treatment options. Had Dr. Campbell sought out such consultations, the preceding errors may have been erased in favour or immediate treatment for Mr. Hasan. Having failed to complete that assessment, Dr. Campbell magnified his errors and fell below the standard of care. I pause here to observe that to this point, any one of the noted errors could have been overcome with a timely neurological consultation. A gait assessment by the neurologist, additional imaging such as a CT angiogram, and even continued observations in hospital would have yielded additional information and set Mr. Hasan on a different diagnostic trajectory.

[139] Instead, Dr. Campbell continued to fall below the standard of care on the following day when Mr. Hasan returned to Trillium, this time by ambulance, paralyzed, and unable to do more than mumble. Although Mr. Hasan was clearly in crisis, Dr. Campbell's clinical judgment remained clouded. Given the expert opinions before the court, I accept that a repeat CT scan was not a wrong first step. However, given Mr. Hasan's cumulative history, Dr. Campbell should have consulted neurology immediately when Dr. Campbell saw Mr. Hasan, and not after two and half hours, when Mr. Hasan experienced respiratory distress and had to be intubated, making a neurology exam of limited value.

[140] In the result, on the evidence at trial, I find on a balance of probabilities that Dr. Campbell did not bring to his task a reasonable degree of skill and knowledge, and did not exercise that degree of care and skill which could reasonably be expected of a normal, prudent practitioner of the same experience and standing. In the result, Dr. Campbell fell below the standard of care in his medical care and treatment of Mr. Hasan on both December 3 and 4, 2011.

CAUSATION

a. Applicable Legal Principles

[141] Having found that Dr. Campbell fell below the standard of care, the legal inquiry moves to causation. Causation is established where the plaintiff proves, on a balance of probabilities, that the defendant caused or contributed to the injury at

issue. The test for causation is the "*but for*" test which is set out in *Clements v. Clements,* 2012 SCC 32, [2012] 2 S.C.R. 181, at para. 8:

> The test for showing causation is the "but for" test. The plaintiff must show on a balance of probabilities that "but for" the defendant's negligent act, the injury would not have occurred. Inherent in the phrase "but for" is the requirement that the defendant's negligence was necessary to bring about the injury – in other words that the injury would not have occurred without the defendant's negligence. This is a factual inquiry. If the plaintiff does not establish this on a balance of probabilities, having regard to all the evidence, her action against the defendant fails.

See also *Athey v. Lionati,* [1996] 3 S.C.R. 458, at paras. 13-17, *Uribe* v. *Tsandelis,* 2021 ONCA 377, at paras. 29-37.

[142] A causation inquiry involves a three-part sequential inquiry: see *Sacks v. Ross*, 2017 ONCA 773, 417 D.L.R. (4th) 387, at para. 47. The first step is to determine what likely happened. The second step is to consider *what would likely have happened* had the defendant not breached the standard of care. The third step is to allocate fault among negligent defendants. In this case there was only one defendant, rendering the third step unnecessary.

[143] The second step, as described in *Sacks*, at para. 48, leads to one of two pathways and outcomes:

> There are two possible outcomes to the trier of fact's imaginative reconstruction of reality at the second step. On the one hand, if the trier of fact draws the inference from the evidence that the plaintiff would likely have been injured in any event, regardless of what the defendant did or failed to do in breach of the standard of care, then the defendant did not cause the injury. On the other hand, if the trier of fact infers from the evidence that the plaintiff would not likely have been injured without the defendant's act or failure to act, then the "but for" test for causation is

> satisfied: but for the defendant's act or omission, the plaintiff would not have been injured. The defendant's fault, which justifies liability, has been established.

[144] When a defendant breaches the standard of care by an omission, the trier of fact must consider the situation as it existed the moment before the breach, and then imagine that the defendant took the action they ought to have taken, to determine whether doing so would have prevented or reduced the injury. "Even though this exercise is bounded significantly by the actual facts, it counts as 'factual' because the task is to consider how the events would have actually unfolded had the defendant taken the action he was obliged to take": see *Sacks*, at para. 47.

[145] In delayed medical diagnosis and treatment actions, a component of the "but for" analysis requires the plaintiff to prove that the delay caused or contributed to the unfavourable outcome. It is not enough to prove that adequate diagnosis and treatment would have led to a chance of avoiding the unfavourable outcome. The chance must go beyond the threshold of "more likely than not": see *Cottrelle v. Gerrard* (2003), 67 O.R. (3d) 737 (C.A.), at paras. 25-26.

[146] The law of negligence does not require scientific evidence of the precise contribution of the defendant's negligence made to the injury. The "but for" test should be applied in a robust, pragmatic, and common-sense fashion, and is essentially a practical question of facts best answered by ordinary common sense, rather than on scientific precision. Medical experts ordinarily determine causation

in terms of certainties. The law requires a lesser standard. See *Donleavy v. Ultramar Ltd.*, 2019 ONCA 687, at paras. 65 and 72; and *Beldycki Estate v. Jaipargas,* 2012 ONCA 537, 295 O.A.C. 100, at para. 39.

[147] The "robust and pragmatic approach" allows the Court to infer causation where the defendant's negligence prevents the plaintiff from demonstrating the link between the injury and its causation. The onus then shifts to the defendant(s) to rebut the inference. Any uncertainty must be resolved in favour of the Plaintiff: see *Adams* v. *Taylor,* 2012 ONSC 4208, at para. 49.

[148] Causation in law must be established on a balance of probabilities, considering all the evidence: factual, statistical, and that which the judge is entitled to presume. Where a fault presents a clear danger and such danger materializes, it may be reasonable to presume a causal link, unless there is a demonstration or indication to the contrary.

[149] Statistical evidence may be helpful in the analysis of causation, but it is not determinative. Where statistical evidence does not indicate causation on a balance of probabilities, causation in law may nonetheless exist where evidence in the case supports such a finding: see *Laferriere c. Lawson,* [1991] 1 S.C.R. 541 at pp. 605-607.

[150] These legal principles translate into the following three questions for consideration:

a. What was the cause of Mr. Hasan's stroke?

b. What would have occurred "but for" the breach of the standard of care by Dr. Campbell? and,

c. Based on what would have occurred, is it more likely than not that Mr. Hasan would have had a good outcome?

Before I turn to each of these questions to arrive at my findings of fact, I make the following overriding observations, that were pervasive in the consideration of all three questions.

[151] First, Dr. Campbell's breach of the standard of care meant that there was no CT angiogram and / or MRI imaging on December 3 and 4, resulting in an evidentiary gap. Apart from the CT scan, with its limited data, the experts did not have any imaging contemporaneous to the stroke progression. That meant they had to work backwards from the CT scans of December 3 and 4 and the CT angiogram and MRI of December 8, taken 5 days after Mr. Hasan exhibited the first clinical symptoms of his evolving injury, to draw inferences on the most likely cause of the stroke. This evidentiary gap was the result of the defendant's negligence. The defence cannot rely on the lack of evidence to support his or her defence: see *Giasi (Litigation Guardian of) v. Singh,* 2018 ONCA 764, at para. 29, and *Goodwin v. Olupana,* 2013 ONCA 259, 305 O.A.C. 245, para. 72-74.

[152] Secondly, the mistakes in Mr. Hasan's assessment did not end with Dr. Campbell's multiple missteps and the implications that followed. A new round of oversights and missed evidence permeated the defence expert opinions, putting their overall reliability into question. Dr. Silver admitted that he overlooked Mr. Hasan's left vertebral artery at C5/C6 when he reviewed the December 8 CT angiogram. He then tried to minimize this oversight by dismissing it as insignificant to his opinion. Dr. Krings admitted that he either overlooked or was unaware of Mr. Hasan's complete clinical presentation. His correlation of this theory to Mr. Hasan's symptomatology was therefore fundamentally flawed.

[153] Third, the experts who testified were all highly accomplished medical scientists in their respective areas of expertise. They presented with outstanding resumes and rich academic and clinical qualifications. They explained their opinions and the underlying science in very clear and accessible terms. It is no wonder that almost all the experts are teachers, and it is not difficult to see why most have received various teaching awards and are recognized as leaders in their respective areas of expertise. Awards and academic accomplishments may be relevant to the assessment of the qualifications of a proposed expert. However, they cannot compensate for flaws in an expert's apprehension of the evidence and the resulting analysis.

[154] Fourth, the plaintiff invited the court to find confirmation bias and unconscious bias in the opinions of Drs. Silver and Krings. Such a finding would disqualify their opinions out of hand and limit my analysis to an assessment of the plaintiff's evidence. Although I identify certain specific concerns that may be explained by the proposed bias, they do not rise to the level of disqualifying the experts out of hand. At the very least, their theoretical explanations and various diagrams contributed significantly to my overall understanding of the science.

Question 1: What was the cause of Mr. Hasan's stroke?

[155] On the totality of the evidence before the court, I conclude, on a balance of probabilities, that Mr. Hasan suffered an embolic stroke that originated with a dissecting aneurysm in the left vertebral artery, at the C5/C6 level. That lesion resulted in the formation of a clot that travelled up to the distal vertebral artery and lodged at the junction with the basilar artery. That caused the blood flow in the basilar artery to reverse, it slowed the flow in the vicinity of the embolus, causing new clot to form, which in turn occluded the proximal basilar artery, a portion of the right vertebral artery and the left vertebral artery at its junction. This sequence compromised the blood flow into certain critical vessels and caused gradual ischemic injury in the medulla, the lower pons, both cerebellar hemispheres and the vermis of the cerebellum. This finding is based on the opinions of Drs.

Gladstone, Mikulis and Cheung, and reflects my overall preference of their expert evidence over that of the defendant experts.

[156] The competing defence theory postulates that the most likely cause for Mr. Hasan's stroke was a perforator ischemia caused by a dissection, with an intramural hematoma that started extracranially at the C1/C2 or atlantal loop level and extended intradurally and up into the basilar artery in a spiralling manner that "knocked out" and occluded selected perforators.

[157] Between the two theories, I prefer the plaintiff's for several reasons. Before I undertake that analysis, it is important to outline the highlights of the various opinions, as they relate to what happened to Mr. Hasan. In doing so, I note that each expert testified between three and four days, and together produced almost 3500 transcript pages of evidence. They reviewed numerous MRI and CT angiogram images and relied on various demonstrative aids to illustrate their explanations. To be clear, the demonstrative aids, both the medical caricatures and one animation, were not admitted into evidence but were used as teaching tools that simplified complex concepts.

a. Plaintiff Expert Opinions

[158] Three experts testified on what happened to Mr. Hasan: Drs. Gladstone, Mikulis, and Cheung. The following are highlights of their testimony.

i. Dr. Gladstone

[159] Dr. Gladstone concluded that Mr. Hasan's stroke was caused by a blockage in his proximal basilar artery and the distal vertebral arteries, specifically at the vertebrobasilar junction, leading to a dissection in the left vertebral artery at the C5-C6 level. He testified that most likely a blood clot or multiple blood clots formed at the site of the dissection that dislodged, moved up, and got stuck at the bottom portion of the basilar artery and vertebrobasilar junction. Over time, the clot propagated with layering of additional clot on the edges, and it could propagate upwards, downwards, and little bit into the top part of the right vertebral artery, until it resulted in the complete occlusion of the proximal basilar artery and the distal vertebral arteries.

[160] The occlusion was limited to the lower part of the basilar artery. The upper part continued to fill with blood because of retrograde flow. Dr. Gladstone explained that Mr. Hasan maintained very good collateral circulation because his two posterior communicating arteries provided blood-flow top to bottom. This nourished the upper part of the brainstem and prevented the clot from extending upwards in the basilar artery. It may have also minimized the chance of clots dislodging at the top of the basilar artery to move upwards, such that flow was maintained through the collateral branches.

[161] According to Dr. Gladstone, Mr. Hasan's gradual symptoms reflected a stepwise pattern of deterioration from the night of December 2 until noon or so on December 4. The clinical presentation "made intuitive clinical sense because it corresponded with the pathophysiological activities in his brain as reflected in the imaging". The cranial nerves associated with Mr. Hasan's developing symptoms, those being, the vertigo and nausea, followed by facial paralysis, and then the intensifying weakness and paralysis in the left arm and leg, ataxia, and difficulties with hearing, have their nuclei in the lower part of the basilar artery, the pons.

[162] The facial nerve nucleus resides in the lower pons. If the right side of the pons were affected that could cause paralysis of the right side of the face. "If a larger area of the pons is affected on the right side, then in addition to the right face being affected, there can also be weakness and paralysis of the left arm and the left leg because the pathways cross and that patter is specific for a pontine lesion". This, according to Dr. Gladstone, is what occurred in Mr. Hasan's case. His symptoms progressed from vertigo and a facial paralysis to weakness and paralysis of his left arm and left leg. As the clot propagated and the blockage enlarged, individual end arteries or perforating arteries or circumferential arteries were individually picked off, resulting in gradual stroke injury and an intensification of Mr. Hasan's symptoms.

[163] By contrast, the cranial nerves associated with cognitive functions, which are situated in the upper part of the brainstem and whose blood flow was maintained and were not affected by the occlusion, corresponded with Mr. Hasan's intact cognitive skills. Mr. Hasan did not suffer any cognitive deficits. This outcome confirmed Dr. Gladstone's theory of the mechanism of Mr. Hasan's occlusion and the fact that his upper brainstem was not affected.

ii. Dr. Mikulis

[164] Dr. Mikulis concluded that Mr. Hasan suffered from an embolic stroke. Referencing Mr. Hasan's symptoms and the various imaging of his neck and head, he explained that a dissecting aneurysm in Mr. Hasan's left vertebral artery, at C5/C6 resulted in the formation of clot that travelled down the length of the vertebral artery to lodge at the junction of the vertebral arteries and the basilar artery. Once that occurred, it altered the blood flow characteristics, "reversing the flow in the basilar artery, slowing the flow in the vicinity of the embolus. New clot formed around the embolus, which occluded the proximal basilar artery and occluded the portion of the right vertebral artery between the PICA origin, and its junction with the left vertebral artery. There was also new clot that formed along the – most of the V3 length of the left vertebral artery." In the result, Dr. Mikulis concluded that "the entire system from the distal vertebral arteries and the proximal basilar artery suffered occlusion that prevented flow in critical vessels that led to

ischemic injury in the medulla, lower pons, and both cerebellar hemispheres and the vermis of the cerebellum."

[165] The essential elements to Dr. Mikulis' opinion rest on his interpretation of Mr. Hasan's imaging, his review of the CT scans of December 3 and 4, his review of the CT angiogram and MRI of December 8, and his interpretation of the "outpouching" in Mr. Hasan's left vertebral artery and vascular anatomy at the C5/C6 level.

[166] The CT scans of December 3 and 4 suggested to Dr. Mikulis that clot-formation was underway. He pointed to the higher attenuation, meaning the whiter than normal areas, in the vertebral arteries and the basilar artery to support that interpretation. Although he acknowledged that the higher attenuation could also be the result of a beam-hardening artifact, calcification in the bones, or embolic material, he favoured the interpretation of clot-formation. The other critical dimension to this imaging was that as of December 3 and 4, the attenuation of the cerebellum looked normal, meaning that he could not detect any infarcts, something that would change in the imaging of December 8. Dr. Mikulis explained that in the face of the December 3 and 4 findings, a CT angiogram would have been essential to obtain to understand what was going on.

[167] The CT angiogram and MRI imaging of December 8 confirmed ischemic injury affecting both cerebellar hemispheres and the medulla suggesting that both

posterior-inferior cerebral arteries (PICAs) and the basilar artery were being affected by local clot. Dr. Mikulis explained that he suspected clot, though the specific images of the injured areas depicted the results of the occlusion and not what caused the occlusion and the infarcts.

[168] Dr. Mikulis identified the source for the cause of Mr. Hasan's injury in his left vertebral artery, at the C5/C6, where a dissecting aneurysm occurred. Pointing to an "outpouching" in the left vertebral artery, at the C5/C6 level he identified it as the "smoking gun" to what caused Mr. Hasan's stroke. Its existence, together with the immediate narrowing of the artery above the outpouching and the visibility of a flap, suggested an acute dissection at that level. When such a dissection occurs, it exposes the constituent elements of the vessel wall, which are thrombogenic, to the bloodstream, making the area susceptible to clot-formation. Absent a resulting occlusion or disruption to the blood flow, the continuing blood flow allows clots to dislodge from the dissection and move with the flow up the artery until it cannot go any further. When that occurs, it will result in a totally different blood-pattern.

[169] Having explained how a clot may result from a dissection and then dislodge to move in the artery to another location, Dr. Mikulis tracked the blood flow in the imaging and the areas of high and low attenuation to show the clot's travel route and its progressive occlusive behaviour. He then contrasted the compromised blood flow below the basilar artery to the upper part of the basilar

artery to explain that the collateral blood flow vessels in the Circle of Willis, from the carotid arteries to the posterior cerebral arteries, maintained the blood flow in that upper part of the basilar artery.

[170] Foundational to Dr. Mikulis' opinion was his assessment of Mr. Hasan's vascular anatomy. According to Dr. Mikulis, the size of Mr. Hasan's vertebral arteries and his basilar artery were atypically small. That explained the path that the clot followed and why it lodged at the junction between the vertebral arteries and the basilar artery, instead of travelling up to the top of the basilar artery. Dr. Mikulis agreed that clots that embolize from a dissection typically move with the blood flow to the tip of the basilar artery and do not lodge at the proximal basilar artery (bottom of the basilar artery). However, in Mr. Hasan's case, with specific reference to Mr. Hasan's imaging, Dr. Mikulis pointed out how the vertebral arteries narrowed and were even smaller after they provided flow to the posterior-inferior cerebral arteries bilaterally than what they would be in the neck area. This narrowing led him to infer that the basilar artery would also be smaller than what would ordinarily be expected.

[171] The reduction in the caliber of Mr. Hasan's arteries at V4 meant that the junction between the vertebral arteries and the basilar artery became "an ideal point for the clot to lodge and obstruct the vertebral and the basilar arteries". Dr. Mikulis agreed that he could not actually take the measurements at the junction

because by the December 8 imaging the junction was fully occluded and presented as a dark area "over" the junction, making impossible to measure. However, with the measurements that he was able to take, he was confident that Mr. Hasan's atypical vascular anatomy explained why the embolus lodged at the junction and did not rise to the top of the basilar artery.

[172] Dr. Mikulis also observed that Mr. Hasan had a variant of collateral blood flow, which meant that as the blood flow from the basilar artery to the large posterior cerebral arteries and occipital lobes was compromised, the carotid arteries took over. This also meant that blood flowed from the carotids to the occipital lobes *via* the posterior communicating arteries. In the result, the posterior communicating arteries from the carotid arteries supplied retrograde blood flow to the posterior inferior cerebral arteries on both sides and to the top of the basilar artery. This significant phenomenon enabled Mr. Hasan to survive the occlusion.

[173] These specific characteristics led Dr. Mikulis to conclude that "the distal left vertebral artery could have occluded very distally after the posterior-inferior cerebellar artery or in the vicinity of it that then reduced the flow". That altered the flow dynamics in the posterior circulation at the junction in a very significant way. With the slowing down of the flow came a predisposition to clot formation.

[174] Dr. Mikulis also concluded that clots formed in the distal right vertebral artery. He attributed that to the change in the blood flow dynamics which made the

vertebral artery prone to developing retrograde clot formation at the junction, between the vertebral arteries, and causing a run-off at the posterior-inferior cerebellar artery.

[175] Dr. Mikulis was clear that what he described was his *theory* of what likely occurred to Mr. Hasan. A timely CT angiogram would have made it possible to see a greater part of the vertebral arteries and the proximal basilar artery and, therefore, take their measurements before their occlusion. Those measurements would have made it possible to verify his theory. In the absence of a timely CT angiogram, Dr. Mikulis was obliged to rely on the visible parts of the imaging, and the blood flow that was evident in the various imaging to arrive at the conclusion that a clot traveled from the C5/C6 dissection and lodged at the identified junction.

[176] In response to the defence's theory, Dr. Mikulis acknowledged that when he first considered what occurred, he had in his mind a differential diagnosis which was "basically between the presence of an embolus with growth of clot, versus a dissection". However, as he studied the available imaging, the evidence of the dissecting aneurysm at Mr. Hasan's C5/C6 level led him to favour the presence of an embolus with the growth of clot over a dissection at the distal vertebral artery. For a dissection to have caused Mr. Hasan's stroke, Dr. Mikulis said he would have wanted to see some evidence of a dissection, such as a dissecting flap, something

that he could not see. Vessel imaging would have also been helpful, but none was taken.

[177] Dr. Mikulis agreed that dissections at the V3/V4 level are common. However, in Mr. Hasan's situation, he could not ignore the visible evidence of an acute dissecting aneurysm at C5/C6 in favour of a competing theory, for which he could see no evidence. Dr. Mikulis acknowledged that the defence's proposed explanation was possible, but he described it as exceptionally rare. He explained that it was not something that he had ever seen in his own experience especially given the length and suggested size of the dissection.

iii. Dr. Cheung

[178] Like Drs. Gladstone and Mikulis, Dr. Cheung concluded that the cause of Mr. Hasan's stroke originated in a tear to his artery at the C5/C6 level which then caused a clot to form. He explained that the clot "broke off and embolized into his brain and blocked off his basilar artery causing a stroke."

[179] Dr. Cheung explained that his initial review of Mr. Hasan's CT scan of December 3 was blind, meaning that he did not consider any other information or history and it looked normal. He then looked at Mr. Hasan's history and, working retrospectively, he identified a hyperdense basilar artery, which he attributed to the existence of a thrombus. In his review of the December 4 CT scan, Dr. Cheung

observed that the left vertebral artery was closed and that the basilar artery was closed in its proximal part at about 50%. He also noted that the distal portion of the right vertebral artery was also closed.

[180] Dr. Cheung explained that on seeing a blockage, the differential diagnosis five possible theories: i. thromboembolus from the heart; ii. thromboembolus from lower down in the neck; iii. a dissection in the upper neck with thromboembolism or clot propagating from the upper neck into the basilar artery; iv. Some weird atherosclerotic lesion; or v. another lesion causing a dissection or causing a narrowing and then a clot. After his review of the December 8 MRI, Dr. Cheung concluded that Mr. Hasan suffered acute strokes predominantly in the brainstem and cerebellum. These corresponded with the occlusions that he saw. He also observed that only the proximal half of the basilar artery was occluded while the distal part remained open.

[181] Dr. Cheung proceeded to look for the cause of the blockage by searching for what he called the classic signs of an intracranial dissection. These would include asymmetry in the vessels, any ectasia, which would suggest the existence of a dilatation, or any enlargement of an artery which would indicate the existence of a pseudoaneurysm. Even with the MRI imaging showing nothing more than a blockage, Dr. Cheung looked to the blockage's shadow to see if he could detect any asymmetry, any ectasia, or any eccentricity. He did not find any such signs.

The blockage shadow looked normal. Working through the MRI imaging, Dr. Cheung pointed to the intracranial arteries and vessels and explained why to him the "the arteries looked fairly symmetric", "the vessels were fairly symmetric", they were "nice and smooth", and the outlines were also smooth.

[182] These results led him to doubt the theory of an intracranial dissection. He then looked below the intracranial area for an explanation. Looking at the CT angiogram of December 8, and specifically, at the C5/C6 level, he noted an abnormality in the left vertebral artery, 2 centimetres in length. He pointed to "a dilatation [or pseudoaneurysm] at the bottom" and several areas of narrowing throughout the artery. Focusing on that specific location, he explained how the artery wall was bumpy and not very dense. Above the dilatation he highlighted the narrowing of the artery. Below the dilatation, the artery had a normal caliber. The dilatation itself was larger than the normal size of the artery, measuring "about 4 by 5 millimetres". Looking at the overall image, Dr. Cheung observed: "So what we see is this alternating dilatation, narrowing, dilatation and then narrowing and then dilatation, and this is what we call a 'string of pearls' sign or a 'string and pearls' sign or 'pearl and string' sign, but it looks like a string of pearls." He then contrasted the left vertebral artery to the right to show how the right side, at the same level of the vertebral artery, was "nice and round and very dense". The left vertebral artery had a problem. The right vertebral artery did not.

[183] The "string of pearls" was the reason that Dr. Cheung concluded that the dissection at the C5/C6 was acute. He explained that one would not see dilatation and narrowing in chronic dissections. He also explained that said pattern was what allowed the clot to form. With endothelial damage in the artery, the artery narrows, causing the blood flow to slow down. The slowing down results in the hypercoagulability of the blood, making it easier for clots to form.

[184] Putting these findings together, Dr. Cheung concluded:

> "The findings are classic, meaning there are very few things that look like that. That is what that means. The differential is limited. Essentially, if you see that, you will call it a dissection and you will call it an acute dissection. There is a string of pearls. There is irregularity. It is at the right location at C5-6 where there is a fulcrum. There is a distal occlusion that is associated with dissections, so we want to look for clots that may have broken off. And that occlusion up high is indicative of a thromboembolus. So you put all those findings together, the diagnosis would be an acute C5-6 vertebral artery dissection on the left side with thromboembolism to the distal left vertebral artery and basilar artery."

[185] Dr. Cheung rejected Dr. Krings' theory that a "tram-track blooming artefact" seen on the MRI was evidence of a peripheral hematoma caused by a dissection in the left vertebral artery in the upper neck. "Peripheral" means "to one side". He explained that the existence of a dissection results in an asymmetry in the wall of an artery because the hematoma would be inside the artery wall. A peripheral hematoma would mean that it was located on the one side of the artery wall. The resulting image would be one black line with a thickened or irregular appearance, caused by the blood inside the wall and not two lines. In contrast to that expected image, Dr. Cheung explained that the specific tram-track image

reflected two parallel, "nice and straight" lines that conform with the shape of a clot inside an artery and not a hematoma in the wall of the artery. The white in between the two black lines reflected a blood clot.

[186] Dr. Cheung was asked to explain what one would see with a "spiralling dissection", another element in the defence theory. He explained that if a dissection were moving up and spiralling, one would see a black line or a track on one side of the wall and as it spiraled to the other side, the black line would be interrupted and seen on the other side. one would then see a black area on the other side. One would not see parallel black lines, as he could see in Mr. Hasan's imaging. Dr. Cheung used the analogy of a monorail to underscore the point that with a dissection there would be one line that was thick and eccentric, instead of two nice and smooth, perfectly shaped straight lines.

[187] Dr. Cheung also took issue with the resulting length of the dissection proposed by the defence. He stated that a dissection at the V3 level, as suggested by the defence, that then dissected all the way up to the basilar would be a very long way for a dissection to travel and was, therefore, very unlikely and improbable. In addition to being unable to find anything in the medical literature to support such a possibility, he explained that such a dissection, would require some flow and upward pressure to push out the tearing wall to the suggested length. But with a lesion at C5/C6, as in Mr. Hasan's case, the severe narrowing of the artery would

decrease the pressure and the blood flow. The addition of a second lesion at V3, as suggested by the defence, would only further decrease the pressure, making it impossible for the dissection to be pushed for the proposed distance; there would simply be virtually no blood flow or pressure to push out the lumen to result in such a large dissection.

b. Defendant Expert Opinions

i. Dr. Silver

[188] Dr. Silver concluded that Mr. Hasan's stroke was caused by a dissection in the left wall of his left vertebral artery that started at the third segment of the vertebral artery, just below the base of the skull at C2-3 and extended up into the basilar and down a little bit into the right distal vertebral artery. This caused multiple branch occlusions and a stepwise progressive stroke with increasing deficits from the multiple infarcts. He also noted the absence of infarcts in the top part of the basilar artery and explained that the upper branches of the basilar, the posterior cerebral, provided retrograde blood flow, leaving the upper brain stem intact.

[189] In his review of the CT scan of December 3, like Drs. Gladstone, Mikulis, and Cheung, Dr. Silver saw a hyperdense sign in the vicinity of the basilar that suggested to him the existence of a thrombus. He also agreed that a CT scan made it impossible to determine if the thrombus was in the lumen or the arterial

wall. In the MRI of December 8, he noted multiple acute infarcts involving the cerebellum, the right ventral pons, the left medial medulla, and the right cerebellum. He explained that the infarcts were located predominantly on the right side of the brain stem and the left side of the medulla, which is supplied by branches coming off the distal vertebral artery. He also noted that the infarcts were within the vascular territories of small side branches, also referred to as the perforating or penetrating arteries, that arise from the distal vertebral arteries and the basilar artery. Dr. Silver described the brain stem infarction as "patchy" which suggested to him that many of the perforating arteries were spared.

[190] Dr. Silver related Mr. Hasan's clinical progression of his symptoms to the various parts in his vasculature to demonstrate a stepwise progression in his deficits. With reference to Figures 7 and 8 of his evidence, Dr. Silver situated Mr. Hasan's first infarct in the right pontine, which is supplied by the paramedian basilar perforating artery, coming off the proximal basilar artery. He situated the last infarct in Mr. Hasan's left medial medulla, which gets its blood flow from the left vertebral artery, in the anterior spinal artery. He agreed that Mr. Hasan's first infarcts occurred higher up in his vasculature, while the last infarct was lower in the vasculature.

[191] Dr. Silver explained that this infarct pattern was typical of an intramural dissection, with blood spiralling up within the arterial wall, picking off some basilar

branches, skipping others. He noted that the stepwise progression in Mr. Hasan's symptoms fit with his theory because "we know as the dissection enters in the layers of the wall and extends up, it tends to spiral, it tends to go from one side to the other; this is very well known. So it fits nicely with that kind of a pattern." He also pointed to two black parallel lines in the basilar artery at the level of the pons, visible in the MRI of December 8, as further confirmation of a clot forming in the wall of the artery. He disagreed with Dr. Cheung's opinion that the two parallel lines were suggestive of an intraluminal clot.

[192] Dr. Silver used various images, certain diagrams, and a demonstrative video to show the progression of an intramural dissection that results in infarcts. With specific reference to the intramural dissection in Mr. Hasan's case, he noted that: "In this case, [the dissection], actually *extended up before it extended down* presumably, because we had involvement of the pons earlier." He then illustrated how the true lumen was not totally blocked, leaving some flow on the left side of the basilar artery and, therefore, allowing for the left branches to remain intact. In contrast, the right side of the basilar artery was blocked which had the effect of blocking the right branches supplying the right side of the pons.

[193] In cross-examination, Dr. Silver's original view that there were no infarcts in the left pons changed. He conceded that there were infarcts there but they were "really tiny and small" when compared to the "extensive infarction on the right side".

When confronted with his statement in his report, that "the left pons was not infarcted", Dr. Silver said that he meant that statement "in a relative sense" and with a comparative reference to the right side where the infarcts were extensive.

[194] Responding to the plaintiff's theory of an intraluminal thrombus as the cause for the occlusion, Dr. Silver testified that for such a theory to hold, there should have been extensive bilateral pontine infarction, which he said, he did not see. Furthermore, with an intraluminal clot, he expected to see fragments of the clot to be breaking loose to flow up to the top of the basilar artery, including the midbrain perforators, thalamo-perforators, and the posterior cerebral arteries. Since Mr. Hasan's MRI did not show any infarcts in those areas, He concluded that it was unlikely that there was a basilar intraluminal thrombus. He also noted that he could not explain why an embolus would lodge at the vertebral and basilar junction and not move up to the tip of the basilar artery.

[195] When, in cross-examination, Dr. Silver was asked to correlate his conclusion that the infarcts occurred from the top to the bottom of Mr. Hasan's vasculature, with his theory that the dissection progressed from the bottom of the left vertebral artery, then immediately to the top, and then back down, Dr. Silver varied his original evidence and stated that the progression of the dissection was not necessarily that simple. Seeking to qualify his original explanation, he said that dissections do not go up uniformly, but they spiral, leaving some segments open

and others blocked. There could therefore still be a profusion from the top of the vertebral artery even if the dissection was running into that segment. He also suggested that since in the average patient there is bilateral blood flow from both vertebral arteries into the spinal artery, the ischemia in the left medulla may not have occurred until the dissection extended down the opposite vertebral artery, knocking out the right supply to it.

[196] When challenged further, Dr. Silver repeated that it was not as simple as saying that the dissection went straight up. Rather, the dissection went in different directions, down one artery and up another, and skipped branches. At one point in the exchange, Dr. Silver suggested that the first symptoms were actually at the back of the pons, suggesting that it involved an occlusion in a longer circumferential branch as opposed to one in a direct perforator. He concluded by saying: "The bottom line is the dissection definitely involved the proximal basilar early on because we have symptoms coming from the proximal basilar early on." Later in his testimony, Dr. Silver said that he could not be certain about the timing of the occlusion in the right vertebral artery because Mr. Hasan did not exhibit symptoms relating to the right cerebellum. The imaging from December 8 indicated that "at some point in time there was a small infarct in the right cerebellum."

[197] Dr. Silver admitted that in his review of Mr. Hasan's imaging, and specifically the CT angiogram of December 8, he did not notice the dissection at

the C5/C6 level which he also referred to as a pseudoaneurysm. He acknowledged that he missed that dissection even though it was highlighted by the reports he received from the treating radiologist, Dr. Gladstone, and other doctors who were asked to review Mr. Hasan's file. Eventually, he came to recognize the dissection at C5/C6 and agreed that missing it amounted to an oversight on his part. However, he insisted that it did not cause him to change his theory of what occurred.

[198] Dr. Silver's offered various explanations why the finding at C5/C6 did not impact his decision evolved over the course of his testimony. He said that he would have reconsidered his opinion if there had been an occlusion at the top of the basilar artery instead of the mid-basilar location. He explained that the aneurysmal dilatation suggested that the dissection at C5/C6 was old. When pressed on the question of "how old", he suggested that it occurred "probably a bit earlier".

[199] To strengthen his explanation, Dr. Silver suggested that 25% of dissections were multiple, the implication being that the dissection at C5/C6 could co-exist with a dissection at the C1 (or V3) level. When challenged on that percentage, he eventually conceded that of the 25%, the majority of multiple dissections were in multiple vessels as opposed to multiple dissections in the same vessel. Although he resisted the suggestion that multiple dissections in the *same* vessel are *rare*, he agreed that this phenomenon occurred in "some" cases. Dr. Silver then suggested that maybe the dissection at the C5 level extended through

V4 all the way up past V3 and up to the mid-basilar but that, at the same time, the vertebral artery appeared completely normal all the way up until the C2 level when the dissection reappeared.

[200] Separate and apart from the potential relationship between the dissection at C5/C6 and the one at C2 (V3 level), Dr. Silver was asked to comment on the likelihood of a dissection extending extradurally from V3, winding its way intracranially and making it all the way up to the mid-basilar. Dr. Silver testified that the literature on the subject documented examples of intradural dissections that extended from the extracranial vertebral, resulting in a basilar occlusion. He eventually agreed that he had never seen a dissection that extended extradurally at V3, winding its way intracranially and *into* the proximal basilar artery himself.

ii. Dr. Krings

[201] Dr. Krings concluded that the most likely cause for Mr. Hasan's strokes was a perforator ischemia. The mechanism of the ischemia was a dissection with an intramural hematoma that led to perforator occlusion. The dissection started extracranially in the left vertebral artery, close to the atlantal loop, or the C1/C2 level, and extended intradurally into the basilar artery in a spiralling manner resulting in the occlusion of selected perforators. The spiralling "knocked out" selected perforators. Dr. Krings rejected the plaintiff's theory that the basilar artery was occluded by a clot. In his view, if that had occurred, it would have led to the

complete infarction of the pons. Instead, Mr. Hasan's infarcts were in disperse areas, "left, right, midline, paramedian on the right side, then on the left side."

[202] Using various images from the MRI and CT angiogram of December 8 and diagrams explaining the progression of a dissection and its impact on perforators, Dr. Krings reviewed the relationship between the basilar artery and the perforators to explain that if there were a clot in the basilar artery, it "would knock out all of the perforators." If perforators were only selectively knocked out, that would indicate perforator occlusions, as opposed to a basilar artery occlusion. Specifically, regarding perforators, he explained that they have a separate origin at the basilar artery and do not communicate with each other, meaning that they do not have a collateral blood supply. They each have a dedicated territory. These specific features mean that once a perforator is occluded, the damage to the brainstem will be done within 2-3 hours, the ischemia tolerance for the brainstem. The further implication of this is that once a perforator is occluded there is no opportunity to regain the function in the territory of the occluded perforator.

[203] Unlike Drs. Silver, Gladstone, Mikulis, and Cheung, Dr. Krings did not believe he saw any hyperdensity within the basilar artery in the CT scans of December 3 and 4. He attributed the hyperdensity to artefact. But even if he were to attribute the hyperdensity to clot, he did not believe that it would offer any clues

on to the pathological mechanism for the clot. There would therefore be no reason to reconsider his opinion.

[204] In his review of Mr. Hasan's December 8 imaging, Dr. Krings identified infarcts in the medulla, the cerebellum, and the pons, although he noted that the infarcts were not in the entire pons. Looking closely at the pons, he highlighted that only a selected number of perforators at different heights of the basilar artery were "knocked out" or infarcted. This to him reflected a classic infarct pattern for perforator ischemia.

[205] Like all the other experts, Dr. Krings agreed that only part of the basilar artery was involved in Mr. Hasan's injury. He also agreed that there were no areas of ischemia upstream in the basilar artery. However, in Dr. Krings' view, this was further evidence that the pathological mechanism of Mr. Hasan's strokes could not be attributed to a clot in the basilar artery. For the mechanism to have been embolic, he would have expected to see infarctions within the distal portion of the posterior circulation, or in other words, all the way to the top of the basilar artery.

[206] An additional feature in the imaging that, in Dr. Krings' view, underscored his theory of a dissection with an intramural hematoma, was the visibility of a flame-shaped tapering of the vertebral artery in the vicinity of the basilar artery. Pointing to specific cross-sectional MRI images, Dr. Krings explained that the crescent-shaped eccentric hypersignal was evidence of a mural hematoma. If there were a

clot in the basilar artery, the signal would be situated more centrally within it. In his view, the signal was clearly peripheral in its location. Although Dr. Krings considered this feature to be especially significant in support of a dissection in the left vertebral artery at the V3 level, he agreed that he did not say anything about it in his first four reports, and only highlighted it in his fifth report, just days before his testimony and several days into the trial.

[207] Finally, Dr. Krings referred to the asymmetric "tram-tracks" signal as further evidence of the mural hematoma. He described one of the tracks as "inferior" and the other as "superior", suggestive of a mural hematoma on one side of the artery.

[208] Dr. Krings was asked to discuss the lesion at C5/C6 and specifically, to explain why he made no mention of it in his first report. In his examination in chief, he said that he saw no need to mention it because he "did not feel it was the causative mechanism of the patient's stroke, given the overwhelming evidence, especially with the flame-shaped tapering of the vertebral artery at the level of the atlantal loop." In cross-examination, he conceded that he did not report a finding that was "potentially acute" and a potential source of clot. Eventually, he agreed that he should have mentioned the C5/C6 lesion. However, he insisted that he considered the abnormality at C5/C6, incidental and, therefore, irrelevant to the cause of the stroke. Accordingly, he omitted it in his report.

[209] Dr. Krings agreed that recirculation, meaning stagnation in the blood flow, could cause an enlargement, or outpouching, of an artery. Such a feature could give rise to the formation of a clot. However, he also added that these were extremely rare situations and outpouching, in and of itself, did not necessarily cause a clot. He agreed with Dr. Silver that a clot could also occur due to slow flow caused by a narrowing of the vessel wall from a dissection. He also agreed with both Drs. Silver and Mikulis that a clot may propagate or grow when blood flow slows or becomes stagnant around the original clot. He also agreed that when a clot propagates or grows, it may not do that in a uniform or systematic way.

c. Assessment of Expert Opinions and Corresponding Findings

[210] There are seven reasons to prefer the plaintiff's theory over the defendant's on what happened to Mr. Hasan. In my review of each of these reasons, I relate my findings and concerns to the competing evidence before the court.

1. Uncontested Evidence

[211] To begin with, the plaintiff's theory rests on several crucial uncontested facts. In light of the following facts, it would therefore be very difficult to prefer the defence theory over the plaintiff:

- Mr. Hasan's treating radiologist diagnosed Mr. Hasan with a highly suggestive left vertebral dissection at the C5-C6 level, with distal thromboembolism.

- The most common cause of an occluded artery, including the basilar artery, is embolus and is the most common cause of stroke when there is a vertebral artery dissection.

- Arterial dissections increase the risk of clot formation and subsequent distal embolism. Clot can form intraluminally because of an irregularity on the wall of the vessel and can also form due to slow flow caused by the narrowing of the vessel.

- A clot can lodge or stop in the proximal or mid-basilar artery. Such occurrences have been reported in the literature.

- A dissection at C5-C6 can cause a thromboembolism at V4 into the basilar artery.

- Clots do not propagate, (grow) in a uniform fashion. Clot propagation is a variable and unpredictable process.

- A stepwise clinical progression / deterioration of a patient, as occurred with Mr. Hasan can occur where a blood clot is propagating.

- Mr. Hasan's right cerebellar infarcts could only be due to clot embolization.

[212] In contrast to these uncontested facts, there was an absence of evidence on some of the critical components of the defence opinions that rendered the defence theory less probable than the plaintiff theory. In particular:

- In their very extensive experience and careers, *none* of the experts have ever seen a such a long dissection that started extracranially and extended intradurally and up to the mid-basilar artery. Although there were passing references to the existence of medical literature that documented dissections starting extracranially and crossing into the dural area and up to the basilar artery, no specific example documented a dissection into the basilar artery.

- *None* of the experts testified that they had ever treated a dissection that started at the V3 segment of the left vertebral artery and spiraled and extended into the mid-basilar artery and then came down into the right vertebral artery.

- A spiralling dissection from one side of the vessel to the other is not something that Dr. Gladstone has ever seen in his practice.
- In his 40-year career, Dr. Mikulis has not seen a spiraling dissection extend from C1 to the lower part of the basilar artery.
- Dr. Krings agreed with Drs. Cheung and Mikulis that it was unlikely that a patient would have more than one dissection at the same time and in the same vessel.

2. Objectivity and Comprehensiveness of the Experts

[213] The plaintiff's experts testified in an objective, forthright, and comprehensive manner. Each reviewed the imaging thoroughly and in great detail. They were impartial in their review and assessment of Mr. Hasan's imaging. They understood their obligations and genuinely tried to assist the court. They gave considered explanations for why they disagreed with competing theories, and they made concessions in cross-examination where that was warranted. Examples of this were reflected in the following specific evidence.

[214] Dr. Mikulis began with a differential diagnosis that had both an embolic clot and a dissection as a cause for Mr. Hasan's stroke. He began to favour an embolic clot over a dissection when he detected the dissecting aneurysm at the C5/C6 level. Very objectively, and without any combativeness in his explanation, Dr. Mikulis agreed that dissections at the V3/V4 level were common, but he could not ignore the visible evidence of an acute dissection aneurysm in Mr. Hasan's neck in favour of a theory for which he could see no evidence.

[215] Dr. Cheung began his investigation with five possible theories in mind. Absent the injury at the C5/C6 level, the differential diagnosis would include thromboembolus from the heart, thromboembolus from lower down in the neck, a dissection in the upper neck with thromboembolism, or clot propagating from the upperneck into the basilar artery. It could also be "some weird atherosclerotic lesion" or some other lesion causing a dissection or narrowing and then the clot. Dr. Cheung then explained why the investigation of such theories would require a full review of a patient's head and neck, and that inevitably lead the investigator to discover the dissecting aneurysm at the C5-C6 level. His explanation of these various theories was detailed, thorough and conveyed to the court a confidence in the depth of his knowledge.

[216] The defendant experts focused principally on their own theory and tended to be absolute in those views. Unlike the plaintiff experts, who were willing to engage with the competing defendant opinions, and explain the reasons for their differentiation, the defendant experts were largely dismissive of any competing theory. The most glaring example was Dr. Silver's candid admission that he did not bother to review Dr. Mikulis' opinion. He also came across as particularly entrenched and became unnecessarily combative with counsel, where a "yes", "no", "I agree", or "I disagree", would have been far more helpful to the court. His attitude introduced to the court an unnecessary source of doubt.

[217] In contrast to Dr. Silver, although Dr. Krings was fixated on his theory, I recognize that his engagement with counsel during his testimony was for the most part beyond reproach. Of all the experts, Dr. Krings was most responsive to the questions that were put to him. He remained focused throughout his testimony and made concessions where it was reasonable for him to do. Where he disagreed with a proposition, he did so elegantly, respectfully and offered an explanation for his view.

3. Defence's Critique of the Plaintiff Experts

[218] A further reason to prefer the plaintiff theory over the defence is that the concerns raised by the defence about the evidence given by Dr. Gladstone and Dr. Cheung were dubious and ill-founded. Although Dr. Gladstone tended to provide more details in his answers than what he was being asked to address, and was generally eager to educate the court, he was neither entrenched nor combative in his responses. For my purpose, what was key to my assessment of the reliability of his opinion was his thorough grasp of Mr. Hasan's evolving clinical presentation and his detailed correlation of those symptoms to the imaging.

[219] Regarding Dr. Cheung's evidence, I reject the defence submission that he brought no particular expertise on the cause of Mr. Hasan's stroke or added benefit to the Court. I also reject the suggestion that his testimony be given no weight because his career path diverged from that of the other experts because

he had fewer awards, recognitions, research, and publications than the defence's highly accomplished doctors. With the greatest respect, awards and publications do not produce opinions; experience produces opinions. The reliability of an opinion is to be evaluated on the strength of the evidence being assessed and the thoroughness of the scientific investigation.

[220] What Dr. Cheung may have lacked in awards and publications, he more than made up by the several thousands of imaging files of heads, necks, and spines that he reviews annually. Dr. Cheung demonstrated a thorough grasp of the imaging that he examined. He took the court through details that, absent his explanation, would have been difficult to understand, and complemented the imaging analysis of Drs. Gladstone and Mikulis by bringing the intricacies and nuances of Mr. Hasan's vasculature, reflected in the imaging, into sharper focus.

[221] The fact that Dr. Cheung corroborated the evidence of Drs. Gladstone and Mikulis on the key features of the plaintiff theory as to the cause of Mr. Hasan's stroke, is not a basis to dismiss his evidence as superfluous or unnecessary. Although some of his evidence overlapped with Dr. Mikulis' evidence, Dr Cheung's primary contribution lay in his detailed imaging review of the dissection at the C5/C6 level, the highlighting of the narrowing of the vertebral artery immediately above the visible outpouching at C5/C6, which he described as the string of pearls, his explanation of how a developing dissection would reduce the blood flow, the

consequences of the slowing blood flow on the thromboembolisms in the distal part of the vertebral artery, the ways that a clot could escape from a dissection, and the unlikelihood of an additional dissection, contemporaneously and above the dissection at C5/C6. Remarkably, neither Drs. Cheung nor Mikulis were challenged on their evidence on these observations and explanations.

4. Methodology of the Experts

[222] The plaintiff experts' methodology, as compared to that followed by the defendant experts was thorough and objective, and gave me greater confidence in the reliability of their opinions. The initial blind review undertaken by Drs. Mikulis and Cheung meant they identified characteristics and features in the imaging without any preconceived notions or theories of what might have occurred. Although it was not clear if Dr. Gladstone's study was entirely blind, the steps he took and the questions he asked as he went about his review of the imaging illustrated a step-by-step approach and an open-minded investigation. The matching up of the imaging with his thorough grasp of the trajectory of Mr. Hasan's evolving clinical symptoms resulted in a comprehensive opinion and a very probable explanation.

[223] The same could not be said about the defendant experts' approach to their investigation and their review of the imaging. Dr. Silver agreed that he developed a theory of what may have occurred, having regard for the location of

the infarcts as reflected in the imaging of December 8, and then sought to prove it. Consequently, Dr. Silver overlooked two critical features in Mr. Hasan's imaging: the C5/C6 outpouching and the existence of infarcts in the left pons. As I will elaborate below, these deficits rendered his opinion less reliable. Having articulated his theory, when, under cross-examination he was asked about specific details he became adversarial; he amplified answers to questions that strengthened his opinion and dismissed those questions that challenged his opinion as insignificant. At times, his attitude suggested confirmation bias and put me on high alert over the reliability of his opinion.

[224] Dr. Krings' methodology was also flawed. His review was not blind. At trial he said he could not recall the content of his exchange with Dr. Silver, but he did *not* dispute that Dr. Silver told him about Mr. Hasan's case and he, in turn, responded that the defendant's lawyer could retain him if he wanted his opinion. Dr. Silver admitted to sharing his theory of what occurred with Dr. Krings. Although nothing in this evidence suggested any deliberate attempt to collude on the evidence or any other improper purpose, I cannot preclude the possibility that the exchange between them may have predisposed Dr. Krings to viewing Mr. Hasan's case in a particular way and resulting in an unconscious or confirmation bias. That concern, combined with the recognition that he did not start his analysis with a blank slate in the way that Drs. Mikulis and Cheung did, heightened my vigilance for bias, something that even Dr. Krings agreed that he could not preclude.

5. C5/C6: The Smoking Gun

[225] The failure by the defendant experts to say anything about the dissection in the left vertebral artery at C5/C6 in their initial opinions, when numerous sources identified it as a feature in the CT angiogram was one of the more problematic aspects of their opinions. It meant that they either worked with incomplete information or chose to ignore it. Their explanations for such failure were not persuasive and instead raised further doubt over the reliability of their respective opinions on causation.

[226] In contrast, the plaintiff experts gave a comprehensive explanation as to why the dissection at C5/C6 was the source for clot formation that eventually resulted in the occlusion of Mr. Hasan's basilar artery. Dr. Mikulis, in particular, offered a thorough and persuasive explanation why the dissection at C5/C6 was the "smoking gun". When the defendant experts were asked to explain their oversight, their explanations were not persuasive. More problematic for this court was their concerted efforts to dismiss the C5/C6 lesion as historic and insignificant. In doing so, they came perilously close to putting into question their objectivity as experts.

[227] To appreciate the significance of this omission, it is necessary to situate the evidence concerning the C5-C6 dissection it its full context. I begin by noting that on the totality of the evidence, I was persuaded by Dr. Mikulis' characterisation

of the C5/C6 dissection as the "smoking gun" to Mr. Hasan's stroke. His evidence, corroborated principally by Dr. Cheung but also Dr. Gladstone, was thorough, comprehensive, and made common sense. His comparative illustration of a healthy and a diseased artery helped me appreciate the significance of the visible bulge in Mr. Hasan's CT angiogram.

[228] Dr. Cheung brought this explanation into greater focus as he explained the features of the narrowing of the left vertebral artery immediately above the bulge and explained the "string of pearls" concept. The diagrams that both Drs. Silver and Krings shared with the court to illustrate how dissections occur, how clots may form, and how they may break away from the dissection, were equally instructive. When combined with the uncontested evidence that:

a) clots may form once a dissection occurs;

b) clots may escape the dissection and move in the direction of the blood flow until they cannot go any further; and

c) the unanimous agreement that a clot is the most common cause of a basilar artery occlusion,

it made sense that a breakaway clot from the dissection at C5/C6 ought to be included in one's differential diagnosis to arrive at an explanation for the occlusion of Mr. Hasan's basilar artery.

[229] In keeping with that assessment, I expected the defence experts, to identify the existence of the dissection at the C5/C6 level. As I heard their evidence,

I remained open to an alternate explanation for the implications of a C5/C6 dissection, and whether or not it could be the "smoking gun", as Dr. Mikulis said. Instead, Dr. Silver readily admitted that he overlooked the C5/C6 dissection and the immediate characteristics surrounding it in his initial review. When challenged on its significance and whether his subsequent recognition of the dissection at C5/C6 impacted his initial opinion, Dr. Silver was dismissive and offered various contradictory explanations whose net effect was to weaken his own credibility.

[230] When I first heard Dr. Silver's explanation, I was prepared to give him the benefit of the doubt that his initial omission was an innocent oversight and I remained open to engaging with and accepting his competing theory. That disposition quickly faded in the face of the curious tension between Dr. Silver's admission that he missed the C5/C6 dissection but that his review of Mr. Hasan's CT angiogram, as well as the reports of Drs. Gladstone and Klimek were thorough. Although I cannot comment on Dr. Klimek's findings because his report was not in evidence, having been educated on the images that depicted the lesion or outpouching at C5/C6, I accept the plaintiff evidence that the C5/C6 dissection would have been front and center to the discerning and attentive eye and mind. It was not buried in a footnote or another obscure reference. An allegedly thorough review was simply incongruent with the allegedly innocent failure to see the C5/C6 dissection.

[231] Dr. Silver's dubious explanation for missing the C5/C6 dissection was compounded by his unconvincing and evolving explanation about why his eventual recognition of the C5/C6 dissection did not cause him to re-evaluate or change his initial opinion. First, he said that he would have reconsidered his original opinion *only if* the resulting occlusion was at the top of the basilar artery. He did not elaborate further, though he appeared to base that view on the typical features of a typical basilar artery occlusion and ignored Mr. Hasan's atypical presentation and the reasons for the clots lodging at the base of the basilar artery.

[232] When challenged on the impact of a C5/C6 dissection on his theory of an acute dissection at V3/V4, Dr. Silver suggested that it did not impact his opinion because he considered it historical and, as such, would not have affected the alleged dissection at the higher level. Had Dr. Silver stopped there, the only issue for consideration would have been to evaluate the strength of his interpretation of the C5/C6 dissection as "historic" and to compare it to the competing views. As a historic dissection, Dr. Silver's explanation made some sense and fit in with his view that the source for the basilar artery occlusion was a dissection above C5/C6, at the V3/V4 level. I could see how it would be very unlikely for a historic lesion to be relevant to the events of December 3-4, because, by definition, the active features of that historic dissection would have resolved and would not have impacted Mr. Hasan's blood flow.

[233] But Dr. Silver undermined his own explanation with a set of admissions and variations to his evidence. In cross-examination, his characterization of the dissection as "historic" morphed from one that "likely occurred in the past", to one that may have occurred the December 2, to an admission that the C5 dissection may have even occurred on December 3, and then finally going as far as to suggest that the dissection at C5/C6 occurred simultaneously with the alleged dissection at V3/V4; although, he then quickly added that he could not be certain.

[234] Finally, when Dr. Silver was confronted with the implication that a historic dissection suited his opinion while an acute dissection compromised it, he distanced himself altogether from his historic/acute distinction and argued instead that multiple dissections could co-exist in one's vessels. He suggested that 25% of reported cases in the medical literature involved multiple dissections in vessels. That last submission became very suspect when Dr. Silver had to admit that his statistical reference referred to multiple dissections in multiple vessels, as opposed to multiple dissections in the same vessel. The dubiousness of this alternate explanation became even more pronounced when Dr. Silver acknowledged that he could not refer to *any* reported case of multiple dissections in the same vessel and agreed that he had never seen such a phenomenon in his own practice.

[235] When I line up that concession with Dr. Mikulis' evidence that in his 40-year career he had never seen multiple dissections in the same vessel, I cannot

see any rational way of preferring Dr. Silver's explanation over Dr. Mikulis'. More significantly, the very fact that Dr. Silver knew that the 25% referred dissections in multiple vessels and not in one alone, but implied otherwise until he was challenged, put his credibility in serious doubt. Although I would like to believe that Dr. Silver's error was the result of being inattentive rather than any deliberate attempt to confuse or mislead, only he could address that concern. For my purposes, this concern gave me one more reason to question the reliability of his opinion.

[236] In addition to these various contradictions, rather ironically, Dr. Silver's video, which illustrated the progression of a dissection and showed that some force would have had to operate to push out the arterial lining, underscored the need to explain how a second dissection could occur at a higher level in the face of the disruption to the blood flow and blood pressure at the lower C5/C6 level. The need for an explanation was amplified further by the defence suggestion that the dissection at V3/V4 was very long dissection and went up and then down. Some force would have had to cause the injury. If the force were disrupted at C5/C6, what residual force would exist to cause the injury at V3/V4? In effect, the diagram served as the perfect illustration of Dr. Cheung's concerns about the effects of a dissection on the blood flow and amplified the need for a corresponding explanation. No such explanation was provided.

[237] In light of these questions, I accept the plaintiff's expert evidence that for the defence theory to hold, something would have had to accelerate the blood flow and pressure to push out the dissection beginning at V3/V4 for the length suggested by the defence. But given the agreement by all experts that a dissection slows blood flow and reduces blood pressure, the acceleration contemplated by the defence theory could not co-exist with the deceleration that the dissection at C5/C6 would have caused. Against such a tension, Dr. Silver's attempt to ignore the dissection at C5/C6 was all but fatal to the reliability and credibility of this part of his opinion.

[238] Turning to Dr. Krings, although he did not flip-flop in his views or try to distance himself from inconvenient contradictions, his engagement with the inconvenient fact of C5/C6 was to simply ignore it and pretend it was not significant, much like putting one's hands over their eyes to avoid an inconvenient fact. Thus, when confronted with the C5/C6 lesion, he sought to reframe the discourse by preferring to describe the lesion at C5/C6 as a fusiform dilatation or an ectasia, instead of calling it a dissection. Although there was some discussion about this distinction, it did not obscure the existence of an irregularity that required an explanation.

[239] Then, rather than engage with the question of whether or not the abnormality at C5/C6 was acute, Dr. Krings deflected the discussion towards the

sufficiency of the CT angiogram for the examination of the subject lesion. His suggestion that a cross-sectional imaging of that location would have been more helpful for the assessment of the abnormality than a CT angiogram may not have been ill-founded. However, his outright rejection of the CT angiogram imaging was tenuous, especially since Drs. Mikulis, Cheung and even Silver, were able to highlight a sufficient number of features to support their respective analyses.

[240] Leaving aside the issue of better imaging, given the uncontested evidence that a CT angiogram would have been the immediate test of choice that Mr. Hasan should have had on December 3-4, following the results of the CT-scan, it was curious that Dr. Krings would not refer to even a single image from Mr. Hasan's CT angiogram in his first report, much less mention any irregularity or abnormality at the C5/C6 level of the left vertebral artery, which would have been clearly visible. As I considered his evidence, I wondered whether he actually studied the CT angiogram of December 8. For somebody so knowledgeable and articulate, it made no sense that he would stay silent on the features in Mr. Hasan's December 8 CT angiogram, especially given the evidence of the other experts. At the very least, he could have explained why the observed irregularities were incidental or not relevant to his assessment. Dr. Krings' eventual agreement, that it would have been best if he had reported on the irregularity at C5/C6, while helpful, was compromised by a certain degree of sarcasm in his additional comment that doing so would have obviated the cross-examination on this point.

[241] Finally, Dr. Krings' eventual suggestion that the overwhelming evidence of a dissection with an intramural hematoma dissection simply eliminated any other explanation and rendered the dissection at C5/C6 "incidental" or "irrelevant" tended to be overstated and far from convincing. For starters, with the aid of all the images before the court and the very helpful orientation of those images by all the experts, the dissection at C5/C6 was just as prominent, if not more so than the suggested "flame" and the "tram-tracks". To say that the outpouching at C5/C6 was irrelevant while the other features were not, simply did not make sense.

[242] Also informing this conclusion is the recognition that the plaintiff experts, and Drs. Mikulis and Cheung in particular, had competing and very divergent explanations from Dr. Krings on how the "flame" and the "train-tracks" might be interpreted to support their opinions. Considered in isolation, the varying interpretations of these features were equally compelling, making it difficult to prefer Dr. Cheung's interpretation of the smooth parallel train tracks with clot in the middle, over Dr. Krings' suggested crescent shape with a mural hematoma. That realization only served to underscore the finding that the features described by Dr. Krings as overwhelming were anything but. The varying descriptions combined with the existence of the lesion at C5/C put into question Dr. Krings suggested explanation and made the evidence as Dr. Mikulis and Cheung that much more attractive. In short neither the "flame" nor the "train-tracks" could explain Dr. Krings' silence over Dr. Mikulis "smoking gun".

6. Mr. Hasan's Vasculature and the Size of Clot at the Junction

[243] The defendant experts' silence over Mr. Hasan's atypical anatomical configuration of the vertebral basilar system was another significant omission in their opinions. In my view, it underscored their less than comprehensive approach to their consideration of causation. Even if Mr. Hasan's atypical presentation may not have been immediately obvious, the defence experts chose to remain silent on the subject even after they would have had the opportunity to review the plaintiff opinions and to comment on that aspect of the plaintiff theory. This was especially problematic given the finding by the plaintiff experts that the dissection at C5/C6 combined with Mr. Hasan's atypical vasculature were the two critical components that explained the occlusion at the proximal basilar artery.

[244] In simple terms, as I understood the evidence, when a clot forms and breaks away, the embolus may vary in size from very small to very large. If it is smaller in diameter than the vessel through which it travels, it will be carried forward by the blood flow until it meets a restriction or obstacle. One such obstacle may be the narrowing of a vessel. As the vessel narrows, the clot will not be able to continue travelling in it but will lodge. Once it lodges, a clot may then propagate and take on an irregular shape.

[245] Having regard for this basic description, Drs. Mikulis and Cheung's conclusion, that Mr. Hasan's vertebral arteries and his basilar artery were much

smaller than those of the average person so that clot could not rise to the top or distal part of the basilar artery, was comprehensive, supported by the imaging, and made sense. With specific reference to Mr. Hasan's imaging, I accept Dr. Mikulis' explanation of how and why the reduction in the caliber of Mr. Hasan's arteries at V4 would make the junction between the vertebral arteries and the basilar artery smaller than what one would expect. Dr. Mikulis' elaboration on why the left vertebral artery became prone to retrograde clot formation at the junction between the two arteries and run-off at the posterior-inferior cerebellar artery and the formation of clot in the distal right vertebral artery was very persuasive.

[246] There was no dispute that in their reports, neither Drs. Silver nor Krings studied Mr. Hasan's vasculature to consider whether its anatomy had anything to do with the cause of his stroke. They also did not comment on, or even try to rebut the views of Drs. Mikulis or Cheung on this subject, even though they were available for their consideration. Dr. Silver admitted that he chose not to review Dr. Mikulis' reports. During his testimony, Dr. Krings suggested that for a clot to lodge in 2 mm vertebral arteries it would have had to be 3 mm large. Apart from that passing reference, he did not offer any other explanation or measurements. For example, he did not comment on the effects of clot propagation, the possibility of multiple clots piling on, and he certainly did not offer any evidence to refute any of Dr. Mikulis' assumptions and conclusions on this particular subject.

[247] The defence's attempt to diminish the significance of Mr. Hasan's arteries size to the overall opinion on causation, by getting Dr. Mikulis to admit in cross-examination that he could not see the junction, or take any specific measurements, was neither effective nor convincing. As a preliminary and overriding observation, none of the experts reviewing the imaging, and specifically the MRI of December 8, could see anything more than the occlusion of the proximal basilar artery. The junction was no more visible to Dr. Mikulis than the suggested dissection at V3/V4 was visible to the defendant experts. The experts had to rely on imaging that was taken five days following Mr. Hasan's initial symptoms instead of imaging contemporaneous to the symptoms, because none was taken. In the result, the experts had to rely on their expertise to draw inferences from what they *could* see, from Mr. Hasan's clinical records, and from the medical literature they chose to study. Dr. Mikulis drew an inference about the junction measurements from what he could see. It was for the defence, through its experts, to rebut that inference but they chose not to do so.

[248] The lack of timely imaging, because of the breach in the standard of care, is exactly the kind of evidentiary gap contemplated in cases such as *Adams,* at para. 49, *Giasi,* at para. 29; and *Goodwin,* at paras. 73-74. The defendant may not rely on such gaps to rebut the plaintiff's evidence. But more to the point, the defendant's criticism of Dr. Mikulis' measurements failed to consider everything that he *could* measure to arrive at his conclusion. His admission that he could not

see or measure the junction was far from fatal to his opinion. Dr. Mikulis provided the court with a very extensive and comprehensive review of the objective evidence he could see on the available imaging to support his theory. Effectively, he walked the court through "all of the roads" leading to the junction to highlight Mr. Hasan's atypical features. He identified the anatomical variant, that is the larger than normal posterior communicating arteries, and explained why in the result the basilar artery would be smaller. He also highlighted the vertebral arteries up until the posterior inferior cerebral arteries and underscored their narrowing, which he explained was caused by the change in the blood flow.

[249] Although Dr. Mikulis readily admitted that he could not see the distribution of the ischemic injury and could not visualize the junction, it stood to reason that if the vertebral arteries were smaller and were narrowing up until the point where one could no longer see them, they would remain small. There was no suggestion by anyone that suddenly the junction would be larger than what was suggested by Dr. Mikulis. In the result, his suggested inference was supported by objective evidence that made sense and I was left with no reason to doubt his evidence.

[250] As a final comment on this point, Dr. Mikulis' candid engagement with questions, especially, though not exclusively, on this subject, reinforced his credibility and reliability. He was neither combative nor reluctant to respond. He did not redirect or avoid the questions that were put to him. By situating the

occlusion in its broader context, he helped the court understand why the junction would be smaller if the arteries leading to the occlusion were also smaller than average, making the lodging of a clot more probable than might otherwise be the case.

[251] Although not essential to this specific issue, Dr. Cheung's opinion on the size of Mr. Hasan's arteries complemented Dr. Mikulis' evidence. His detailed descriptions of Mr. Hasan's vascular anatomy enriched the court's overall understanding of what was depicted in the imaging and its significance. Dr. Cheung's approach was systematic, thorough, and validated the treating radiologist's conclusion that Mr. Hasan's basilar artery had a small caliber. Dr. Cheung was not challenged on this evidence or his findings.

7. Clinical Symptoms and Infarct Correlations

[252] Both defendant experts failed to appreciate the progression of Mr. Hasan's symptoms. That put their respective opinions into serious doubt. Dr. Krings' misapprehension of the progression of Mr. Hasan's symptoms was fatal to his appreciation of what occurred and undermined his opinion on causation. Dr. Silver's explanation was not nearly as problematic as Dr. Krings', but he too demonstrated an incomplete appreciation of Mr. Hasan's infarcted areas. The differences in the respective understandings of Mr. Hasan's symptomatology

meant that the two defendant experts gave conflicting views on how the infarcts would have occurred following the suggested dissection at V3/V4.

[253] Dr. Krings told the court that it was of utmost importance to correlate a patient's imaging with their clinical presentation and status to arrive at a diagnosis. Having reviewed the available imaging, he then explained that in his review and understanding of Mr. Hasan's clinical presentation he noted "progressive *ascending* neurological deficits" that pointed to different areas of the brainstem "sequentially being knocked out". This in his view served to confirm his theory that the dissection, which started at the atlantal loop and spiraled upwards, caused the ascending neurologic injury contemporaneous with the dissection's upward movement.

[254] Except that, Dr. Krings' conclusion was beset by two flaws. First, in characterizing the hyperdensity in the CT scans of December 3 and 4 as "artefact", and not as "clot" as the other experts did, Dr. Krings foreclosed the consideration of a competing theory. Even if Dr. Krings did not detect the clot in his initial observations, his failure to comment on the competing theory once he reviewed the opinions of the other experts, reinforced the concern that Dr Krings' orientation in favour artefact was shaped by either confirmation or unconscious bias and limited his scope of inquiry.

[255] Second and more seriously, Dr. Krings misapprehended the progression of Mr. Hasan's neurological deficits. In short, Dr. Krings understood Mr. Hasan's symptoms in the reverse order than the way they occurred. Contrary to Dr. Krings' understanding, Mr. Hasan's injury did not ascend from V3/V4 upwards but it descended. Dr. Krings readily admitted that he was unaware of Mr. Hasan's right facial palsy on December 3. He conceded that he was unaware of Mr. Hasan's complaint of left hearing loss, left neck pain, and his ataxic gait all of which were symptoms that defined the descending direction in the progression of the neurological deficits from the basilar artery downwards.

[256] Dr. Silver's explanation and correlation between the movement of the dissection and progressing infarcts revealed a deliberate attempt to fit the clinical symptoms into his theory that a dissection caused the stroke. As he explained what was admittedly a very intricate subject, he ran into some contradictions in his own analysis. In his examination in chief, Dr. Silver explained that the first infarcts occurred higher up in his vasculature while the last infarct was lower. He also explained that the infarct pattern was typical of an intramural dissection, with blood spiralling up within the arterial wall, picking off some basilar branches and skipping others.

[257] In cross-examination, when challenged over the location of the infarcts, Dr. Silver's explanation evolved. He suggested that the progression of the

dissection was not necessarily as simple as moving uniformly upwards, but that the spiralling meant that the dissection went in different directions, down one artery, up another, and skipped branches. Following an extensive exchange over where Mr. Hasan's first symptoms were situated, Dr. Silver sought to shut-down the debate by saying: "The bottom line is the dissection definitely involved the proximal basilar early on because we have symptoms coming from the proximal basilar early on." Although I tried to overlook the combative exchanges between Dr. Silver and plaintiff's counsel, his evolving answers on the characteristics of the dissection and his revised view away from the original absolute view that there were "no infarcts in the left pons" in favour of a concession that there were infarcts in the left pons, though they were "really tiny and small" when compared to the "extensive infarction on the right side", did not inspire any confidence.

[258] In contrast to these concerns, Dr. Gladstone provided the court with the more persuasive and reliable explanation of what occurred to Mr. Hasan. Although at times he tended to be verbose and sought to give more detail than he was asked to give, he correlated Mr. Hasan's clinical stepwise progression to the very imaging and pathophysiology in a very careful, deliberate and accurate manner and demonstrated a thorough grasp of Mr. Hasan's symptomatology. That correlation captured the descending order in which Mr. Hasan's neurological deficits presented, making the plaintiff's theory of what happened most plausible and convincing.

Question 2: What would have occurred to Mr. Hasan "but for" the breach of the standard of care by Dr. Campbell?

[259] Before I go further, it is crucial to note that my findings on what caused Mr. Hasan's stroke is inextricably linked to the answers to both the "but for" question and the question on the likelihood of success. Had I accepted the defence's theory on the cause of Mr. Hasan's stroke my analysis would come to a conclusion and the plaintiff's claim would be dismissed because there was little dispute that it would have been very difficult to treat Mr. Hasan, much less to obtain a positive outcome. In the same vein, the experts agreed that on the plaintiff's theory, treatments would have been available. *But for* the breach in the standard of care, Mr. Hasan would have received some form of treatment. The defendants however were tentative on the extent of the available treatments as well as the likelihood of their success and the degree of Mr. Hasan's recovery. The analysis that follows is therefore grounded on the plaintiff's theory that Mr. Hasan's stroke was embolic.

[260] Turning to my answer to this question, I am satisfied on a balance of probabilities that *but for* Dr. Campbell's breach of the standard of care, Mr. Hasan would have been assessed by a neurologist at Trillium at some point between 4:30 pm on December 3 and the morning of December 4; he would have had a CT angiogram that would probably have identified the activity underway in his left

vertebral artery, and with that information, the specialists would be able to identify the appropriate options and timing for treatment.

[261] I arrive at this conclusion for the following reasons. First, Mr. Hasan would have been subjected to a fuller neurological assessment. Second, the treating physicians would have had a range of treatments to consider, and third, Mr. Hasan presented at Trillium early enough so that he could have received a timely medical intervention to allow for a successful outcome.

[262] On the totality of the evidence, there was no dispute that if there were no breach to the standard of care, the next step in Dr. Campbell's assessment would have been to order a CT angiogram or alternatively to consult a neurologist. In all likelihood, the neurologist would have obtained a CT angiogram immediately or he may have monitored the patient to identify the appropriate next steps and timing for further diagnostic imaging. The imaging results would have allowed the neurologist to evaluate the options for treatment and the timing of that treatment.

[263] As to the timing of the CT angiogram, Dr. Gladstone's very lively description of two possible scenarios that may have unfolded had Mr. Hasan received timely care. He underscored the sense of urgency. In the first scenario, the neurologist would have requested an immediate CT angiogram which would have revealed the occlusion underway and resulted in Mr. Hasan's immediate hospitalization in a closely monitored unit and the initiation of some form of

treatment. In the second scenario, the neurologist would admit Mr. Hasan for further evaluation, with a CT angiogram being requested later in the day or evening and treatment to follow to try to open his basilar blockage, extract the clot, and prevent the worsening of his situation. On either scenario, Mr. Hasan would be poised to receive treatment without delay.

[264] As to treatment options and their availability in 2011, this issue is relevant both to the *but for* analysis and the likelihood of success. The most helpful evidence on available treatments in 2011 was provided by Dr. Gladstone, who spoke generally about incrementally invasive options. He explained that the first line of treatment is typically aspirin, which is a mild blood thinner, although he did not believe that this would have assisted Mr. Hasan. A related option would have been to prescribe Heparin, a stronger blood thinner given through an intravenous infusion or injection and that aims to prevent the extension of the thrombosis or the dislodging of the blood clots. Heparin is used as a stroke preventative treatment to stabilize patients with critical blockages, like a near occlusion, where there is slow flow and a high risk for clotting.

[265] Dr. Gladstone did not comment on the potential effectiveness of Heparin for Mr. Hasan. Rather, he said that "the most effective and most direct treatment" that had the greatest chance to help Mr. Hasan would have been a recanalization

treatment, meaning opening up a blockage, which could be accomplished in different ways. He then outlined several such treatments for consideration.

[266] The first recanalization option would have been to administer a clot-dissolving drug, called a "tissue plasminogen activator" or tPA, intravenously in the ED or in the ICU. Mr. Hasan did not have any contraindications to the use of tPA. Subject to concerns about the timing for such a treatment, this would have been one approach to treatment.

[267] A second recanalization treatment available in 2011 "and arguably would have been the most effective and safest treatment for Mr. Hasan to have received" would have been to use a catheter to pull out the clot. This mechanical thrombectomy procedure is referred to as "endovascular thrombectomy" or EVT. Dr. Gladstone described a MERCI retriever device, akin to a corkscrew, that was in use in 2011 and would be inserted into and up the artery to pull out the clot. Other EVT interventions could involve vacuum suction and stent retrievers. Such a treatment could be safer than using tPA which could cause bleeding.

[268] A third recanalization option would be to inject a smaller dose of tPA directly into the clot through the tip of the catheter. He explained that sometimes such a procedure may be done as a combination approach where intra-arterial tPA and the mechanical extraction of the clot may be used in combination.

[269] In contrast to Dr. Gladstone, although the defence experts agreed that a stroke, caused by a thromboembolic occlusion could be treated, they were negatively disposed to the availability of treatment options, particularly in relation to EVT options. Their attempt at an explanation was not satisfactory. Leaving aside their concerns over the efficacy of such treatments, their evidence in chief left me with the impression that in 2011 the recanalization treatment modalities, including EVT options, would not have been available for consideration. Comments to the effect that EVT was not standard of care treatment, or that these were very dangerous procedures, suggested that in 2011 the viability of such treatments, separate and apart from the extent of their success, would not have even been considered.

[270] This negative disposition was not helpful to the court, particularly when in cross-examination, both Drs. Silver and Krings were obliged to concede that tPA *would* have been an option for consideration, as would the use of the MERCI device for a mechanical thrombectomy. For example, Dr. Krings agreed that he was performing EVT and tPA at Toronto Western Hospital outside of clinical trials *prior* to December 2011 and that clot removal with retrievable stents, mechanical disruption of thrombus, micro guide wires, and angioplasty were also available at the time.

[271] Like Dr. Gladstone, Dr. Cheung was of the view that treatments for Mr. Hasan could have included IV clot-busting therapy with tPA or a combination of drugs infused directly into the clot, and/or endovascular therapy. Alternatively, catheters and wires could be used to disrupt the clot mechanically, or the clot could be removed using various devices, including a MERCI device or a retrievable stent. Dr. Cheung's absence from the operating room for several years did cause me to pause before I accepted his evidence on this particular issue. Recognizing that Dr. Krings had the most current experience with interventions, I was especially cautious. That said, significant parts of Dr. Cheung's evidence coincided with the evidence of Drs. Gladstone and Krings. Accordingly, on this subject, I rely on Dr. Cheung's evidence only where it coincided with the evidence of the other experts.

[272] With the latter qualification in mind, I am satisfied that in 2011, the potential available treatments that the neurologist and the interventional neuroradiologist would have had at their disposal for *consideration* would have been the following:

- Aspirin and/or heparin as a first line of treatment;
- Intravenous (IV) administration of tPA;
- Intra-arterial administration of tPA whereby tPA would be administered directly into the clot; and / or
- Endovascular therapy, EVT, which would involve the mechanical removal of the clot using a retrieval device such as the MERCI retriever, and possible stents and wires.

[273] Per the parties' Agreed Statement of Facts, these treatments were available at Trillium in December 2011, and they were performed by the same interventional neuroradiologist, who continues to hold privileges at Trillium. Fourteen EVTs were conducted at Trillium between 2008 to 2011 as follows:

> In 2008, three (3) EVT procedures were performed;
> In 2009, five (5) EVT procedures were performed;
> In 2010, five (5) EVT procedures were performed; and
> In 2011, one (1) EVT procedure was performed.

[274] Considering the evidence by both Drs. Gladstone and Krings on how a neurologist and an interventional neuroradiologist would interact and collaborate to identify the best course of treatment for a patient, I am satisfied that *but for* Dr. Campbell's breach in the standard of care, such an interaction would have occurred, paving the way for the identification of a treatment plan for Mr. Hasan.

[275] I disagree with the defence proposition that Dr. Gladstone's opinion was focused only on EVT and that he did not provide a comprehensive opinion on how each of the other treatment options may have evolved. Dr. Gladstone mapped out the decision-making process that would be employed by a capable stroke neurologist in consultation with interventional neuroradiology to determine the appropriate approach to treatment decisions. He also outlined the treatments that

would be reasonable to consider and related each of them to the corresponding time windows for their success. He went as far as to explain that in his experience, he will often be in the suite adjacent to the procedure room watching the interventional neuroradiologist doing a procedure so that, if necessary, a procedure may be adjusted on the spot to respond to what the interventionalist might encounter. This demonstrated how dynamic each case may be and how it would be exceptionally difficult for anyone to foresee all the various treatment options and their associated intricacies, in advance of the actual procedure. In light of that recognition, it would be wrong to require any expert to map out further details and hypothetical scenarios.

[276] At the risk of being repetitive, it cannot be forgotten that other than the CT scans, which were of limited value for the determination of treatment options, none of the experts had the benefit of imaging contemporaneous to December 3 or 4. They all were obliged to project backwards from the damage seen in the MRI and CT angiogram five days after the injury to arrive at their conclusions. Apart from agreeing that no clot burden was visible on the CT scans of December 3 and 4, none of the experts could say *what* the neurologists would be looking at on December 3 in the afternoon, later that day and into the evening, or in the morning of December 4 to pronounce definitively on the appropriate intervention and its timing. It is, therefore, rich for the defendant to criticize Dr. Gladstone's opinion for not mapping out exactly how each treatment option would have unfolded. In my

view, with the paucity of evidence on Mr. Hasan's evolving condition on December 3 and 4, to require Dr. Gladstone to go beyond the description of the available treatments would be to require him to speculate. More to the point, the most significant takeaway from the evidence is that various treatment options were available for the treating physicians to consider and deploy.

[277] Finally, in response to the defence's argument that Dr. John's, failure to order a CT angiogram could be relied on by the court to question whether in fact a CT angiogram would ever have been ordered, that submission was misguided and unhelpful. Dr. John, a neurologist at Trillium, entered the picture in the afternoon on December 4, *after* Mr. Hasan collapsed and had to be intubated. She could not take any meaningful history from him, and what she did obtain was wrong. As noted by Dr. Sayal, Mr. Hasan's circumstances on December 3 and the morning of December 4, before his collapse, were very different from what they became later.

[278] Keeping in mind that Dr. John's involvement on December 4 was very different from what it might have been on December 3 or the morning of December 4, there is no evidence to suggest that Mr. Hasan would not have been treated. Such a conclusion would be inconsistent with even the defence agreement that in the period between the afternoon of December 3 and the morning of December 4,

Mr. Hasan had not suffered any infracts. It would therefore not have been too late for any treatment.

Question 3: Based on what would have occurred, is it more likely than not that Mr. Hasan would have had a good outcome?

[279] In my review of the evidence, I am satisfied on a balance of probabilities that had Mr. Hasan been attended to and treated in a timely manner, he would have had a successful recanalization and, therefore, a good outcome. As already noted, this finding is connected to my conclusion that Mr. Hasan's stroke was embolic and that the task at hand would have been to deal with the clot formation and clot removal to accomplish recanalization.

[280] On the evidence before me, I accept that if the cause for the stroke were a dissection at V3/V4, Mr. Hasan would have received the most conservative care, consisting of supportive medical care, designed to prevent secondary complications of stroke related to aspiration pneumonia, deep vein thrombosis from being immobilized, and urinary tract infections, as outlined by Dr. Silver. Although I am not entirely persuaded that nothing could be done to address Mr. Hasan's developing dissection, there was no evidence before the court to suggest any promising prospects.

[281] My conclusion, that one of the treatment options would have recanalized Mr. Hasan's arteries and resulted in a good outcome, meaning a Rankin score of

3 or less, rests principally on the evidence of Dr. Gladstone, although I also considered Dr. Cheung's evidence as well as the cautions raised by Drs. Silver and Krings. The specific evidence I considered for this question touched on a. Mr. Hasan's imaging and clinical presentation; b. Mr. Hasan's unique anatomical characteristics; and c. the recognition in the medical literature that there were a number of recanalization treatment modalities which were shown to be beneficial to patients.

a. Mr. Hasan's Imaging and Clinical Presentation

[282] Beginning with Mr. Hasan's imaging, given the overall evidence before me, I place significant weight on the uncontested evidence that Mr. Hasan's clot burden was unknown on December 3 and 4. As already noted, this evidentiary gap resulted from the defendant's failure to obtain a contemporaneous CT angiogram. That, combined with the unanimous evidence that no ischemia was detected on the CT scans of December 3 and 4, leads me to conclude that recanalization efforts would not have been futile. To the contrary, when considered together with his clinical presentation, I accept Dr. Gladstone's evidence that on those critical dates Mr. Hasan's condition was not severe. Therefore, it makes good sense that the prospects of a positive outcome following treatment were strong.

[283] I specifically accept Dr. Gladstone's explanation that the stepwise progression of Mr. Hasan's symptoms would have made it possible to successfully

treat Mr. Hasan's symptoms with early intervention. Given the significance of the view by all experts that "time is brain", Dr. Gladstone's direct correlation between Mr. Hasan's unique clinical presentation and the time-window for treatment was compelling and persuasive.

[284] I expressly highlight his extensive explanation offered in both his examination in chief and his cross on how the stepwise progression of symptoms and the corresponding stepwise growth of the clot substantially extended the window for treatment beyond the traditional view of a four-and-a-half-hour window from the onset of symptoms. Apart from the observation that the traditional view was not specific to basilar artery occlusions, his conclusion that the "sweet spot" for treatment would have been in the night, between December 3 and 4, when Mr. Hasan was "neither too mild to treat or too severe to treat" was very compelling and consistent with all the evidence before the court.

[285] Equally compelling was the way Dr. Gladstone framed the opportunities for treatment, with December 3 and December 4 being the two extreme points. His description of how Mr. Hasan might be observed by a neurologist to develop an appropriate treatment approach, prevent further damage, and possibly even reverse the damage to lessen the severity of any pre-treatment deficits was entirely consistent with the cautious approach to treatment options that Dr. Krings described for his patients.

[286] In preferring Dr. Gladstone's evidence on the prospects of a successful outcome over Drs. Silver and Krings' guarded, if not outright negative disposition, I rely first and foremost on Dr. Gladstone's robust understanding of Mr. Hasan's clinical progression and his reliance on that evidence to formulate his opinion. For the reasons already discussed, Dr. Krings' misapprehension of that progression inevitably compromised his understanding of Mr. Hasan's clinical presentation on December 3 and 4. That, in turn, undermined completely his opinion on Mr. Hasan's prospects. Without a clear understanding of "what" Dr. Krings would be treating or "how" such a treatment might proceed, I cannot place any weight on his conclusion that no treatment for Mr. Hasan could result in a positive outcome.

[287] In Dr. Silver's evidence, although he spoke of the stepwise progression of Mr. Hasan's symptoms for the reasons already discussed, his understanding of that progression had its own flaws. Moreover, he did not explain why he doubted the success of any recanalization treatment specifically for Mr. Hasan. To be fair to Dr. Silver, his focus on treatment options was connected to his underlying theory of what caused the stroke. He did not give any detailed evidence on how an embolic stroke could be treated. In cross-examination, Dr. Silver differentiated between successful recanalizations and successful outcomes. He referred to medical literature to support the contention that the former was not equivalent to the latter and to explain that if the damage were too far gone, recanalization could not reverse damage to infarcted areas.

[288] I had no difficulty understanding the distinction and I do think that it was a fair one to make. However, in my consideration of this issue, the critical evidence concerned the observation and agreement by all that there was no evidence to support a finding that on December 3 and the morning of December 4, Mr. Hasan was too far gone or that he had already suffered irreversible damage, to support the conclusion that recanalization would have failed. The experts agreed that there was no clot burden on December 3. Having regard for Dr. Gladstone's grasp of Mr. Hasan's clinical progression, his discussion of the appropriate timeframe for treatment, as compared to the flaws in the defence opinion, was compelling and persuasive, giving me the confidence to conclude that had Mr. Hasan been treated in a timely manner, the recanalization would have succeeded, and he would have had a successful outcome and recovery.

[289] Dr. Krings' elaborate explanation on the mechanisms of the various treatments, the challenges with EVT, the limitations of MERCI devices, particularly back in 2011, and the very delicate manoeuvres that must be considered when using catheters and undertaking thrombectomies was thorough and reliable. However, Dr. Krings made the treatments sound so impossible that if I were to accept his evidence, the logical extension of that would be to conclude that difficult cases would not have been treated because they could never result in successful outcomes. Such a conclusion would simply not make sense and was unsupported by the evidence. Specifically in relation to Mr. Hasan, there was nothing in the

evidence to support the automatic conclusion that EVT could not be used to treat Mr. Hasan successfully.

[290] The more pronounced difficulty with the opinions of Drs. Silver and Krings is that they relied on the MRI of December 8 and projected backwards, to speculate on how far-gone Mr. Hasan's injury must have been on December 3 and 4. That projection was tainted by their flawed understanding of the progression of Mr. Hasan's symptoms. They therefore did not have any actual evidence of how far-gone Mr. Hasan was on December 3 or on the morning of December 4. The MRI of December 8 recorded the results of the untreated injury *five and a half days after the initial symptoms*. It could not offer any evidence on the progression or propagation of clot formation. In addition, there was no available evidence to track the progression of the dissection at C5/C6. For example, would the outpouching have been as pronounced on December 3 or 4, as it was on December 8, and what implications would that have had on the approach to recanalization? Or would the flap in the lining, seen by Dr. Mikulis, been as pronounced on December 3 or 4 to possibly prohibit intervention with a MERCI device, as Dr. Krings suggested? In the absence of answers to these and other related considerations, I prefer Dr. Gladstone's approach to the identification of treatment options, his open-mindedness, and his evidence on why the proposed treatments were viable options over the limited evidence of over Drs. Krings and Silver.

b. Mr. Hasan's Unique Characteristics

[291] In addition to Mr. Hasan's clinical presentation, I accept Dr. Gladstone's assessment that Mr. Hasan had unique and good prognostic features to support a successful recanalization and a positive recovery. For example, Mr. Hasan was not burdened by other medical conditions that could impede a successful recovery. He was only 40 years old, and he did not have any pre-existing serious health conditions. In addition, his atypical vasculature ensured good collateral blood flow from his posterior communicating arteries and only part of the basilar artery was occluded. These features, combined with his initial mild clinical symptoms and a Posterior Circulation Aspect score exceeding 8, were good prognostic factors for a good recovery.

[292] Dr. Gladstone's explanation that a neurologist, working in close association with his interventional neuroradiologist, would design an appropriate strategy to treat Mr. Hasan was comprehensive, compelling, and consistent with the approach that Dr. Krings described. Considering Mr. Hasan's characteristics, Dr. Gladstone's conclusion that Mr. Hasan was a case to be as "aggressive as can be" in terms of "offering him the maximum medical therapy that has a chance to help open up the blockage", reflected a robust analysis. Most compelling was Dr. Gladstone's express recognition that when considering a range of treatment options,

> "we don't do this for everybody with a stroke, and I don't want my comments to be misinterpreted. We don't do it for most patients with stroke, to be honest, but when you have these set of circumstances, this the type of patient that you would want to be aggressive with and, you know do everything possible to give him the best fighting chance."

To me, this demonstrated caution but also a very considered analysis, responsive to Mr. Hasan's specific situation.

[293] In contrast to Dr. Gladstone, neither Drs. Silver nor Krings referenced Mr. Hasan's positive prognostic factors in their contrasting opinion on the prospects of successful treatment. All the experts demonstrated extreme caution and admitted to the challenges with basilar artery occlusions. But they parted ways on the implications of those concerns on Mr. Hasan's circumstances. Against that recognition, and having regard for the evidence before me, I am satisfied that on a balance of probabilities, if treated, Mr. Hasan would have had a positive outcome.

c. Medical Literature on Recanalization and Successful Outcomes

[294] The medical literature presented to the court supports Dr. Gladstone's opinion that Mr. Hasan's several unique characteristics, together with the availability of various recanalization treatment modalities (tPA and EVT), would have resulted in a successful outcome. To put this part of the analysis in its proper context, although much time was spent on the presentation of medical literature to support specific propositions, distilled to their essence, the differences of opinion

between Drs. Gladstone, Silver, and Krings did not lie in any competing interpretations of that literature or varying views about how successful a recanalization treatment might be. Rather, their differences lay over whether Mr. Hasan's condition was too far gone for *any* treatment to be successful. The consideration of this question therefore had more to do with the experts' appreciation of Mr. Hasan's clinical condition, discussed in the preceding section and to what extent his profile aligned, (or not), with the profiles recorded in the various medical studies.

[295] That said, given Dr. Gladstone's reliance on certain medical articles to form his opinion and the extensive cross-examination concerning his conclusions, I find it necessary to make the following observations. All the experts agreed that a successful outcome would depend on a successful recanalization and the minimization, if not the elimination of Mr. Hasan's deficits. They also agreed that recanalization treatment modalities had a greater than 50% success rate and that in 2011 the use of the MERCI had a success rate between 60-70%. That left the following question for consideration: Were Mr. Hasan's deficits on December 3 and the morning of December 4 too far gone to allow for their minimization, if not their reversal?

[296] In response to this last question, Dr. Gladstone's evidence was far more persuasive than the evidence of his defence colleagues. Having regard for all the

evidence before the court, I accept Dr. Gladstone's assessment that Mr. Hasan did not have severe deficits at the time he should have been treated. In light of that finding, I also accept Dr. Gladstone's conclusion on a balance of probabilities that recanalization using one of the available treatment modalities, (tPA or EVT), would have been successful. Dr. Gladstone was very deliberate and diligent in the way he distinguished Mr. Hasan from the various profiles in the studies. In his conclusion that Mr. Hasan had much to gain from a timely treatment he was very convincing. Contrary to the suggestion by the defence that he exaggerated the prospect of success and that he cherry-picked from the medical literature, in my review of the medical literature, I was satisfied that the concerns raised by the defence were unfounded.

[297] I recognize that the defence experts engaged with the various studies and percentages of success. However, they did not relate that data to Mr. Hasan's specific clinical presentation. Dr. Krings seemed to agree that Mr. Hasan did not have severe deficits on December 3 and 4. It therefore did not make sense that he would be so guarded over Mr. Hasan's prospects. Dr. Silver was keen to point out that many patients in the recanalization studies did not have successful outcomes, implying that one could not conclude on a balance of probabilities that treatment for Mr. Hasan would have been successful. But he was not as keen to compare or relate the medical profiles or clinical burdens of those patients to Mr. Hasan's specific condition and overall presentation. Albeit reluctantly, he eventually agreed

that 73.8% of patients with a pre-aspect score exceeding 8 achieved a good outcome following recanalization. It is undisputed that Mr. Hasan's score exceeded 8 on December 3.

[298] Complementing these findings is the very compelling submission by Mr. Hasan's counsel that nothing in the medical literature supported the view that if Mr. Hasan had received recanalization treatment in a timely manner and before his deficits progressed, his condition would have worsened. This observation is especially critical because all the experts agreed that Mr. Hasan was not too far gone on December 3 and the morning of December 4. Time was still on Mr. Hasan's side. This finding, together with the preceding findings lead me to conclude on a balance of probabilities that with timely intervention Mr. Hasan would have had a very positive outcome. Even if the mild deficits could not be reversed, they would not have progressed. On the evidence before the court, such an outcome would result in a Level 1 score on the Rankin scale, meaning minimal deficits.

DISCOUNT ON DAMAGES

DISCOUNT: If liability is established, should damages be reduced to reflect Mr. Hasan's "but for" the incident position and, if so, by what percentage.

[299] There was no evidence before the court to be able to engage meaningfully with this issue. Dr. Campbell asks this court to reduce the damages already agreed

to in advance of the trial on a 100% global basis by 25% to reflect the difference between Mr. Hasan's current condition and the condition he would have reached, but for the negligence.

[300] The defence based this submission on the essential and most basic principle of tort law outlined in *Athey v. Leonati*, [1996] 3 S.C.R. 458, at para. 32, which holds that a plaintiff must be placed in the position that they would have been absent the defendant's negligence but they must not be placed in a position better than their original position. Counsel then argued that per Dr. Gladstone,

a) Mr. Hasan's condition was at 75% when he entered the reasonable treatment window;

b) treatment for Mr. Hasan would more likely have prevented further deteriorating but would not have reversed the damage already done; and

c) Mr. Hasan's condition might go up the Rankin scale from level 4 to level 1 or 2, but not all the way up to zero,

The global damages should therefore be discounted to reflect a less than full recovery. The defendant did not lead any other evidence to support the suggested discount or to relate the deficits to a possible discount.

[301] The plaintiff opposed this submission in its entirety. Counsel submitted that deficits should not be conflated with the legal assessment of damages. More importantly, the defendant had the onus to review the various heads of damages and to lead the appropriate evidence to link the deficits to a specific discount in

damages. Counsel submitted that even if Mr. Hasan had recovered to Level 2 on the Rankin scale, he would have been able to work, he would have been able to walk, and to take care of his immediate personal needs. In short, the court did not have any evidence to relate any anticipated deficits to an appropriate discount.

[302] In my review of this issue, I find that there can be no automatic correlation between the level of a potential deficit and the global damages. This idea of a fast and loose correlation might be the kind of reasoning that might be resorted to in a negotiation for settlement purposes, but there is no legal foundation for a legal determination.

[303] To begin with, deficits in abilities, depending on their degree, might not result in any discount. For example, at Level 0 or 1 of the Rankin scale, the deficits would likely be negligible. At Level 2 or 3, there could be a discount, but its measure would have to be connected to a specific head of damage. This would require expert evidence on the nature and scope of the deficits and the corresponding impact on one's life activities. For example, deficits assessed at 25% may preclude any ability for employment, depending on the nature and extent of the deficits and the corresponding employment skills or vocational requirements. Similarly, the determination of general damages, may or may not be impacted. No such evidence was before the court to enable any engagement with the subject.

Accordingly, I see no basis to conclude that 25% in suggested deficits would automatically translate in a 25% discount in damages.

[304] Finally, having been advised that the parties arrived at a global settlement on damages, I have no way of knowing whether potential discounts were considered by the parties in negotiations and whether some discount might have been embedded in the settlement.

[305] In short, while the legal principles were correctly stated, in the absence of a fulsome evidentiary record concerning the heads of damages, the related calculations, and the related correlation with the suggested deficits, there is no way to make any credible and legal determination.

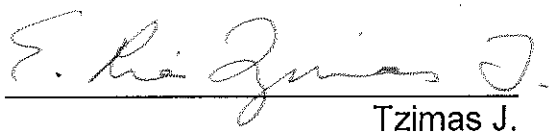
CONCLUSION

[306] In the result, I find on a balance of probabilities that Dr. Campbell fell below the standard of care of an emergency physician, that Mr. Hasan suffered an embolic stroke, and that but for Dr. Campbell's breach of the standard of care Mr. Hasan would have been assessed and received treatment in a timely manner, which in turn would have resulted in a successful treatment and recovery.

[307] I am advised that the parties have settled damages. For the aforementioned reasons, I dismiss the defendant's claim for a 25% discount on

account of the contention that Mr. Hasan would not have had a full recovery even if he were treated in a timely manner.

[308] Turning to costs, it is my sincere hope that the parties will be able to settle, much as they settled damages. However, if the parties are unable to arrive at an agreement, they may write jointly to me to propose a timetable for their respective filing of written submissions, such submissions not to exceed five pages double-spaced, in addition to a supporting Bill of Costs.



Tzimas J.

Released: July 7, 2022

CITATION: Hasan v. Trillium Health Centre Mississauga, 2022 ONSC 3988
COURT FILE NO.: CV-13-5376
DATE: 20220707

ONTARIO
SUPERIOR COURT OF JUSTICE

BETWEEN:

SYED JADWAL HASAN, SHAZIA HASAN, SYEDA KISA ZEHRA, SYED ZAIN JAFFARY, SYEDA ARUBA ZEHRA

Plaintiffs

- and -

TRILLIUM HEALTH CENTRE MISSSSAUGA DR. VERITY JANE JOHN, DR. ALAN CAMPBELL, DR. CHIKKAHANUMAIAII DEVARAJ, JOHN DOE and JANE DOE

Defendants

REASONS FOR JUDGMENT

TZIMAS J.

Released: July 7, 2022

SCHEDULE "A"

JOINT MEDICAL GLOSSARY

i. **Relevant Anatomical terms:**

a. **Central Nervous System:** The part of the nervous system consisting of the brain and spinal cord.

b. **Brain Stem**: The funnel-like group of brain structures between the cerebral hemispheres and the spinal cord, including the midbrain, pons and medulla oblongata;

c. **Vertebral Arteries**: The left and right vertebral arteries are located in the neck and lower part of the head. They join to become the basilar artery. Each vertebral artery is comprised of four segments proximal (lower) to distal (higher): V1, V2, V3, and V4. V1 is approximately from the subclavian to the C5-C6 vertebrae. V2 is the segment where the artery travels within the spine between the C6 and C3 vertebrae. V3 is where the artery loops around the upper cervical vertebra, which is the segment with the most arterial stretching and compression from neck movements. V4 is the intracranial segment after the artery pierces the opening that allows the spinal cord to exit the skull.

d. **Basilar Artery**: The artery that arises from the midline union of the left and right vertebral arteries. It provides blood flow to the brain stem, cerebellum, occipital lobes, medial temporal lobes and thalamus.

e. **Distal Basilar Artery**: The top end of the basilar artery, where it divides into the two posterior cerebral arteries.

f. **Proximal Basilar Artery**: The bottom end or trunk of the basilar artery.

g. **Cerebellum**: A part of the brain, below the cerebral hemisphere, located in the posterior fossa. It controls learned, skilled motor functions like balance walking, standing and coordination.

h. **Occipital Region**: The back and lower part of the skull.

i. **Medulla Oblongata**: The lowest part of the brain stem. It controls functions like breathing, circulation, heartbeat, swallowing, vomiting, and digestion.

j. **Pons**: The middle part of the brain stem. It controls the functions like sleep, breathing, swallowing, bladder control, hearing, equilibrium, eye movement, taste, facial expressions, facial sensation and posture.

k. **Pontine**: Relating or affecting the pons of the brain stem.

l. **Posterior Fossa**: A shallow, bowl-like area of the skull located at rear and bottom which contains the cerebellum and brain stem.

m. Perforator Arteries: Small arteries that arise from larger cerebral arteries that run outside the brain in the subarachnoid space, These small arteries penetrate the brain to supply blood to a small region of brain tissue.

n. Lumen (true): The normal channel of blood flow within an artery. If the artery is like a tube, the lumen is the inside of the tube.

o. Lumen (false): The abnormal channel of blood flowing within the wall of an artery after a dissection.

p. Intima: The inner layer of the wall of a blood vessel.

q. Adventitia: The outer layer of the wall of a blood vessel.

r. Intramural: Within the arterial wall.

s. Intraluminal: Within the true lumen (inside cavity) of an artery,

t. Thrombus: A blood clot formed within a blood vessel and remaining attached to its place of origin.

u. Embolus: A blood clot formed within the heart or a blood vessel that detaches from its point of origin and moves downstream.

v. Embolism: The sudden obstruction of a blood vessel by an embolus, which may lead to brain ischemia and or infarction.

w. Lesion: An area of pathology within an anatomical structure (e.g., brain or artery) due to injury or disease.

x. **Hematoma:** A collection of blood within body tissues or compartments that can cause an expanding mass.

y. **Stenosis:** Narrowing or constricting of the true lumen of a blood vessel.

z. **Dissection:** A tear

aa. **Occlusion:** The blockage of a blood vessel.

bb. **Infarct (cerebral)**: An area of irreversible tissue injury (cell death) in the brain resulting from severe or prolonged brain ischemia (insufficient blood flow).

cc. **Ischemic Stroke:** A type of stroke that results from an insufficient blood supply to a part or parts of the brain.

dd. **Intracranial Hemorrhage:** Bleeding from a ruptured blood

ee. **Embolic occlusion:** One mechanism of ischemic stroke, which can be secondary to arterial dissection. A thrombus forms within the true lumen of the dissected artery at the site of the vessel wall tear, within the false lumen, or it forms because of slow, turbulent blood flow related to arterial narrowing or occlusion. The thrombus can then either embolize, or also serve as the source of emboli, that travel downstream and block distal arteries.

ff. **Basilar Artery Occlusion: [insert]**

gg. **Vertebral Artery Dissection:** A tear of the inner layer (intima) of the arterial wall, which allows blood to track up within the layers of the arterial wall. A vertebral artery dissection may create abnormalities may create abnormalities in the size and caliber of the artery and/or develop a hematoma or thrombus/embolus.

hh. **Perforator Artery Occlusion:** One mechanism of ischemic stroke. In the case of a dissection, an intramural hemorrhage may track up within the layers of the arterial wall blocking perpendicular side branches, causing ischemia and loss of neurologic function specific to the brain territory supplied by each branch.

ii. **Subarachnoid Hemorrhage:** Bleeding in the space that surrounds the brain.

jj. **Hemodynamic Occlusion:** A mechanism of brain ischemia that results from an occlusion or narrowing of a proximal artery leading to insufficient blood flow to distal arterial beds. This implies there are inadequate collateral blood vessels to compensate with the reduction in blood caused by the narrowed or blocked proximal segment.

In the case of an arterial dissection with an intramural hemorrhage, the hemorrhage expands within the vessel wall until it narrows or totally obstructs the true lumen.

In the case of an arterial dissection with a thrombus/embolus, a clot may propagate until it narrows or totally obstructs the true lumen.

ii. **Symptoms**

a. **Ataxia:** Impaired voluntary coordination of muscle movements, which may result in limb incoordination or gait unsteadiness.

b. **Gait:** The manner of walking or moving on foot.

c. **Bell's Palsy:** An acute paralysis of one side of the face secondary to damage of the facial nerve, usually of unknown cause and presumed to be post-viral. It is a peripheral facial palsy.

d. **Guillain-Barre Syndrome:** A neurological disorder in which the body's immune system mistakenly attacks part of the true peripheral nervous system, causing progressive muscle weakness and sometimes paralysis.

e. **Nystagmus:** An abnormal rhythmic oscillation of the eyes.

f. **Diplopia:** Double-vision.

g. **Vertigo:** A type dizziness characterized by a hallucination of movement, typically described as the room spinning or a sense of tilting or swaying.

h. **Central Vertigo:** Vertigo caused by a pathological lesion within the brain and/or brain stem.

i. **Peripheral Vertigo:** Vertigo caused by dysfunction of the inner ear.

j. **Tinnitus:** A sensation of noise, ringing, or roaring in the ears a person experiences, without extreme stimuli.

iii. **Diagnostic Tests**

a. **CT Head Scan:** Computerized Tomography (CT) of the head and brain. A CT scan creates a series of x-rays to provide detailed, cross-sectional images of the blood vessels and brain.

b. **CT Angiogram:** A CT imaging test used to visualize the blood vessels of the neck and brain by injecting contrast dye into a large arm vein.

c. **MRI Brain:** Magnetic Resonance Imaging of the brain. Magnetic and radio waves create detailed 3D images of the blood vessels and brain.

d. **CTAS:** Canadian Triage and Acuity Scale: A standardized method of assessing urgency and relative priority of patients presenting to the Emergency Department. It has 5 levels: resuscitation (1), emergent (2), urgent (3), less urgent (4), and non-urgent (5).

e. **GCS:** Glasgow Coma Scale. A 15 point scale used to assess the level of consciousness based on eye opening, best verbal response, and best motor response. It was developed to rapidly assess patients with head injuries. 15/15 is a normal score.

f. **Modified Rankin Scale:** A 6-point scale to quantify patient recovery after a stroke. (The six points are discussed below in relation to legal causation).

iv. **Treatments**

a. **Heparin:** An anticoagulant medication that can be given intravenously or by subcutaneous injection to inhibit clotting.

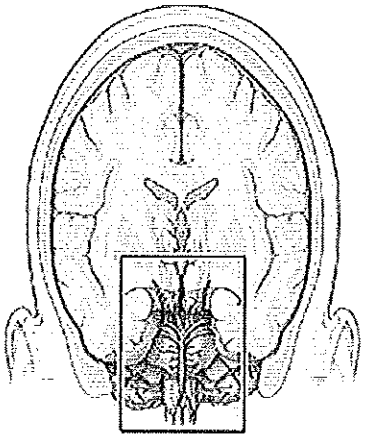
b. **tPA:** Tissue plasminogen activator. A medication that can be given to help dissolve intraluminal clots in selected patients with acute ischemic stroke, based on established criteria. tPA can administered intravenously or intra-arterially.

c. **Endovascular Treatments (EVT):** A procedure whereby a highly specialized neuroradiologist, neurologist, or neurosurgeon advances special catheters from the femoral artery (groin) or the

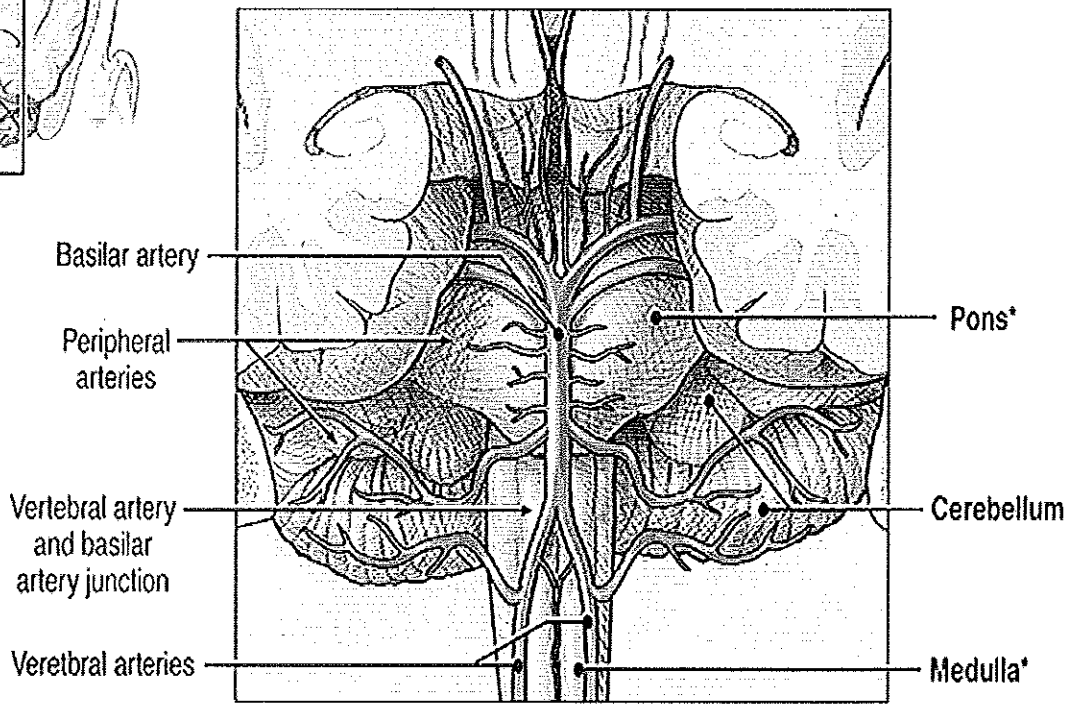
radial artery (arm) to the blocked cerebral arteries within the brain. The treatment may include either mechanical disruption of the blockage, or the administration of medication, or both.

d. **Recanalization:** Re-opening of a partially or totally occluded blood vessel.

BLOOD SUPPLY TO THE BRAINSTEM AND CEREBELLUM



FRONT SECTIONAL VIEW



Illustrations constructed from medical literature

*Components of brainstem