Workplace Safety and Insurance
Appeals Tribunal

Tribunal d'appel de la sécurité professionnelle
et de l'assurance contre les accidents du travail

Stroke

Discussion paper prepared for

The Workplace Safety and Insurance Appeals Tribunal

February 2003

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WSIAT literature search reviewed by Dr. D. Rowed in 2011, who is of the opinion that this paper still provides a balanced overview of the medical knowledge in this area.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

Introduction

Because of the aging of the population and the steep association of stroke with age, the incidence of stroke is expected to increase dramatically in the near future. The Heart & Stroke Foundation of Ontario forecasts an increase of 32% between 1996 and 2006, and a doubling by 2016.

There have been major advances recently in the treatment of acute stroke with the “clot-buster” tPA (tissue plasminogen activator), and also in the prevention of stroke. Controlling high blood pressure can reduce the incidence of stroke by nearly half, antiplatelet agents (ASA and new super-ASA drugs such as clopidogrel) can reduce stroke by about 30%, and an operation to clean out narrow carotid arteries (endarterectomy) reduces stroke and death from 26% to 9% in two years, in patients with symptomatic severe narrowing of the carotid arteries.

The purpose of this review is to discuss stroke in relation to the workplace.

What is a stroke?

The word ‘stroke’ means the sudden onset of a focal disturbance of brain function from a brain hemorrhage or a blocked blood vessel to a part of the brain. However, about 15% of patients presenting to an emergency room with stroke-like symptoms turn out to have something other than vascular disease: stroke mimics include brain tumors, abscesses, blood clots under the lining ofthe brain (subdural hematomas), migraine and paralysis following epileptic seizures.

Once such non-vascular causes are eliminated, then vascular stroke must be sorted out into its subtypes. Vascular strokes can either be due to blockage ofan artery in the brain, leading to loss of blood supply (ischemic strokes, approximately 89% of all strokes), or can be due to rupture of blood vessels, leading to hemorrhage into the brain (hemorrhagic strokes, approximately 20% of all strokes). Within each of those two major categories there are subtypes, shown in Table 1.

Table 1. Types of stroke

Hemorrhagic	Ischemic
Subarachnoid	Lacunar
Intracerebral	Atherosclerotic
- amyloid angiopathy	- occlusion
- Arterio-venous malformation (AVM)	- embolization of platelet clumps
- hypertensive	- embolization of atheromatous debris
2º cerebral vein thrombosis	Cardioembolic
	- recent myocardial infarction
	- atrial fibrillation
	- ventricular aneurysm
	- paradoxical
	Embolization of passengers in the blood
	- air embolism
	- fat embolism
	Vasculitis
	- giant cell arteritis
	- lupus (SLE)

Hemorrhagic Strokes

Subarachnoid Hemorrhage. The outer lining of the brain is called the arachnoid membrane; hemorrhage under that lining is called subarachnoid hemorrhage. Such hemorrhages are usually due to rupture of an aneurysm, at a branch point on one of the main arteries at the base of the brain, where all four main arteries join together to form a natural bypass system, similar to a ring road around the base of the brain. This ring of arteries was discovered in the 17th century by Thomas Willis, it is called the Circle of Willis (Figure 1). Such aneurysms (like small blisters) are thought to be due to an underlying weakness in the wall of the artery, and get larger with age until they reach a size at which they may rupture. High blood pressure does not cause aneurysms to form or enlarge, but high blood pressure may cause them to rupture.

Figure 1 - Base of the brain showing principal arteries (the circle of Willis).

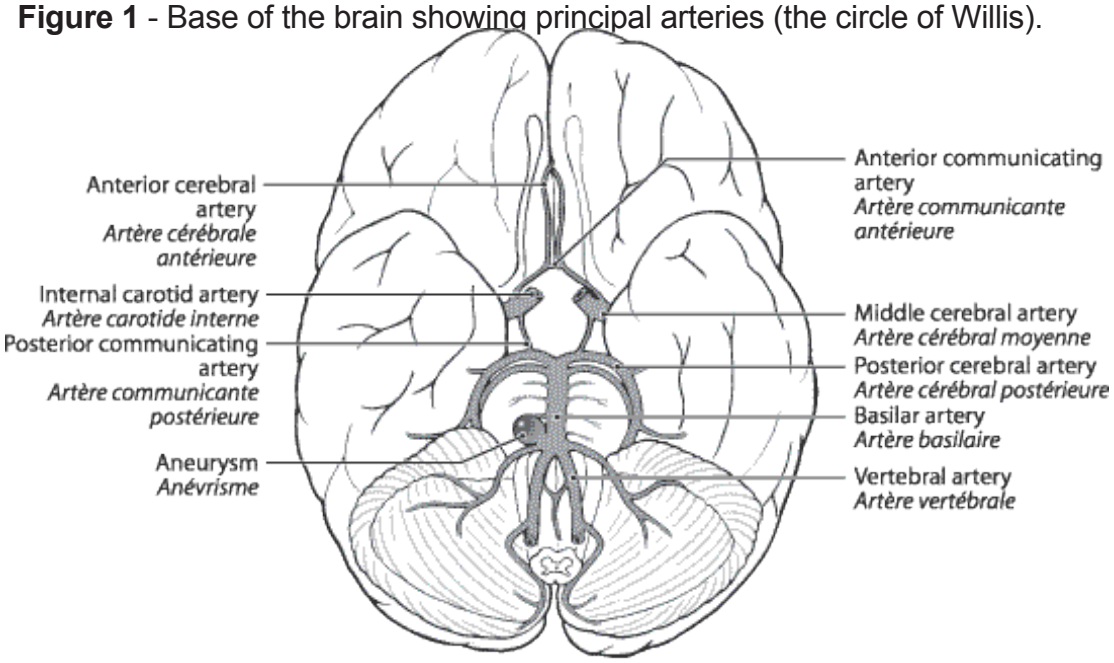


Figure 1. Base of the brain showing principal arteries (the circle of Willis). An aneurysm is shown on the basilar artery.

Figure 1. Base du cerveau montrant les artères principales (le cercle de Willis). Un anévrisme est montré sur l'artère basilaire.

An aneurysm is shown on the basilar artery. There are two possible ways in which subarachnoid hemorrhage may be related to workplace activities. One is that a sudden rise in blood pressure may contribute to the rupture of an existing aneurysm. It is possible that such a sudden rise in blood pressure may be work-related, and this issue will be discussed below.

The second is that an injury to the vertebral artery (discussed below) may cause the layers of the artery lining to separate (so called "dissection"). A dissection of the wall of the vertebral artery may extend inside the skull, constituting a 'false aneurysm' and rupture of this pseudoaneurym may lead to subarachnoid hemorrhage.

Other causes of hemorrhagic strokes are (1) hypertensive hemorrhage (Figure 2), which is hemorrhage due to rupture of a small blood vessel that has been damaged by high blood pressure (2) hemorrhage due to amyloid angiopathy (usually in the elderly), which is a degenerative condition in which blood vessels are weakened by deposition of a protein called amyloid (this isnot caused by hypertension, but high blood pressure may contribute to rupture of the weakened vessels); and (3) hemorrhage due to rupture of an existing congenital abnormality of the arteries or veins. The latter can be (a) an arteriovenous malformation (AVM), in which blood flows directly from arteries into veins,

instead of going through the usual tiny network of capillary vessels (this is similar to a red birthmark on the skin), or (b) a venous abnormality called a cavernous angioma (cavernoma).

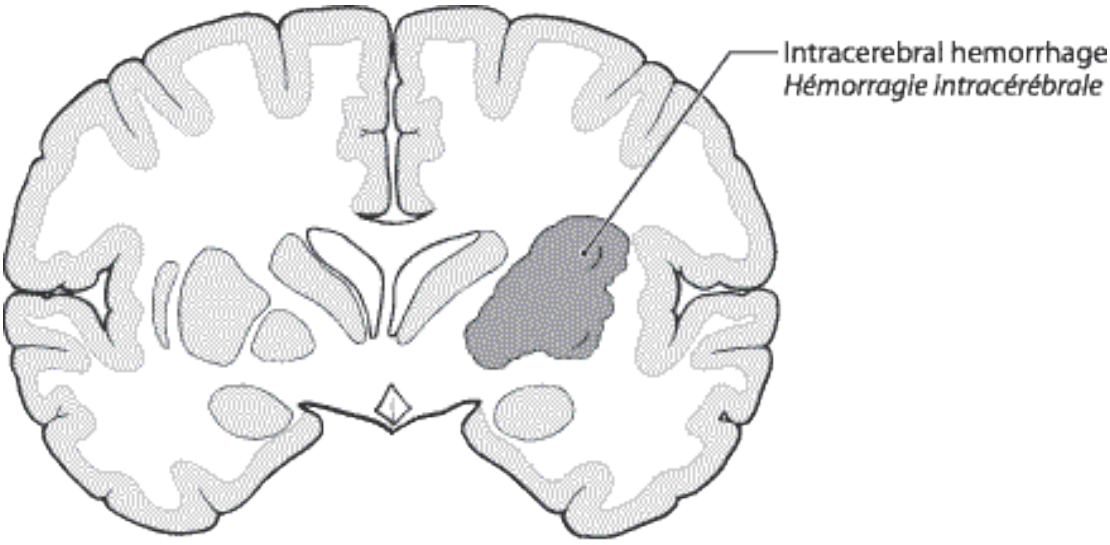


Figure 2. Intracerebral hemorrhage
Figure 2. Hémorragie intracérébrale

Figure 2 - Intracerebral hemorrhage

These types of hemorrhages occur in different locations in the brain. Hypertensive hemorrhages occur in a particular distribution, in the deep parts and at the base of the brain, where short straight arteries with few branches transmit the high pressure from the large arteries right through to the resistance vessels, which are called arterioles. These hemorrhages affect the basal ganglia, internal capsule, brainstem and cerebellum. The high pressure damages the arterioles, and leads either to hemorrhage when the vessels rupture, or to small ischemic strokes called lacunar strokes when the arterioles occlude.

Hemorrhages from amyloid angiopathy tend to occur at the junction between the cortex and white matter, in the parietal area, well up in the hemispheres. Arteriovenous malformations and cavernomas can occur in many locations. It is possible that an acute elevation of blood pressure may provoke hemorrhage in cases of amyloid angiopathy and possibly cavernoma, but probably less likely in hemorrhage from AVM's.

Ischemic strokes:

Ischemia means loss of blood flow, and infarction means death of tissue due to loss of blood flow. Ischemic stroke has many causes. An infarct may be fairly large, if a major artery is blocked, or very small. Lacunar infarcts are very small infarcts (2 to 15 mm diameter) in the deep portion of the cerebral hemispheres or brain stem, due to blockage of very small artery branches (so-called ‘perforating’ branches).

An artery may occlude because of disease in the artery wall, or may be occluded by a chunk of material that originates upstream, and lodges in a branch that is too small for to permit further advance; the latter mechanism is called embolism (the chunk is an embolus; the mechanism is called embolic stroke).

Diseases in the artery wall that can lead to occlusion include atherosclerosis (hardening of the arteries), hypertensive small vessel disease, as described above, and inflammation (vasculitis).

Examples of vasculitis include lupus, which usually affects young people, and giant cell arteritis (also called temporal arteritis because it commonly causes headaches with tender arteries in the temple area), which mainly affects the elderly. These causes would not be work-related, unless the lupus is caused by workplace exposure to certain chemicals, or by a work-related burn (very rare).

Ischemic strokes due to hypertension occur in the same distribution mentioned above for hypertensive hemorrhages: the base of the brain, for example the basal ganglia, internal capsule, brainstem and cerebellum.

Atherosclerosis causes ischemic stroke by two mechanisms: a plaque (athickening in the artery wall due to a growth of smooth muscle cells, infiltration of macrophages and accumulation of oxidized LDL cholesterol andinflammatory cells) may rupture into the lumen of the artery, leading to occlusion of the artery followed by thrombosis (clotting), or there may be embolism from a plaque in the heart, aorta or carotid arteries, with either chunks of plaque or platelet clumps that have formed on a rough plaque breaking off and embolizing into the brain arteries. Chunks of plaque often contain cholesterol crystals; such emboli are called atheromatous debris. Some such strokes may be prevented by removing the plaque (carotid endarterectomy). Platelet clumps (called white thrombus, because they are actually white) form on roughened plaques, and the formation of such platelet clumps is reduced by ASA and other antiplatelet agents

Cardio-embolic stroke:

About a quarter of ischemic strokes are due to blood clots from the heart, which break off and embolize to the brain. These thrombi are usually red thrombus, which forms in areas of stasis (pooling) of blood. Clotting proteins form a polymer mesh similar to cotton wool, which entraps platelets and red blood cells, so they look red. Such strokes may be prevented by drugs that prevent the formation of the clotting proteins, drugs called anticoagulants, and which include heparin (given intravenously or by subcutaneous injection) and coumadin (warfarin).

Clots commonly form in the heart in the setting of a heart rhythm disturbance called atrial fibrillation, in which the upper chambers of the heart, instead of beating rhythmically, wiggle like a bag of worms. An appendage of the left atrium called the auricle, similar to an ear sticking out of the side of the atrium, is an area of blood stasis in which red clots form. In addition to anticoagulants, removal of the auricle, or blockage of the auricle with a prosthesis are two new treatments being explored for stroke prevention in atrial fibrillation.

Clots may also form in the ventricles of the heart if the heart muscle is damaged. If the ventricle functions very poorly, either because of muscle diseases called cardiomyopathies, or because part of the heart muscle hasbeen damaged by a heart attack, there may be stasis of blood and clot formation. About 8% of ischemic strokes occur as the result of a recent heart attack, and the heart attack in such cases has often not been recognized. (About 30% of heart attacks occur without chest pain; these are called “silent” heart attacks.) Clots may also form on artificial heart valves; occasionally they also form on heart valves that have been damaged by infection, rheumatic heart disease, or mitral valve prolapse.

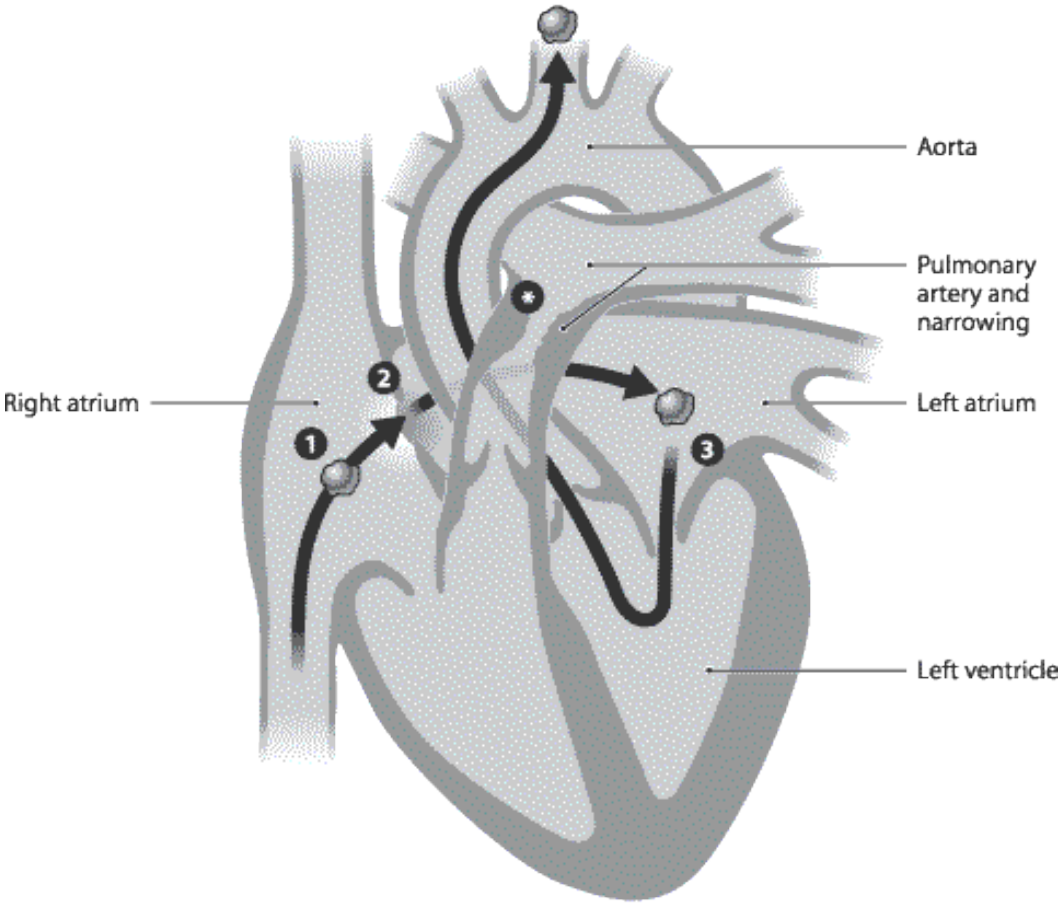
It is possible that such strokes could be work-related if a cardiomyopathy results from exposure to toxins in the workplace, but this would be very rare.

Paradoxical embolism:

A particular type of embolic stroke that could be work-related is paradoxical embolism (Figure 3). This happens when a blood clot that has formed in a vein, for example a leg vein in a patient with deep vein thrombosis, and breaks off, making its way through an abnormal passage into the arteries, and then to the brain. The name ‘paradoxical’ comes from the unexpectedness of a venous clot going directly to the brain, instead of being filtered out in the lungs.

There are two ways for a blood clot to get from the right side of the heart into the arterial circulation: a pulmonary arteriovenous fistula (about 2% of strokes in young people), or a hole in the dividing wall between the right and left side of the heart. The commonest is called an atrial septal defect, which is a hole in the dividing wall between the left and right atrium.

Figure 3. Paradoxical embolism.



1. Embolus (blood clot) from a vein in leg or pelvis enters right atrium.
2. Embolus passes through defect in septum between right and left atria, and enters left atrium.
3. Embolus enters left ventricle, and is then pumped into the aorta and thence into the brain, causing a stroke.

* Narrowing of the pulmonary artery causes increased pressure differential between right and left side of heart, expediting passage of embolus from right to left atrium.

Figure 3 - Paradoxical embolism

Once thought to be very rare, this cause of stroke is increasingly recognized through new imaging modalities including trans-esophageal echocardiography and transcranial Doppler. It is likely that about 4% of ischemic stroke is due to this mechanism.

There are several ways in which this type of stroke may be work-related. If aworker experiences an injury that results in immobilization and deep vein thrombosis in the legs or pelvis, then a stroke due to this mechanism would be work-related. Also, certain occupations such as caisson workers and divers would be more susceptible to this kind of stroke because of embolization of nitrogen bubbles into the brain during decompression, or Valsalva maneuvers (high-pressure expiration of breath) during diving. High G-forces in pilots might also be related to such strokes.

Figure 4 gives a breakdown of the relative frequency of types of ischemic stroke.

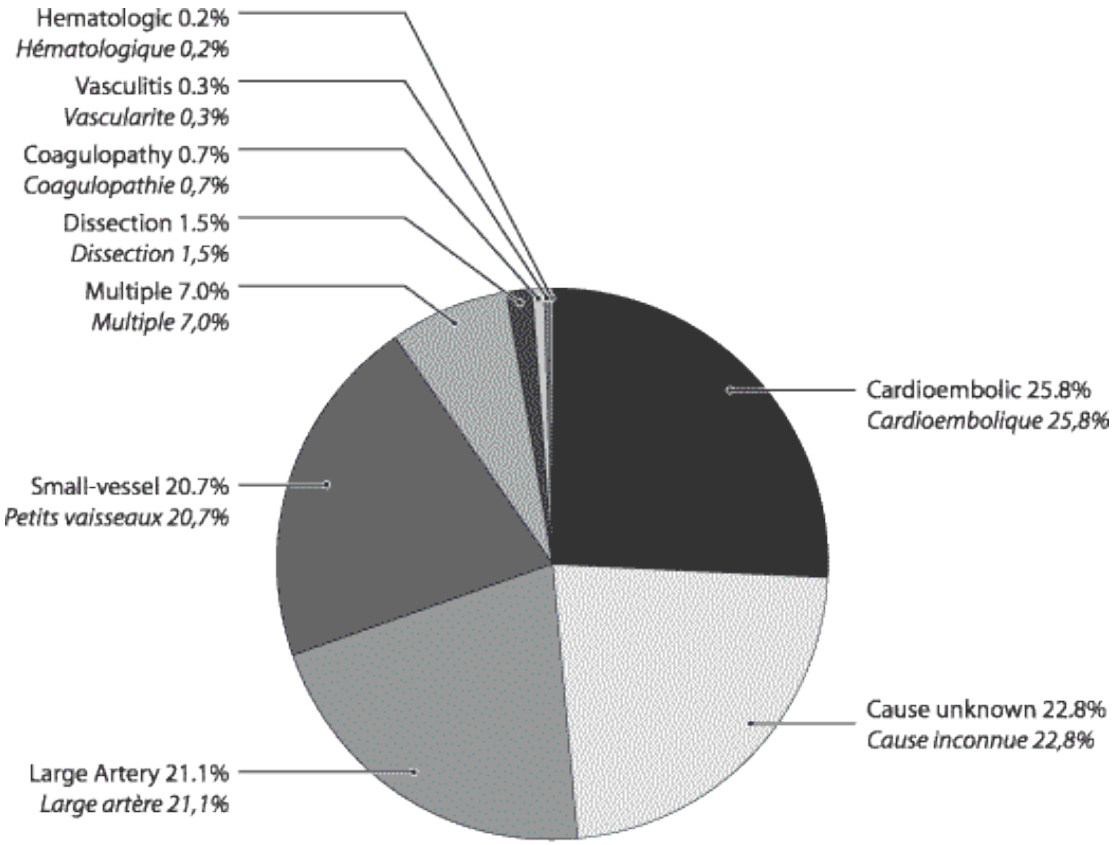


Figure 4. Subtypes of ischemic stroke.
Figure 4. Sous-types d'accidents ischémiques cérébraux.

Figure 4 - Subtypes of ischemic stroke

Symptoms of stroke in the territory of the carotid arteries:

The carotid arteries supply the cerebral hemispheres (the upper and front part of the brain) and the eyes. Symptoms of stroke depend on what part of the brain is involved. If the blood flow to the eye is interrupted, there will be loss of vision in one eye. Loss of blood flow in the cortex on one side may lead to numbness and/or weakness on the other side of the body, sometimes with thickening of speech. If the dominant hemisphere (usually the left hemisphere) is involved, there may be loss of language function; either inability to comprehend speech (receptive aphasia) or inability to produce speech (motor aphasia), or a combination of both (global aphasia). Besides speech other cognitive processes such as calculation may be lost. A large infarction, particularly in the right hemisphere, sometimes leads to severe neglect of the opposite side of the body; this is very disabling. In this condition, the patient may not even recognize their own hand if it is held up in front of their face.

Injury to Arteries in the Neck

Carotid Arteries

The carotid arteries may be injured in the neck, near the base of the skull, by indirect injuries such as sudden violent extension/rotation of the neck, with resultant dissection of the layers of the artery wall. Clot is laid down on the injured part of the carotid artery, and emboli are released into the branches of the carotid, resulting in a stroke in the part of the brain supplied by the carotid artery.

Vertebral Arteries

A particular type of stroke that may be work-related is stroke due to injury of a vertebral artery in the neck, near the base of the skull. The vertebral arteries are susceptible to injury at the C1-2 level of the spinal column, due to their precarious position within the bones at that level. They may be injured during violent extension and rotation of the neck. The mechanism of stroke in such cases probably involves embolization of thrombus material from the site of the injured vertebral artery into the brain, and there is usually a delay between the injury and the stroke. The best known injury of this kind is chiropractic manipulation of the neck, which has been much in the news lately. In my opinion chiropractic stroke is much underestimated because of the assumption that the manipulation must occur shortly before the stroke inorder to be causally related. Among more than 80 cases of traumatic vertebrobasilar ischemia that I have seen, a delay of months between the injury and the stroke is not

uncommon. Often when there is a long delay between the injury and the stroke, the relationship may not be recognized. In addition to chiropractic treatment, work-related injuries to the head and neck, face-mask injuries in football and motor vehicle accidents can also cause stroke from an injured vertebral artery. Figure 5 shows the mechanism of injury to the vertebral arteries.

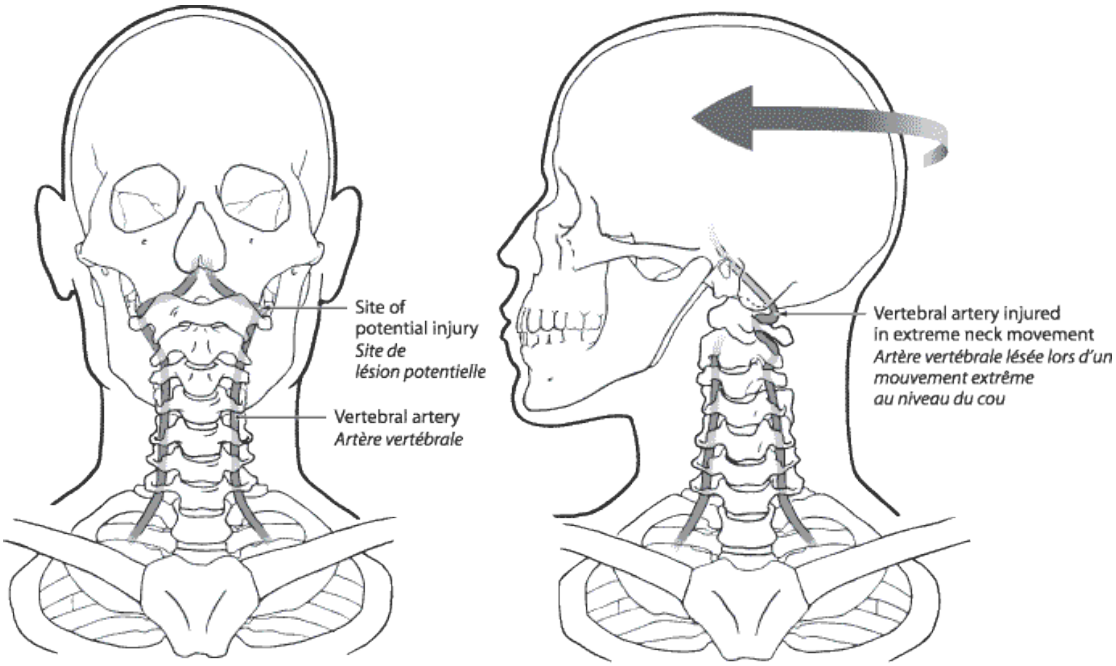


Figure 5. Mechanism of injury in traumatic vertebrobasilar stroke
Figure 5. Méchanisme de lésion dans les accidents vertébrobasilaires traumatiques.

Figure 5 - Mechanism of injury in traumatic vertebrobasilar strokeThe vertebral arteries join at the base of the brain to form the basilar artery, which supplies the brainstem and cerebellum; the basilar artery then divides to form the posterior cerebral arteries (see figure 1). The posterior cerebral arteries (see figure 6) supply the occipital lobes, where visual function is located, and the inferior mesial temporal lobe, which is vital to short-term memory.

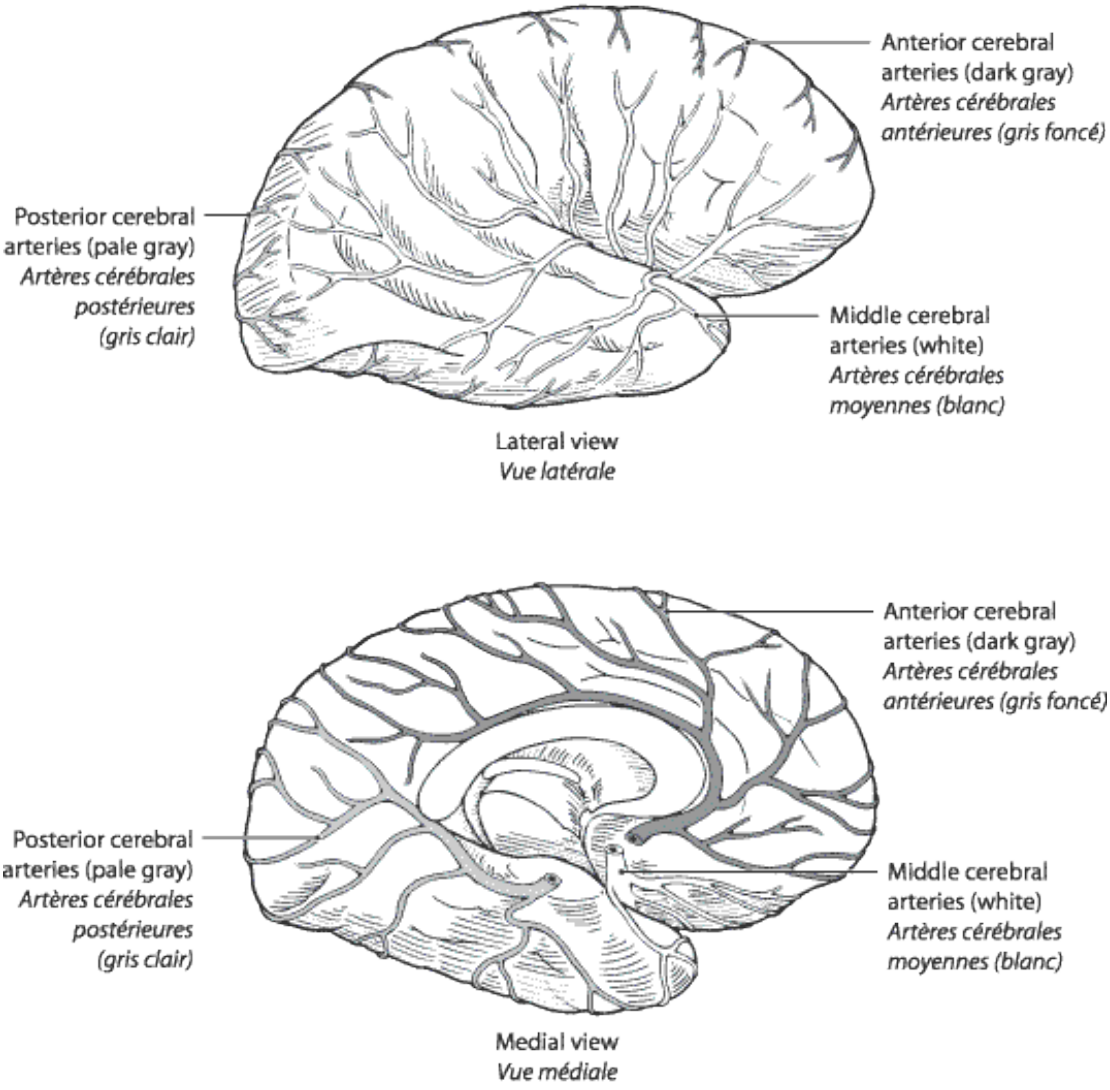


Figure 6. Blood supply of the cerebral hemispheres.
Figure 6. Approvisionnement en sang des hémisphères cérébraux.

Figure 6 - Blood supply of the cerebral hemispheres

Symptoms of vertebrobasilar ischemia:

Symptoms of vertebrobasilar ischemia may vary, depending on what part of the brain or brainstem is involved. If the occipital lobes are involved, there may be visual symptoms (flashing lights, zig-zag lines, loss of vision, orimpaired visual processing - for example becoming lost in familiar surroundings). If the mesial temporal lobe is involved, there may be impairment of short-term memory that can present as transient global amnesia, or if there is actual infarction,

permanent impairment of short-term memory. Involvement of the midbrain will commonly present with double vision; involvement in the pons commonly presents with vertigo; involvement in the medulla may lead to dysarthria (thickening of speech), and trouble with swallowing. Numbness around the mouth, or numbness and weakness on one side of the body or both sides of the body may occur. Involvement of the cerebellum can cause clumsiness, and staggering.

Workplace stress and hypertension:

It can be expected that from time to time a claim will be made that stroke isdue to stress at work. This would rarely be justified. One mechanism by which such a claim may be justified would be if the stroke were due to a marked rise in blood pressure during a particular stressful event, in which the worker experiences, literally, apoplexy. However, the kind of stroke that could be related to such an event would only be a hemorrhage due to rupture of apreviously existing berry aneurysm, or a stroke due to hypertensive small vessel disease (either a hypertensive intracerebral hemorrhage, or a lacunar infarction), in the part of the brain in which such strokes commonly occur (see above).

We have shown that there is a wide range of blood pressure responses tomental stress. A frustrating cognitive task called the Stroop colour-word interference task can raise blood pressure by as much as 54mmHg systolic and 27 diastolic; an angry encounter can raise blood pressure even more (on average by twice as much). However, some subjects actually experience a drop in blood pressure during mental stress. A rise from 140/80 to 194/107 (within the observed range described above) could be enough to cause a stroke. *(Barnett PA, Spence JD, Manuck SB, Jennings JR. Psychological stress and the progression of carotid artery disease. J Hypertension 1997; 15(1):49-55.)*

Increased blood pressure levels during mental stress is related to faster progression of thickening of the heart muscle over two years *(Spence JD, Bass M, Robinson HC, Cheung H, Melendez LJ, Arnold JMO, Manuck SB. Prospective study of ambulatory monitoring and echocardiography in borderline hypertension. Clin Invest Med 1991;14(3):241-50.)*, and to thickening of the wall of the carotid artery over two years *(Barnett PA, Spence JD, Manuck SB, Jennings JR. Psychological stress and the progression of carotid artery disease. J Hypertension 1997; 15(1):49-55.)*.

It could thus be argued that chronic workplace stress might therefore also berelated to vascular disease and thus stroke; this case would be hard to make, because other risk factors such as diet, cholesterol, and smoking are so important. To evaluate such a claim it would be useful if possible to evaluate whether the individual has a marked blood pressure rise during mental stress.

Unusual physical activity would not usually be regarded as a precipitant of stroke, unless the activity led to a myocardial infarction with a subsequent stroke due to embolization of a clot from the heart, or unless the activity led to a marked rise in blood pressure.

Smoking, stroke and the workplace:

A potentially serious issue in the workplace is stroke related to second-hand smoke. Earlier studies of second-hand smoke have seriously underestimated the importance of tobacco smoke as a risk factor for stroke because they failed to properly account for second-hand smoke. When non-smokers regularly exposed to second-hand smoke are lumped together with non-smokers who are not exposed, the risk of smoking is underestimated. Bonita et al (Bonita R, Duncan J, Truelsen T, Jackson RT, Beaglehole R. Passive smoking as well as active smoking increases the risk of acute stroke. Tob Control. 1999;8(2):156-60.) recently showed that smoking increases stroke risk 6-fold, and passive smoking nearly doubles stroke risk. This far outweighs the 30% increase in lung cancer attributable to passive smoke.

Conclusion:

Stroke is the third leading cause of death, and the leading cause of disability, among Canadian adults. Because of the aging of the population, stroke is expected to double in Canada by 2016. Evaluating whether a stroke might be work-related depends importantly on the type of stroke, as certain types of stroke, (including those due to neck trauma and chiropractic manipulation), are more likely to be work-related.