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An anatomical assessment of brain infarcts: A MRI Study

Janéane Potgieter

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**Supervisors: Mrs. N. Briers (University of Pretoria)
Prof. M. C. Bosman (University of Pretoria)**

DECLARATION

I, JANEANE POTGIETER, hereby declare that the work on which this thesis is based is original (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it, has been, or is to be submitted for another degree at this or any other university, tertiary institution, or examining body.

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J POTGIETER

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DATE

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Love and blessings to all!

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Abstract

An infarct is an area which has lost its blood supply due to obstruction, thrombosis or embolism. It is the third leading cause of death in the Western world, following non-cerebral cardiovascular disease and cancer. This research study focused on determining the infarct prevalence according to age, sex and brain areas most affected by infarcts. The prevalence of different infarct types was also determined. Brain MRI statistics were obtained from a Private Radiology practice in Pretoria for a 13-month period. A total of 1844 brain MRI examinations were evaluated, of which 299 patients presented with infarcts. Their age and sex were noted and their individual reports were obtained to record the anatomical structures and brain lobes that were infarcted. The infarct types were also noted. Diffusion-weighted images were used to measure new infarcts, while FLAIR images were used to measure old infarcts. Results showed an overall incidence of 16.10% and vascular structures accounted for 26.63% of these. Most infarcts were new (56.80%) and mainly affected patients aged 70–79 years (31.36%). Normal cerebral infarcts (72.49%) and embolic infarcts (14.50%) were the most common. The parietal lobe (34.91%) and right middle cerebral artery (11.54%) presented with the most infarcts. The right hemisphere (34.91%) presented with slight infarct predominance, but this was not significant when compared to the left (31.95%) hemisphere (Chi square $p>0.05$). No significant difference was found concerning the overall male to female ratio (Chi square $p>0.05$). Females aged 18–39 years of age presented with three times more infarcts than their male counterparts. This may possibly be due to their use of oral contraceptives and pregnancy, which increases the risk of thrombosis and embolism. Females over 80 years also presented with higher infarct prevalence, which is expected, since men die at earlier ages due to other co-morbidities such as cancer.

Opsomming

Infarksie is die uiteinde van geblokkeerde bloedvoorsiening as gevolg van trombusformasie, embolisasie of obstruksie en is tans, na kardiovaskulêre siekte en kanker, die derde grootste oorsaak van dood in die Westerse wêreld. Hierdie navorsingsprojek het daarop gefokus om die infark insidensie te bepaal ten opsigte van ouderdom, geslag en mees geaffekteerde breinarea. Die insidensie van die verskillende tipes infarkte is ook bepaal. Brein MRI statistieke is verkry vanaf ‘n privaat radiologie praktyk in Pretoria oor ‘n 13-maande periode. ‘n Totaal van 1844 MRI ondersoeke is geëvalueer waarvan 299 pasiente met infarkte gepresenteer het. Hul ouderdomme en geslag is noteer en individuele verslae is verkry om die anatomiese strukture, geaffekteerde brein lobbe en infark tipes te noteer. Diffusie-beelde is gebruik om nuwe infarkte te meet en die FLAIR beelde om ou infarkte te meet. Die resultate het ‘n insidensie van 16.10% opgelewer waarvan die vaskulêre strukture die meeste geaffekteer was (26.63%). Die meeste infarkte was nuut (56.80%) en het meestal mense tussen 70–79 jaar oud geaffekteer (31.36%). Normale serebrale infarkte (72.49%) en emboliese infarkte (14.50%) was die mees opvallendste. Die pariëtale lob (34.91%) en die regter middel serebrale arterie (11.54%) het die meeste infarkte opgelewer. Die regter hemisfeer (34.91%) het met ‘n effense hoër infark-insidensie as die linker een (31.95%) gehad, maar die verskil was nie betekenisvol nie (Chi square $p>0.05$). Geen betekenisvolle verskil is aangaande die manlike tot vroulike verhouding opgemerk nie (Chi square $p>0.05$). Vrouens 18–39 jaar het met drie maal meer infarkte presenteer as mans van dieselfde ouderdom. Dit kan wees as gevolg van swangerskap en die gebruik van orale kontrasepsie wat die risiko van trombose en embolisasie verhoog. Vrouens ouer as 80 jaar het ook ‘n hoër infark-insidensie getoon. Dit is verstaanbaar aangesien mans op vroeër ouderdomme sterf as gevolg van ander oorsake soos kanker.

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1. Introduction

Chronic cerebral infarction is the third leading cause of death in the Western world, following non-cerebral cardiovascular disease and cancer. It is also a major cause of long-term disability. One in five patients that present with first-ever stroke will survive to ten years. In the United States 760 000 – 780 000 people are annually affected by cerebral infarction and it contributes to 150 000 deaths per year. [1, 2]

Currently no studies exist regarding the exact prevalence of brain infarction amongst South Africans. European research reports that the most common age group affected is above 55 years of age.[2] This study was therefore conducted to determine the exact prevalence of infarcts amongst the general South African population according to age and sex. It was also performed to determine if infarcts are more prone to occur in specific areas of the brain, i.e. a brain hemisphere, lobe, structure or arterial supply system.

Infarct size on a particular level is not measured as a standard protocol by radiologists. The volume of infarcts can also as yet not be determined due to the lack of specialized software. This is attributed to the fact that no need currently exists for these measurements to be taken, since the residing neurologists do not request it. However, this study includes an observation on the presenting size of infarcts when looking at length, width and circumference.

The study is limited to one institution in Pretoria and only patients that presented with infarcts on MRI examinations were used for observation. As stated above, only the length, width and circumference of the infarcts could be measured, since volume measurement programs are not yet available or in use in South Africa.

2. Aims

The aims of the research study were to determine:

(i) The proportion/percentage of infarcts found in the brain according to the following sub-divisions:

a. Dominant brain hemisphere, i.e. left or right.
b. Dominant brain lobe, i.e. temporal lobe and/or parietal lobe and/or frontal lobe and/or occipital lobe.
c. Affected arterial distribution area, i.e. middle, anterior and posterior cerebral arteries, internal and external carotid arteries or the vertebral arteries
d. Anatomical structures affected, i.e. putamen, centrum semi-ovale, caudate nucleus etc.

(ii) The distribution of infarct type predominance according to the following categories:

a. Different infarct types, i.e. venous, watershed, pointy, embolic, haemorrhagic, lacunar, etc.
b. Each type of infarct was categorized according to age (10 year interval groups) and sex (male or female) to determine individual prevalence's

(iii) Distribution of infarct size (old/new) on presentation, in accordance to length, width and circumference measurements.

3. Literature review

3.1 Normal anatomy

3.1.1 Circle of Willis

The brain receives its arterial supply from the two internal carotid arteries and the two vertebral arteries that form part of the circle of Willis. The circle of Willis is situated in the interpeduncular cistern and it surrounds the optic chiasm, the neural infundibular stem of the cerebral hypophysis and other related structures in the interpeduncular fossa (See Figure 1).[3]

The anterior cerebral arteries, which are medially directed branches of the internal carotid artery, are joined anteriorly by the anterior communicating artery. The basilar artery divides into the two posterior cerebral arteries that, in turn, are joined via the posterior communicating artery to the ipsilateral internal carotid artery.[3]

This description of the circle of Willis applies to a minority of patients since its vessels vary in diameter and are often only partially developed or even absent. Therefore 60% of all circles present with anomalies.[3]

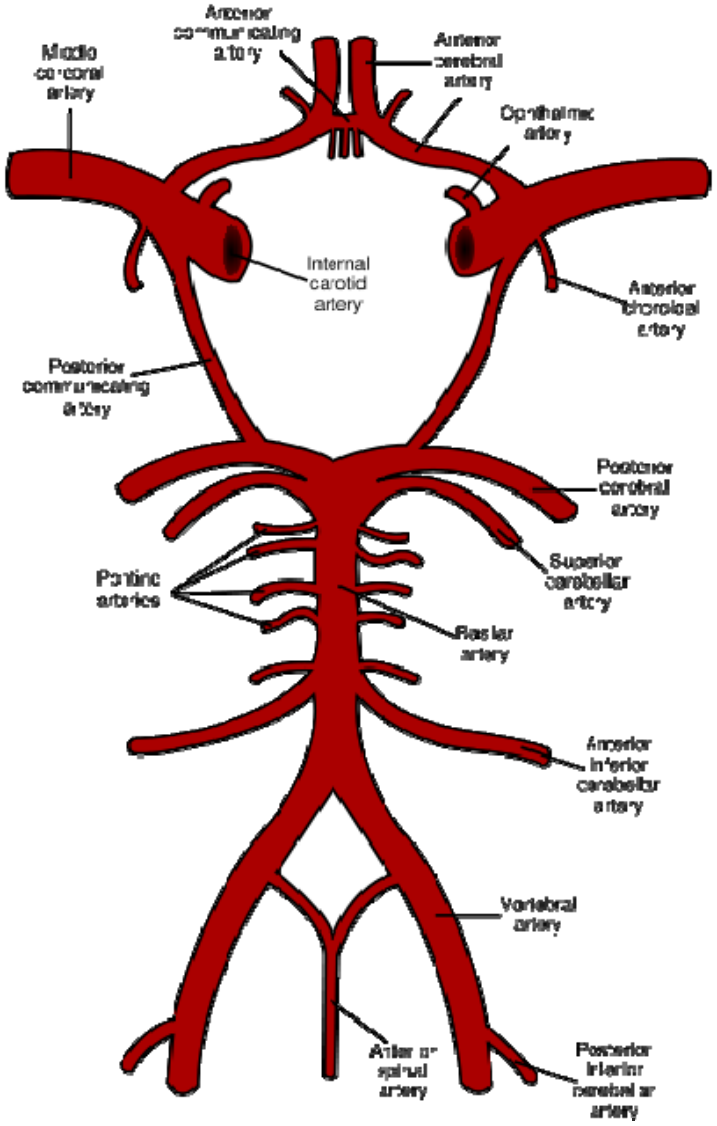

Figure 1: Circle of Willis[3]

3.1.2 Variations: Circle of Willis

The variations of the circle of Willis have been studied extensively. Such studies include Windle's account in 1888[4] of 200 specimens, Fawcett and Blachford (1906)[5] of 700 dissections, Riggs and Rupp's (1963)[6] of 994 dissections and Puchades-Orts *et al* (1976)[7] of 62 dissections. Fields *et al* (1965)[8] have summarized these studies as follows: Anterior and posterior cerebral and communicating arteries can all be absent, double or triple, or variably

hypoplastic. A complete circular channel is present in 90% of cases with at least one sufficiently narrowed vessel to reduce its role in a collateral route.[8]

The calibre of the communicating arteries usually disturbs the haemodynamic balance. The variations occur in the segments of the anterior and posterior cerebral arteries that extend from their point of origin to their junctions with the communicating arteries. This is especially evident in the anastomotic relationship of the posterior cerebral artery and the posterior communicating artery. When the pre-communicating part of the posterior cerebral artery has a larger diameter than the posterior communicating artery, the occipital lobe will mainly be supplied by the vertebro-basilar system. When the diameter of the pre-communicating part of the posterior cerebral artery is smaller than the posterior communicating artery, the occipital lobe will receive its arterial supply from the internal carotid arteries via the posterior communicating arteries (Van Overbeeke *et al* 1991).[9] The latter's' frequency ranges from 6% (McCormick 1969)[10] to 40% (Zeal and Roton 1978)[11] and is referred to as the embryonic configuration. This is opposed to the standard adult configuration. Studies conducted by Abbie[12], Williams[13] and Kaplan[14] proved that this can be attributed to the ontogenetic and phylogenetic association of the posterior cerebral and internal carotid arteries. Van Overbeeke *et al* (1991)[9] has recently shown that the embryonic configuration is not more common in human foetuses than in adults.

Agenesis or hypoplasia of the initial anterior cerebral segment of the anterior part of the arterial circle is more frequent than anomalies in the anterior communicating part. This leads to a more common cause of defective circulation. Sedzmir[15] indicated via angiographic evidence that such defective or absent circulations exists in about a third of individuals. An effective arterial circle can never be assumed to exist and therefore surgical procedures have to be preceded by angiography, during which radio-opaque substances are

injected into the internal carotid or vertebral arteries to monitor the condition of their intracranial branches.[3]

3.1.3 Venous drainage of the brain

The venous system of the brain has no valves and is extremely thin, due to the absence of muscular tissue. The veins pierce the meningeal layer of the dura mater and the arachnoid membrane to open into the cranial venous sinuses. These veins are categorized as cerebral and cerebellar veins. The cerebral veins are subdivided into internal and external cerebral veins, due to the fact that they either drain the outer, or the inner parts of the brain hemispheres. The external veins consist of superior, inferior and middle cerebral veins.[3]

There are eight to twelve superior cerebral veins that drain the superior, lateral and medial surfaces of the brain. They are lodged in the sulci of the brain, between or over the gyri, and open into the superior sagittal sinus. The anterior veins run at right angles to the sinus, whilst the posterior larger veins run directly obliquely forward, and open into the sinus in a direction opposed to the current blood flow. The middle cerebral vein starts on the lateral aspect of the hemisphere and runs along the lateral cerebral fissure, to end either in the cavernous or the spheno-parietal sinus. It is connected to the superior sagittal sinus and the transverse sinus via the superior anastomotic vein of Trolard and the inferior anastomotic vein of L'Abbe respectively. The inferior cerebral veins are small veins that drain the under surfaces of the brain hemispheres. The veins on the orbital surface of the frontal lobe join the superior cerebral veins and open into the superior sagittal sinus. The veins of the temporal lobe anastomose with the middle cerebral and basal veins, to join the cavernous, spheno-parietal, and superior petrosal sinuses.[3]

The basal vein (see Figure 2) is formed at the anterior perforated substance by the union of the small anterior cerebral vein, deep middle cerebral vein and the inferior striate veins. The small anterior cerebral vein accompanies the anterior cerebral artery and drains the medial surface of the frontal lobe by the fronto-basal vein. The deep middle cerebral vein (deep Sylvian vein), receives

tributaries from the insula and neighboring gyri, and runs in the lower part of the lateral cerebral fissure. The inferior striate veins leave the corpus striatum through the anterior perforated substance. The basal vein passes backward around the cerebral peduncle, and ends in the great cerebral vein (vein of Galen); it receives tributaries from the interpeduncular fossa, the inferior horn of the lateral ventricle, the hippocampal gyrus, and the mid-brain.[3]

The two internal cerebral (see Figure 2) veins drain the deep parts of the brain hemispheres and are formed by the union of the terminal and choroid veins. They run backward parallel with one another, between the layers of the tela chorioidea of the third ventricle and beneath the splenium of the corpus callosum. Here they unite with the great cerebral vein, which is also the receiving point of the corresponding basal vein. The terminal vein receives numerous veins from the corpus striatum and the thalamus. It unites behind the crus of the fornix with the choroid vein, to form one of the internal cerebral veins. The choroid vein runs along the length of the choroid plexus and receives veins from the hippocampus, fornix and the corpus callosum. The great cerebral vein (see Figure 2) is formed by the union of the two internal cerebral veins. It curves backwards and upward around the splenium of the corpus callosum and ends in the straight sinus.[3]

The superior and inferior cerebellar veins are found on the surface of the cerebellum. The superior cerebellar veins pass across the superior vermis to end in the straight sinus and the internal cerebral veins. The inferior cerebellar veins are larger and end in the transverse, superior petrosal and occipital sinuses.[3]

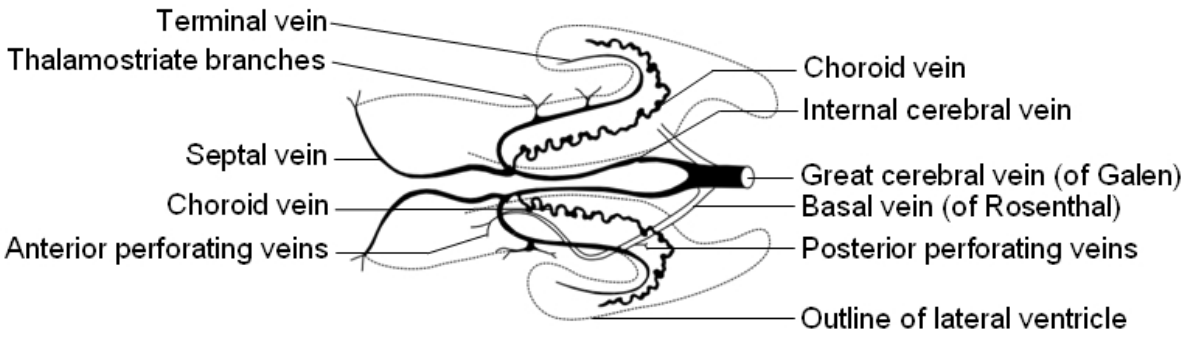

Figure 2: Venous drainage of the brain

3.2 Stroke (Cerebrovascular incident)

Stroke occurs due to ischaemia or haemorrhage and presents as an abrupt onset of focal or global neurological symptoms that lasts longer than 24 hours. If the symptoms resolve within 24 hours, the event is known as a transient ischaemic attack (TIA). Stroke is mainly caused by thrombosis, cerebral embolism, ischaemia and cerebral haemorrhage. Thrombosis occurs when a blood clot enters a blood vessel of the brain or neck. It is caused by atherosclerosis or slowing of the cerebral circulation. Atherosclerosis occurs when plaque builds up on the arterial walls.[16]

Cerebral embolism occurs when a blood clot or any other material is carried to the brain. Emboli may originate in the left side of the heart due to infective endocarditis, rheumatic heart disease, myocardial infarction and pulmonary infections. The embolus usually lodges itself within the middle cerebral artery or its branches, where it disrupts the cerebral circulation. Ischaemia is the result of decreased blood flow to a level where either temporary or permanent loss of organ function occurs,[17] due to atheromatous constriction of the arteries that supply the brain.[16]

Global ischaemia occurs when there is significant diminished blood flow to all parts of the brain. Regional or focal ischaemia therefore occurs when there is diminished blood flow to only selected areas of the brain. Cerebral haemorrhage occurs when a cerebral blood vessel ruptures and blood accumulates in the brain tissue or spaces surrounding the brain.[16]

3.2.1 Risk factors of stroke

The risk of stroke increases with advancing age. The risk more than doubles for each decade of life after the age of 55 years.[1] It does however affect people of all ages – sometimes even children.[18] Stroke is more common in men than women, although more than half of total stroke deaths occur in women. More

women than men die of stroke, irrespective of their age.[1,18] This could partially be due to their use of oral contraceptives and pregnancy. Contraceptives and oestrogen therapy often cause venous thrombosis and, on rare occasions, intimal hyperplasia and thrombosis of the cerebral and extra-cerebral arteries.[19] A hospital-based study[18] indicated that increased age is the strongest determinant of stroke. Men were found to be at a slightly higher risk than women, but women presented with higher stroke prevalence due to the fact that men die earlier as a result of other co-morbidities. It was found that diabetes mellitus increased the stroke risk four-fold, and hypertension three-fold. Cardiac impairment was found to increase the stroke risk by 2 to 4 times. Smokers also show a 1.5 to 2.9 higher risk when compared to non-smokers.[18]

Stroke is hereditary and a patient's stroke risk therefore increases if a parent, grandparent, sister or brother has had a stroke. African-Americans are also at a greater risk than Caucasians of death from stroke due to the fact that they are more susceptible to high blood pressure, diabetes and obesity. More than 80% of African-Americans over the age of 65 suffer from hypertension.[20] Twenty five percent of adults in the U.S. suffer from high blood pressure.[21] Hypertension increases the workload of the heart and damages the inner lining of the blood vessels, which increases the formation of atherosclerotic plaques. The heart and arteries are therefore more prone to injury[22] The Northern Manhattan multi-ethnic community cohort study[20] indicated that 18% (158 / 892) of the studied subjects presented with infarcts. African-Americans accounted for 24% of the infarct cases noted, compared to a mere 18.1% in whites. It was also found that age, male sex and hypertension were independently associated with sub-cortical infarction. Infarction increased with age: <65: 9.7%, 65–75: 16.4% and > 75 years: 26.1%. Men presented with a 21.3% infarct incidence compared to only 15.2% in women.[20]

Transient ischaemic attacks (TIA's) are small strokes that last for only a few hours. It is a warning sign that a more disabling event might follow.[23] Thirty percent of all people who suffer from TIA's will develop a stroke within five years.[24] The internal carotid artery is a major supplier of blood to the brain. If a carotid artery is narrowed by fatty deposits from atherosclerosis, it may become blocked by a thrombus. This is known as carotid artery disease or carotid artery stenosis. Seventy five percent of diabetics die of some form of heart or blood vessel disease. Heart disease death rates of diabetic patients are 2 to 4 times higher when compared to the general population without diabetes. Atrial fibrillation is a heart rhythm disorder where the atria of the heart quiver instead of beating effectively. The blood can therefore pool and clot. If a clot breaks off, and enters the bloodstream as an embolism and lodges in an artery that supplies the brain, a stroke results.[18,20]

High levels of LDL (low density lipo-protein) cholesterol increase the development of atherosclerosis. The cholesterol deposits itself in the arterial walls and increases the build-up of plaque. Low levels of HDL (high density lipo-protein) cholesterol also raise the risk of stroke. This is because HDL cholesterol carries cholesterol back to the liver where it is removed from the body.[21] Diets high in saturated fats and cholesterol raises blood cholesterol levels, whilst the blood pressure is increased with a high sodium (salt) diet. High calorie diets, combined with a lack of exercise, contribute to obesity.[18] Inactivity also increases the risk of developing heart disease and stroke. Exercise helps to control blood cholesterol, diabetes and especially obesity and it even lowers blood pressure. Obesity increases the heart's workload. Blood pressure and blood cholesterol levels are therefore raised and the risk to develop diabetes is also higher in obese people.[21]

Tobacco smoke injures blood vessel walls and speeds up the hardening process of arteries. People who smoke have a 2-4 times higher risk of

developing cardiovascular disease than non-smokers.[21] Nicotine increases the blood pressure, heart rate and blood flow from the heart for a short period, whilst carbon monoxide reduces the amount of oxygen that the blood can carry. Together, these tobacco substances create an imbalance between the demand for oxygen by the cells and the amount of oxygen the blood can supply. Other tobacco components damage the arterial walls, increasing the risk of atherosclerosis. Smoke also makes blood platelets stickier which leads to easier blood clot formation. Alcohol abuse can lead to multiple medical complications including stroke. Drug abuse, especially cocaine, amphetamines and heroin have been associated with an increased stroke risk.[18,21]

Midlife migraines increase the risk of ischaemic stroke. Launer *et al.* [25] performed an international study that indicated that more women than men suffer from migraines. Midlife migraines (age 50) were reported in 18% of their female participants versus only 5% in the male population. It was also found that headaches with visual aura had a significant increase on late-life (25 years after headaches: age 75) infarcts.[25]

3.2.2 Pathogenesis of stroke

a. Cerebral blood flow thresholds

Normal cerebral blood flow (CBF) is approximately 50-60ml/100g of brain tissue/min. Cerebral infarction is a dynamic process that starts when an alteration in blood flow occurs. Neuronal dysfunction is noted electro-physiological when flow decreases to 20-40ml/100g/min. Irreversible neuronal tissue damage occurs when levels fall below 10-15ml/100g/min. A 30–35% reduction from normal CBF leads to functional suppression (failure of synaptic transmission). Reductions of 15–20% leads to the failure of the energy-requiring ion pump mechanisms in the affected area of the brain.[1]

b. Time Dependence

Near-total CBF reductions, i.e. cardiac arrest, can lead to neuronal damage within minutes. During long periods of reduced CBF, i.e. thrombo-embolic stroke, permanent injury may be avoided due to possible retained collateral circulation.[2]

c. Selective Vulnerability

Ischaemic damage depends on the sensitivity of different cell types. Neurons are the most sensitive to these ischaemic changes. They are followed by astrocytes, oligodendroglia, microglia and endothelial cells.[2]

Neurons, however, also have a hierarchy of sensitivity to ischaemic changes. Neurons in the area of the hippocampus, neocortical layers III, V and VI, and the neostriatum are the most vulnerable.[26] The neocortex is the superficial layer of the cerebral hemisphere, is 2-4 mm thick, and made up of six layers that lie parallel to the surface. These layers are arranged from superficial to deep in the following manner: (I) molecular or plexiform, (II) external granular, (III) external pyramidal, (IV) internal granular, (V) internal pyramidal and (VI) multiform layer.[16] The corpus striatum is functionally related to the subthalamus of the diencephalon and substantia nigra of the midbrain. The corpus striatum consists of the lentiform and caudate nuclei which are separated by the internal capsule. The lentiform nucleus is subdivided into the medial globus pallidus and the lateral putamen, which are separated by the external medullary lamina. The putamen appears pruned from the caudate nucleus.[27] The corpus striatum is divided into the neostriatum, which consists of the caudate nucleus and the putamen, the ventral striatum (ventral parts of the caudate nucleus and putamen) and the paleo-striatum (globus pallidus). Together they form part of the basal nuclei and control the primary motor routes. Each cerebral hemisphere consists of an external stratum of neurons (cortex) and an internal

mass of neuronal processes (centrum semi ovale) within the basal nuclei and a lateral ventricle.[3]

d. Collateral Supply

Cerebral blood flow changes with a number of factors and therefore the consequences of arterial occlusions vary greatly. The clinical outcome of the patient is influenced by the duration of the reduction in CBF, the location, as well as the available collateral circulation.[2]

3.3 Infarction

Infarct is the term used to describe a specific area which has lost its blood supply. The arteries of internal organs that have imperfect anastomoses are referred to as end-arteries. Obstructions of these vessels lead to ischaemia and cause necrosis of that specific part or tissue. An infarcted tissue has therefore already undergone ischaemic necrosis. Infarction is a result of the acute occlusion of an artery either by a thrombosis or an embolism. While these are common causes of brain infarcts, it can also occur due to hypotension.[16,17]

When an occluding thrombus or embolus breaks, blood leaks from collateral vessels and necrotic capillaries and reperfuses the infarcted area.[28] Retrograde flow or anastomosing small vessels engorge the small blood vessels in the dying tissue with blood. These engorgements, with its accompanying haemorrhages, are referred to as red or haemorrhagic infarcts. They are common in the intestines and lungs where some anastomoses can be found.[29] Autopsies revealed that 18–48% of arterial brain infarcts displayed haemorrhagic components.[16]

3.3.1 Types of Infarcts

a. Haemorrhagic

Superficially a haemorrhagic infarct resembles a haematoma,[30] showing confluent larger haemorrhages, especially within the necrotic grey matter.[16] These infarcts mainly consist of red blood cells and fibrin strands. The intrinsic architecture of the affected tissue, is however preserved.[30]

b. Pale

When little blood enters the dying tissue a pale infarct will develop. This mainly occurs in organs or parts where little or no anastomoses can be found, for example the heart and the kidneys. The dead tissue becomes slightly soft and

swollen and there is loss of definition between the grey and white matter. Ischaemic necrosis of the neurons is also visible upon histological examination. White or anaemic infarcts are mainly composed of blood platelets.[3]

c. Lacunar

Lacunar infarcts are small infarcts (smaller than 15mm in diameter)[2] that are found in the basal nuclei, thalamus and the corona radiata.[2,31] They can be located in the deep nuclei especially the putamen, thalamus, internal capsule, caudate nuclei and the pons. Lacunar infarcts (see Figure 3) are commonly found in patients older than 55 years,[16] with the first stroke presenting at about age 65.[2,31] An increased lacunar frequency is noted in African-Americans and in men. Twenty percent of all strokes can be attributed to this type of infarct. Lacunar infarcts are caused by small vessel disease, which is a term used to describe a vascular lesion seen in cases of hypertension, diabetes and old age. The small penetrating arteries and arterioles which supply the basal nuclei, thalamus, deep white matter and the brainstem, thicken. Their normal wall components are replaced by collagen and proteins.[32] Lacunar infarcts can develop abruptly within 3 hours (1/3 of cases), or gradually over 2–3 days (1/3 of cases). It is preceded by a transient ischaemic attack and it is the most common cause of multi infarct dementia syndrome.[33]

In cases of hypotension, small vessel disease can be attributed to endothelial injury and plasma protein leakage in and around the vessels. In cases of diabetes, it is caused by protein glycation and diffuse basement membrane thickening. This leads to tortuosity and narrowing of the lumen, which in effect lengthens the distance that the blood has to travel to perfuse its targets. Ischaemia follows and there is a diffuse loss of tissue density in the white matter. The vessels will become fragile due to loss of elasticity from the destruction of the smooth muscle fibres. Micro-bleeds and large catastrophic haemorrhages can occur spontaneously or after trivial trauma.[28]

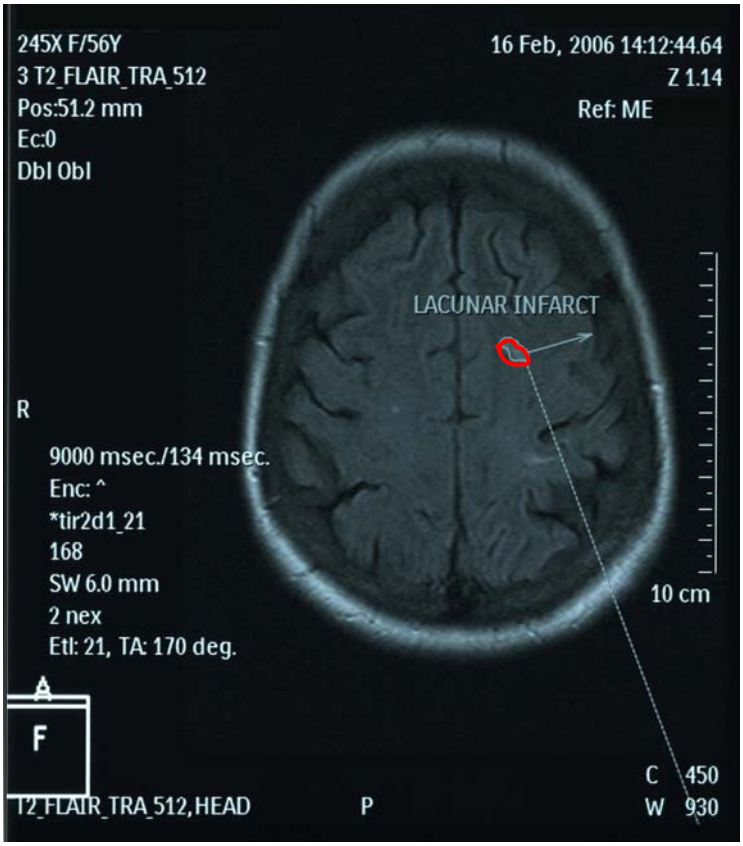

Figure 3: Lacunar infarcts

d. Venous

Venous infarcts (see Figure 4) occur when the venous sinuses and their tributaries are blocked due to thrombosis. This leads to congestion, haemorrhage and brain tissue necrosis. These infarcts are caused by the use of oral contraceptives, cancer and dehydration. The latter is especially relevant in infants.[28]

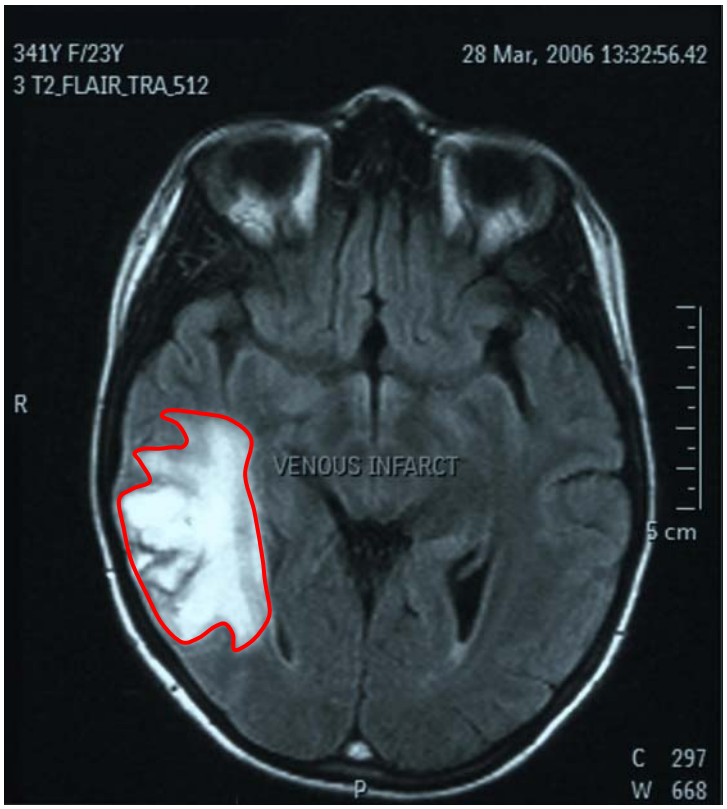

Figure 4: Venous infarct

e. Multi Infarct Dementia (MID)

MID (see Figure 5) also known as vascular dementia, is characterized by deteriorated mental function that occurs due to a series of small strokes. “Multi-infarct” means that multiple brain areas have been injured due to a lack of blood supply.[34] MID’s are silent strokes, since there are no clinically presenting symptoms and individuals are thus not aware of the strokes that are occurring. Over time symptoms will start to appear, but only after multiple areas of the brain have been affected and several blood vessels are blocked.[35] It is responsible for 10-20% of dementia cases and is more common in men than women, since men are more prone to suffer from diabetes, high blood pressure, heart disease and smoking, which are all risk factors for MID.[36] It usually affects people over the age of 55, with an average onset at age 65. Symptoms however, are more likely to appear after the age of 70.[34] MID is the third common cause of dementia, following Alzheimer’s disease and dementia of the

Levy bodies.[35] Since it is very similar to the latter, it is difficult to diagnose and co-existence makes it severely under-diagnosed. [36]

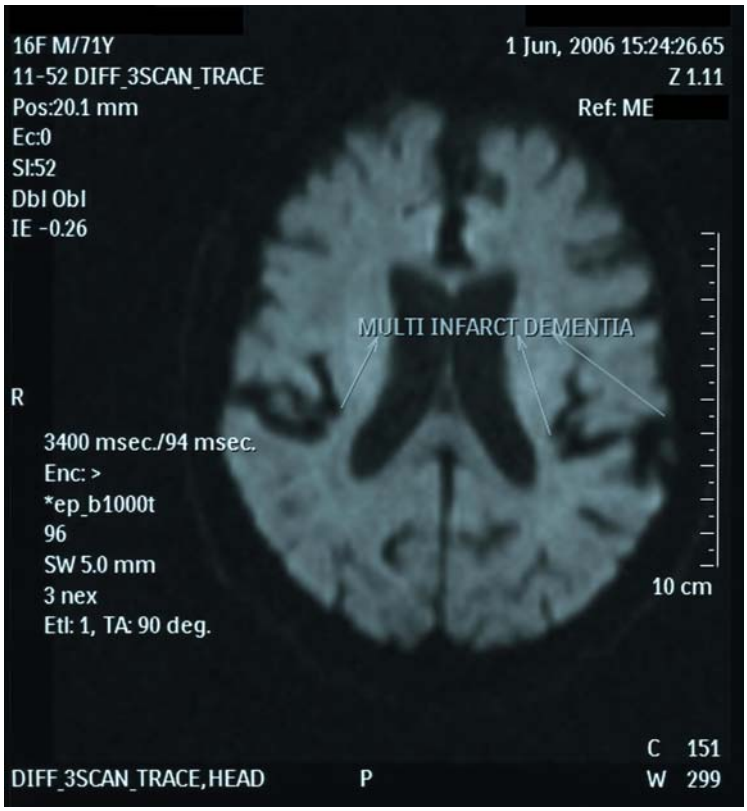

Figure 5: Multi Infarct Dementia

3.3.2 Features of infarcts

Infarcts of the brain are caused by a severe reduction of blood flow usually due to atheroma of the cerebral or internal carotid arteries (in the brain) or vertebral arteries (in the neck). The causes of impaired blood flow can be due to an arterial occlusion, thrombus formation, embolus, stenosis or even severe hypotension.[1,16]

The infarction site depends on the cause of ischaemia. Major cerebral artery blockage will lead to an infarction within the territory supplied by it. Blockages of the internal carotid or vertebral arteries, as well as hypotensive episodes will result in infarctions of the boundary zones.[16] Borderzone (boundary) infarcts are

ischaemic lesions that occur at the junctions of 2 or 3 arterial territories and is also referred to as watershed infarcts (see Figure 6). Ten percent of all brain infarcts are located in borderzone areas,[37] which are located in the most distal part of the perfusion area of the cerebral arteries, or between the superficial and deep supply area of the middle cerebral artery.[38] Risk factors include systemic hypotension, carotid stenosis, occlusion and micro-emboli. [37]

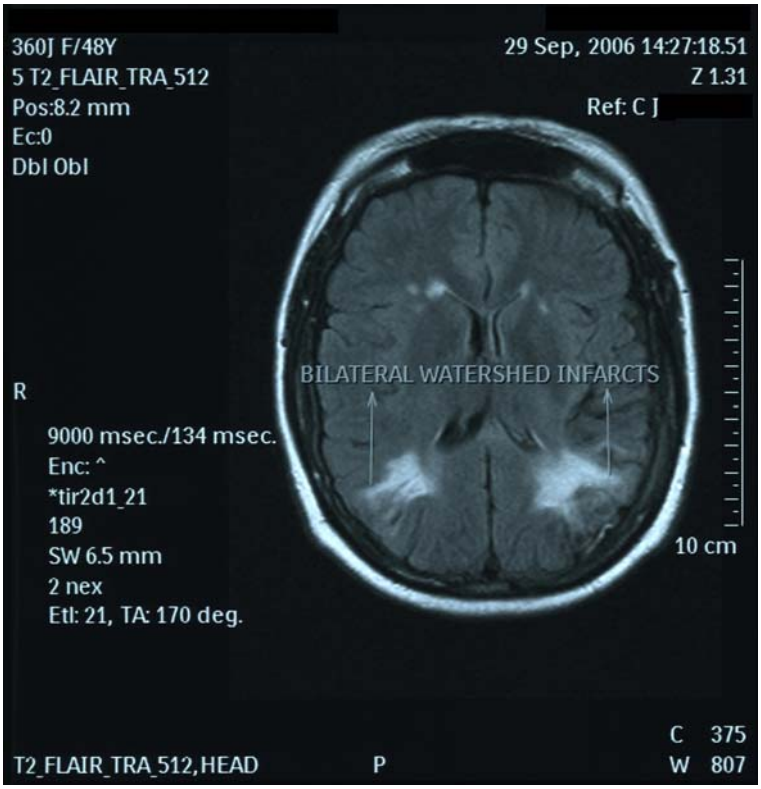

Figure 6: Bilateral watershed infarcts

Every infarct has a central core that consists of total ischaemia and necrosis, which is irreversible. This area is surrounded by a zone of borderline ischaemic tissue (ischaemic penumbra) which receives collateral circulation. Penumbral ischaemia can cause electrical failure, but it cannot result in irreversible structural damage. Prompt injection, within 3-4 hours with thrombolytic agents, such as tPA, to restore circulation inside of the penumbra, may prevent any structural damage from occurring and possibly also penumbral necrosis.[39,40]

PLAT (tPA/ Tissue plasminogen activator) is a thrombolytic agent that dissolves blood clots.[39] It is used in diseases such as myocardial infarction and stroke. But in order for it to be effective it has to be administered intravenously within 3 hours of the event or within 6 hours to the site of the occlusion via an arterial catheter.[40] In 1996 the U.S. Food and Drug Administration (FDA) approved tPA administration as a method of treatment for stroke.[41] Merino JG *et al*[42] conducted a study in which 43.4% (23/53) patients presented with a smaller baseline infarct lesion when compared to the chronic lesion after tPA treatment. After three months of receiving treatment, 60.4% (32/53) patients had an excellent clinical outcome.[42]

Prior to tPA administration, a CT scan of the brain must be performed to exclude haemorrhage in cases of a haemorrhagic stroke. If administered in such cases, the patient could bleed to death since tPA adds to bleeding and intracranial pressure.[39,43] There are two types of haemorrhagic transformation. Haemorrhagic infarction is caused by diapedesis of red blood cells into the infarcted tissue. It is not associated with any deterioration in neurological signs, since the bleeding does not take up mass and the tissue affected is already dead and non-functional. Parenchymal haemorrhage however, is a discrete mass that develops within the brain tissue and is a life-threatening condition.[29]

The size of an infarct depends on the efficiency of the potential collateral circulation through arteries on the surface of the brain and the circle of Willis.[29] The circle of Willis is regarded as the major source of collateral blood flow in patients presenting with severe carotid artery disease. Patients that present with an occluded internal carotid artery have post-occlusive diminished arterial pressure. Collateral blood flow via the circle of Willis in these patients is important in order to maintain cerebral perfusion at a level sufficient to accommodate metabolic demands. The circle of Willis can use the anterior communicating or the ipsilateral posterior communicating arteries as a collateral

pathway to redistribute blood flow to the deprived side of the brain. When collateral compensation mechanisms fail, obstructive internal carotid artery lesions will decrease cerebral perfusion pressure, leading to the presence of borderzone infarcts in the brain.[38]

3.3.3 Clinical Findings of infarcts

Ischaemic infarcts cause focal neurological deficits. These deficits appear abruptly in cases of embolic infarcts and evolve over a period of time (hours) in patients presenting with athero-thrombotic infarcts. The latter is often preceded by TIA's.[16]

Cerebral oedema is caused by the release of osmotically active substances such as arachidonic acid, electrolytes and lactic acid. The condition is aggravated by vascular injury and the leakage of proteins within the interstitial space. All of the interstitial fluid will accumulate within and around the infarct within 3-4 days. This is the most dangerous period of a cerebral infarct. Cerebral oedema and herniations lead to massive hemispheric infarcts that, in turn, is fatal to the patient.[16]

3.3.4 Post infarct

A cerebral infarct is also referred to as a softening, due to the dead tissue that starts to break down within a few days. The necrotic tissue is gradually removed by phagocytes, which results in a cystic shrunken area in the brain. Microscopic examination will reveal phagocytes that are filled with globules of lipid that is produced during the breakdown of myelin. After the necrotic tissue is removed, the phagocytes will become scanty and fibrillary gliosis will occur. This is when the lesion will ultimately shrink and become cystic. The adjacent lateral ventricle will enlarge due to this shrinkage.[16]

3.3.5 Infarct phases & preferred imaging modalities

Acute cerebral ischaemic infarctions are a result of interrupted blood flow to the brain that presents with variable neurological deficits. They mainly occur in one or more of the vascular territories or at the border zones. The size of these infarcts depends solely on the ability of the collateral circulation to restore blood flow. The affected area appears wedge-shaped when the grey matter is involved, but the shape varies when the white matter is affected. These infarcts are best demonstrated when using FLAIR, diffusion and perfusion MR imaging.[44]

Subacute infarctions follow about 2–14 days after the initial ischaemic event took place. They are usually located within the cerebral hemispheres, the brainstem, cerebellum and/or their vascular territories. These wedge-shaped abnormalities vary from focal to global ischaemia. These images are best visualized using diffusion weighted imaging.[44]

Chronic cerebral infarction is the end result of prolonged cerebral ischaemia that affects the cerebral hemispheres, brainstem and cerebellum. Territorial infarctions involve the brain tissues supplied by the specific cerebral artery. For example, supra-tentorial infarcts involve the middle, anterior, or posterior cerebral arterial distribution, whilst infra-tentorial infarcts involve the basilar, or posterior cerebral arterial distribution. Watershed infarcts involve the vascular territories, whilst lacunar infarcts affect the deep arterial distributions such as the basal nuclei, thalami and white matter. Normal MR or CT imaging is sufficient to visualize these lesions, but diffusion-weighted imaging is recommended to visualize lacunar infarcts.[44]

3.4 Computed Tomography (CT)

3.4.1 Computed tomography techniques

During the computed tomography procedure anatomical structures are separated at different depths within the body. The source/detector make a complete 360 degree rotation about the subject to obtain a complete set of data from which images are reconstructed. Multi slice scanners have more than one detector. Scanners with 4, 8, 16, 32, 40 and 64 detector rings and increasing rotation speeds are currently available and in use in South Africa. Current models have up to 3 rotations per second and an isotropic resolution of 0.35mm voxels with z-axis scan speeds of up to 18 cm/s. The major benefit of multi-slice CT is the increased speed of volume coverage. Large volumes can therefore be scanned at the optimal time of intravenous contrast administration.[45]

During a computed tomography procedure, the computer stores a large amount of data (attenuation values) from a pre-selected region of the body i.e. the brain, thorax, abdomen etc. The computed tomogram consists of a matrix of attenuation values that are depicted in various shades of grey, to create a spatial image of the scanned image. Attenuation is measured by the detectors which are aligned behind the patient, opposite to the x-ray source.[45]

A rotation scanner with stationary detectors consists of an angled fan x-ray beam than covers the entire test object. The source rotates inside or outside a stationary ring detector array with 300 to 4000 detectors in order to scan the test object. The scan time ranges from 3 to 8 seconds.[45]

3.4.2 Image reconstruction

Breathing, peristalsis and heartbeat causes motion artefacts which can be eliminated with short scanning times. The attenuation values for each projection are registered in the computer to reconstruct the CT image by means of a

complex computational process. The finite number of attenuation values is organized in matrix form. These numbers are translated into various analogous grey levels, to create a visual image of the scanned cross-sectional image. Internal structures have different absorbing capacities and are therefore identifiable on the picture image. The image matrix size depends on the number of individual projections and it therefore influences the quality of image resolution.[45]

3.4.3 Density Value

Each element has a numerical value (attenuation value) that corresponds with the amount of radiation that is absorbed by the tissue. CT density is directly proportional to the attenuation coefficient. After internal calibration, the CT density of water is set at 0 and air at -1000 Hounsfield units (HU). The various types of body tissues are therefore assigned values relative to the Hounsfield scale. See Table below.[45]

Table 1: Tissue type in Hounsfield units [45]

TISSUE TYPE	HU
BONE	> 250
THYROID	70
LIVER	65
MUSCLE	45
SPLEEN	45
PANCREAS	40
KIDNEY	30
FAT	- 65
BLOOD	80

3.4.4 CT usage

CT can be used when patients present with an acute neurological deficit to exclude haemorrhage and stroke mimickers,[1] such as cerebral oedema, vascular malformations, neoplasm's, infections, inflammatory disorders and toxic-metabolic disorders.[45]

3.4.5 CT Effectiveness

CT improvements now allow this modality to detect 75% of hyper-acute infarcts (infarcts less than 6 hours old). However, failing to discern 25% of infarcts within the first six most important hours still questions its reliability.[46] MRI is therefore considered to be the imaging modality of choice since it detects 82% of ischaemic infarcts within the first 24 hours when compared to 58% when using CT.[47] Brott *et al*[48] used CT to estimate the volume of brain damage caused by an infarct and found a correlation between the infarct volume and the neurological deficit experienced. This was also supported by Hertanu *et al*[49] who determined that the bigger the infarction size, the greater the chance of dependency. Temporal infarction, especially, is associated with a small chance of successful rehabilitation, as was demonstrated on a normal CT. Infarct size is therefore inversely related to the patient's rehabilitation ability.[49]

3.5 Magnetic Resonance Imaging (MRI)

3.5.1 Image Formation

Magnetic Resonance Imaging is a cross-sectional tomographic imaging modality. Radio waves and atomic nuclei in the body interact in the presence of a magnetic field to produce an image. Protons have charge and spin, which, when they are placed in a large magnetic field, align to the external field. When exposed to radio frequency from the gradient coils, the protons tip and precess (change of axis direction of a rotating object). This changes the magnetic field and thus a signal is received from the MRI and, due to the help of computer analysis, an image can be generated. MRI has higher contrast resolution in comparison to CT.[50] MRI allows differentiation between ischaemic and infarcted brain tissue provides excellent anatomical detail and helps to exclude intracranial haemorrhage.[44]

3.5.2 Diffusion-Weighted Imaging

MRI Diffusion-Weighted Imaging (DWI) is more than 95% sensitive when detecting infarcts within the first 6 hours from the onset of symptoms and is currently the most reliable method for infarct identification.[51,52] Diffusion weighted magnetic resonance imaging does not only detect infarcts of the brain within the first hour after the onset of symptoms, but it also allows differentiation between acute and chronic lesions. DWI can also reveal multiple acute brain infarcts which, in turn, improve our knowledge of the aetiology of stroke and development of preventative strategies in patients presenting with lacunar strokes.[53] MR images, produced with this technique, depend on the motion of water molecules in the brain.[54] Normally, extracellular fluid water motion is unrestricted and randomly oriented. This produces signal loss on diffusion-weighted images, resulting in a uniform grey appearance to normal tissue. Diseases restrict this motion of water molecules and therefore produce high signal-intensity images. During acute cerebral infarction, water molecules shift

from the extracellular fluid to the intracellular space. High signal intensity is produced on diffusion weighted images due to the severely restricted motion of water molecules confined within the cells.[51]

MRI Diffusion-Weighted Imaging is ideal when evaluating ischaemic strokes, since it is highly sensitive to the pathological changes that occur within the lesion. DWI enhancement appears within 5-10 minutes after the onset of a stroke and lasts for up to two weeks. This is a major breakthrough when compared to CT, since the latter can only detect acute infarct changes after 4-6 hours. CT is therefore used to rule out haemorrhagic stroke which is a contra-indication for the use of tPA.[39,43]

When compared to CT, DWI is 33% more sensitive in the detection of middle cerebral artery involvement when identifying acute infarctions.[54] Diffusion imaging is extremely valuable due to its ability to reliably detect hyper-acute ischaemic infarcts when other imaging modalities are still normal. It also assists with determining the age of the infarct and to distinguish between small lacunar infarcts and chronic micro-vascular changes.[53]

3.5.3 Perfusion Imaging

During MRI Perfusion Imaging, the brain is imaged whilst a bolus of Gadolinium contrast medium is injected into a peripheral vein. The passing bolus distorts the local magnetic field around the blood vessel, causing decreased signal intensity on MR images. The cerebral blood flow level is proportional to the degree of signal intensity change. The signal intensity change is mapped due to the dynamically acquired data obtained as contrast passes through the brain. Perfusion measurements can be obtained by manipulating the data mathematically, i.e. relative cerebral blood flow (rCBF) and relative cerebral blood volume (rCBV: the relative amount of blood that enters the brain).[1,50,55]

MR diffusion and perfusion imaging techniques can significantly improve the clinical decision making in, and care of, stroke patients due to its ability to identify and characterize acutely ischaemic tissue.[1]

3.5.4 FLAIR Imaging

FLAIR (Fluid-attenuated inversion-recovery) imaging has proved to be extremely valuable in the diagnosis of acute ischaemic infarctions.[51] Noguchi *et al*[56] proved that FLAIR imaging can detect infarctions that are only 2 to 3 hours old. FLAIR depicts areas of tissue T2 prolongation whilst it suppresses the signal of CSF (cerebrospinal fluid). This proves to be a very sensitive method of detecting lesions. Acute strokes can be detected after 4 to 6 hours, when T1-weighted images and T2-weighted images are still normal.[50] FLAIR imaging can therefore compete with diffusion-weighted imaging to diagnose hyper-acute cerebral infarcts. Diffusion-weighted imaging does however demonstrate more infarcts than FLAIR images and it has proved to be consistently better regarding lesion conspicuity.[51,57]

3.5.5 MRI Findings

Cerebral and cerebellar infarct MRI features depend on the age of the infarct relative to the time of the examination. An infarct that is less than 12 hours old will demonstrate an iso-intense signal to normal brain on T1-weighted images and T2-weighted images will demonstrate localized oedema. Decreased diffusion will be apparent due to cytotoxic oedema and arterial flow will be absent. Images will also demonstrate vascular infarct distribution enhancement.[58]

Infarcts aged 12 to 24 hours will present with an intermediate signal on the T1-weighted images, whilst the T2-weighted images will present with a high signal to normal brain. Localized oedema will be visible as well as signal abnormalities of the cerebral cortex and the sub-cortical white matter and/or basal nuclei. One

to three day old infarcts will present with a low or intermediate signal on the T1-weighted images, whilst the T2-weighted images will present with a high signal. Localized oedema and associated haemorrhage will become visible and enhancement might be present.[58]

Infarcts aged 4 days to 2 weeks present with a low to intermediate signal T1-weighted image, whilst the T2- weighted images present with a high signal, due to resolving oedema, or the diminishing mass-like effect. Associated haemorrhage can be seen and enhancement might be present. Two week- to 2 month-old infarcts show a low to intermediate signal on the T1-weighted images, whilst the T2-weighted images present with a high signal. In this instance oedema has been resolved and enhancement will show a steady declining appearance.[58]

Infarcts older than two months will again present with a low T1-weighted signal and a high T2–weighted image. Encephalomalacic changes will become visible and calcification might be present.[58]

Table 2: Distinguishing characteristics of new and old infarcts [59]

	New infarct	Old infarct
Aetiology	An arterial occlusion that effectively cuts off nutrients and oxygen to the affected area by means of a thrombus or embolus.	Occluded artery of circle of Willis or any of its branches or a vein by means of a thrombus or embolus.
Pathogenesis	Cerebral or arterial atherosclerosis due to age, smoking, hypertension, serum lipids, cholesterol and diabetes.	Atherosclerosis thickens and partially or completely occludes the arterial lumina. Thrombosis follows due to decreased flow or rupture of the atherosclerotic plaque. Heart diseases and abnormal heart valves give rise to emboli.
Epidemiology	Patients age 55 and over are at an increased risk of death and disability due to stroke.	Cerebro-vascular disease is the third leading cause of death following heart disease and cancer.
Gross Description	A pale infarct will present with tan discoloration, swelling and softening of the affected area. A haemorrhagic infarct's grey matter will present with multiple haemorrhages in the affected area.	Old infarcts resemble cystic spaces that vary in size, depending on the size of the arterial occlusion. It contains portions of blood vessels and glial fibres.
Microscopic Appearance	The cerebral grey and white matter of the affected area will appear pale. Neutrophils will appear within 24 hours. Macrophages will appear within 3 days to phagocytose necrotic tissue. Astrocyte borders will present with swelling or reactive change as if to wall it off.	A cystic appearance containing remnants of blood vessels and macrophages. Surrounding tissues present with myelinated fibre and neuron loss.
Clinical Correlation	Infarct patients will experience a sudden onset of focal neurological deficit, i.e. hemiparesis. This will worsen with the accompanying oedema and then stabilize. In cases of stroke with oedema, herniation and death will follow.	Sudden loss of function may appear in the form of hemiparesis. Depending on the size and the location of the old infarct, the patient may present with a fixed deficit, or present with no symptoms.

3.6 Previous studies

Population-based studies reported a 7.2% prevalence of asymptomatic brain infarcts in the general population. A 5.6% lacunar infarct prevalence was noted, whilst the rest of the infarcts were attributed to cortical infarcts. Observations noted that the incidence of asymptomatic brain infarcts increases with age.[60] Studies regarding the treatment methods of infarct patients, such as tPA (see 6.7: Treatment), especially regarding its administration site and the timeframe during which it should be administered in order to be effective, as well as indications and contra-indications have also been conducted.[29,39,40,42,43]

Several investigations[2,61] evaluated imaging modalities such as CT, MRI's perfusion and diffusion imaging, FLAIR imaging and the quickest modalities and techniques available to demonstrate brain infarcts. Both found that MRI is the best imaging modality currently to demonstrate infarcts and that diffusion and perfusion imaging can significantly improve the care of stroke patients due to its ability to identify and characterize acutely ischaemic tissue. It was also noted that FLAIR imaging is equally effective and sensitive after 48 hours of the onset of stroke symptoms to demonstrate infarcts when compared to diffusion and perfusion imaging.

Billelo *et al*[62] manually segmented DWI images and spatially transformed and registered them to a common co-ordinate system. This computed probalistic map, showed mild left-sided predominance of brain infarcts, which likely represents asymmetry in eloquence of the brain regions. No other studies have however been conducted to confirm these conclusions. Several studies[29,61] have been conducted regarding specific areas of infarct locations, but not to determine if infarcts are bound to a specific area or are more prone to occur in specific structures, i.e. centrum semi ovale, or the middle cerebral artery. Hendrikse et al [38] investigated the collateral ability of the circle of Willis when

infarcts interrupt blood flow. They concluded that insufficient collateral blood flow leads to the formation of borderzone infarcts and they provided a more in-depth investigation of borderzone infarct location and incidence.

The focus of the above-mentioned studies was confined to very specific infarct components and characteristics. A lack of knowledge exists however regarding more specific infarct information i.e. current age and sex incidence's, specific infarct locations and infarct measurements.

3.7 Treatment

Thrombolytic therapy with intravenous tPA prevents death and functional dependence. tPA administration has definite benefits, but in 30-40% of patients treated, arterial revascularization was not achieved and life-threatening haemorrhagic complications occurred in 5-10% of patients.[63] Future challenges for thrombolytic therapy includes increased administration and use of this therapy in order to increase revascularization rates, whilst decreasing post-treatment bleeding. An extended therapeutic window to include more candidates is also necessary.[64]

Boehringer Ingelheim[40,65] has announced that ACTILYSE should be used as the first and only treatment method of acute ischaemic stroke. ACTILYSE is the first recombinant human tissue-type plasminogen activator and it is a fibrinolytic agent. Its use has been approved in South Africa for patients suffering from either stroke or acute pulmonary embolism. The European Health Authorities state that ACTILYSE can reduce the disabling effects of an acute stroke and that it can improve the patients' quality of life significantly.[40,65]

Massive cerebral infarction with oedema was considered as life-threatening, untreatable and fatal. Hemi-craniectomy (decompressive surgery) has however proved to significantly reduce mortality when compared to maximal conservative treatment alone.[66] It effectively lowers the increased intracranial pressure and prevents brain herniation.[67] However, all studies performed were retrospective with low patient numbers.[66] Surgical management is aimed to improve cerebral perfusion and to prevent ischaemic damage and mechanical brain compression against the intracranial structures.[67]

Cytotoxic and vasogenic oedema due to infarction leads to swelling of the brain. Cytotoxic oedema is caused by ischaemia, which disturbs the regulatory mechanism within the cell membrane, resulting in intracellular fluid accumulation. Sometimes blood flow is restored via the collateral circulation or via fragmentation and moving of emboli. This accelerates the enlargement of the forming mass and promotes soft tissue swelling, which, in turn, increases intracranial pressure. Cerebral perfusion pressure and blood flow is further reduced resulting in further infarction. Decompressive surgery breaks this cycle.[67]

4. Materials and Methods

4.1 Methodology

This study was a longitudinal, retrospective, non-interventive, observational quantitative study.

4.2 Sample Size

Patients that were referred for brain MRI examinations at a private radiology practice in Pretoria and that presented with old or new brain infarcts, as well as chronic ischaemic changes for the period 1 January 2006 until 31 January 2007 were included in the study. A total number of 2 588 patients were referred for brain MRI's during this period. Children (newborn to 17 years) were excluded from the study, since MRI examinations are only performed on children to investigate specific brain pathologies such as tumours, hydrocephalus and sinusitis. During the above-mentioned timeframe, 454 children were examined and 290 (13.58%) patient records could not be found and were therefore excluded from this study. The total sample size for the study was therefore 1 844 patients (population examined minus children and records not found). A total of 338 (15.84%) infarcts were observed for the 13 month time period stated above.

The study sample is limited, since only patients from one practice in Gauteng was used and only patients that presented with infarcts were used for the actual study. No bias with regard to age, sex or race however exists, since all available patients were used regardless of determinants.

4.3 Methods

MRI's were carried out at a private radiology practice with a whole-body 1.5 T MRI-system (Symphony/Avanto: Siemens; General Electric). Continuous 5mm sliced T1-weighted (TR, 500 milliseconds; TE, 10 milliseconds), T2-weighted

(TR, 4 000 milliseconds; TE, 89 milliseconds), FLAIR (TR, 9 000; TE, 125 milliseconds), and Diffusion-Weighted (TR, 3100 milliseconds; TE, 89 milliseconds) scans were acquired with the fast spin-echo technique for diagnostic purposes.

All MRI examinations were noted and recorded in a logistics book by the operating personnel for reference purposes and statistics. These books were used to compile individual lists of each month, to determine the exact amount of brain MRI's performed for each individual month, for the time period 1 January 2006 until 31 January 2007. Each month's list was then analyzed and sorted according to sex (male & female) and age. Patients were then categorized according to their ages, by using 10-year interval groups i.e. 18–29 years, 30–39 years etc. This determined the most common age group and sex being affected.

The report for each brain MRI examination was obtained by using the Promed Viking System (Promed Computer Services: 1997 – 2005; Build 4.0.0.0). The reports are in a standard format and normally indicate whether or not the patient presented with a normal brain MRI examination, or with pathology. Patients were sorted according to the diagnosis made on the reports, i.e. normal, infarct, and other pathology (sinusitis, tumour, hydrocephalus, etc). Patients that presented with old or new brain infarcts or acute or subacute ischaemic incidents were noted as infarcts for this study. This allowed the calculation of the number of infarcts present for each individual month as well as for the total time period.

Each report also stipulates the exact location of the infarct, i.e. new left frontal lobe and left middle cerebral artery infarct. It further specifies the type of infarct noted, i.e. new, embolic, cerebral infarct or old lacunar infarct in right parietal lobe. The patients were subdivided into old and new infarct categories,

according to age and sex. Each patient's infarct type and location was noted. This allowed the calculation of the infarct prevalence, according to the most dominant brain lobe, anatomical structure and infarct type.

The MR brain images of the patients that presented with brain infarcts or acute ischaemic incidents were obtained from the MRI Department at Burger Radiologists by writing these images onto CD's. The Diffusion and FLAIR images were used. Diffusion images allow visualization of new brain infarcts, whilst FLAIR images allow the visualization of old brain infarcts. A measuring program (DICOM) is automatically included when writing these images to discs. DICOM allows measuring of length, width, circumference and density (ROI).

The Diffusion-Weighted Images (B-100-T) were used to identify the brain infracts as well as the acute ischaemic lesions (see Figures 7 & 8). New infarcts appear as a high signal (white) on the images and old infarcts appear as a low signal (dark).

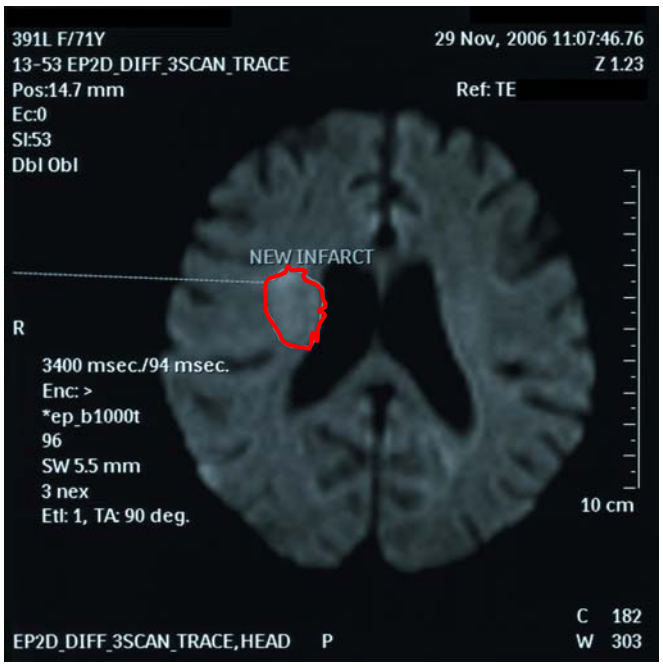

Figure 7: New infarcts appear as white densities on an MRI

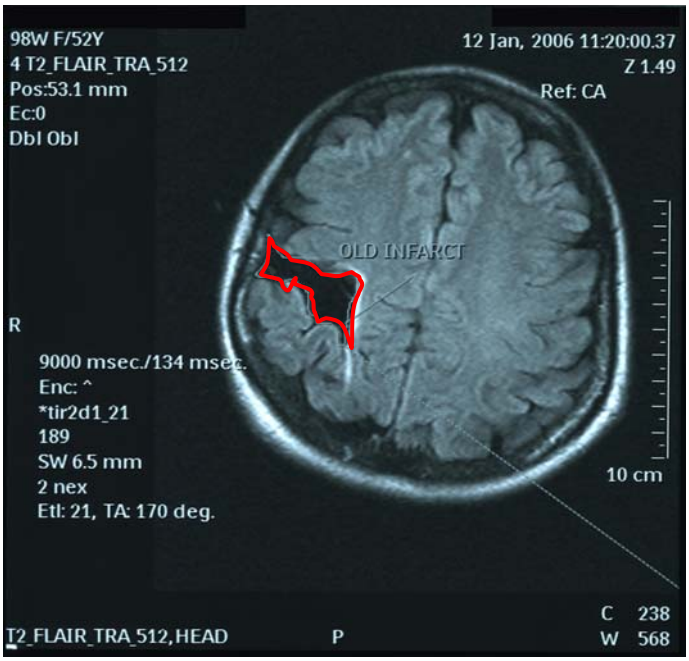

Figure 8: Old infarcts appear as black densities on an MRI

The measuring program (Syngo FastView standalone viewing tool for DICOM images from Siemens: AG 2004; Build 1.0.0.4, Berlin & München) was used to measure the areas presenting with infarcts or acute ischaemic changes. Infarcts vary greatly in size and shape. They can be microscopic or even affect an entire hemisphere. Infarcts also vary in size on each image of an MRI. All of the images of a patient were viewed to find the image that demonstrated the infarct best (see Figures 11). Only this image was used to measure the longest length, width and circumference (see Figures 9 & 10). An example is shown below. These measurements were noted for evaluation of this research study.

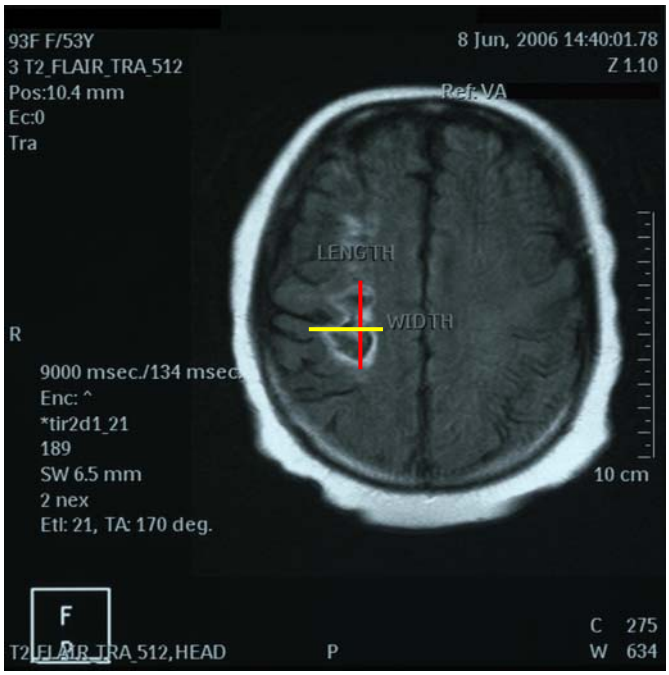

Figure 9: Measurement method (length & width)

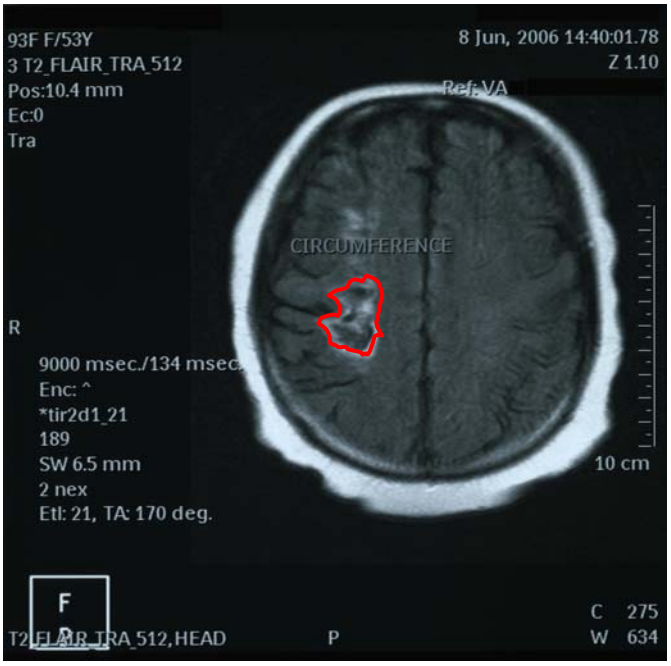

Figure 10: Measurement method (circumference)

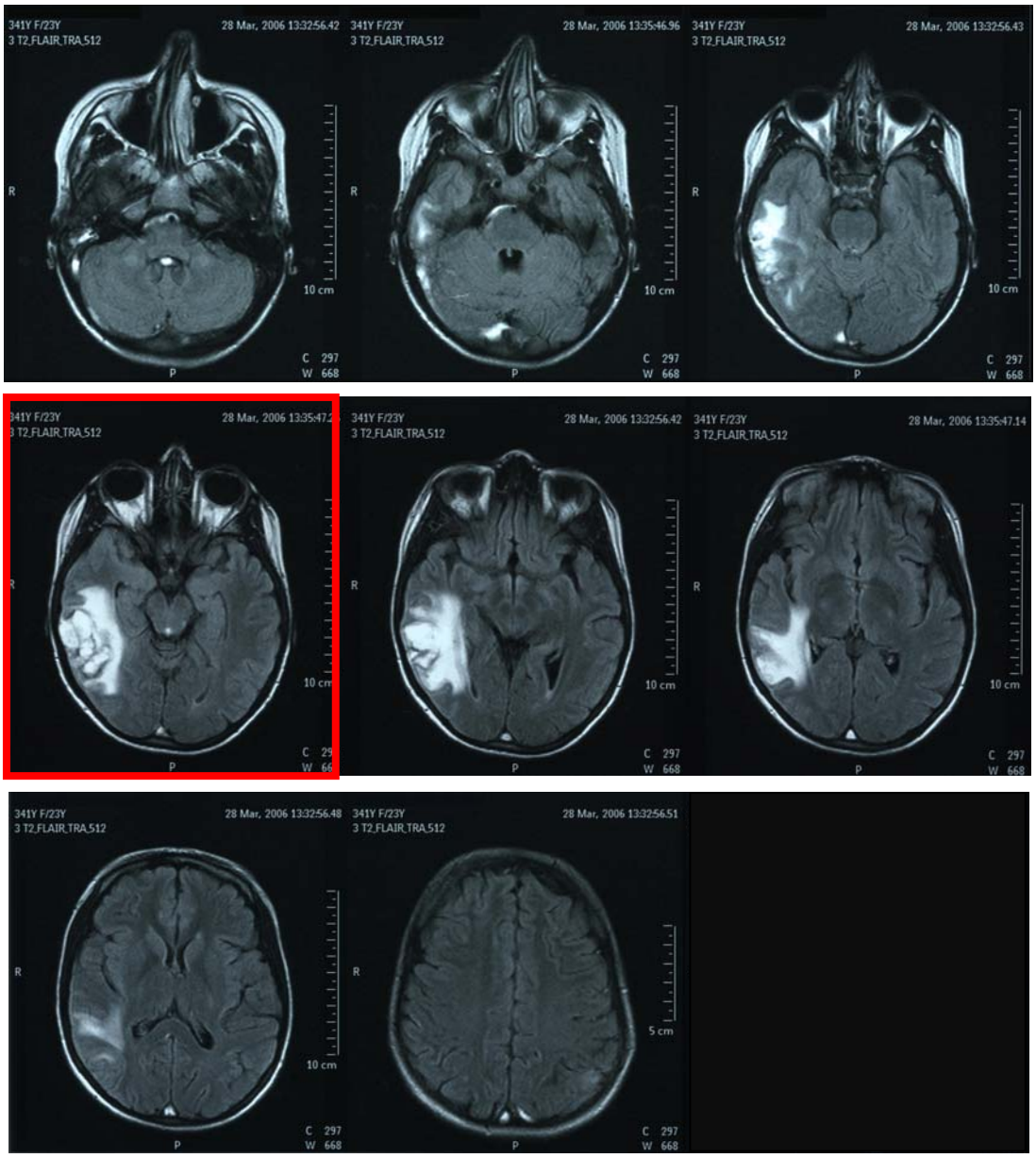

Figure 11: A typical MRI film showing the MR images that were evaluated to obtain the image where the infarct is best present

The volume of the infarcts could not be determined, due to the fact that no software is currently available. Saunders *et al*[61] made use of the ANALYZE image analysis software package to measure the infarct volume on T2-weighted MRI images. This technique was found to be highly accurate and reproducible. The package/software contains a volume estimator algorithm that randomly superimposes markings over the entire image in a three-dimensional array of

known size. The operator then selects the marks that he or she perceives to be within the infarct. This method is particularly valuable in measuring the volume of cerebral infarction in stroke where the border is irregular and follows the lines of a sulcus. Their study showed that infarct volume and site have a major influence on outcome. Patients with infarct volumes of less than 80 cm^3 had a better outcome compared to patients with larger volumes.[61]

Martel *et al.*[68] used an adaptive segmentation technique to automatically determine the brain and infarct volumes. This technique is based on the Interactive Conditional Modes (ICM) method. It allows the relationship between adjacent voxels to be taken into account during segmentation. Contrast on Diffusion-Weighted Images occur due to the mobility of water molecules in tissue, i.e. acute cerebral ischaemia causes cell damage which results in oedema (accumulation of intracellular water). Infarcts therefore show up as a region of increased signal intensity. The result is an image with three distinct regions: air (intensity=0), normal brain (intermediate grey scale) and infarcted tissue (high signal). Some infarcts are however too small or the contrast between the normal brain tissue and infarcts are too low to separate the normal brain tissue from the infarcted tissue. This limits the use of this technique severely. Also, distorted images occur due to patient motion (a symptom in stroke patients) and thus interfere with the process of correctly registering the images in order to correct it.[68]

Unfortunately, as stated previously, the software used by Saunders[61] and Martel[68] is not available for use in South Africa and therefore no volume measurements could be done for this study.

4.4 Ethical Considerations

This was a retrospective study that only made use of the patient records of patients that already presented with brain infarcts at a private radiology practice. No patient names were mentioned or used. Permission was obtained from the head of the practice to make use of the patient examinations. Ethical clearance was obtained from the Student Ethics Committee of the Faculty of Health Science at the University of Pretoria (S115/2007).

4.5 Statistics

The MRI images and reports of the patients that presented with infarcts and ischaemic changes were collected and analyzed. The images were evaluated and measurements were taken of the affected areas in question, as described above.

Categorical data was summarized and presented as frequencies, percentages, 95% confidence intervals, cross-tables, pie-charts and bar charts. Infarct sizes were summarized using descriptive statistics mean, standard deviation, median, minimum, maximum and histograms. Age and sex categories were analyzed with respect to the infarct size (width, length and circumference) using a two-way analysis of variance (ANOVA) with interaction term included. Testing was done at the 0.05 level of significance.

All data was statistically evaluated with the help of Prof PJ Becker, Chief Specialist Scientist of the MRC and Extraordinary Professor at the University of Pretoria.

5. Results

5.1 Pilot Study

5.1.1 Materials and Methods

A pilot study was performed on the patients that presented for an MRI brain examination at a private institution for the month of June 2006. A total of 248 brain MRI examinations were performed during that period. Of these, 42 examinations were of children aged newborn to 17 years. Children were excluded from the study since they mainly present with brain tumours, sinusitis, or hydrocephalus. Thirty patients' results could not be found. A total of 176 patients were therefore used as the sample size for the pilot study.

The population (total n = 176) was then divided into 2 groups: male (n = 73) and female (n = 103).

The male and female patients were further subdivided according to our respective age groups for the study namely: 18–29, 30–39, 40–49, 50–60, 61–70 and 71 years and above.

Table 3: Sample population

Age groups (years)	Male	Female
18–29	7	15
30–39	13	16
40–49	14	20
50–59	17	14
60–70	10	16
70 & >	12	22
Totals	**73**	**103**

5.1.2 Pilot study results

The male and female age groups were evaluated according to their individual reports and classified as follows: Normal, Infarct and Other pathology. The preliminary results of male pathologies are demonstrated in Table 4 and the female pathology results in Table 5.

Table 4: Pathology classification of males

Age groups (years)	Normal	Infarct	Other pathology
18–29	3	0	4
30–39	3	2	8
40–49	4	2	8
50–59	4	4	9
60–70	4	4	2
70 & >	1	6	5
Totals	**19**	**18**	**36**

Table 5: Pathology classification of females

Age groups (years)	Normal	Infarct	Other pathology
18–29	2	0	13
30–39	7	2	7
40–49	7	4	9
50–59	2	1	11
60–70	2	4	10
70 & >	2	10	10
Totals	**22**	**21**	**60**

The male patients presented with 24.65% (n = 18/73) infarcts, whilst the female patients presented with 20.38% (n = 21/103) infarcts. Both male and female groups had the highest amount of infarct patients in the age group 70 years and above. The male patients above 70 presented with 33.3% of the total infarcts (n = 6/18) compared to 47.6% of the female patients above 70 (n = 10/21) (See Table 2 & 3).

The patients that presented with infarcts were then subdivided to differentiate between old and new infarcts and Multi Infarct Dementia (MID). The male infarct classification is demonstrated in Table 6 and the female infarct classification in Table 7.

Table 6: Infarct classification in males

Age groups (years)	Multi Infarct Dementia	Old infarcts	New infarcts	Old and new infarcts
18–29	0	0	0	0
30–39	1	0	1	0
40–49	1	0	1	0
50–60	0	3	2	0
61–70	0	1	1	0
70 & >	1	1	6	0
Totals	**3**	**5**	**11**	**0**

Table 7: Infarct classification in females

Age groups (years)	Multi Infarct Dementia	Old infarcts	New infarcts	Old and new infarcts
18–29	0	0	0	0
30–39	0	0	2	0
40–49	0	1	3	0
50–60	0	0	1	0
61–70	0	1	1	0
70 & >	2	2	6	2
Totals	**2**	**4**	**13**	**2**

As demonstrated in Tables 6 & 7 respectively, male patients presented with 61% new infarcts (n = 11/18) and the female patients presented with 61.90% new infarcts (n = 13/21). Old infarcts accounted for 27.77% in male patients (n = 5/18) and 19% in female patients (n = 4/21). The age group 70 years and above again presented with the most new infarcts for both male and female patients.

The MRI reports were then evaluated to determine the specific areas of occurrence and to determine if hemispheric dominance exists. A summary of the infarct locations is demonstrated in Table 8.

Table 8: Infarct location dominance

Brain Region	Number of patients with infarct sites
Frontal lobe	9
Parietal lobe	6
Occipital lobe	4
Cerebral arteries	4
Centrum semi ovale	6
Cerebellum	6
Pons	5
Brainstem	2
Caudate nucleus	1
Totals	**43**

Eight patients presented with infarcts that occurred in the left hemisphere, compared to a total of twenty patients that presented with infarcts in the right hemisphere. Only two patients presented with bilateral hemispheric infarcts. Further evaluation indicated that most infarcts, 20.98% (9/43) occurred in the frontal lobe of the brain and the least amount, 2.33% (1/43) occurred in the caudate nucleus. The parietal lobe, cerebellum and centrum semi ovale (CSO) presented with a steady amount, 13.95% (6/43) of infarcts (see Table 8).

5.1.3 Pilot study measurements

MRI images were obtained of patients that presented with old and new infarcts and Multi Infarct Dementia and their respective infarcts were measured. The maximum length, width and circumference were noted, as well as the number of infarcts. Male infarct measurements are demonstrated in Table 9 and female infarct measurements in Table 10.

The ANOVA statistical analysis was used to analyze the presenting size of infarcts. The age groups were modified from six groups to three groups, since the sample size used was too small to give an accurate ANOVA analysis. This was done for both the male and female populations. Ages 40-59 years, 60-69 years and 70 years and above were grouped together, while the age group 18-39 years were excluded for the pilot study, due to the small number of individuals in the group. Thus, only one male patient and two female patients were excluded for this test.

Table 9: Comparison of the presenting size of infarcts according to age groups in males

Male age	Number of presenting infarcts	Hemisphere	Length (in mm)	Width (in mm)	L x W (mm²)	Circumference (mm)
40-59	14	Right	16.2	14.2	230	63.4
		Right	12.9	17.1	220.6	54.4
		Right	8.1	8.6	69.7	64.7
		Right	10.4	10.1	105	43.2
		Right	13.2	14.8	195.4	50.7
		Right	14.9	5.9	87.9	55.5
		Right	11.6	11.3	131	41.9
		Right	15.4	16.0	246	54.0
		Right	9.0	12.9	116	30.1
		Left	14.3	8.1	115.8	38.0
		Left	71.7	37.3	2674.4	227.2
		Left	40.3	26.5	1068	116.7
		Left	25.2	12.6	317.5	71.8
		Left	58.7	35.6	2089.7	163.4
60-69	5	Right	21.4	20.4	436.6	107.5
		Right	39.7	21.9	869.4	121.9
		Right	30.8	19.1	588.3	94.8
		Left	23.7	21.4	507.2	111.7
		Left	54.9	28.9	1586.6	228.3
70 & >	21	Right	22.4	23.9	535.4	113.7
		Right	30.5	2.4	73.2	108.5
		Right	45.7	12.0	548.4	129.5
		Right	6.4	6.4	41	30.1
		Right	8.1	6.6	53.5	29.6
		Right	6.4	3.8	24.3	25.0
		Right	57.7	2.4	138.5	118.0
		Right	22.4	10.8	241.9	70.9
		Right	11.6	13.3	154.3	53.1
		Right	11.5	12.0	138	43.0
		Right	20.7	14.8	306.4	76.0
		Right	10.2	8.3	84.7	33.5
		Right	20.3	8.0	162.4	45.0
		Right	7.5	4.6	34.5	19.7
		Right	10.2	9.9	101	34.3
		Right	9.9	7.8	79.6	29.9
		Right	9.4	8.3	78	34.7
		Left	18.2	22.6	411.3	97.5
		Left	54.5	24.2	1319	148.4
		Left	18.1	14.9	269.7	54.4
		Left	9.2	9.6	88.3	40.0

Table 10: Comparison of the presenting size of infarcts according to age groups in females

Female age	Number of presenting infarcts	Hemisphere	Length (in mm)	Width (in mm)	L x W (mm²)	Circumference (mm)
40-59	3	Right	4.6	4.6	21.2	13.4
		Right	5.8	6.7	38.9	30.5
		Right	8.1	8.2	66.4	41.2
60-69	2	Left	22.9	25.9	593	89.9
		Left	19.9	8.5	169.2	61.3
70 & >	18	Right	13.7	10.2	139.7	49.4
		Right	33.5	25.5	854.3	113.6
		Right	8.1	9.8	79.4	27.5
		Right	36.8	25.3	931	122.1
		Right	17.4	20.8	361.9	72.0
		Right	57.8	31.5	1820.7	165.8
		Right	13.3	13.3	176.9	58.6
		Right	17.2	14.4	247.7	57.4
		Right	13.1	11.3	148	44.8
		Right	21.2	10.6	224.7	44.6
		Left	14.6	20.2	294.9	64.6
		Left	17.0	24.6	418.2	78.5
		Left	34.6	14.2	491.3	102.3
		Left	6.4	10.0	64	31.4
		Left	9.5	8.2	77.9	35.0
		Left	6.1	8.2	50	25.5
		Left	28.5	13.6	387.6	92.9
		Left	96.2	29.1	2799.4	386.4

A total of 5 patients' images were not found and were thus not included in the statistical analysis.

A Chi-square[2] Goodness-of-fit test was used to compare the frequency of infarcts in the left and right hemispheres of the brain. It was hypothesized that the left hemisphere of the brain will present with a mild dominance.[17] A significant deviation from the hypothesized hemisphere was found (Chi-square[2] (5) = 0.018, $p<0.05$), with the right side showing more infarcts.

The mean lengths of infarcts (as present on the MRI images) were compared between and within the respective male and female groups using a one-way ANOVA. No significant difference was found ($F(5,52) = 1.253$, $p>0.05$).

However, a significant difference was found with regard to the width of the presenting infarcts ($F(5,52) = 3.255$, $p<0.05$).

The surface area (L x W) and the circumference between males and females and their respective age groups were also compared using the ANOVA test. Statistically no significant difference was found regarding the surface area of infarcts ($F(5,57) = 1.32$, $p>0.05$). Men appeared to have a larger brain infarct surface area when compared to their female counterparts. The standard deviation was also high, which indicate that infarcts vary greatly in size.

A significant difference was found regarding the circumference of the presenting infarcts ($F(5,57) = 1.6$, $p<0.05$). Men aged 60-69 years, appeared to have a significantly larger circumference with respect to the 70 & above age group, as well as the younger females (40-59 years).

The same test was conducted to compare the incidence of brain infarcts in males and females according to the age groups as stipulated above. A significant difference was found amongst men aged 40-59 years and males aged 70 years and above (7.72 ± 3.06 – mean ± sd) ($p=0.015$). Older men aged 70 years and above, presented with the highest infarct incidence when the male age groups were compared. Women aged 40-59 years also presented with more infarcts when compared to their male counterparts (12.97 ± 5.11) ($p=0.014$).

Women aged 70 years and above, also presented with more infarcts when compared to women aged 40-59 years (-10.6 ± 3.82) ($p=0.008$). Men aged 70 years and above, however, still presented with more infarcts when compared to their female counterparts (5.4 ± 2.50) ($p=0.037$).

5.1.4 Conclusion of pilot study

This pilot study's preliminary conclusion indicated that infarcts mainly occur in the right hemisphere of the brain. Significance also exists concerning the width of infarcts when comparing male and female patients. Men aged 60-69 years also appeared to have a significantly larger circumference with respect to their older male counterparts (70 & above) as well as the younger females (40-59 years).

Men aged 70 years and above presented with more infarcts than their female counterparts. Women aged 40-59 years presented with more infarcts when compared to their male counterparts of the same age. Women aged 70 years and above still presented with higher infarct prevalence when compared to the other female age groups. Men aged 70 years and above therefore presented with the highest number of infarcts.

5.2 Prevalence

5.2.1 Total infarct prevalence

A total of 2 588 Brain MRI examinations were performed at Unitas Hospital's MRI department for the period 1 January 2006 until 31 January 2007. Of these, 290 patient records could not be found and a total of 454 patients were children (newborn to 17 years). The reports that could not be found were due to administrative errors. The reports were hand-written by the doctors and never captured on the system. The sample size for this study was therefore 1 844 patients.

Two hundred and ninety nine patients from the resulting sample size presented with infarcts. This gives an infarct prevalence of 16.10% for the 13 month period. Of these, 21 patients presented with more than one infarct, i.e. an old and a new infarct simultaneously. A further 18 patients presented with infarcts that were classified as old and lacunar. The total number of infarcts was therefore 338. The individual monthly statistics are provided in Appendix B.

5.3 Infarct categories (old, new, other)

5.3.1 Prevalence of infarct categories

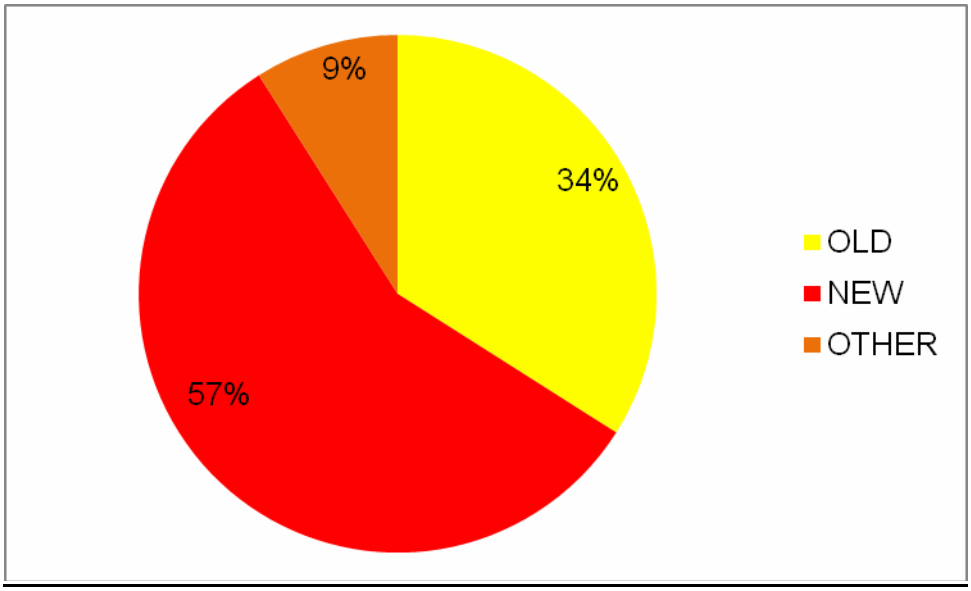

Figure 12: The prevalence of infarct categories

The pie chart (Figure 12) shows that from the 338 infarcts noted, 56.80% were new, 33.73% were old and 9.47% other types of infarcts i.e. lacunar, venous, watershed etc.

5.3.2 Prevalence of infarct categories according to age

Table 11: Prevalence of infarct categories according to age

Age groups	New		Old		Other		Total	
	n	%	n	%	n	%	n	%
18–29	7	3.65	2	3.13	1	2.96	10	2.96
30–39	19	9.9	9	7.89	-	-	28	8.28
40–49	16	8.33	15	13.16	2	6.25	33	9.76
50–59	37	19.27	21	18.42	6	18.75	64[b]	18.93
60–69	32	16.67	20	17.54	-	-	52[b]	15.38
70–79	51	26.56	32	28.07	23	71.88	106[a,b]	31.36
> 80	27	14.06	14	12.28	-	-	41	12.13
Unknown	3	1.56	1	0.88	-	-	4	1.18
Total	**192**	**56.80**	**114**	**33.73**	**32**	**9.47**	**338**	**100%**

a **Age group most affected by infarcts**

b **Age groups presenting with highest infarct prevalence's**

It is clear that people aged 70–79 years of age presented with the highest prevalence (31.36%)[a] of infarcts in total, as well as in the individual categories (new: 26.56%, old: 28.07%, other: 71.88%).

When looking at the infarct totals, it is noted that age groups 50–59 years (18.93%)[b], 60–69 years (15.38%)[b] and 70–79 years of age (31.36%)[a] are most affected by infarcts. A decrease of presence is noted in the age groups > 80 years (12.13%), 30–39 years (8.28%) and 40–49 years (9.76%). The age group 18–29 years presented with the least amount of infarcts (new: 3.65% & old: 3.13%).

Table 11 above also illustrates that other infarct types (venous, lacunar, etc.) mainly affect the elderly population (70–79 years)[a]. Age groups 30–39 years, 60–69 years and > 80 years of age were not affected by these types of infarcts. Age groups 50–59 years (18.75%), 40–49 years (6.25%) and 18–29 years (2.96%) also presented with other types of infarcts.

New infarcts mainly affected age groups 70–79 years (26.56%), 50–59 years (19.27%), 60–69 years (16.67%) and above 80 years of age (14.06%). Age groups 40–49 years presented with 16 new infarcts, 30–39 years with 19 infarcts and age group 18–29 years with only 7 infarcts.

Old infarcts were least noted in patients categorized in age groups 18–29 years (3.13%), 30–39 years (7.89%), above 80 years of age (12.28%) and 40–49 years of age (13.16%). Most of the old infarcts presented in patients in age groups 70–79 years (28.07%), 50–59 years (18.42%) and 60–69 years (17.54%).

5.3.3 Prevalence of infarct categories according to sex

Table 12: Prevalence of new infarcts according to sex

Age groups	Male		Female		Total	
	n	%	n	%	n	%
18–29	3	3.03	4	4.30	7	3.65
30–39	6	6.06	13	13.98	19	9.09
40–49	12	12.12	4	4.30	16	8.33
50–59	23	23.23	14	15.05	37	19.27
60–69	16	16.16	16	17.02	32	16.67
70–79	32	32.32[a]	19	20.43[b]	51	26.56
> 80	7	7.07	20	21.51	27	14.06
Unknown	-	-	3	3.23	3	1.56
Total	**99**	**51.56**	**93**	**48.44**	**192**	**100**

a Male age group most affected by new infarcts

b Female age group most affected by new infarcts

The number of males and females that presented with new infarcts was almost equal (99 *versus* 93) and all of the age groups were involved.

Males in age groups 70–79 years (32.32%)[a], 50–59 years (23.23%), 60–69 years (16.16%) and 40–49 years (12.12%) were mainly affected when only observing new infarcts. Age groups 30–39 years (6.06%) and above 80 years of age (7.07%) showed a much lower prevalence, whilst males in age group 18–29 years (3.03%) presented with the least amount of new infarcts.

Females however, showed a higher prevalence in the elderly population. Age groups above 80 years (21.51%)[b], 70–79 years (20.43%)[b], 60–69 years (17.2%), 50–59 years (15.05%) and 30–39 years (13.98%) showed the highest incidence's. The least amount of new infarcts noted was in age groups 18–29 years and 40–49 years (4 females in each group).

Table 13: Prevalence of old infarcts according to sex

Age groups	Male		Female		Total	
	n	%	n	%	n	%
18–29	-[b]	-	2	23.23	2	21.75
30–39	-[b]	-	9	14.52	9	97.89
40–49	8	15.38	7	11.29	15	13.16
50–59	12	23.08	9	14.52	21	18.42
60–69	10	19.23	10	16.13	30	26.32
70–79	16	30.77[a]	16	25.81[a]	32	28.07[a]
> 80	6	11.54	8	12.90	14	12.28
Unknown	-	-	1	1.61	1	0.88
Total	**52**	**45.61**	**62**	**53.39**	**114**	**100**

a Age group most affected by old infarcts

b Males aged 18–39 years did not present with any old infarcts

Table 13 illustrates that more females (62/114 patients) than males (52/114 patients) presented with old infarcts. All female age groups were affected, but males in age groups 18–39 years[b] were not affected. In both males and females, the highest old infarct prevalence was noted in age groups 70–79 years (females [f]: 25.81%, males [m]: 30.77%)[a] and 60–69 years (f: 16.13%, m: 19.23%) of age.

Males categorized in age groups 40–49 years (15.38%), 50–59 years (23.08%) and above 80 years of age (11.54%) were also affected. Females showed a much lower prevalence in the other age groups affected when compared to the categories 60–69 years and 70–79 years. Age groups 18–29 years (3.23%), 30–39 years (14.52%), 40–49 years (11.29%), 50–59 years (14.52%) and above 80 years of age (12.9%) all presented with old infarcts.

Table 14: Prevalence of other infarct types according to sex

Age groups	Male		Female		Total	
	n	%	n	%	n	%
18–29	-	-	1	4.55	1	3.13
30–39	-	-	-	-	-	-
40–49	-	-	2	9.09	2	6.25
50–59	1	10	5	22.73	6	18.75
60–69	-	-	-	-	-	-
70–79	9	90[a]	14	63.64[a]	23	71.88[a]
> 80	-	-	-	-	-	-
Unknown	-	-	-	-	-	-
Total	**10**	**31.25**	**22**	**68.75**	**32**	**100**

a Age groups most affected by other infarct types

Males only accounted for 31.25% of the other infarct type population as demonstrated in Table 14. Only males in age groups 50–59 years (1 male) and 70–79 years (9 males)[a] were affected.

Females contributed 68.75% of the other infarct type population affected. Only age groups 18–29 years (1 female), 40–49 years (2 females), 50–59 years (4 females) and 70–79 years (14 females: 68.75%)[a] were affected.

5.4 Different infarct types (embolic, lacunar etc.)

5.4.1 Prevalence of different infarct types

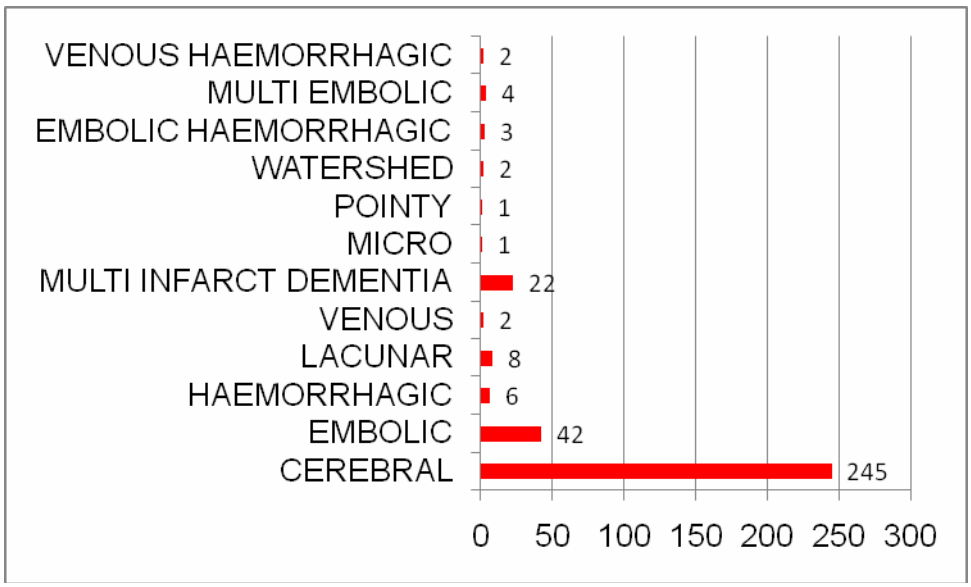

Figure 13: Prevalence of different infarct types

Figure 13 indicates that cerebral infarcts are the most common infarcts found (72.49%). Some patients presented with more than one type of infarct i.e. embolic & haemorrhagic, etc. These figures were thus added to the separate infarct types as well, to get an accurate prevalence number. An example is demonstrated below:

Embolic infarcts: 42 (embolic only) + 3 (embolic & haemorrhagic) + 4 (multi embolic infarcts)
= 49 patients in total
= 14.5%

The prevalence of the different types of infarcts is therefore as follows:
Embolic infarcts were present in 14.5% of the infarct population. MID (Multi Infarct Dementia) syndrome was seen in 7.7% of the total population and haemorrhagic infarcts were found in 3.25% of the population. Lacunar infarcts followed with a prevalence of 2.37%, venous infarcts with 1.18% and watershed

infarcts with a mere 0.59%. The least number of infarcts was seen in the micro and pointy infarct type categories (1 patient each).

5.4.2 Prevalence of different infarct types according to infarct categories

Table 15: Prevalence of different infarct types

Infarct types	New		Old		Other		Total	
	n	%	n	%	n	%	n	%
Cerebral	141	73.44	104	91.23	-	-	245	72.49
Embolic	42	21.88	-	-	-	-	42	12.43
Haemorrhagic	4	2.08	2	1.75	-	-	6	1.78
Lacunar	2	1.04	5	4.39	1	3.13	8	2.37
Venous	-	-	1	0.88	1	3.13	2	0.59
Multi	-	-	-	-	22	68.75	22	6.51
Micro	-	-	-	-	1	3.13	1	0.30
Pointy	1	0.52	-	-	-	-	1	0.30
Watershed	-	-	-	-	2	6.25	2	0.59
Embolic & haemorrhagic	1	0.52	2	1.75	-	-	3	0.89
Multi embolic	-	-	-	-	4	12.5	4	1.18
Venous haemorrhagic	1	0.52	-	-	1	3.13	2	0.59
Total	**192**	**56.80**	**114**	**33.73**	**32**	**9.47**	**338**	**100**

Cerebral infarcts accounted for 72.49% of all infarcts noted. Of these, 57.55% were new infarcts and 42.45% old infarcts.

Embolic infarcts only presented as new infarcts and accounted for 21.88% of the total number of new infarcts noted. Haemorrhagic infarcts presented with old (2 cases) and new (4 cases) infarcts. Lacunar infarcts also presented with old (5 cases) and new (2 cases) infarcts.

Only one old venous infarct was noted and one new pointy and venous haemorrhagic infarct was noted. Embolic haemorrhagic infarcts presented with old (2 cases) and new (1 case) infarcts.

MID, micro-, watershed- and multi embolic infarcts did not present with old or new infarcts.

5.4.3 Prevalence of different infarct types according to age groups

Table 16: Prevalence of different infarct types according to age groups

Age groups	18–29		30–39		40–49		50–59		60–69		70–79		> 80		Total
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	
Cerebral	5	2.04	25	10.2	27	11.02	45	18.37	44	17.96	63[a]	25.71	32	13.06	245
Embolic	1	2.38	3	7.14	2	4.76	7	16.67	5	11.90	18[b]	42.86	6	14.29	42
Haemorrhagic	1		-	-	1		2		1		1		-	-	6
Lacunar	-	-	-	-	-	-	4	50[c]	1		1		2		8
Venous	2		-	-	-	-	-	-	-	-	-	-	-	-	2
Multi	-	-	-	-	-	-	-	-	-	-	22[d]		-	-	22
Micro	-	-	-	-	-	-	-	-	-	-	1		-	-	1
Pointy	-	-	-	-	1		-	-	-	-	-	-	-	-	1
Watershed	-	-	-	-	2		-	-	-	-	-	-	-	-	2
Embolic & haemorrhagic	-	-	-	-	-	-	1		1		-	-	1		3
Multi embolic	-	-	-	-	-	-	4[e]		-	-	-	-	-	-	4
Haemorrhagic venous	1		-	-	-	-	1		-	-	-	-	-	-	2
Total	**10**		**28**		**33**		**64**		**52**		**106**		**41**		**338**

a Age group most affected by cerebral infarcts

b Age group most affected by embolic infarcts

c Age group most affected with lacunar infarcts

d Only age group that presented with multi infarct dementia

e Only age group presenting with multi embolic infarcts

Table 16 indicates that most cerebral infarcts (63/245)[a] are found in the 70–79 year age group. Embolic infarcts were seen in all of the age groups, but presented with the highest prevalence (18/42)[b] also in the 70–79 year age group. Haemorrhagic infarcts showed an even distribution (1-2 per age group) throughout the age groups, but was absent in the age groups 30–39 years and above 80 years of age.

Lacunar infarcts only presented in patients aged 50 years and older and was completely absent in younger patients. The highest lacunar infarct prevalence (4/8)[c] was seen in the 50–59 year age group.

Venous infarcts were only found in the 18–29 year age group. MID syndrome only affected patients in age group 70–79 years (22 patients)[d]. Only 1 patient presented with a micro infarct and was in the age group 70–79 years. A pointy infarct was also only found in 1 patient in the 40–49 year age group. Two

patients presented with watershed infarcts and were both in the 40–49 year age group.

Embolic haemorrhagic infarcts were seen in the age groups 50–69 years and above 80 years of age, whilst venous haemorrhagic infarcts affected patients in the 18–29 and 50–59 year age groups only. Multi embolic infarcts were only present in the 50–59 year age group (4 patients)[e].

5.4.4 Prevalence of different infarct types according to sex

Table 17: Prevalence of embolic, haemorrhagic, lacunar and venous infarcts according to age & sex

	Embolic infarcts				Haemorragic infarcts				Lacunar infarcts				Venous infarcts			
Age groups	**Male**		**Female**		**Male**		**Female**		**Male**		**Female**		**Male**		**Female**	
	n	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**
18–29	1	4.76	-	-	-	-	1	50	-	-	-	-	-	-	2	100[e]
30–39	1	4.76	2	9.53	-	-	-	-	-	-	-	-	-	-	-	-
40–49	2	9.53	-	-	1[c]	25	-	-	-	-	-	-	-	-	-	-
50–59	2	9.53	5	23.81	1[c]	25	1	50	2	40	2	66.67	-	-	-	-
60–69	1	4.76	4	19.05	1[c]	25	-	-	1	20	-	-	-	-	-	-
70–79	12[a]	57.14	6[b]	28.57	1[c]	25	-	-	-	-	1	33.33	-	-	-	-
> 80	2	9.53	4	19.05	-	-	-	-	2	40	-	-	-	-	-	-
Unknown	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Totals	21		21		4		2		5[d]		3		-		2	

a Male age group most affected by embolic infarcts

b Female age group most affected by embolic infarcts

c Haemorrhagic infarcts had an equal prevalence throughout age groups 40–79 years

d Males presented with more lacunar infarcts compared to their female counterparts

e Only 2 females aged 18–29 years presented with venous infarcts

According to Table 17, the most common age group, regardless of sex, that presented with embolic infarcts was patients in age group 70–79 years (18/42 patients). The total number of males that were affected was equal to the number of females. The male population was affected throughout all of the age groups. Age group 70–79 years presented with 57.14%[a] embolic infarcts in males, followed by 9.52% in males categorized in age group 40–59 years and above 80 years of age. A mere 4.76% of embolic infarcts in males were seen in age groups 18–39 years and 60–69 years. Females aged 18–29 years and 40–

49 years presented with no embolic infarcts. The highest embolic infarct prevalence in females was noted in the age group 70–79 years (28.57%)[b]. This was followed by age groups 50–59 years (23.81%) and 60–69 & > 80 years (19.05%). A prevalence of 9.52% was noted in females categorized in age group 30–39 years.

Haemorrhagic infarcts mainly presented in the age group 50–59 years of age (2/6 patients).The number of males affected was double the number of females (4 *versus* 2). Males in age groups 18–39 years and above 80 years were completely unaffected by haemorrhagic infarcts. The rest of the male age groups (40–79 years) presented with an equally distributed 25%[c] prevalence in each 10-year interval group. Only females in age groups 18–29 years and 50–59 years presented with haemorrhagic infarcts and each group had only one patient each.

Fifty percent (4/8 patients)[d] of the lacunar infarct population affected was patients of age group 50–59 years. In both sexes, age groups 18–49 years were unaffected, as well as males in age group 70–79 years and females of age group 60–69 years. Males accounted for (5/8)[d] of the lacunar population compared to (3/8) females. Two males presented with lacunar infarcts in age group 50–59 years and above 80 years and only one male aged 60–69 years. Female lacunar infarcts were mainly found in patients categorized in age group 50–59 years (2 patients) *versus* only one female in age group 70–79 years.

Venous infarcts were infrequently found[e] as seen in Table 17 and only affected females in the 18–29 year age group.

Table 18: Prevalence of MID, micro, pointy and watershed infarcts according to age & sex

	Multi Infarct Dementia		Micro infarcts		Pointy infarcts		Watershed infarcts	
Age groups	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
18–29	-	-	-	-	-	-	-	-
30–39	-	-	-	-	-	-	-	-
40–49	-	-	-	-	1	-	-	2
50–59	-	-	-	-	-	-	-	-
60–69	-	-	-	-	-	-	-	-
70–79	8[a]	14[a]	1	-	-	-	-	-
> 80	-	-	-	-	-	-	-	-
Unknown	-	-	-	-	-	-	-	-
Totals	**8**	**14**	**1**	**-**	**1**	**-**	**-**	**2**

a MID only affected 70–79 year age group

As seen from Table 18, MID only affected patients of age group 70–79 years. Eight males (36.36%) *versus* 14 (63.64%) females presented with this type of infarct. Micro infarcts were only noted in one male patient in the 70–79 year age group and only one male patient in the 40–49 year age group presented with a pointy infarct. Two females in the 40–49 year age group presented with watershed infarcts.

Table 19: Prevalence of embolic haemorrhagic, multi embolic and venous haemorrhagic infarcts according to age & sex

	Embolic haemorrhagic infarcts		Multi embolic infarcts		Haemorrhagic venous infarcts	
Age groups	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
18–29	-	-	-	-	-	1
30–39	-	-	-	-	-	-
40–49	-	-	-	-	-	-
50–59	-	1	-	4[a]	-	1
60–69	1	-	-	-	-	-
70–79	-	-	-	-	-	-
> 80	1	-	-	-	-	-
Unknown	-	-	-	-	-	-
Totals	**2**	**1**	**-**	**4**	**-**	**2**

a Multi embolic infarcts only affected females in the 50–59 year age group

Table 19 shows that embolic, haemorrhagic infarcts only affected one female aged 50–59 years and two males aged 60–69 years and above 80 years of age. Multi embolic infarct dementia was only noted in four females and all were in age group 50–59 years.

Haemorrhagic venous infarcts only affected two females, one in age group 18–29 years and the other in the 50–59 years age group.

5.5 Hemispheric predominant infarcts (left, right, bilateral)

5.5.1 Prevalence of infarcts according to hemispheric predominance

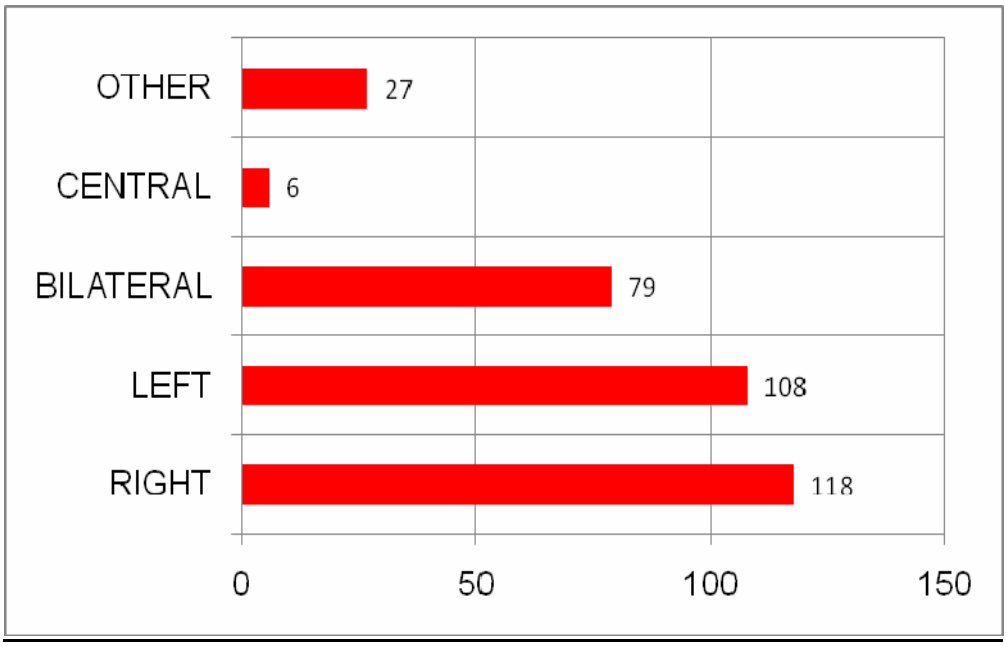

Figure 14: Prevalence of infarcts according to hemispheric predominance

Figure 14 demonstrates that infarcts are more or less evenly distributed between the left and right hemispheres. The right hemisphere presented with 118 patients (34.91%) and the left with 108 patients (31.95%). The left to right hemisphere infarct ratio was calculated at 1:1.09. Seventy nine patients (23.37%) presented with infarcts that affected both of the hemispheres. Centrally located structured infarcts were found in 6 patients (1.78%), and other infarct types, such as watershed, lacunar, etc., accounted for the remaining 7.99%.

5.5.2 Prevalence of hemispheric predominant infarcts according to infarct categories

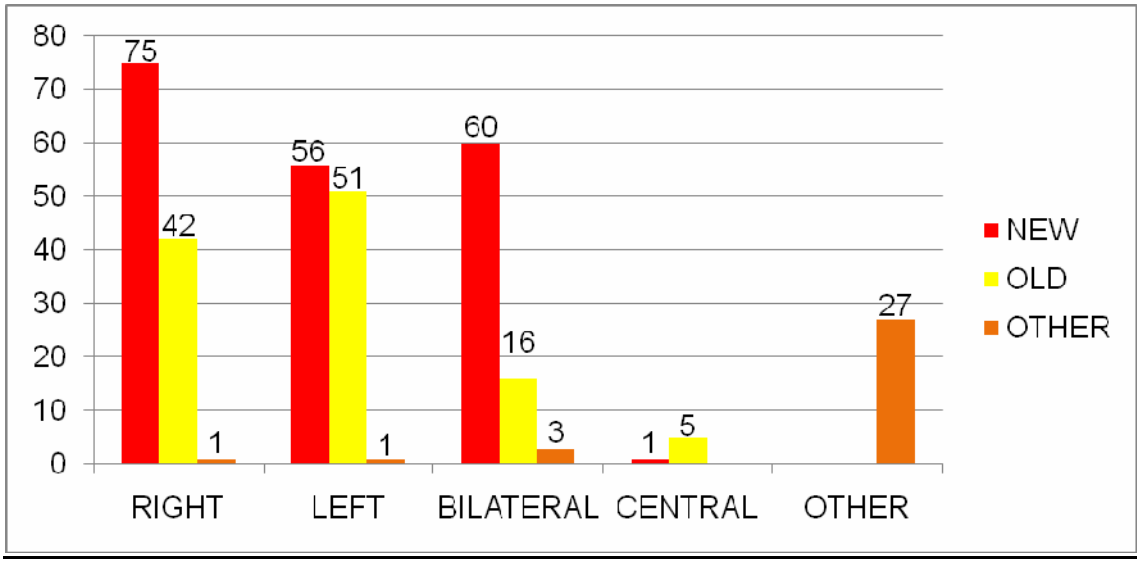

Figure 15: Prevalence of hemispheric predominant infarcts according to infarct categories

Figure 15 demonstrates the hemispheric predominance of infarcts according to infarct categories: old, new or other types of infarcts. Right hemispheric infarcts presented with 75 new infarcts *versus* 42 old infarcts. Left hemispheric infarcts presented with 56 new infarcts and only 51 old infarcts. The left and right hemispheres only presented with 1 patient each when observing other infarct types. Bilateral hemispheric infarcts presented with more (60) new infarcts than old infarcts (16). Other types of infarcts, i.e. embolic, haemorrhagic, etc. accounted for 27 of the infarcts.

5.5.3 Prevalence of hemispheric predominant infarcts according to age

Table 20: Prevalence of hemispheric predominant infarcts according to age

Age groups	Right	Left	Both	Central	Other	Total
18–29	4	4	2	-	-	10
30–39	15	7	6	-	-	28
40–49	13	11	8	1	-	33
50–59	24	26	10	-	4	64
60–69	19	15	16	2	-	52
70–79	25	32	23	3	23	106
> 80	17	11	13	-	-	41
Unknown	1	2	1	-	-	4
Total	**118**	**108**	**79**	**6**	**27**	**338**

Observation of Table 20 clearly indicates that all of the age groups present with infarcts concerning the left and right hemispheres. Below, each hemisphere is illustrated and discussed according to the age groups.

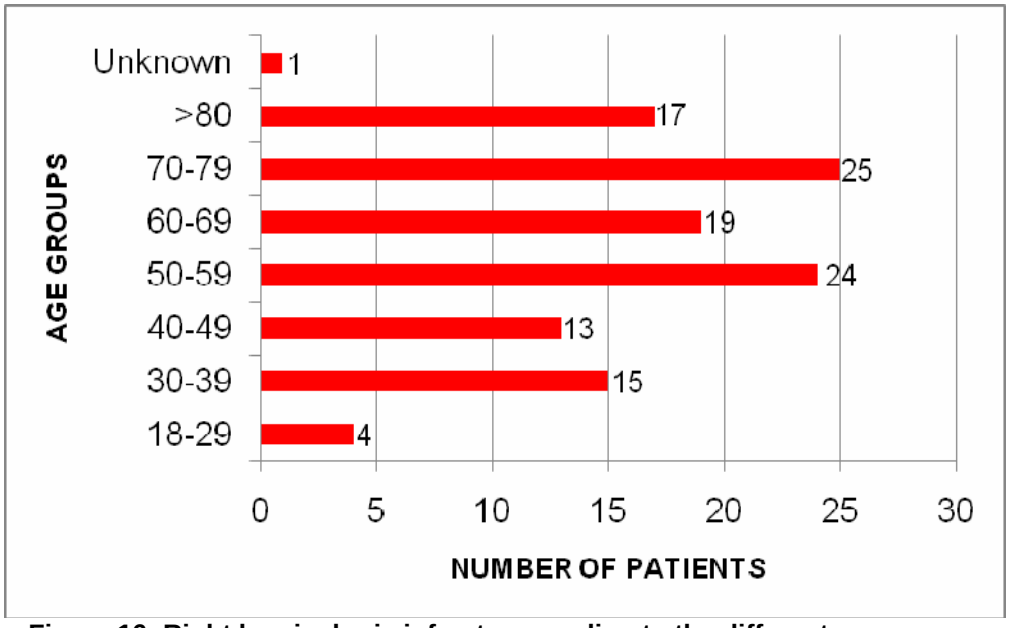

Figure 16: Right hemispheric infarcts according to the different age groups

The right hemisphere mainly affected patients in age groups 70–79 years (21.19%) and 50–59 years (20.34%). Age groups 30–39 years had 15 patients, 40–49 years had 24 patients, 60–69 years had 19 patients and > 80 years had 17 patients. The age group 18–29 years presented with only 3.39% (4/118) infarcts.

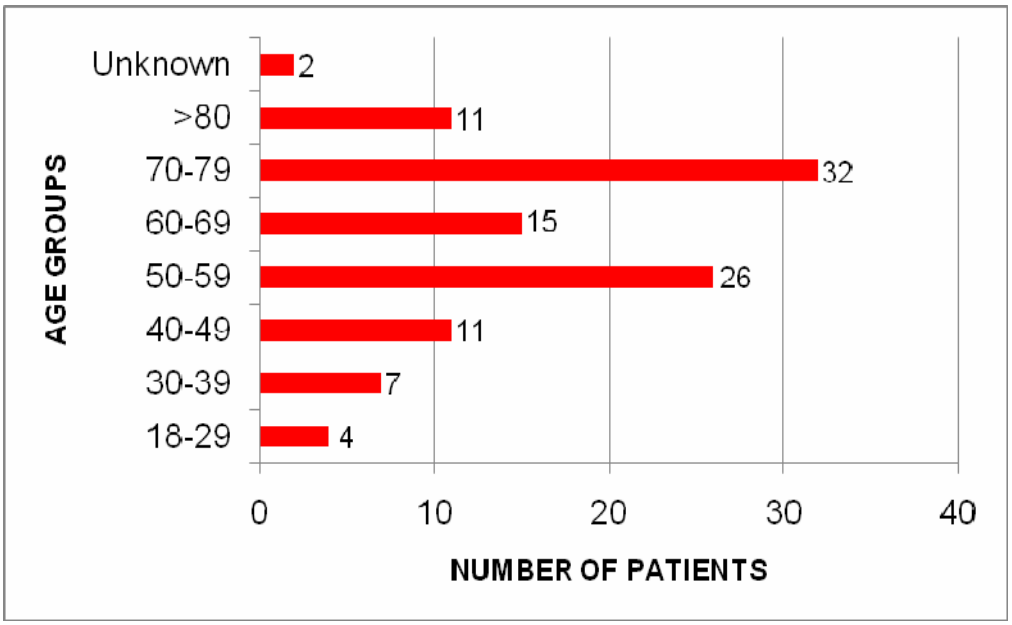

Figure 17: Left Hemispheric infarcts according to the different age groups

The age group 70–79 years (29.63%) showed the highest number of infarcts in the left hemisphere. The age group 50–59 years followed with a prevalence of 24.07%. Age groups 60–69 years presented with 15 patients, 40–49 years and above 80 years of age with 11 patients and age group 30–39 years with only 7 patients. Age group 18–29 years presented with the least number (3.7%) of infarcts.

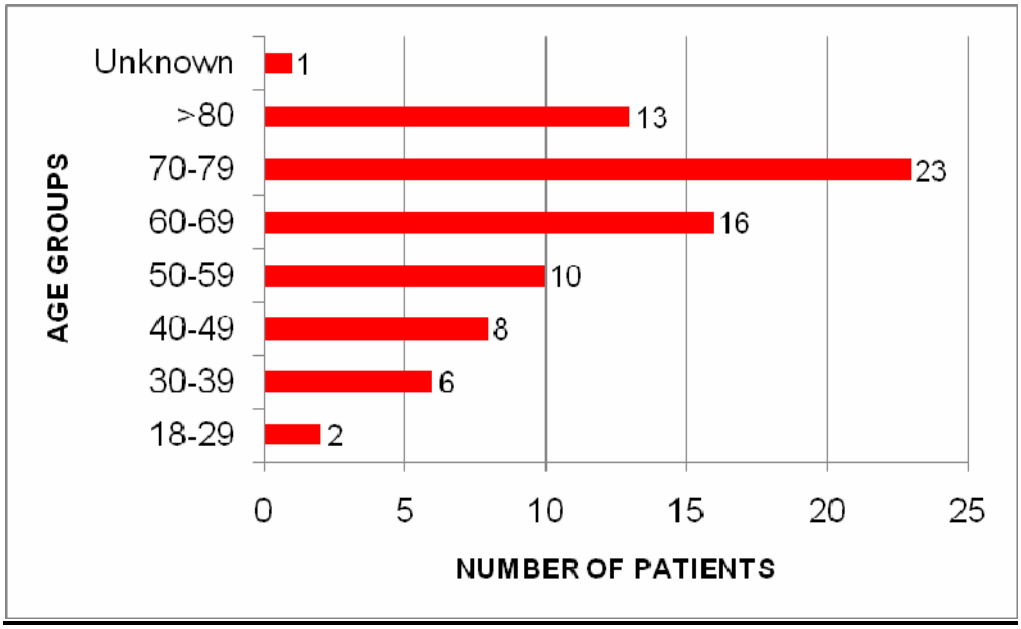

Figure 18: Bilateral hemispheric infarcts according to the different age groups

Bilateral hemispheric infarcts mainly affected the 70–79 year age group (29.11%). The age group 60–69 years presented with 20.25% infarcts that affected both hemispheres. Age groups above 80 years presented with 16.46% infarcts, whilst the age group 50–59 years presented with a mere 12.66%. Only 8 patients in the 40–49 year age group presented with bilateral hemispheric infarcts *versus* only 6 patients aged 30–39 years. The least number of infarcts was noted amongst 18–29 year olds, which accounted for 2.53%.

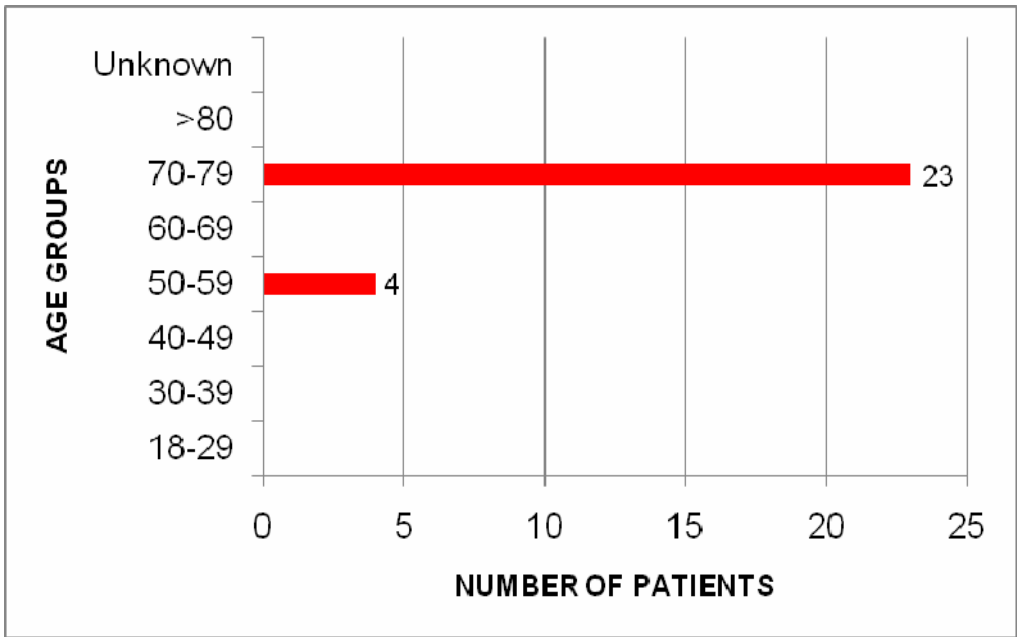

Figure 19: Other infarct types according to age groups

From Figure 19 it is noted that other infarct types (i.e. MID, etc.) only affected two age groups. These were patients in age groups 50–59 years and 70–79 years.

5.5.4 Prevalence of hemispheric infarct predominance according to sex

Table 21: Prevalence of right hemispheric infarcts according to sex

Age groups	Male (M)		Female		M & F Total	
	n	%	n	%	n	%
18–29	1	2.04	3	4.35	4	3.39
30–39	3	6.12	12[c]	17.39	15	12.71
40–49	8	16.33	5	7.25	13	11.02
50–59	12[a]	24.49	12[c]	17.39	24	20.34
60–69	8	16.33	11	15.94	19	16.10
70–79	12[a]	24.49	13[b]	18.84	25	21.19
> 80	5	10.20	12[c]	17.39	17	14.41
Total	**49**	**41.53**	**69**	**58.47**	**118**	**100**

a Male age groups most affected by right hemispheric infarcts

b Female age group presenting with most right hemispheric infarcts

c Female age groups presenting with an equal constant right hemispheric infarct prevalence

Right hemispheric infarcts were more prevalent in the female population (58.47%) than the male population (41.53%), as seen from Table 21. The male to female ratio was 1:1.41.

The male population that presented with right hemispheric infarcts was categorized according to age groups. Males in age groups 50–59 years and 70–79 years showed the highest prevalence (24.49%)[a]. Males of age groups 40–49 years and 60–69 years followed with 16.33%. The age groups 18–29 years (2.04%) and 30–39 years (6.12%) showed the lowest prevalence.

Right hemispheric infarcts in females showed a more equally distributed prevalence throughout the age groups affected. Females categorized in age groups 30–39 years, 50–59 years and above 80 years of age presented with an infarct prevalence of 17.39%[c]. The highest figure was noted in the category 70–79 years (18.84%)[b]. Females in age groups 60–69 years showed 15.94% prevalence. Age groups 18–29 years (4.35%) and 40–49 years (7.25%) presented with the least amount of right hemispheric infarcts.

Table 22: Prevalence of left hemispheric infarcts according to sex

Age groups	Male (M)		Female (F)		M & F Total	
	n	%	n	%	n	%
18–29	2	3.23	2	4.35	4	30.70
30–39	1	1.61	6	13.04	7	6.48
40–49	8	12.90	3	6.52	11	10.19
50–59	19	30.65	7	15.22	26	24.07
60–69	8	12.90	7	15.22	15	13.89
70–79	21[a]	33.87	11[b]	23.91	32	29.63
> 80	3	4.84	8	17.39	11	10.19
Total	**62**	**57.41**	**46**	**42.59**	**108**	**100**

a Male age group most affected by left hemispheric infarcts

b Female age group that presented with highest left hemispheric infarct prevalence

Table 22 illustrates that males (57.41%) presented with more left hemispheric infarcts than their female (45.59%) counterparts. The male to female ratio was 1:1.35.

The male population showed the highest left hemispheric prevalence in the age groups 70–79 years (33.87%)[a] and 50–59 years (30.65%). Age groups 40–49 years and 60–69 years followed with a prevalence of 12.90%, whilst only 1.61% of infarcts were noted in the males age groups 30–39 years and 3.23% in age group 18–29 years.

Females showed a more equally distributed left hemispheric infarct pattern. Age group 70–79 years presented with 23.91%[b] infarcts, followed by 17.39% in the age group above 80 years. Age groups 50–69 years showed 15.22% infarct prevalence's respectively. The least amount of infarcts was noted in the categories 18–29 years (4.35%) and 40–49 years (6.52%).

Table 23: Prevalence of bilateral hemispheric infarcts according to sex

Age groups	Male		Female		M & F Total	
	n	%	n	%	n	%
18–29	-	-	2	4.76	2	2.53
30–39	2	5.41	4	9.52	6	7.60
40–49	3	8.12	5	11.90	8	10.13
50–59	5	13.51	5	11.90	10	12.66
60–69	10	27.03	6	14.29	16	20.25
70–79	12[a]	32.43	11[b]	26.19	23	29.11
> 80	5	13.51	8	19.05	13	16.46
Total	**37**	**46.84**	**42**	**53.16**	**79**	**100**

a Male age group most affected by bilateral hemispheric infarcts

b Female age group most affected by bilateral hemispheric infarcts

Females (53.16%) presented with more bilateral hemispheric infarcts when compared to their male counterparts (46.84%), as demonstrated in Table 23. The male to female ratio was 1:1.14)

Bilateral hemispheric infarcts mainly affected males of age group 70–79 years (32.43%)[a] and 60–69 years (27.03%). Males in age groups 50–59 years and above 80 years had a prevalence of 13.51% each. The age group 18–29 years was unaffected, whilst men aged 30–39 years had a prevalence of 5.41% and 40–49 years a prevalence of 8.11%.

Females, unlike the males, were affected at all ages. Females however, showed a higher prevalence amongst the older females, aged 70–79 years (26.19%)[b] and above 80 years (19.05%). Females of age groups 40–59 years had a prevalence of 11.90%. The least number of bilateral hemispheric infarcts in females was noted in the age groups 18–29 years (4.76%) and 30–39 years (9.52%).

Table 24: Prevalence of central structured infarcts according to sex

Age groups	Males (M)	Females (F)	M & F Total
18–29	-	-	-
30–39	-	-	-
40–49	1	-	1
50–59	-	-	-
60–69	-	2	2
70–79	3	-	3
>80	-	-	-
Total	4	2	6

Centrally situated infarcts include infarcts that are found in the pons, medulla oblongata, etc., that do not include the left or right hemisphere. Males accounted for 4/6 of centrally structured infarcts in comparison to only 2/6 females.

Table 24 indicated that only males of age groups 40–49 years (1/4) and 70–79 years (3/4) were affected, whilst only females (2/6) in the age group 60–69 years were affected.

Table 25: Prevalence of other types of infarcts according to sex

Age groups	Males (M)	Females (F)	M & F Total
18–29	-	-	-
30–39	-	-	-
40–49	-	-	-
50–59	-	4[b]	4
60–69	-	-	-
70–79	9[a]	14[b]	23
>80	-	-	-
Total	**9**	**18**	**27**

a Only males aged 70 – 79 years presented with other infarct types

b Only females aged 50 – 59 years and 70 – 79 years presented with other infarct types

Other infarct types consist of MID, pointy, micro, watershed, venous and lacunar infarcts. Of the population affected, 18/27 of the patients were female, compared to 9/27 males. The male to female ratio was 1:2. Only males in the 70–79 year group were affected (9 patients). Females affected, were distributed between ages 50–59 years (4/18) and 70–79 years (14/18).

5.6 Brain Lobes

5.6.1 Prevalence of infarcts according to brain lobes and infarct categories

A total of 338 infarcts were noted of which 192 were new, 114 old and 32 other types of infarcts. Other infarcts were categorized as either old lacunar or new venous infarcts, but the brain lobes were not given in the reports and were therefore not included in the following section. The overall prevalence of the right and left sides of the brain lobes were therefore added and divided by the total number of infarcts.

a. Parietal Lobe

Table 26: Prevalence of parietal lobe infarcts

	Right side		Left side	
	n	**%**	**n**	**%**
Old infarcts	13	11.40	16	14.04
New infarcts	51	26.56	38	19.79
Totals	**64**	**18.93**	**54**	**15.98**

The right parietal lobe displayed 18.93% infarcts and left parietal lobe 15.98%. There was not a significant difference between the left and right parietal lobes (p=0.2450). The right parietal lobe presented with 26.56% new infarcts and 11.40% old ones. New left parietal lobe infarcts had a prevalence of 19.79%, *versus* 14.04% old infarcts.

b. Frontal Lobe

Table 27: Prevalence of frontal lobe infarcts

	Right side		Left side	
	n	**%**	**n**	**%**
Old infarcts	10	8.77	8	7.02
New infarcts	33	17.19	37	19.27
Totals	**43**	**12.72**	**45**	**13.31**

A total of 13.31% infarcts were found in the left frontal lobe of the brain. Of these, 19.27% were new infarcts and 7.02% were old infarcts. Right frontal lobe infarcts had a prevalence of 12.72%, of which 17.19% were new and 8.77%

were old infarcts. The difference between the left and right frontal lobe was however not significant (p=0.7681).

c. Occipital Lobe

Table 28: Prevalence of occipital lobe Infarcts

	Right side		Left side	
	n	**%**	**n**	**%**
Old infarcts	9	7.89	15	13.16
New infarcts	21	10.94	20	10.42
Totals	**31**	**9.17**	**35**	**10.36**

Table 28 shows that old left occipital lobe infarcts had a prevalence of 13.16%, compared to new infarcts of 10.42%. That brings the total left occipital lobe infarct prevalence to 10.36%. Infarcts present in the right occipital lobe accounted for 9.17% of which 10.94% of the infarcts were new and 7.89% were old. The difference between the left and right occipital lobe was not significant (p=0.5465).

d. Temporal Lobe

Table 29: Prevalence of temporal lobe Infarcts

	Right side		Left side	
	n	**%**	**n**	**%**
Old infarcts	8	7.02	8	7.02
New infarcts	14	7.29	8	4.17
Totals	**22**	**6.51**	**16**	**4.73**

Right temporal lobe infarcts had a prevalence of 6.51%. Old infarcts contributed 7.02% and new infarcts 7.29%. The left temporal lobe (4.73%) had the lowest overall brain lobe infarct prevalence. These were divided between 4.17% new infarcts and 7.02% old infarcts. No significant difference was noted between the left and right temporal lobes (p = 0.3173).

5.6.2 Prevalence of infarcts according to brain lobes in terms of age and sex

a. Right Parietal Lobe

Table 30: Prevalence of right parietal lobe infarcts according to age and sex

Age groups	Male (M)		Female (F)		M & F Total	
	n	%	n	%	n	%
18–29	-	-	2	6.06	2	3.13
30–39	2	6.45	2	6.06	4	6.25
40–49	2	6.45	3	9.09	5	7.81
50–59	7	22.58	8	24.24[c]	15	23.44
60–69	6	19.35	5	15.15	11	17.19
70–79	11	35.48[b]	5	15.15	16	25[a]
> 80	3	9.68	8	24.24[c]	11	17.19
Unknown	-	-	-	-	-	-
Total	**31**	**48.44**	**33**	**51.56**	**64**	**100**

a. Age group most affected by right parietal lobe infarcts

b. Male age group most affected by right parietal lobe infarcts

c. Female age groups most affected by infarcts

According to Table 30 the main age groups that presented with right parietal lobe infarcts were 70–79 years (25%)[a], 50–59 years (23.44%), 60–69 years (17.19%) and above 80 years of age (17.19%). The least number of infarcts was noted in age groups 40–49 years (5 patients), 30–39 years (4 patients) and 18–29 years (2 patients).

Males in age group 18–29 years were unaffected, but 35.48%[b] in the age group 70–79 years and 22.58% in the age group 50–59 years were affected. A much lower prevalence was noted throughout the other male age groups: 60–69 years (19.35%), above 80 years (9.68%) and 18–39 years (6.45% each).

Females of all ages were affected. The highest prevalence was seen in the age groups 50–59 years and above 80 years (24.24%)[c]. The age groups 60–79 years presented with 15.15% infarcts and age groups 18–39 years with 6.06%. Females aged 40–49 years of age presented with 9.09% right parietal lobe infarcts.

b. Left Parietal Lobe

Table 31: Prevalence of left parietal lobe infarcts according to age and sex

Age groups	Male (M)		Female (F)		M & F Total	
	n	%	n	%	n	%
18–29	-	-	1	3.70	1	1.85
30–39	1	3.70	3	11.11	4	7.41
40–49	1	3.70	4	14.81	5	9.26
50–59	10	37.04[b]	5	18.52[c]	15	27.78[a]
60–69	6	22.22	5	18.52[c]	11	20.37
70–79	7	25.93	5	18.52[c]	12	22.22
> 80	2	7.41	3	11.11	5	9.26
Unknown	-	-	1	3.70	1	1.85
Total	**27**	**50**	**27**	**50**	**54**	**100**

a. **Age group most affected by left parietal lobe infarcts**
b. **Male age group presenting with highest left parietal lobe infarct prevalence**
c. **Female age groups most affected by left parietal lobe infarcts**

The age groups most affected by left parietal lobe infarcts were 50–59 years (27.78%)[a], 70–79 years (22.20%) and 60–69 years (20.37%). The other age groups showed a much lower prevalence. Age group 18–29 years only had 1 patient; 30–39 years had 4 patients and age groups 40–49 years and above 80 years had 5 patients each.

An equal amount of males and females were affected when analyzing left parietal lobe infarcts. Males in age group 18–29 years of age were unaffected. The highest male prevalence's were noted in age groups 50–59 years (37.04%)[b] and 70–79 years (25.93%). Age groups 18–39 years had 1 patient in each category; 60–69 years had 6 patients, and two men above 80 presented with such infarcts.

Females were affected at all ages. Females had the highest left parietal lobe prevalence amongst age groups 50–79 years (18.52% each)[c]. Age groups 30–39 years and above 80 years of age presented with 3 patients each. The lowest prevalence was noted in age group 18–29 years (only 1 patient).

c. Left Frontal Lobe

Table 32: Prevalence of left frontal lobe infarcts according to age and sex

Age groups	Male (M)		Female (F)		M & F Total	
	n	%	n	%	n	%
18–29	-	-	1	4.35	1	2.22
30–39	-	-	2	8.70	2	4.44
40–49	1	4.55	2	8.70	3	6.67
50–59	7	31.82[b]	3	13.04	10	22.22
60–69	7	31.82[b]	4	17.39	11	24.44
70–79	7	31.82[b]	5	21.74	12	26.67[a]
> 80	-	-	6	26.09[c]	6	13.33
Unknown	-	-	-	-	-	-
Total	**22**	**48.89**	**23**	**51.11**	**45**	**100**

a. **Age group with highest left frontal lobe infarct prevalence**
b. **Male age groups with most left frontal lobe infarcts**
c. **Female age group most affected by left frontal lobe infarcts**

Table 32 shows that left frontal lobe infarcts mainly presented in patients aged 70–79 years (26.67%)[a], 60–69 years (24.44%) and 50–59 years (22.22%). Age groups 18–49 years showed a gradual increase from one to three patients in each category. Patients above 80 years of age presented with a prevalence of 13.33%.

Males in the 18–39 years and above 80 years age groups were completely unaffected, whilst females of all ages presented with left frontal lobe infarcts. Males in age groups 50–79 years presented with a steady 31.82%[b] incidence. One male categorized in age group 40–49 years was affected.

The highest female prevalence was seen in age groups 70–79 years (21.74%), 60–69 years (22.22%) and above 80 years (26.08%)[c]. A gradual increased prevalence was noted throughout female age groups 18–59 years (1-3 patients).

d. Right Frontal Lobe

Table 33: Prevalence of right frontal lobe Infarcts according to age and sex

Age groups	Male (M)		Female (F)		M & F Total	
	n	%	n	%	n	%
18–29	-	-	-	-	-	-
30–39	2	8.70	4	20[c]	6	13.95
40–49	1	4.35	1	5	2	4.65
50–59	4	17.39	4	20[c]	8	18.60
60–69	6	26.09	4	20[c]	10	23.26
70–79	8	34.78[b]	4	20[c]	12	27.91[a]
> 80	2	8.70	2	10	4	9.30
Unknown	-	-	1	5	1	2.33
Total	**23**	**53.49**	**20**	**46.51**	**43**	**100**

a. Age group most affected by right frontal lobe infarcts

b. Males most affected by right frontal lobe infarcts

c. Female age groups presented with an equal infarct prevalence

From Table 33 it is apparent that age groups 70–79 years (27.90%)[a] and 60–69 years (23.26%) showed the highest right frontal lobe prevalence (27.9%). Age group 18–29 years was not affected. Patients categorized in age groups 30-39 years presented with six infarcts, 40–49 years with 2 infarcts, 50–59 years with 8 infarcts and patients above 80 years with only 4 infarcts.

Right frontal lobe infarcts were absent in both males and females in age group 18–29 years of age. The male population had a prevalence of 34.78%[b] in the age group 70–79 years and 26.09% in age group 60–69 years. Males in age group 30–39 years and above 80 years presented with 2 infarcts each. Only one male was found in the category 40–49 years and 4 infarcts in age group 50–59 years.

Females presented with a prevalence of 20%[c] in age groups 30–39 years and 50–79 years respectively. One female in age group 40–49 years and 2 above 80 years were affected.

e. Left Occipital Lobe

Table 34: Prevalence of left occipital lobe infarcts according to age and sex

Age groups	Male (M)		Female (F)		M & F Total	
	n	%	n	%	n	%
18–29	-	-	-	-	-	-
30–39	1	4.76	2	14.29	3	8.57
40–49	3	14.29	1	7.14	4	11.43
50–59	5	23.81	1	7.14	6	17.14
60–69	1	4.76	2	14.29	3	8.57
70–79	8	38.10[b]	4	28.57[c]	12	34.29[a]
> 80	3	14.29	4	28.57[c]	7	20
Unknown	-	-	-	-	-	-
Total	**21**	**60**	**14**	**40**	**35**	**100**

a. **Age group most affected by left occipital lobe infarcts**
b. **Males most affected by left occipital lobe infarcts**
c. **Female age groups most affected with left occipital lobe infarcts**

Patients categorized in age group 70–79 years had a left occipital lobe infarct prevalence of 34.29%[a], regardless of sex, as demonstrated in Table 34. Patients above 80 years presented with 20% infarcts, 50–59 years with 17.14% infarcts and 40–49 years with 11.43% infarcts. Three patients were found in age groups 30–39 years and 60–69 years.

The total population of left occipital lobe infarcts consisted of 40% females *versus* 60% males. The male to female ratio was 1.5:1. Both males and females in age group 18–29 years were unaffected. The highest male prevalence was seen amongst the age group 70–79 years (38.10%)[b]. Males in age group 40–49 years and above 80 years presented with 3 infarcts each and age groups 30–39 years and 60–69 years with only one infarct each.

Females in age group 70 years and above presented with 4 infarcts[c] each *versus* only I infarct each in categories 40–59 years. Two infarcts were noted in age groups 30–39 years and 60–69 years.

f. Right Occipital Lobe

Table 35: Prevalence of right occipital lobe Infarcts according to age and sex

Age groups	Male (M)		Female (F)		M & F Total	
	n	%	n	%	n	%
18–29	-	-	-	-	-	-
30–39	2	11.76	3	21.43	5	16.13
40–49	2	11.76	2	14.29	4	12.90
50–59	3	17.65	1	7.14	4	12.90
60–69	1	5.88	1	7.14	2	6.45
70–79	8	47.06[b]	5	35.71[c]	13	41.94[a]
> 80	1	5.88	2	14.29	3	9.68
Unknown	-	-	-	-	-	-
Total	**17**	**54.84**	**14**	**45.16**	**31**	**100**

a. **Almost half of the right occipital lobe infarcts were noted in age group 70 – 79 years**
b. **Almost half of the male population affected were found in age group 70 – 79 years**
c. **Females most affected**

Table 35 indicates that age group 70–79 years (41.94%)[a] presented with the highest infarct prevalence overall. Patients in age groups 40–59 years presented with 4 infarcts each, 30–39 years with 5, above 80 years with 3 and 60–69 years with only 2 infarcts.

Males and females in age group 18–29 years were unaffected. The highest male prevalence was seen in age group 70–79 years (47.06%)[b]. One infarct was noted in age groups 60–69 years and above 80 years of age. Males in age groups 30–49 years of age had 2 infarcts each and age group 50–59 years had 3 infarcts.

One female presented with a right parietal lobe infarct in age groups 50–69 years of age respectively, and two females in age groups 40–49 years and above 80 years of age. The highest prevalence was noted in age group 70–79 years of age (35.71%)[c].

g. Right Temporal Lobe

Table 36: Prevalence of right temporal lobe infarcts according to age and sex

Age groups	Male (M)		Female (F)		M & F Total	
	n	%	n	%	n	%
18–29	1	16.67[b]	2	12.5	3	13.64
30–39	1	16.67[b]	3	18.75[c]	4	18.18
40–49	1	16.67[b]	1	6.25	2	9.09
50–59	1	16.67[b]	3	18.75[c]	4	18.18
60–69	-	-	3	18.75[c]	3	13.64
70–79	-	-	1	6.25	1	4.55
> 80	2	33.33	3	18.75[c]	5	22.73[a]
Unknown	-	-	-	-	-	-
Total	**6**	**27.27**	**16**	**72.73**	**22**	**100**

a. Age group most affected by right temporal lobe infarcts

b. Males aged 18–59 years presented with a constant infarct prevalence

c. Females most affected presented with an equal infarct prevalence

From Table 36 it is apparent that, of the entire population affected by right temporal lobe infarcts, the age group most affected was above 80 years (22.73%)[a]. Age groups 30–39 years and 50–59 years had a prevalence of 18.18% respectively. Patients in age group 18–29 years presented with 3 infarcts, 40–49 years with 2 infarcts and 70–79 years with only 1 infarct.

Females accounted for 72.73% of the population affected *versus* a male population of only 27.27%. The male to female ratio was 1:2.67. The male population was more or less equally distributed with a mean prevalence of 16.67%[b] for ages 18–59 years. Males aged 60–79 years were unaffected and only 2 infarcts were noted for males over 80 years.

Females were affected at all ages and showed a prevalence of 18.75%[c] in the categories 30–39 years, 50–69 years and above 80 years of age. One infarct was noted in the age categories 40–49 years and 70–79 years. Females in age group 18–29 years presented with 2 infarcts.

h. Left Temporal Lobe

Table 37: Prevalence of left temporal lobe infarcts according to age and sex

Age groups	Male (M)		Female (F)		M & F Total	
	n	%	n	%	n	%
18–29	1	25	-	-	1	6.25
30–39	1	25	2	16.67	3	18.75[a]
40–49	1	25	1	8.33	2	12.5
50–59	1	25	2	16.67	3	18.75[a]
60–69	-	-	3	25	3	18.75[a]
70–79	-	-	3	25	3	18.75[a]
> 80	-	-	1	8.33	1	6.25
Unknown	-	-	-	-	-	-
Total	**4**	**25.0**	**12**	**75.0**	**16**	**100**

a. **Age groups most affected by left temporal lobe infarcts**

Patients aged 30–39 years and 50–79 years showed an overall prevalence of 18.75%[a] each as seen from Table 37. Patients in age group 40–49 years presented with 2 infarcts and 18–29 years with only 1 infarct.

Males accounted for 25% of infarcts noted *versus* 75% females. The male to female ratio was 1:3. Males aged 60 years and above were unaffected. Males presented with one infarct each throughout the age groups 18–59 years of age.

When observing the female population, it is noted that the age groups 18–29 years and the unknown age group, was unaffected. Age groups 30–39 years and 50–59 years presented with 16.67% left temporal infarcts, whilst the highest number of infarcts were noted in the age groups 60–79 years (25% each). Only one infarct was found in women of age groups 40–49 and above 80 years of age.

Please see appendix C for a complete summary of the brain lobe infarct prevalence.

5.7 Anatomical structures

5.7.1 Prevalence of infarcts according to anatomical structures

a. *Middle cerebral artery* (MCA)

Table 38: Prevalence of MCA infarcts

	Right side		Left side	
	n	**%**	**n**	**%**
Old infarcts	11	9.65	9	7.89
New infarcts	28	14.58	21	10.94
Totals	**39**	**11.54**	**30**	**8.88**

The right MCA presented with a new infarct prevalence of 14.58%, whilst only 9.65% of presenting infarcts were old. The overall infarct prevalence noted in the right middle cerebral artery was 11.54%. New left MCA infarcts accounted for 10.94% *versus* 7.89% old infarcts. The overall left middle cerebral artery prevalence accounted for 8.88% of all cases. No significant difference between the amount of infarcts noted in the left and right MCA was seen (p=0.2568).

b. *Lentiform nucleus*

Table 39: Prevalence of lentiform nucleus infarcts

	Right side		Left side	
	n	**%**	**n**	**%**
Old infarcts	1	0.88	3	2.63
New infarcts	6	3.13	9	4.69
Totals	**7**	**2.07**	**12**	**3.55**

The right lentiform nucleus indicated an overall prevalence of 2.07%. Of these, 3.13% of the patients presented with new infarcts and only 0.88% with old infarcts. New infarcts in the left lentiform nucleus contributed to 4.69% infarct prevalence *versus* 2.63% old infarcts. The overall left lentiform nucleus infarct prevalence was calculated at 3.55%. The difference between the amount of infarcts noted in the left and right lentiform nucleus was not significant (p=0.1655).

c. Internal capsule

Table 40: Prevalence of internal and external capsular infarcts

	Internal capsule		External capsule	
	n	**%**	**n**	**%**
Old infarcts	-	-	-	-
New infarcts	10	5.21	3	1.56
Totals	**11**	**3.25**	**3**	**0.89**

No old infarcts were noted in the internal or external capsule. New infarcts presented in 5.21% of patients and other infarct types, i.e. watershed, lacunar etc., presented with 3.13% of infarcts noted in the internal capsule. The overall prevalence was 3.25%. New infarcts in the external capsule consisted of 1.56% of cases and an overall prevalence of 0.89%.

d. Posterior cerebral artery (PCA)

Table 41: Prevalence of PCA infarcts

	Right side		Left side	
	n	**%**	**n**	**%**
Old infarcts	-	-	-	-
New infarcts	8	4.17	6	3.13
Totals	**8**	**2.37**	**6**	**1.78**

The PCA only presented with new infarcts. The right PCA presented with 4.17% new infarcts and the overall prevalence was 2.37%. Left PCA infarcts presented with 3.13% new infarcts and had an overall prevalence of 1.78%. The infarct prevalence between the left and right PCA was not significantly different (p=0.4795).

Centrum semi ovalis (CSO)

Table 42: Prevalence of CSO infarcts

	Right side		Left side	
	n	**%**	**n**	**%**
Old infarcts	2	1.75	2	1.75
New infarcts	21	10.94	17	8.85
Totals	**23**	**6.80**	**19**	**5.62**

Old right CSO infarcts accounted for 1.75% of infarct cases *versus* 10.94% new infarct cases. The overall prevalence was calculated at 6.80%. Left CSO infarcts presented with 1.75% old and 8.85% new infarct cases. The total prevalence for this region was 5.62%. No significant difference between the left and right CSO was noted (p=0.3711).

e. White matter

Table 43: Prevalence of infarcts in the white matter

	Subcortical white matter		Periventricular white matter	
	n	**%**	**n**	**%**
Old infarcts	2	1.75	4	3.51
New infarcts	6	3.13	6	3.13
Totals	**8**	**2.37**	**10**	**2.96**

Infarcts presenting in the subcortical white matter region attributed to 1.75% old cases noted and 3.13% new cases. Overall, infarcts in this region were calculated at 2.37%. Periventricular white matter infarcts accounted for 2.96% of cases. Of these 3.13% were new and 3.51% were old.

f. Caudate nucleus

Table 44: Prevalence of caudate nuclear infarcts

	Right caudate nucleus		Left caudate nucleus	
	n	**%**	**n**	**%**
Old infarcts	-	-	1	0.88
New infarcts	4	2.08	7	3.65
Totals	**4**	**1.18**	**8**	**2.37**

New right caudate nucleus infarcts were noted in 2.08% of cases and the total prevalence was 1.18%. New left caudate nucleus infarcts were noted in 3.65% of cases *versus* 0.88% old infarcts. The prevalence was calculated at 2.37%.

No significant difference was noted between the number of infarcts noted in the left and right caudate nucleus (p=0.1025).

g. Vertebral artery

Table 45: Prevalence of left vertebral arterial infarcts

	n	%
New infarcts	2	1.04
Old infarcts	-	-
Other infarcts	-	-
Total	2	0.59

The left vertebral artery infarct prevalence was 0.59%. Only new infarcts (1.04%) were found in this region. Even though no infarcts were noted in the right vertebral artery, the difference regarding the infarct prevalence was not significant (p=0.1573).

h. Medulla Oblongata

Table 46: Prevalence of infarcts in the medulla oblongata

	n	%
New infarcts	6	3.13
Old infarcts	-	-
Other infarcts	-	-
Total	6	1.78

Only new infarcts (3.13%) were noted in the medulla oblongata. The total prevalence was 1.78%.

i. Pons

Table 47: Prevalence of pontine infarcts

	n	%
New infarcts	11	5.73
Old infarcts	5	4.39
Other infarcts	-	-
Total	16	4.73

Old infarcts noted in the area of the pons contributed to 4.39% of cases, whilst new infarcts accounted for 5.73% of cases. The overall prevalence was calculated at 4.73%.

j. Lateral ventricle

Table 48: Prevalence of lateral ventricular infarcts

	n	%
New infarcts	13	6.77
Old infarcts	7	6.14
Other infarcts	-	-
Total	20	5.92

Lateral ventricular infarcts accounted for 5.92% of cases. Old cases accounted for 6.14%, *versus* 6.77% of new infarcts noted.

k. Lateral fissure

Table 49: Prevalence of infracts in the lateral (Sylvian) fissure

	n	%
New infarcts	5	2.60
Old infarcts	6	5.26
Other infarcts	-	-
Total	11	3.25

Old infarcts as noted in the lateral fissure presented with a prevalence of 5.26% and new infarcts with 2.60%. The overall prevalence was calculated at 3.25%.

l. Internal carotid artery (ICA)

Table 50: Prevalence of ICA infarcts

	Right side		Left side	
	n	%	n	%
Old infarcts	1	0.88%	4	3.51%
New infarcts	7	3.65%	4	2.08%
Totals	**8**	**2.37%**	**8**	**2.37%**

Right ICA infarcts accounted for 2.37% of cases. Of these, 0.88% was old infarcts and 3.65% was new. The left ICA also presented with an overall infarct prevalence of 2.37%. Old infarcts accounted for 3.51% of cases and new infarcts for 2.08%. The difference between the number of infarcts noted in the left and right ICA was not significant (p=1.0000).

m. Cerebellum

Table 51: Prevalence of cerebellar infarcts

	n	%
New infarcts	8	4.17
Old infarcts	5	4.39
Total	13	3.85

The cerebellar infarct prevalence was noted as 3.85%. Of these, 4.17% were new infarcts and 4.39% were old infarcts.

n. Vermis

Table 52: Prevalence of infarcts in the vermis

	n	%
New infarcts	3	1.56
Old infarcts	-	-
Total	3	0.89

Only new infarcts (1.56%) were found in the vermis. The overall prevalence was 0.89%.

o. Brainstem

Table 53: Prevalence of infarcts in the brainstem

	n	%
New infarcts	4	2.08
Old infarcts	6	5.26
Total	10	2.96

New brainstem infarcts accounted for 2.08% of cases, *versus* 5.26% new infarcts. The total brainstem infarct prevalence was 2.96%.

p. Globus pallidus

Table 54: Prevalence of infarcts in the globus pallidus

	n	%
New infarcts	1	0.52
Old infarcts	1	0.88
Total	2	0.59

The prevalence of infarcts noted in the region of the globus pallidus was calculated at 0.59%. Of these, 0.52% was new and 0.88% was old infarcts.

q. Midbrain

Table 55: Prevalence of infarcts in the midbrain

	n	%
New infarcts	1	0.52
Old infarcts	3	2.63
Total	4	1.18

New midbrain infarcts accounted for 0.52% and old infarcts for 2.63% of the patients affected. The overall infarct prevalence was 1.18%.

r. Cerebral peduncle

Table 56: Prevalence of cerebral peduncular infarcts

	n	%
New infarcts	-	-
Old infarcts	1	0.88
TOTAL	1	0.30

Only 1 patient (0.88%) presented with an infarct in the cerebral peduncle. The prevalence was therefore 0.30%.

s. Basal nuclei

Table 57: Prevalence of infarcts in the basal nuclei

	n	%
New infarcts	11	5.73
Old infarcts	2	1.75
Total	13	3.85

Basal nuclei infarcts consisted of 1.75% old infarcts and 5.73% new infarcts. The overall prevalence was 3.85%.

t. Corona radiata

Table 58: Prevalence of infarcts in the corona radiata

	n	%
New infarcts	5	2.60
Old infarcts	2	1.75
Total	7	2.07

Infarcts in the region of the corona radiations accounted for 2.07% of infarct cases noted. Of these, 1.75% was old infarcts and 2.60% were new infarcts.

u. Thalamus

Table 59: Prevalence of thalamic infarcts

	n	%
New infarcts	6	3.13
Old infarcts	-	-
Other infarcts	1	3.13
Total	7	2.07

The thalamic region only had new infarcts (3.13%). Other types of infarcts (i.e. lacunar, embolic, etc.) also presented with 3.13% of infarcts in this structure. The overall prevalence was calculated at 2.07%.

v. *Anterior cerebral artery* (ACA)

Table 60: Prevalence of right anterior cerebral arterial infarcts

	n	%
New infarcts	4	2.08
Old infarcts	-	-
Total	4	1.18

New right ACA infarcts were noted in 2.08% of infarct cases and the overall prevalence was calculated at 1.18%. Even though the left ACA presented with no infarcts, no significant difference between the left and right ACA was noted (p=0.4795).

x. *Precentral gyrus*

Table 61: Prevalence of precentral gyrus infarcts

	n	%
New infarcts	2	1.04
Old infarcts	-	-
Total	2	0.59

Two patients presented with infarcts in the precentral gyrus, which brought the overall prevalence to 0.59%.

y. Optic tract

Table 62: Prevalence of optic tract infarcts

	n	%
New infarcts	1	0.52
Old infarcts	-	-
Total	1	0.30

One new infarct was noted in the optic tract and the prevalence was therefore 0.30%.

z. Corpus callosum

Table 63: Prevalence of infarcts in the corpus callosum

	n	%
New infarcts	-	-
Old infarcts	2	1.75
Total	2	0.59

Only old infarcts (1.75%) were found in the corpus callosum. The prevalence was 0.59%.

aa. Putamen

Table 64: Prevalence of infarcts in the putamen

	n	%
New infarcts	3	1.56
Old infarcts	-	-
Total	3	0.89

New putamen infarcts were found in 1.56% of cases studied and the prevalence was 0.89%.

ab. Frontal gyrus

Table 65: Prevalence of frontal gyrus infarcts

	n	%
New infarcts	1	0.52
Old infarcts	-	-
Total	1	0.30

One new infarct was noted in the frontal gyrus, with a prevalence of 0.30%.

ac. Postcentral sulcus

Table 66: Prevalence of infarcts in the postcentral sulcus

	n	%
New infarcts	1	0.52
Old infarcts	-	-
Total	1	0.30

One new infarct was found in the postcentral sulcus, which brought the prevalence to 0.30%.

5.7.2 Prevalence of infarcts according to anatomical structures in terms of age & sex

a. *Right middle cerebral artery* (MCA)

Table 67: Prevalence of right MCA infarcts according to age & sex

Age groups	Males		Females		Totals	
	n	%	n	%	n	%
18–29	1	6.25	-	-	1	2.56
30–39	1	6.25	4	17.39	5	12.82
40–49	4	25[b]	2	8.70	6	15.38
50–59	4	25[b]	3	13.04	7	17.95
60–69	1	6.25	4	17.39	5	12.82
70–79	4	25[b]	4	17.39	8	20.51[a]
> 80	1	6.25	5	21.74[c]	6	15.38
Unknown	-	-	1	4.35	1	2.56
Totals	**16**	**41.03**	**23**	**58.97**	**39**	**100**

a. **Highest right MCA infarct prevalence acc to age groups**
b. **Male age groups presenting with highest and equal infarct prevalence**
c. **Female age group presenting with most right MCA infarcts**

Table 67 demonstrates that patients that presented with infarcts in the right MCA, was mainly noted in the age group 70–79 years (20.51%)[a]. Age group 50–59 years presented with an infarct prevalence of 17.95%, whilst age groups 40–49 years and above 80 years of age presented with 15.38% of infarcts. The age groups 30–39 years and 60–69 years presented with 12.82% infarcts. Age group 18–29 years presented with a mere 2.56% right MCA prevalence.

The male right MCA infarct population presented with 41.03% infarcts, when looking at the entire population affected. All of the male age groups were

affected. The highest male prevalence's (5 infarcts each: 25%)[b] were noted in the age groups 40–59 years and 70–79 years of age. All of the other age groups presented with 6.25% infarcts (1 patient in each group).

Females accounted for 58.97% of the right MCA cases noted. The female age group 18–29 years of age was unaffected. The highest female prevalence was noted in the age group above 80 years (21.74%)[c] of age. Age groups 30–39 years and 60–79 years of age presented with 17.39% of infarcts noted. Patients aged 50–59 years presented with 13.04% of infarcts, whilst patients aged 40–49 years of age presented with only 8.70%.

b. Left middle cerebral artery (MCA)

Table 68: Prevalence of left MCA infarcts according to age & sex

Age groups	Males		Females		Totals	
	n	%	n	%	n	%
18–29	-	-	-	-	-	-
30–39	-	-	-	-	-	-
40–49	3	18.75	-	-	3	10.0
50–59	3	18.75	2	14.29	5	16.67
60–69	2	12.50	5	35.71[c]	7	23.33
70–79	8	50.0[b]	4	28.57	12	40.0[a]
> 80	-	-	2	14.29	2	6.67
Unknown	-	-	1	7.14	1	3.33
Totals	**16**	**53.33**	**14**	**46.67**	**30**	**100**

a. **Age group most affected by left MCA infarcts**
b. **Half of the males presenting with left MCA infarcts were seen in age group 70–79 years**
c. **Female age group presenting with highest left MCA infarct prevalence**

From Table 68 it is apparent that age groups 18–39 years of age did not present with any infarcts in the left MCA. The highest infarct prevalence was noted in the age group 70–79 years (40%)[a] of age. Age group 60–69 years presented with 23.33% of infarcts, whilst ages 50–59 years presented with 16.67%. Patients in age group 40–49 years presented with 10% of the infarcts noted. The age group above 80 years of age presented with the lowest (6.67%) infarct prevalence.

The male population accounted for 53.33% of left MCA cases noted. Age groups 18–39 years and above 80 years of age were unaffected. Fifty percent (8/16 infarcts)[b] of infarcts were noted in the age group 70–79 years of age. Males in age group 40–59 years of age presented with 18.75% infarcts and age group 60–69 years with only 2 infarct cases each.

Females accounted for 46.67% of infarcts noted in the left MCA, but were unaffected throughout age groups 18–49 years. The highest female prevalence (35.71%)[c] was noted in the age group 60–69 years and was closely followed by females in age group 70–79 years (28.57%). Age groups 50–59 years and above 80 years, presented with a mere 14.29% left middle cerebral artery infarct prevalence.

c. Lentiform nucleus

Table 69: Prevalence of lentiform nuclear infarcts according to age & sex

	Right side				Left side			
Age groups	**Male**		**Female**		**Male**		**Female**	
	n	**%**	**n**	**%**	**n**	**%**	**n**	**%**
18–29	-	-	-	-	-	-	-	-
30–39	-	-	-	-	-	-	-	-
40–49	-	-	1	25.0	1	11.11	-	-
50–59	2	66.67[a]	-	-	3	33.33[b]	2	66.67[c]
60–69	1	33.33	-	-	2	22.22	-	-
70–79	-	-	1	25.0	3	33.33[b]	1	33.33
> 80	-	-	2	50	-	-	-	-
Unknown	-	-	-	-	-	-	-	-
Totals	**3**	**42.86**	**4**	**57.14**	**9**	**75.0**	**3**	**25.0**

a. **Two males aged 50–59 years presented with right lentiform nucleus infarcts**
b. **Three males presented with left lentiform nucleus infarcts aged 50–59 years & 70–79 years**
c. **Two females presented with left lentiform nucleus infarcts aged 50–59 years**

Right lentiform nucleus infarcts did not affect age groups 18–39 years of age, as seen from Table 69. The highest infarct prevalence (28.57%) was noted throughout age groups 50–59 years and above 80 years. Age groups 40–49 years and 60–79 years presented with an infarct prevalence of 14.29%. Males accounted for 42.86% of infarcts noted and females for 57.14%. Only males in age groups 50–69 years were affected. Females were unaffected throughout age groups 18–39 years and 50–69 years.

Infarcts noted in the left lentiform nucleus did not affect the age groups 18–49 years and above 80 years. Age group 50–59 years presented with 41.67% of infarcts and 70–79 years with 33.33%. Age group 60–69 years presented with 16.67% of infarcts and 40–49 years with 8.33%. Males accounted for 75% of the infarct population affected, but were unaffected throughout 18–39 years and above 80 years. The highest male prevalence (33.33%)[b] was noted in the age groups 50–59 years and 70–79 years. Age groups 60–69 years presented with 22.22% of infarcts and age group 40–49 years with 11.11%. Females accounted for 25% of infarcts noted in the left lentiform nucleus. Only females in age groups 50–59 years (66.67%)[c] and 70–79 years (33.33%) were affected.

d. Internal & external capsule

Table 70: Prevalence of capsular infarcts according to age & sex

	Internal capsule				External capsule			
Age groups	**Male**		**Female**		**Male**		**Female**	
	n	**%**	**n**	**%**	**n**	**%**	**n**	**%**
18–29	-	-	-	-	-	-	-	-
30–39	1	20.0	-	-	-	-	-	-
40–49	-	-	-	-	1	50.0[c]	-	-
50–59	2	40.0[a]	1	16.67	1	50.0[c]	-	-
60–69	-	-	1	16.67	-	-	-	-
70–79	1	20.0	2	33.33[b]	-	-	-	-
> 80	1	20.0	2	33.33[b]	-	-	1	100
Unknown	-	-	-	-	-	-	-	-
Totals	**5**	**45.45**	**6**	**54.55**	**2**	**66.67**	**1**	**33.33**

a. **Forty percent of males presenting with internal capsular infarcts were aged 50–59 years**
b. **Two females each presented with internal capsular infarcts aged 70 years and above**
c. **Only two males aged 40–59 years presented with external capsular infarcts**

Table 70 demonstrates that internal capsular infarcts did not affect age groups 18–29 years and 40–49 years. Age groups 50–59 years and 70 years and above presented with 27.27% of infarcts, whilst age groups 30–39 years and 60–69 years with only 9.09%. Males accounted for 45.45% of infarcts and females for 54.54%. Males in age groups 18–29 years, 40–49 years and 60–69 years of age were unaffected. Forty percent[a] of male infarcts were noted in the age group 50–59 years. Age groups 30–39 years and 70 years and above presented with the remaining 20% of infarcts each. Females in age groups 18–

49 years were not affected. Females aged 70 years and above presented with 33.33%[b] of infarcts noted and age groups 50–69 years of age with 16.67%.

External capsular infarcts affected two males[c] in the age groups 40–59 years age group, and one female above 80 years of age.

e. *Posterior cerebral artery* (PCA)

Table 71: Prevalence of PCA infarcts according to age & sex

	Right side				Left side			
Age groups	**Male**		**Female**		**Male**		**Female**	
	n	**%**	**n**	**%**	**n**	**%**	**n**	**%**
18–29	-	-	-	-	1	50.0	1	25.0
30–39	1	50.0	2	33.33	-	-	1	25.0
40–49	-	-	-	-	-	-	-	-
50–59	-	-	-	-	-	-	-	-
60–69	-	-	2	33.33	-	-	2	50.0
70–79	1	50.0	2	33.33	1	50.0	-	-
> 80	-	-	-	-	-	-	-	-
Unknown	-	-	-	-	-	-	-	-
Totals	**2**	**25.0**	**6**	**75.0**	**2**	**33.3**	**4**	**66.67**

Right PCA infarcts did not affect the age groups 18–29 years, 40–59 years and above 80 years of age, as demonstrated in Table 71. The highest infarct prevalence (37.5%) was noted in the age groups 30–39 years and 70–79 years of age. The remaining 25% of infarcts were noted in the age group 60–69 years of age. Males only accounted for a mere 25% (2/8) of infarcts noted in the right PCA as compared to 75% (6/8) in females. Male age groups 18–29 years, 40–69 years and above 80 years of age were not affected. Age groups 30–39 years and 70–79 years of age presented with only 1 patient in each category. Females aged 30–39 years and 60–79 years presented with an infarct prevalence of 33.33% each. The other female age groups were not affected.

Patients aged 18–29 years and 60–69 years presented with 33.33% left PCA infarct prevalence's and age groups 30–39 years and 70–79 years with 16.67% infarcts. The other age groups were not affected. Males presented with 33.33% of the infarcts noted in the left PCA and females with 66.67%. Males only

presented with a single patient each in the age groups 18–29 years and 70–79 years. Females presented with 2 patients in the age group 60–69 years and only 1 patient in the categories 18–39 years each.

f. Centrum semi ovalis (CSO)

Table 72: Prevalence of CSO infarcts according to age & sex

	Right side				Left side			
Age groups	**Male**		**Female**		**Male**		**Female**	
	n	**%**	**n**	**%**	**n**	**%**	**n**	**%**
18–29	-	-	-	-	-	-	-	-
30–39	-	-	3	21.43	-	-	2	25.0
40–49	2	22.22	1	7.14	-	-	1	12.5
50–59	1	11.11	2	14.29	2	18.18	1	12.5
60–69	4	44.44	3	21.43	5	45.45	2	25.0
70–79	2	22.22	3	21.43	3	27.27	-	-
> 80	-	-	2	14.29	1	9.09	2	25.0
Unknown	-	-	-	-	-	-	-	-
Totals	**9**	**39.13**	**14**	**60.87**	**11**	**57.89**	**8**	**42.11**

Table 72 shows that patients aged 18–29 years of age did not present with any infarcts in the right CSO. Patients aged 60–69 years presented with the most (30.43%) infarcts, closely followed by the age group 70–79 years (21.74%). Patients aged 30–59 years of age all presented with 13.04% infarcts in each category. Patients above 80 years of age presented with the least (8.70%) amount of infarcts. Males accounted for 39.13% (9/23) of the right CSO infarct population *versus* 60.87% (14/23) females. Males aged 18–39 years and above 80 years of age were unaffected. The highest male prevalence (44.44%) was found in the age group 60–69 years. Male age groups 40–49 years and 70–79 years accounted for 22.22% of the infarcts noted *versus* only 11.11% in the age group 50–59 years. Females showed a more evenly distributed infarct prevalence throughout the affected age groups. Age group 18–29 years was unaffected. The highest female prevalence (21.43%) was found in age groups 30–39 years and 60–79 years. Age groups 50–59 years and above 80 years of age presented with 2 patients each. The least amount of infarcts was found in the category 40–49 years of age (7.14%).

Left CSO infarcts were mainly found (36.84%) in the age group 60–69 years. The age group 18–29 years was unaffected. Age groups 50–59 years and 70 years and above presented with an infarct prevalence of 15.79%. The least amount of infarcts was noted in the categories 30–39 years (10.53%) and 40–49 years (5.26%) of age. Males presented with 57.90% of the total number of infarcts found in the left CSO *versus* 42.10% in females. Males aged 18–49 years presented with no infarcts. The highest infarct prevalence (45.45%) was noted in the age group 60–69 years. Age groups 70–79 years (27.27%), 50–59 years (18.18%) and above 80 years of age (9.09%) also presented with infarcts. Females aged 18–29 years and 70–79 years were unaffected. The highest female prevalence was found (25%) in age groups 30–39 years, 60–69 years and above 80 years of age. The remaining 12.5% was found in the categories 40–59 years of age respectively.

g. White matter

Table 73: Prevalence of white matter infarcts according to age & sex

	Subcortical white matter				Periventricular white matter			
Age groups	**Male**		**Female**		**Male**		**Female**	
	n	**%**	**n**	**%**	**n**	**%**	**n**	**%**
18–29	-	-	-	-	-	-	-	-
30–39	-	-	-	-	-	-	1	25.0
40–49	1	16.67	1	50.0	1	16.67	-	-
50–59	-	-	-	-	3	50	2	50.0
60–69	3	50	-	-	1	16.67	-	-
70–79	2	33.33	-	-	1	16.67	-	-
> 80	-	-	1	50.0	-	-	1	25.0
Unknown	-	-	-	-	-	-	-	-
Totals	**6**	**75.0**	**2**	**25.0**	**6**	**60.0**	**4**	**40.0**

Subcortical white matter infarcts were not found throughout the age groups 18–39 years and 50–59 years of age, as noted in Table 73. An infarct prevalence of 37.5% was noted in the age group 60–69 years of age *versus* 25% in the age groups 40–49 years and 70–79 years and only 12.5% in the age group above 80 years of age. Males accounted for 75% (6/8) of the total infarct population. Half of the affected males were found in the category 60–69 years of age. Categories 70–79 years and 40–49 years of age presented with 33.33% and

16.67% infarcts respectively. Age groups 18–39 years, 50–59 years and above 80 years of age were unaffected. Females only accounted for 25% (2/8) of the infarct population. The two patients involved, were found in the age groups 40–49 years and above 80 years of age.

Half of the infarcts noted in the periventricular white matter were in patients aged 50–59 years. Ten percent of infarcts were noted in each category of ages 30–49 years and 60 years and above. Patients aged 18–29 years were unaffected. Males contributed 60% of the total number of infarcts. Half of the male population was found in the category 50–59 years *versus* only 16.67% in the age groups 40–49 years and 60–79 years. Categories 18–39 years and above 80 years of age were not affected. Forty percent of infarcts noted were attributed to the female population. Half of them were found in the age group 50–59 years and 25% in the categories 30–39 years and above 80 years of age each.

h. Vertebral artery

Table 74: Prevalence of left vertebral arterial infarcts according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	1	1
40–49	-	-	-
50–59	-	-	-
60–69	-	-	-
70–79	-	-	-
> 80	-	1	1
Unknown	-	-	-
Totals	-	2	2

Left vertebral arterial infarcts only affected two females, one in the age group 30–39 years and the other in the category above 80 years.

The right vertebral artery did not present with any infarcts.

i. Medulla Oblongata

Table 75: Prevalence of infarcts in the medulla oblongata according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	2	-	2
50–59	-	-	-
60–69	-	-	-
70–79	2	-	2
> 80	1	1	2
Unknown	-	-	-
Totals	5	1	6

Infarcts noted in the medulla oblongata were divided between the age groups 40–49 years and 70 years and above (33.33%). All of the other age categories were unaffected (See Table 75).

The male population accounted for 83.33% (5/6) of the total number of infarcts. They were divided amongst the age groups 40–49 years, 70–79 years (40%) and above 80 years of age (20%).

The only female affected was found in the age group above 80 years of age.

j. Pons

Table 76: Prevalence of pontine infarcts according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	1	-	1
50–59	2	-	2
60–69	1	4	5
70–79	3	1	4
> 80	3	1	4
Unknown	-	-	-
Totals	10	6	16

Table 76 indicate that patients aged 18–39 years of age were not affected by infarcts in the pons. Pontine infarcts were found in the age groups 60–69 years

(31.25%), 70 years and above (25% in each category), 50–59 years (12.5%) and 40–49 years (6.25%).

The male population accounted for 62.5% (10/16) of all pontine infarcts noted *versus* only 37.5% (6/16) females. Thirty percent of the males affected were found in age group 70 years and above. The remaining infarcts were found in the categories 50–59 years (20%) and 40–49 years and 60–69 years (10%).

Females in age groups 18–59 years were completely unaffected. An infarct prevalence of 66.67% was noted in the age group 60–69 years of age and the remaining 16.67% in the age groups 70 years and above.

k. Lateral ventricle

Table 77: Prevalence of lateral ventricular infarcts according to age & sex

Age groups	Males	Females	Totals
18–29	-	1	1
30–39	1	1	2
40–49	1	-	1
50–59	1	2	3
60–69	-	2	2
70–79	4	3	7
> 80	2	2	4
Unknown	-	-	-
Totals	9	11	20

Table 77 demonstrated that all of the age groups were affected when looking at ventricular infarcts. The highest number of infarcts (35%) were noted in the age group 70–79 years, followed by the age groups above 80 years (20%), 50–59 years (15%), 30–39 years and 60–69 years of age (10%). The least number of infarcts (5%) was spotted in the category 18–29 years of age.

Males accounted for 45% of infarcts noted, *versus* 55% female lateral ventricular infarcts. The highest male prevalence (44.44%) was found in the category 70–79 years of age. The age group above 80 years of age presented with 22.22% of the infarcts, whilst the age groups 30–59 years of age presented

with 11.11% each. Age groups 18–29 years and 60–69 years of age were unaffected.

The 40–49 years age group were the only female age category that presented with no infarcts. The highest female lateral ventricular infarct prevalence was noted in the category 70–79 years (27.27%). Female age groups 50–69 years and above 80 years of age presented with18.18% infarcts and age groups 18–39 years with 9.09% each.

l. Lateral Fissure

Table 78: Prevalence of infarcts in the lateral fissure according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	1	1	2
50–59	2	-	2
60–69	-	1	1
70–79	1	2	3
> 80	1	2	3
UNKNOWN	-	-	-
TOTALS	5	6	11

Infarcts presenting in the lateral fissure did not affect the age groups 18–39 years of age, as seen from Table 78. The highest infarct prevalence was noted in the category 70 years and above 80 yaers of age (27.27%), followed by age groups 40–59 years (18.18%) and 60–69 years (9.09%) of age.

Males (45.45%) and females (54.54%) were almost equally affected by lateral fissure infarcts. Males aged 18–39 years and 60–69 years of age were unaffected. Forty percent of the male infarcts were found in the category 50–59 years of age and 20% in the categories 40–49 years and 70 years and above.

Females aged 18–39 years and 50–59 years were unaffected. The highest female prevalence (33.33%) was found in the categories 70 years and above and 16.67% in the age groups 40–49 years and 60–69 years of age.

m. *Internal carotid artery* (ICA)

Table 79: Prevalence of ICA infarcts according to age & sex

	Right side				Left side			
Age groups	**Male**		**Female**		**Male**		**Female**	
	n	**%**	**n**	**%**	**n**	**%**	**n**	**%**
18–29	-	-	-	-	-	-	-	-
30–39	-	-	-	-	-	-	-	-
40–49	-	-	-	-	1	20.0	-	-
50–59	1	25.0	1	25.0	4	80.0	1	33.33
60–69	1	25.0	1	25.0	-	-	1	33.33
70–79	2	50.0	1	25.0	-	-	-	-
> 80	-	-	1	25.0	-	-	-	-
UNKNOWN	-	-	-	-	-	-	1	33.33
TOTALS	**4**	**50.0**	**4**	**50.0**	**5**	**62.5**	**3**	**37.5**

Left internal carotid arterial (ICA) infarcts were absent throughout age groups 18–39 years and 70 and above (see Table 79). The highest infarct prevalence was noted in age group 50–59 years (62.50%). Categories 40–49 years and 60–69 years presented with one patient each. Males accounted for 62.5% of the infarct population affected *versus* 37.5% females. The male population only presented with infarcts in the age categories 50–59 years (4 infarcts noted) and 40–49 years (only 1 infarct noted). Females in age goups 18–49 years and 70 years and above did not present with any left ICA infarcts. One infarct was however noted in age groups 50–69 years.

Only 8 out of 338 infarcts noted presented in the right ICA. Two infarcts each were found in categories 50–69 years of age and only 1 infarct in the category above 80 years of age. The highest prevalence (3 infarcts) was noted in the age group 70–79 years. An equal number of males and females were affected (4 infarcts each). Two males in age group 70–79 years presented with infarcts and 1 male was found in categories 50–69 years respectively. One female presented with an infarct throughout categories 50 years and above respectively.

n. Cerebellum

Table 80: Prevalence of cerebellar infarcts according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	1	-	1
50–59	2	-	2
60–69	2	-	2
70–79	2	3	5
> 80	-	3	3
Unknown	-	-	-
Totals	7	6	13

A total of 13 patients presented with cerebellar infarcts. Of these, 5 infarcts were found in category 70–79 years, 3 infarcts in the category above 80 years of age and 1 infarct in category 40–49 years. Age groups 50–69 years presented with 2 infarcts in each category.

The male *versus* female ratio, concerning cerebellar infarcts, was almost equal (7:6). Males aged 18–39 years and above 80 years were not affected. Two infarcts each were found throughout age groups 50–79 years of age. Age group 40–49 years presented with one infarct.

Females aged 18–69 years of age did not present with any cerebellar infarcts. Three infarcts were noted in each category of patients aged 70 years and above.

o*. Vermis*

Table 81: Prevalence of infarcts in the vermis according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	1	1
40–49	-	-	-
50–59	-	-	-
60–69	-	-	-
70–79	2	-	2
> 80	-	-	-
Unknown	-	-	-
Totals	2	1	3

Only two males (aged 70–79 years) and one female (aged 30–39 years) presented with an infarct in the vermis.

p. Brainstem

Table 82: Prevalence of infarcts in the brainstem according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	-	1	1
50–59	-	-	-
60–69	1	1	2
70–79	2	1	3
> 80	3	1	4
Unknown	-	-	-
Totals	6	4	10

Table 82 illustrates that a gradual increase is noted throughout the affected age groups. Age groups 18–39 years and 50–59 years did not present with any infarcts in the brainstem. One infarct was noted in age group 40–49 years, two in age group 60–69 years, three in age group 70–79 years and four infarcts in age group 80 years and above.

Sixty percent of the population affected was male *versus* only 40% females. Males aged 18–59 years were not affected. The age group above 80 years of age presented with 50% of the male infarct prevalence noted. The remainder was shared amongst age groups 70–79 years (2 infarcts) and 60–69 years (1 infarct).

One female was noted in each category aged 40–49 years and 60 years and above. The other age groups did not present with any infarcts in the brainstem.

q. Globus pallidus

Table 83: Prevalence of infarcts in the globus pallidus according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	1	-	1
50–59	1	-	-
60–69	-	-	-
70–79	-	-	-
> 80	-	-	-
Unknown	-	-	-
Totals	2	-	2

Only 2 males in age groups 40–49 years and 50–59 years presented with an infarct in the globus pallidus.

r. Midbrain

Table 84: Prevalence of infarcts in the midbrain according to age & sex

Age groups	Males	Females	Totals
18–29	-	1	1
30–39	-	-	-
40–49	-	-	-
50–59	-	-	-
60–69	-	-	-
70–79	2	1	3
> 80	-	-	-
Unknown	-	-	-
Totals	2	2	4

Two males (in age group 70–79 years) and two females (1 in age group 18–29 years, 1 in age group 70–79 years) presented with an infarct in the midbrain.

s*. Cerebral Peduncle*

Table 85: Prevalence of infarcts in the cerebral peduncle according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	-	-	-
50–59	-	-	-
60–69	-	-	-
70–79	1	-	1
> 80	-	-	-
Unknown	-	-	-
Totals	1	-	1

Only one cerebral peduncular infarct was noted in a male patient (category 70–79 years).

t. Basal Nuclei

Table 86: Prevalence of infarcts in the basal nuclei according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	2	2
40–49	-	-	-
50–59	4	3	7
60–69	1	-	1
70–79	2	1	3
> 80	-	-	-
Unknown	-	-	-
Totals	7	6	13

Table 86 illustrates that patients aged 50–59 years presented with a basal nuclei infarct prevalence of 53.85%. Age groups 70–79 years (23.08%), 30–39 years (15.39%) and 60–69 years (7.69%) also presented with infarcts in this region.

Males accounted for 53.85% of the population affected *versus* 46.15% females. The highest male prevalence was noted in age group 50–59 years. Two infarcts were noted in age group 70–79 years and one in age group 60-69 years.

Fifty percent of the female population affected was noted in age group 50–59 years. Age groups 70–79 years (1 infarct) and 30–39 years (2 infarcts) also presented with infarcts.

u. Corona Radiata

Table 87: Prevalence of infarcts in the corona radiata according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	1	-	1
40–49	-	1	1
50–59	-	1	1
60–69	-	-	-
70–79	2	-	2
> 80	1	1	2
Unknown	-	-	-
Totals	4	3	7

Age groups 70 years and above presented with an infarct prevalence of 28.57% *versus* 14.29% throughout age groups 30–59 years (See Table 87).

Males accounted for 57.14% of the population affected *versus* 42.86% females. Two infarcts were noted in male age group 70–79 years and one infarct each in age groups 30–39 years and above 80 years of age.

Only three females presented with infarcts in the corona radiata. One infarct each was noted in age categories 40–59 years and above 80 years of age.

v. Thalamus

Table 88: Prevalence of thalamic infarcts according to age & sex

Age groups	Males	Females	Totals
18–29	1	1	2
30–39	-	-	-
40–49	-	-	-
50–59	2	1	3
60–69	-	-	-
70–79	1	-	1
> 80	1	-	1
Unknown	-	-	-
Totals	5	2	7

The highest thalamic prevalence was noted in age group 50–59 years (42.86%), as seen from Table 88. Age group 18–29 years presented with a

28.57% prevalence and age groups 70 years and above with 14.29% infarcts each.

The male population accounted for 71.43% of the affected thalamic infarct population *versus* only 28.57% females. Males in age groups 30–49 years and 60–69 years did not present with any infarcts. Age groups 18–29 years and 70 years and above each presented with one infarct and age group 50–59 years with two infarcts.

Only two females (aged 18–29 years and 50–59 years) were affected.

w. Anterior cerebral artery (ACA)

Table 89: Prevalence of right ACA infarcts according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	1	-	1
50–59	1	-	1
60–69	1	-	1
70–79	-	1	1
> 80	-	-	-
Unknown	-	-	-
Totals	3	1	4

Table 89 illustrates that a total of four right ACA infarcts were noted. One infarct each was noted throughout age groups 40–79 years. Three males aged 40–69 years were affected. One infarct was noted in each category. Only one female patient presented with a right ACA infarct in age group 70–79 years.

No infarcts were noted in the left ACA.

x. *Caudate nucleus*

Table 90: Prevalence of caudate nuclear infarcts according to age & sex

Age groups	Right side				Left side			
	Male		Female		Male		Female	
	n	%	n	%	n	%	n	%
18–29	-	-	-	-	-	-	-	-
30–39	-	-	1	50.0	-	-	-	-
40–49	-	-	-	-	-	-	-	-
50–59	1	50.0	-	-	2	40.0	-	-
60–69	1	50.0	-	-	1	20.0	-	-
70–79	-	-	1	50.0	1	20.0	1	33.33
> 80	-	-	-	-	1	20.0	2	66.67
Unknown	-	-	-	-	-	-	-	-
Totals	**2**	**50.0**	**2**	**50.0**	**5**	**62.5**	**3**	**37.5**

Table 90 demonstrate that two males (in age group 50–69 years) and 2 females (in age groups 30–39 & 70–79 years) presented with infarcts in the right caudate nucleus.

The left caudate nucleus affected males aged 50 years and above. Two infarcts were seen in the category 50–59 years. Females presented with three left caudate nucleus infarcts in age group 70 years and above.

y. *Precentral gyrus*

Table 91: Prevalence of precentral gyrus infarcts according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	-	-	-
50–59	-	-	-
60–69	1	-	1
70–79	1	-	1
> 80	-	-	-
Unknown	-	-	-
Totals	2	-	2

Two males in age categories 60–79 years of age (1 male in each category) presented with infarcts in the precentral gyrus.

z. Optic tract

Table 92: Prevalence of optic tract infarcts according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	-	-	-
50–59	-	-	-
60–69	-	-	-
70–79	1	-	1
> 80	-	-	-
Unknown	-	-	-
Totals	1	-	1

Optic tract infarcts only affected one male patient in the group 70–79 years of age.

aa. Corpus callosum

Table 93: Prevalence of corpus callosum infarcts according to age & sex

Age groups	Males	Females	Totals
18–29	-	1	1
30–39	-	-	-
40–49	-	-	-
50–59	-	-	-
60–69	-	-	-
70–79	1	-	1
> 80	-	-	-
Unknown	-	-	-
Totals	1	1	2

One male (in age group 70–79 years) and one female (in age group 18–29 years) presented with a single infarct each, in the corpus callosum.

ab. Putamen

Table 94: Prevalence of infarcts in the putamen according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	1	-	1
50–59	1	-	1
60–69	-	-	-
70–79	1	-	1
> 80	-	-	-
Unknown	-	-	-
Totals	3	-	3

Only 3 patients presented with infarcts. All were male and they were in the age groups 40–59 years and 70–79 years.

ac. Frontal gyrus

Table 95: Prevalence of frontal gyral infarcts according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	-	-	-
50–59	-	-	-
60–69	-	-	-
70–79	-	-	-
> 80	-	-	-
Unknown	-	1	1
Totals	-	1	1

One female patient presented with an infarct in the frontal gyrus, but her age was not available.

ad. Postcentral sulcus

Table 96: Prevalence of infarcts in the postcentral sulcus according to age & sex

Age groups	Males	Females	Totals
18–29	-	-	-
30–39	-	-	-
40–49	-	-	-
50–59	-	-	-
60–69	-	-	-
70–79	1	-	1
> 80	-	-	-
Unknown	-	-	-
Totals	1	-	1

Only one patient presented with an infarct in the postcentral sulcus. The patient was male and in the age group 70–79 years.

A summary of the anatomical infarct prevalence's is available in Appendix D

5.8 Harvard system

5.8.1 Harvard Classification

Table 97: Harvard Classification

LOCATION	STRUCTURES
Basal Nuclei	• Caudate nucleus • Putamen • Globus pallidus
White Matter	• Corona radiata • Centrum semi ovale • Corpus callosum • Internal capsule
Midbrain	• Superior & Inferior colliculus • Red nucleus • Substantia nigra
Brainstem	• Pons • Medulla
Cerebellum	• Vermis
Diencephalon	• Thalamus • Hypothalamus • Optic tract
Ventricular System	• Lateral ventricle
Vascular Structures	• Internal carotid artery • Anterior / Posterior/ Middle cerebral artery • Posterior communicating artery • Vertebral artery

5.8.2 Infarct prevalence according to the Harvard system

The Harvard system was used to group the anatomical structures together, in order to summarize dominant infarct location. This was done to determine whether or not infarcts are more likely to occur within a specific area or system in the brain. The Harvard system is a classification method that is used by Harvard University to group anatomical structures together in a logical system.

The frequency column in Table 98 therefore indicates the total number of infarcts noted in a specific Harvard classified area.

Table 98: Infarct prevalence according to the Harvard system

Area	No of infarcts	%
Sulci	15	4.44%
Basal nuclei	31	9.17%
White matter	62	18.34%[b]
Brainstem	27	7.99%
Cerebellum	15	4.44%
Diencephalon	7	2.07%
Ventricular system	20	5.92%
Vascular system	90	26.63%[a]
Midbrain	4	1.18%
Totals	**338**	**100%**

a. **Vascular structures presented with the most infarcts**

b. **White matter presented with the second most infarcts overall**

Table 98 shows that the vascular system presented with the highest number (26.63%)[a] of infarcts. The white matter presented with the second highest (18.34%)[b] infarct frequency. The basal nuclei presented with 9.17% infarcts, the brainstem with 7.99% and the ventricular system with 5.92%. The brain sulci and the cerebellum each presented with an infarct prevalence of 4.44%. The diencephalon (2.07%) and the midbrain (1.18%) presented with the least amount of infarcts.

5.8.3 Harvard system classification according to age & sex

a. Sulci

Table 99: Sulcal prevalence according to age & sex

Age groups	Male		Female		Totals	
	n	%	n	%	n	%
18–29	-	-	-	-	-	-
30–39	-	-	-	-	-	-
40–49	1	12.5	1	14.29	2	13.33[a]
50–59	2	25.0	-	-	2	13.33[a]
60–69	1	12.5	1	14.29	2	13.33[a]
70–79	3	37.5[b]	2	28.57[c]	5	33.33[a]
> 80	1	12.5	2	28.57[c]	3	20
Unknown	-	-	1	14.29	1	6.67
Totals	**8**	**53.33**	**7**	**46.67**	**15**	**100**

a. **The highest sulci infarct prevalence was noted in age group 70–79 years**

b. **The male age group presenting with most sulci infarcts**

c. **Female age groups with most sulci infarcts**

Table 99 demonstrates that age groups 70–79 years (33.33%)[a] and above 80 years (20%), presented with the highest sulcal infarct prevalence's. Age groups 40–69 years presented with infarct prevalence's of 13.33% each. Age groups 18–39 years were not affected.

Males presented with 8 infarcts noted in the sulci *versus* 7 in females. Both males and females aged 18–39 years were not affected. Male age group 70–79 years presented with the highest (37.5%)[b] infarct prevalence. Age group 50–59 years presented with 25% infarcts and age groups 40–49 years, 60–69 years and above 80 years of age presented with an infarct prevalence of 12.5% each.

Females aged 70 years and above presented with 28.57%[c] infarcts in each category. Age groups 18–39 years and 50–59 years were not affected. Age groups 40–49 years and 60–69 years presented with 14.29% infarcts.

b. Basal nuclei

Table 100: Basal nuclei prevalence according to age & sex

Age groups	Male		Female		Totals	
	n	%	n	%	n	%
18–29	**-**	-	**-**	-	**-**	-
30–39	-	-	2	15.38	2	6.45
40–49	1	5.56	1	7.69	2	6.45
50–59	7	38.89[b]	4	30.77[c]	11	35.48[a]
60–69	3	16.67	-	-	3	9.68
70–79	6	33.33	2	15.38	8	25.81
> 80	1	5.56	4	30.77[c]	5	16.13
Unknown	**-**	-	**-**	-	**-**	-
Totals	**18**	**58.06**	**13**	**41.94**	**31**	**100**

a. The highest basal nuclei infarct prevalence was noted in age group 50–59 years

b. Males aged 50–59years was most affected

c. Females most affected by basal nuclei infarcts

Basal nuclei infarcts did not affect the age group 18–29 years as seen from Table 100. The highest prevalence was found in age group 50–59 years (35.48%)[a], followed by 70–79 years (25.81%) and above 80 years (16.13%) of age. Age group 60–69 years presented with 9.68% infarcts and age groups 30–49 years with 6.45% each.

Males affected by basal nuclei infarcts accounted for 58.06% (18/31) of the population affected and females for 41.94% (13/31). Males aged 18–39 years were not affected. The highest male infarct prevalence was noted in age group 50–59 years (38.89%)[b]. Age group 70–79 years presented with 33.33% infarcts and age group 60–69 years with 16.67%. The least amount (5.55%) of infarcts was noted in age groups 40–49 years and above 80 years of age.

Female age groups 18–29 years and 60–69 years were not affected by basal nuclei infarcts. The highest prevalence (30.77%)[c] was noted in age groups 50–59 years and above 80 years of age. Age groups 30–39 years and 70–79 years presented with 15.38% infarcts each. The lowest infarct prevalence was noted in age group 40–49 years (7.69%).

c. White matter

Table 101: White matter prevalence according to age & sex

Age groups	Male		Female		Totals	
	n	%	n	%	n	%
18–29	-	-	1	3.57	1	1.6
30–39	2	5.88	4	14.29	6	9.68
40–49	5	14.71	3	10.71	8	12.90
50–59	8	23.53[d]	5	17.86[e]	13	20.97[b]
60–69	9	26.47[c]	5	17.86[e]	14	22.58[a]
70–79	8	23.53[d]	5	17.86[e]	13	20.97[b]
> 80	2	5.88	5	17.86[e]	7	11.29
Unknown	-	-	-	-	-	-
Totals	**34**	**54.84**	**28**	**45.16**	**62**	**100**

a. Age group most affected by infarcts

b. Age groups with high white matter infarct prevalence's

c. Male age group with highest infarct prevalence

d. Male age groups with high white matter infarct prevalence's

e. **Female age groups most affected by infarcts: note equal distribution**

White matter infarcts affected patients of all ages. Ages 60–69 years (22.58%)[a], 50–59 years and 70–79 years (20.97%)[b] showed the highest infarct prevalence's. Patients aged 40–49 years (12.90%), above 80 years (11.29%) and 30–39 years (9.68%) followed thereafter. The least amount of infarcts was noted in age group 18–29 years (1.61%).

Males (54.84%) were more affected than females (45.16%), when observing white matter infarcts. Males aged 18–29 years were not affected. The highest male prevalence was noted in age groups 60–69 years (26.47%)[c], 50–59 years and 70–79 years (23.53%)[d]. Patients aged 40–49 years showed a prevalence of 14.71%. The lowest male white matter infarct prevalence (5.88%) was noted in age groups 30–39 years and above 80 years of age.

Females of all ages were affected. Female categories 50 years and above showed a steady infarct prevalence of 17.86%[e] each. Age groups 30–39 years (14.29%) and 40–49 years (10.71%) were also affected. Only 1 female patient in age group 18–29 years presented with an infarct in the white matter of the brain.

d. Brainstem

Table 102: Brainstem prevalence according to age & sex

Age groups	Male		Female		Totals	
	n	%	n	%	n	%
18–29	-	-	-	-	-	-
30–39	-	-	-	-	-	-
40–49	3	16.67	1	11.11	4	14.81
50–59	2	11.11	-	-	2	7.41
60–69	2	11.11	4	44.44[c]	6	22.22
70–79	6	33.33[b]	2	22.22	8	29.63[a]
> 80	5	27.78	2	22.22	7	25.93
Unknown	-	-	-	-	-	-
Totals	**18**	**66.67**	**9**	**33.33**	**27**	**100**

a. **Age group with highest brainstem infarct prevalence**
b. **Males most affected**
c. **Female age group most affected: almost half of affected population**

Table 102 demonstrates that no patients aged 18–39 years were affected when observing infarcts noted in the brainstem. The highest overall prevalence's were noted in age groups 70–79 years (29.63%)[a], above 80 years of age (25.93%) and 60–69 years (22.22%). Age group 40–49 years presented with 14.81% infarcts *versus* only 7.41% in age group 50–59 years.

Males affected were double (66.67%) the amount than females. Males aged 18-29 years presented with no infarcts. The highest number of infarcts were noted in age groups 70–79 years (33.33%)[b], above 80 years of age (27.78%) and 40–49 years (16.67%). Age groups 50–69 years showed an infarct prevalence of 11.11% respectively.

Females aged 18–39 years and 50–59 years were not affected. Only 1 female aged 40–49 years and 2 females aged 70 years and above were affected. Age group 60–69 years presented with 44.44%[c] infarcts.

e. Cerebellum

Table 103: Cerebellum prevalence according to age & sex

Age groups	Male		Female		Totals	
	n	%	n	%	n	%
18–29	-	-	-	-	-	-
30–39	-	-	1	14.29	1	6.67
40–49	1	12.5	-	-	1	6.67
50–59	2	25.0	-	-	2	13.33
60–69	2	25.0	-	-	2	13.33
70–79	3	37.5[b]	3	42.86[c]	6	40.0[a]
> 80	-	-	3	42.86[c]	3	20.0
Unknown	-	-	-	-	-	-
Totals	**8**	**53.33**	**7**	**46.67**	**15**	**100**

a. **Highest cerebellar infarct prevalence was noted in age group 70–79 years**
b. **Male age group most affected**
c. **Female age groups with highest cerebellar infarct prevalence's**

Patients aged 18–29 years were not affected when observing cerebellar infarcts as set out in Table 103. Only 1 patient each was noted throughout categories 30–49 years and 2 patients throughout age groups 50–69 years. Patients above 80 years of age presented with a prevalence of 20% infarcts *versus* 40%[a] in age group 70–79 years.

The male (53.33%) *versus* female (46.67%) ratio was almost equal. Males aged 18–39 years and above 80 years of age were not affected. The highest prevalence was noted in age groups 70–79 years (37.5%)[b] and 50–69 years (25%). Only 1 male patient in age group 40–49 years was affected.

Females aged 18–29 years and 40–69 years were not affected. Only 1 female patient was noted in age group 30–39 years, *versus* 42.86%[c] throughout age groups 70 years and above.

f. Diencephalon

Table 104: Diencephalon infarct prevalence according to age & sex

Age groups	Males	Females	Totals
18–29	1	1	2
30–39	-	-	-
40–49	-	-	-
50–59	2	1	3
60–69	-	-	-
70–79	1	-	1
> 80	1	-	1
Unknown	-	-	-
Totals	**5**	**2**	**7**

Patients aged 30–49 years and 60–69 years did not present with any infarcts. Seven infarcts were noted in the diencephalon, of which 3 were found in the category 50–59 years. Age groups 18–29 years (2 infarcts) and 70 years and above (1 infarct each) were also affected.

Males accounted for 71.43% of the infarct population *versus* 28.57% females. One infarct was noted in the male categories 18–29 years and 70 years and above. Two males presented with infarcts in the age group 50–59 years.

Only 2 females presented with infarcts, in the age groups of 18–29 years and 50–59 years.

g. Ventricular system

Table 105: Ventricular system prevalence according to age & sex

Age groups	Male		Female		Totals	
	n	%	n	%	n	%
18–29	-	-	1	9.09	1	5.0
30–39	1	11.11	1	9.09	2	10.0
40–49	1	11.11	-	-	1	5.0
50–59	1	11.11	2	18.18	3	15.0
60–69	-	-	2	18.18	2	10.0
70–79	4	44.44[b]	3	27.27	7	35.0[a]
> 80	2	22.22	2	18.18	4	20.0
Unknown	-	-	-	-	-	-
Totals	**9**	**45.0**	**11**	**55.0**	**20**	**100**

a. **Age group 70–79 years presented with the most infarcts**

b. **Males aged 70–79 years were most affected**

Table 105 demonstrates that the highest ventricular system prevalence was noted in age groups 70–79 years (35%)[a], above 80 years of age (20%) and 50–59 years (15%). Age groups 30–39 years and 60–69 years each presented with two infarcts each, whilst age groups 18–29 years and 40–49 years only presented with one infarct each.

The male ventricular system infarct prevalence accounted for only 45% of the population affected, *versus* 55% females. Males aged 18–29 years and 60–69 years were not affected. One infarct each was noted in age categories 30–59 years. Two infarcts were seen in males over 80 years of age. The highest prevalence was noted in the category 70–79 years (44.44%)[b].

Females aged 18–39 years of age presented with a single infarct in each category. No infarcts were seen in the age group 40–49 years. Age groups 50–69 years and above 80 years of age, each presented with 18.18% infarcts. The highest female infarct prevalence was noted in age group 70–79 years (3 infarcts).

h. Vascular system

Table 106: Vascular system prevalence according to age & sex

Age groups	Male		Female		Totals	
	n	%	n	%	n	%
18–29	2	4.55	1	2.17	3	3.33
30–39	2	4.55	6	13.04	8	8.89
40–49	8	18.18	2	4.35	10	11.11
50–59	13	29.55	7	15.22	20	22.22
60–69	4	9.09	9	19.57	13	14.44
70–79	14	31.82[b]	10	21.74[c]	24	26.67[a]
> 80	1	2.27	8	17.40	9	10
Unknown	-		3	6.52	3	3.33
Totals	**44**	**100**	**46**	**100**	**90**	**100**

a. **Age group 70–79 years presented with the most infarcts overall**
b. **Males aged 70-79 years were most affected**
c. **Females aged 70–79 years were most affected**

The highest vascular system infarct prevalence's were noted in age groups 70–79 years (26.67%)[a], 50–59 years (22.22%), 60–69 years (14.44%) and 40–49 years (11.11%), as seen from Table 106. Eight infarcts were noted in age group 30–39 years and 9 infarcts in patients over the age of 80. Age group 18–29 years presented with 3.33% infarcts.

The male (48.89%) to female (51.11%) ratio was almost equal. Males and females of all ages were affected. The highest male infarct prevalence's were noted in age groups 70–79 years (31.82%)[b], 50–59 years (29.55%) and 40–49 years (18.18%). Two infarcts each were noted in categories 18–39 years and four infarcts in category 60–69 years. Only one infarct was seen in patients over 80 years.

Only one female aged 18–29 years presented with an infarct in the ventricular system and two females aged 40–49 years. Age groups 70–79 years (21.74%)[c], 60–69 years (19.57%), above 80 years (17.39%) and 50–59 years (15.22%) presented with the most infarcts. Females in age group 30–39 years had six infarcts cases.

i. Midbrain

Table 107: Midbrain prevalence according to age & sex

Age groups	Males	Females	Totals
18-29	-	1	1
30-39	-	-	-
40-49	-	-	-
50-59	-	-	-
60-69	-	-	-
70-79	2	1	3
> 80	-	-	-
Unknown	-	-	-
Totals	**2**	**2**	**4**

Only four infarct cases were noted in the midbrain. Two males in age group 70–79 years and two females (1 in age group 18–29 years, 1 in age group 70–79 years) were affected.

5.9 Measurements

5.9.1 Circumference of infarcts according to age & sex

$$\textbf{Circumference: } \frac{2\,(L+W)}{C} \textbf{ x 2 (L+W)}$$

For the measurements, the age groups were grouped together in order to get bigger sample sizes. The 10–year interval age groups used thus far were combined into three larger groups as follows: 18–49 years, 50–69 years & 70 years and above. These age groups are referred to as the new age groups (see Appendix F).

Circumference is usually determined using the formula: length plus width plus length plus width (adding all sides of a square). Since infarcts vary greatly in size and shape the circumference formula was determined as follows: The normal circumference formula for a square was divided by the actual circumference (C) obtained when the infarcts were measured. This figure was then multiplied by the circumference formula of a square.

It was found that the circumference of infarcts vary greatly. This can be attributed to the considerable variation in the size and shape of infarcts which indicate that it can either be small or affect an entire hemisphere.

5.9.2 Circumference according to ANOVA

A one-way ANOVA was computed to compare the measurements of the infarcts according to sex and age. Male and female age groups were grouped together as follows: 18–39 years, 40–59 years, 60–69 years, 70–79 years and above 80 years were grouped together. The younger age groups were grouped together in order to get bigger sample sizes, since they presented with less infarcts. Four females of unknown age and four males with centrally structured infarcts were not included in this test. A total of 17 patients images were not found and therefore also not included in the test.

A one-way ANOVA was conducted on the left and right hemispheric infarct measurements separately, whilst looking at the width, length and circumference measurements respectively. The means of the measurements in the age groups were compared to obtain a mean difference value. It was also noted whether or not the mean difference value was significant or not. The standardized mean difference is the effect size generally recommended in clinical trials and other studies assessing treatment effects on outcomes measured on a continuous scale.[69]

From the ANOVA test conducted it was noted that males aged 70 years and above presented with significantly different right hemispheric infarct width measurements ($F(9,269)=1.074$) than females aged 18–39 years. Men aged 70–79 years (6.5±2.92) and > 80 years (8.17±3.89) presented with larger right hemispheric infarct width measurements than females aged 18–39 years of age.

Males aged 40–59 years (16.64±7.22) and 70–79 years (15.88±7.32) presented with significantly larger left hemispheric infarct length measurement ($F(9,247)=1.150$) than females aged 18–39 years.

Males aged 18–39 years ($F(9,247)= 1.866$) had significantly larger left hemispheric infarct width measurements than males aged 60–69 years (13.28±6.42) and females aged 18–39 years (15.54±6.45) and 40–59 years (12.08±6.17).

Males aged 40–59 years presented with significantly larger left hemispheric infarct width measurements than males aged 60–69 years (6.60±3.13) and females aged 18–39 years (8.87±3.20) and 40–59 years (5.41±2.61).

Males aged 70–79 years presented with significantly larger left hemispheric infarct width measurements than males aged 60–69 years (6.54±3.18) and females aged 18–39 years (8.80±3.24) and 40–59 years (5.35±2.66).Females aged 18–39 years of age had a significantly larger left hemispheric width infarct measurement than females aged 70–79 years of age (7.09±3.40).

Males aged 40–59 years presented with significantly larger left hemispheric infarct circumference measurements (F(9,246=1.413) than males aged 60–69 years (41.94±21.10) and females aged 18–39 years of age (54.20±21.53). Males aged 60–69 years presented with significantly larger left hemispheric infarct circumference measurements when compared to males aged 70–79 years (41.18±21.41). Males aged 70–79 years of age presented with significantly larger left hemispheric infarct circumference measurements when compared to females aged 18–39 years (53.43±21.83).

5.9.3 Surface Area of infarcts according to age & sex

$$\textbf{Surface Area: } \frac{2(L+W)}{C} \times (L \times W)$$

For the surface area measurements, the age groups were grouped together in order to get bigger sample sizes. The 10-yr interval age groups combined and grouped as follows: 18–49 years, 50–69 years & 70 years and above. These age groups are referred to as the new age groups (See Appendix G).

The surface area formula was determined as follows: When dividing the circumference formula of a square (2 x (L+W)) by the actual circumference of the infarcts measured, the ratio was always 1. Thus, the circumference formula developed was simply multiplied by the surface area formula of a square (LxW).

The results showed that the surface area of the infarcts is highly variable. This can be attributed to the considerable variation of infarcts in size and shape, which indicate that infarcts can be either small or affect an entire hemisphere.

A one–way ANOVA test could not be conducted, since the shape of an infarct is highly variable. The surface area formula of a square (L x W) could therefore not be used.

6. Discussion

6.1 Pilot study

A pilot study was performed on patients that presented for brain MRI examinations at a private institution in Pretoria for the month of June 2006. A total of 248 brain MRI examinations were performed during that period, of which 42 patients were children and 30 patient records could not be found. These patients were thus excluded from the pilot study and the sample size therefore consisted of 176 patients. The reports of the examinations were obtained using the Promed Viking System. The procedures followed were similar to the actual research study conducted.

Table 108: A comparison of results between the pilot study and actual research study

Study	Sample size	No of infarcts	Infarct prevalence	Age group most affected	Dominant brain lobes	Dominant structures affected
Pilot study	176	38	21.59%	70–79 yrs	• Frontal • Parietal • Occipital	• CSO • Cerebellum • Pons
Actual research study	1 844	338	16.10%	70-79 yrs	• Parietal • Frontal • Occipital	• MCA • Lentiform nucleus • Internal capsule

Table 109: A comparison of infarct prevalence's between the pilot and actual research studies

	Infarct prevalence		Old vs New infarcts		Hemispheric predominance	
Study	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
Pilot study	18	21	Old: 5 New: 11	Old: 4 New: 13	Right: 29 Left: 11	Right: 13 Left: 10
Actual research study	161	177	Old: 52 New: 99	Old: 62 New: 93	Right: 49 Left: 62	Right: 69 Left: 46

Table 110: A comparison of the measurement outcomes of the pilot and actual research studies

	Pilot study	Actual research study
Measurement outcomes	• Men showed larger infarct surface area compared to female counterparts. • Men aged 60–69 years presented with larger infarct circumferences compared to other males and all female age groups. • Mean and standard deviation figures varied greatly due to size and shape of infarcts.	• Surface area measurements could not be determined using ANOVA one-way analysis • Left hemispheric infarcts presented with significantly different sizes (length, width , circumference) • Right hemispheric infarcts were more stable in size (length, width, circumference) • Mean and standard deviation figures varied greatly due to size and shape of infarcts.

6.2 Actual Research Study

6.2.1Total Infarct Prevalence

This study was performed over a period of thirteen months. During this period 2 588 brain MRI examinations were performed. Of these patients, 299 presented with infarcts. The infarct prevalence for the time period 1 January 2006 until 31 January 2007 was 16.10%. This prevalence is almost double the number of other studies. A population-based study in the Netherlands (Rotterdam) reported an infarct prevalence of 7.2%,[60] whilst a retrospective analysis study reported a stroke prevalence of 6.4% (796 patients presented with stroke 12 454 cases) in Karachi (Pakistan).[70] Vermeer *et al.*[23] conducted a population-based cohort study on the Rotterdam community among 1077 elderly people that presented for brain MRI scans during 1995–1996. Of these, 57 participants (6%) presented with infarcts.[23] The high infarct prevalence noted in this study (16.10%) can be due to the fact that the other studies were performed on European populations and not on the South African population. The 1996 Global Burden of Disease[71] study revealed that cardiac and cerebral vascular disease surpassed infectious and parasitic disease to become the leading causes of death in the developing world, India and Sub-Saharan Africa. In South Africa, stroke accounted for 8–10% of all reported deaths and 7.5% of deaths among people of prime working age, 25–64 years old.[72,73] The higher infarct prevalence in South Africa can be due to the increased incidence of hypertension, lifestyle and HIV/AIDS. The prevalence of hypertension was 28% in North America and 44% in Western Europe.[74]

In South Africa, the most common risk factor for stroke was found to be atherosclerosis. Other stroke risk factors are also prevalent in the South African population, whether looking at young or old patients. Giovanni and Fritz[75] investigated 75 young patients seen at the Johannesburg Hospital and compared them to older patients in terms of risk factors and causes of stroke in South Africa. Younger patients were more likely to have migraine, mitral valve

proplapse, valvular heart disease and used oral contraceptives, whilst older patients were more likely to have hypertension, ischaemic heart disease, peripheral vascular disease and had a previous history of smoking.

Lifestyle has an enormous effect on the development of stroke and increases the risk factors causing stroke. Hoffman[76] analyzed 320 young stroke patients in the Durban Stroke Register and found that endemic disease and population affinity were important determinants of the underlying cause and risk factor profile. Whites presented with more cases of hypertension (71%), alcohol abuse (20%) and smoking (9%) whilst Blacks/Africans were more prone to suffer stroke after HIV infection, tuberculosis vasculitis, bilharzia and cerebral venous thrombosis. It was also found that patients aged 35–54 years of age had a higher stroke incidence.[76] The Southern Africa Stroke Prevention Initiative (SASPI)[77] found that stroke prevalence in rural South Africa is much higher than previously documented in Africa, but that it is lower than in high-income countries. They concluded that South Africa suffers from a huge burden of HIV/AIDS and diseases due to poverty and violence (2004).[77] Thirty-six million people are currently infected with HIV/AIDS, of which 25 million people are found in Sub-Saharan Africa. In Botswana, 36% of adults have the HIV virus and in South Africa 20%[78] (See Table 119 for AIDS statistics 2001).[79] HIV and the body's response to it, alter the walls of blood vessels, making them less responsive to blood pressure changes and therefore more prone to rupture, causing haemorrhagic stroke. HIV can also damage the endothelial lining of the blood vessels, which triggers blood clots, causing ischaemic strokes.[80] This supports the perception that population affinity, age and lifestyle increases the risk of developing stroke.

Table 111: Regional AIDS statistics, 2001[79]

Region	People with HIV/AIDS	Newly infected	Adult prevalence rate
Sub-Saharan Africa	28.1 million	3.4 million	8.4%
North Africa & Middle East	440, 000	80, 000	0.2%
South & South-east Asia	6.1 million	800, 000	0.6%
East Asia & Pacific	1 million	270, 000	0.1%
Latin America	1.4 million	130, 000	0.5%
Caribbean	420, 000	60, 000	2.2%
Eastern Europe & Central Asia	1 million	250, 000	0.5%
Western Europe	560, 000	30, 000	0.3%
North America	940, 000	45, 000	0.6%
Australia & New Zealand	15, 000	500	0.1%
Total	**40 million**	**5 million**	**1.2%**

6.2.2 Prevalence of infarct categories according to age & sex

The sample size of this study consisted of 338 infarcts. Of these, 56.80% were new, 33.73% were old and 9.47% were other types of infarcts. No studies have been performed up to date to determine the specific prevalence of the different infarct categories i.e. new, old and other infarcts (lacunar, embolic etc).

When observing the infarct categories, the highest infarct prevalence was noted in age group 70–79 years (new: 26.56%, old: 28.07%, other: 71.88%), while the age group 18–29 years was least affected. This can be due to the fact that infarcts mainly tend to affect the elderly population.

All age groups presented with infarcts, but a decrease was observed in patients above 80 years of age. This might be due to mortality, since the average expected lifespan of men at birth is 72 years compared to women which is 79 years.[81]

The total number of males (51.56%) that presented with new infarcts was almost equal to that of females (48.44%). Males and females were affected throughout all of the age groups. Females (54.39%), presented with more old infarcts than their male (45.61%) counterparts. All female age groups were affected, but males aged 18–39 years of age did not present with any old infarcts. This is due to the fact that women present with infarcts at young ages (18–39 years) due to pregnancy and the use of oral contraceptives,[19,82,83] whilst young males are unaffected by infarcts during these ages. More women therefore present with old infarcts, since they already suffered from infarction when they were younger. Fewer males (31.25%) presented with other types of infarcts than females (68.75%). Only males aged 50–59 years and 70–79 years of age were affected, whilst females aged 30–39 years and 60–69 years of age were not affected. Other types of infarcts occurred in the age group 70–79 years of age. There were no infarcts in the age groups 30–39 years, 60–69 years and above 80 years of age.

6.2.3 Prevalence of infarcts according to age

As expected, the most common age group affected by infarcts, was patients aged 70–79 years of age. A prevalence of 31.36% was noted. Age groups 50–59 years (18.93%), 60–69 years (15.38%) & above 80 years of age (12.13%) also presented with high infarct prevalence's. The MRC/Wits Rural Public Health and Health Transition Research Unit,[84] found that for Limpopo province (1992–1995), stroke was the most common cause of death in the 55–74 yr old age group. In age group 35–54 years and above 75 years of age, stroke was the second most common cause of death after assault and congestive heart failure.[84]

Literature stipulates that infarcts mainly affect patients over the age of 55 years and that the stroke risk increases with advancing age.[2] No studies have however been conducted to target the exact age group and infarct prevalence's

noted. The least number of infarcts were noted in age groups 18–29 years (2.96%), 30–39 years (8.28%) and 40–49 years of age (9.76%).

Ueda Y *et al*.[85] found that patients younger than 50 years had a higher prevalence of smoking (68% *versus* 48%, P=0.001) and obesity (42% *versus* 15%, P=0.0001). When comparing patients younger than 40 years to 40–50 year-olds they found the following difference: smoking: 88% *versus* 62%, P=0.05).[85]

6.2.4 Prevalence of infarcts according to sex

Males presented with a total of 161 infarcts, whilst females presented with 177. The male to female ratio, regardless of age, was therefore 1:1.10, which is not a significant difference. The Agincourt Health and Population Unit (AHPU)[86] of the University of Witwatersrand, has been monitoring the causes of deaths, births and migration by means of a health and demographic surveillance system in a population of ± 70 000 people since 1992.[86] The Southern Africa Stroke Prevention Initiative (SASPI) reported a male to female ratio of 1:1.8.[87]

6.2.5 Prevalence of infarct types according to infarct categories, age & sex

Normal cerebral infarcts accounted for 72.49% of the infarcts noted. The other infarct type prevalence's were as follows: embolic: 14.50%; Multi Infarct Dementia syndrome: 7.7%; haemorrhagic: 3.25%; lacunar: 2.37%; venous: 1.18% and watershed: 0.59%.

Cerebral infarcts (72.49%) presented with 57.55% new infarcts and 42.45% old infarcts and mainly affected patients aged 70–79 years (59.43%). Embolic infarcts only presented with new (21.88%) infarcts and also mainly affected the 70–79 year (16.98%) age group. The total number of males that presented with embolic infarcts was equal to the number of females affected. Males of all ages were affected, but mainly in the age group 70–79 years (57.14%). Females aged 18–39 years and above 80 years of age did not present with any embolic

infarcts. The highest female embolic prevalence was noted in age group 70–79 years of age (28.57%).

Haemorrhagic infarcts showed a more evenly distributed infarct presentation throughout the age groups, but did not affect age groups 30–39 years and above 80 years of age. Males presented with double the number of haemorrhagic infarcts when compared to their female counterparts. Literature states that haemorrhagic infarcts are mainly caused by embolism, which occurs due to the use of thrombolytic agents or anti-coagulants. Men are more prone to use anti-coagulants as treatment for cardiac disease.[87] Our research figures therefore supports these statements. Males aged 40–79 years of age presented with an equal number of infarcts in each age group. The other age groups, 18-39 years and above 80 years of age was not affected. Only one female aged 18–29 years of age and 50–59 years of age presented with a haemorrhagic infarct. Embolic haemorrhagic infarcts only affected patients aged 50–69 years of age and above 80 years of age. Venous haemorrhagic infarcts only affected patients aged 18–29 years and 50–59 years of age.

Lacunar infarcts only affected patients over the age of 50 years and targeted more men (62.50%) than women (37.50%). This correlates with literature that assert that the mean age of a first lacunar stroke is 65 and that men present with higher lacunar infarct prevalence than women.[2,88] The mean male age groups affected were 50–59 years and above 80 years of age (40%). Only females aged 50–59 years (66.67%) and 70–79 years (33.33%) were affected. Lacunar infarction occurs due to haemodynamic impairment of the microcirculation in the perforators. Metabolic disruption, due to cerebral ischaemia, breaks down the cell membrane sodium-potassium homeostasis. The intracellular fluid increases osmotically at the expense of water in the extracellular space. This correlates with cytotoxic oedema. Vasogenic oedema follows, which is a suspected progressive stroke factor. The perforating arteries

vary greatly in size and have many branches. DWI scans indicate that the haemodynamics of microcirculation in the perforating territory continually change in the acute stage of the lacunae.[89]
Autopsy studies also revealed that small lacunae are mainly caused by hypertensive small-artery disease, whilst large lacunar infarcts are caused by atheromatous or embolic perforator occlusion.[90]

Venous infarcts were only noted in females aged 18–29 years of age. The main cause of venous infarction is said to be venous thrombosis, which is more than often associated with the use of oral contraceptives and pregnancy.[19,82,83] During pregnancy blood is more likely to clot since the changes that occur in blood clotting are designed to reduce bleeding at the time of delivery. A dramatic reduction in blood flow speed also occurs in the veins, which carry blood from the legs back to the heart. This is due to the effect of the pregnancy hormones on the veins and also to the increased size of the uterus. Blood flow reduction becomes more obvious during week 16, and it becomes still more sluggish closer to full-term as the body prepares for delivery. Blood flow does not return to normal until 6 weeks after delivery. It is due to this reduction in blood flow, combined with the increased clotting tendency while pregnant, that can result in thrombosis.[82]

MID syndrome only affected patients aged 70–79 years. Fourteen females and eight males were affected. These figures contradict literature stats that more men than women suffer from MID. MID is caused by atherosclerosis, during which fatty deposits occur in the inner arterial lining. Platelets clump around the injured area. Cholesterol and fat collect at the site to form a mass within the arterial lining. Atherosclerotic plaques form multiple scattered blood clots (thrombi) that block off the small blood vessels, thereby preventing blood flow and oxygen supply to localized areas of the brain.[35,36] Men are more likely to present with high cholesterol, but due to our changing lifestyles, women are

evolving and adapting to present with the same problems. Multi-embolic infarcts only affected patients aged 50–59 years. Only one male patient aged 70–79 years of age presented with a micro infarct. Two females aged 40–49 years of age presented with watershed infarcts and only one male aged 40-49 years of age presented with a pointy infarct.

6.2.6 Brain Lobe infarct prevalence according to infarct categories, age & sex

No studies regarding brain lobe infarct predominance have been conducted. From this study it is noted that no significant difference exists between the left and right brain lobes when observing infarct prevalence (See Appendix C). The parietal lobe presented with the highest infarct prevalence (34.91%), followed by the frontal lobe (26.03%), occipital lobe (19.53%) and finally the temporal lobe (11.24%). This might be due to the fact that the parietal lobe is supplied by the MCA.[3] The MCA is the largest of all the cerebral arteries and the vessel that is most affected by cerebrovascular disease. Slater *et al*.[91] reported that MCA-territory infarcts affected 80 per 100 000 patients. The infarction site depends on the site of occlusion. An embolus will always remain in the blood vessel with the biggest diameter i.e. MCA.

When observing the lobes separately according to infarct categories, it is seen that the parietal (new: 46.35%, old: 25.44%) and frontal lobe (new: 36.46%, old: 15.79%) presented with double the number of new infracts than old infarcts. The occipital lobe however demonstrated an equal old *versus* new infarct distribution, whilst the temporal lobe (old: 14.04%, new: 11.46%) presented with more old than new infarcts.

When looking at the male *versus* female ratio in each lobe, it was found that the parietal lobe (males: 58; females: 60) and the frontal lobe (males: 45; females: 43) presented with an almost equal male to female ratio. The occipital lobe however, presented with more males (38) than females (28), whilst the temporal

lobe presented with more females (28) than males: (10). The overall male *versus* female ratio was however almost equal (males: 151, females: 159).

The brain lobes all presented with the highest infarct prevalence in the age group 70–79 years of age. This was closely followed by age groups 50–59 years and 60–69 years.

When observing infarcts in the brain lobes according to age and sex the following was noted: In age groups 18–49 years and above 80 years of age, more females than males were affected. This can be due to the fact that women tend to use oral contraceptives and fall pregnant at young ages.[19,82,83] Contraceptives and oestrogen therapy commonly causes venous thrombosis, which is a risk factor for developing infarcts. The risk of thrombosis is up to 9 times higher in oral contraceptive users. Haemodynamic studies found a slight dilation of veins in the legs of oral contraceptive users. Histological studies found that a lack of fibrinolytic activators, which causes thrombosis, was however not a characteristic of contraceptive users. A change in blood lipid levels have also been observed in users, but this is not conducive to thrombosis. It is believed that some of these combining factors as well as unknown reasons are the cause of thrombosis in oral contraceptive users.[19] Age groups 50–59 years and 70–79 years had more males, whilst age group 60–69 years presented with equal numbers of males and females. Females over 80 years presented with more infarcts when compared to their male counterparts. This can be due to the fact that women tend to live longer than men,[81] since men die at younger ages due to other co-morbidities such as heart attacks and cancer.

The “Initial burden of disease estimates for SA”, 2000, found that stroke was the most important non-communicable disease which caused death in females, compared to ischaemic heart disease in males.[87]

Three times more men aged 15–24 years die than women annually, due to reckless and violent behaviour, motor vehicle accidents, homicide, suicide and drowning. This is due to the so-called “testosterone-storm”, which increases aggression and the competitive behaviour in men. Testosterone also increases bad cholesterol (low-density lipoprotein) which increases their incidence of heart disease and stroke. Men aged 55–64 years of age are four times more likely to commit suicide and two times more likely to be in a motor vehicle accident than their female counterparts. The male death-rate due to diseases related to alcohol consumption and smoking is also higher and heart disease kills 5 in every 1000 men in this age group annually. Women generally die due to arthritis, osteoporosis and diabetes, since childbirth procedures are much safer. It is said that men die of disease, whilst women live with them. Oestrogen decreases bad cholesterol and increases good cholesterol, whilst oestrogen therapy also decreases the risk of dying from heart disease. Oestrogen regulates liver enzyme activity that’s involved in cholesterol metabolism and it acts as an anti-oxidant, which rids the body of free radicals. The average expected lifespan of men at birth is 72 years, compared to women, which is 79 years.[81]

6.2.7 Hemispheric predominance

The right hemisphere presented with 34.91% infarcts and the left hemisphere with 31.95%. This is contradictory to a previous conducted study that reported a mild left hemispheric infarct predominance. Billelo *et al*.[62] manually segmented Diffusion-Weighted Images and registered them to a common co-ordinate system. This computed probalistic map, showed mild left-sided predominance of brain infarcts, which likely represents asymmetry in eloquence of the brain regions.[62] It has also been found that right parietal lobe lesions can cause a patient to neglect the left side of the world, i.e. they do not groom their left side, they only draw the right side of their world in drawings and they constantly bump into things with the left side of their body. They are thus completely unable to integrate visual and somatosensory input from the left side. A left

parietal lobe lesion however, does not have this effect on patients, suggesting that the brain is asymmetrical.[92]

Bilateral hemispheric infarcts accounted for 23.37% of infarct cases. The rest was divided amongst other infarct types (7.99%) and central structured infarcts (1.78%). The right hemisphere presented with almost twice as many new infarcts than old infarcts (ratio of 1.79:1). The left hemisphere demonstrated an almost equal old *versus* new infarct distribution (ratio of 1:1.09). Bilateral hemispheric infarcts presented with almost four times as many new than old infarcts (ratio of 3.75:1). All hemispheric infarcts were most prevalent in age groups 70–79 years, followed by 50–59 years and 60–69 years. The figures for the male *versus* female ratio of the right, left and bilateral hemispheric infarcts were not significant.

Centrally situated infarcts presented with double the amount of males than females and other types of infarcts presented with double the amount of females than males.

6.2.8 Anatomical structures

The right MCA presented with the highest infarct prevalence (11.54%) when observing infarcts according to anatomical structures affected. It also presented with more new than old infarcts (2.5:1) and affected males and females of all age groups. The male to female ratio was the same except in age groups 30–39 years (M:F = 1:4) and above 80 years of age (M:F = 1:5). Females aged 18–39 years of age presented with twice as many infarcts when compared to their male counterparts. This is due to pregnancy[82] and the use of oral contraceptives[19,83] as discussed earlier. The left MCA presented with the second highest infarct prevalence (8.88%) and consisted of three times more new than old infarcts. In the age group 70–79 years of age, males presented with double the amount of infarcts than females. To summarize the MCA, it was

noted that the artery presented with more new than old infarcts and with no other types of infarcts (lacunar, etc.). The male to male ratio (right *versus* left) was the same but females presented with more left MCA infarcts than right MCA infarcts (1.64:1).

The high MCA infarct prevalence was expected, since the most common reported infarct location is the MCA (75% of cases).[17] The MCA arises as a single trunk, is 18–26 mm long with a diameter of ± 3 mm. Perforating branches consist of 15 to 17 small lenticulostriate arteries that supply the putamen, lentiform nucleus, internal capsule and the caudate nucleus. The MCA then bifurcates into superior and inferior divisions. The superior division supplies the prefrontal and orbitofrontal cortex, whilst the inferior branch supplies the middle and polar temporal regions.[91]

The right lentiform nucleus presented with six times more new *versus* old infarcts and had an infarct prevalence of (2.07%). The left lentiform nucleus presented with three times more new *versus* old infarcts and had an infarct prevalence of 3.55%. The male to female ratio was 3:1. The lentiform nucleus as a whole therefore presented with more new than old infarcts and no other types of infarcts. The female ratios were the same, whilst the males were more affected in the right nucleus than the left (3:1).

The internal capsule (2.37% infarct prevalence) only presented with new infarcts. The globus pallidus (M:F = 2:0), putamen (M:F = 3:0), thalamus (M:F = 2.5:1), subcortical white matter (M:F = 3:1) and the medulla oblongata (M:F = 5:1) affected more males than females. All of the abovementioned structures are supplied by the MCA. MCA infarct location depends largely on the size of the embolic mass. Stem occlusion is rare and requires embolic matter of about 3 to 5 mm. Emboli arise because of intravascular rigid foreign matter, such as large thrombi with bacteria, large plaques due to direct or internal carotid artery

trauma, or puncture.[91] It therefore explains the infarct development of these structures, since they are all supplied by the MCA.[3]

The right PCA only presented with new infarcts and had an infarct prevalence of 2.37% and a male to female ratio of 1:3. The left PCA also only presented with new infarcts but had an infarct prevalence of 1.78% and a male to female ratio of 1:2. The PCA therefore only presented with new infarcts and affected a lot more females (1.5:1), when comparing the left and right PCA.

In most individuals the PCA stems from the basilar artery and sometimes from the ipsilateral ICA, via the posterior communicating artery. The posterior arteries supply the occipital and temporal lobes of the cerebral hemispheres. When infarction occurs in the PCA, it is usually secondary to embolism from lower segments of the heart or the vertebrobasilar system.[3]

The right CSO presented with 10.5 times more new than old infarcts and had an infarct prevalence of 6.80%. The left CSO presented with 8.5 times more new than old infarcts and had an infarct prevalence of 5.62%. When comparing the amount of females affected in the left and right CSO, it was noted that more females were affected in the right CSO (1.75:1).

The left vertebral artery did not present with any infarcts, whilst the right vertebral artery only presented with two infarcts in two female patients. The vertebral artery arises from the subclavian artery and is 3 to 5 mm in diameter. In 6% of cases the left vertebral artery arises directly from the aortic arch. The vertebral artery consists of four parts. The first part enters the transverse foramina at the level of C5 or C6. The second part courses within the intervertebral foramina. As it exists behind the atlas going into the foramen magnum, part three starts. The last part is formed when the artery pierces through the dura and arachnoid mater at the base of the skull to form the

midline basilar artery. In 15% of healthy patients one vertebral artery is atretic (<2 mm in diameter). In 50% of cases the left vertebral artery is the dominant branch as compared to the right vertebral artery that was found to be the dominant branch in 25% of cases. In the other 25% of cases the left and right vertebral arteries are similar in caliber.[93] The left vertebral artery sometimes appears as a direct branch of the aorta or as a normal branch of the subclavian artery. The left subclavian artery is also a direct branch of the aorta, whilst the right subclavian artery branches from the brachiocephalic trunk. The left subclavian artery therefore has better blood flow than the right side due to its origin from the aorta.[3]

The left anterior cerebral artery was also not affected by infarcts, whilst the right anterior cerebral artery had an infarct prevalence of 1.18% and a male to female ratio of 3:1.

6.2.9 Harvard Classification infarct prevalence

The vascular structures (26.63%) and white matter (18.34%) were most affected by infarcts. This correlates with the high infarct prevalence noted in the right parietal lobe and the middle cerebral artery. The main cause of infarction is the acute occlusion of an artery[1] – especially the middle cerebral artery.[17] No significant difference between the number of males and females could be found, except in the brainstem where double the number of males appeared, when compared to their female counterparts. This correlates with the figures found that indicated that the number of males that presented with central structured infarcts (i.e. the midbrain, pons and medulla oblongata are part of the brainstem) were double the number of females affected. The brainstem is the final pathway between the cerebral structures and the spinal cord. Stroke in the brainstem usually occurs due to basilar occlusion. The basilar artery is formed at the base of the skull from the vertebral arteries.[3] Infarcts in this region therefore cause headaches; dizziness and difficulty in swallowing. Infarcts in this region can be very serious or even fatal, since it's the main structure that is responsible for vital functions, such as respiration, heartbeat and blood

pressure. Since men suffer more from hypertension,[94] it explains the higher male than female ratio found.

In age groups 18–39 years of age, young males presented with three times less infarcts when compared to the number of females affected. This again, as stated before, is due to women's use of oral contraceptives and pregnancy, which often causes embolism and venous thrombosis.[19,82,83] It appears that plasma from oral contraceptive users are resistant to the anticoagulant action of activated protein C (APC). A cross-over study also found that increased fibrinolytic activity is present in oral contraceptive users, but that it is counterbalanced by increased fibrinolytic activity during protein activity that participates in the inhibition of fibrinolysis.[83] The vascular system is the only system that was affected in all of the age groups when looking at both sexes. The white matter followed closely, but was absent in men aged 18–29 years.

6.2.10 Measurements

The mean and standard deviation circumference and surface area measurements were very high. This was expected since infarcts vary greatly in size and shape. It is recommended that a follow-up study be performed using a volume measuring program. Up to date no program has been developed that can be incorporated in practice.

An ANOVA one-way analysis was performed on the circumference measurements of infarcts. Left hemispheric infarcts presented with significant differences, indicating that left hemispheric infarcts vary greatly in size (length, width and circumference). Right hemispheric infarcts appeared to be more stable in size, which might indicate that the damage is more predictable.

7. Conclusion

In summary, this study indicated that women presented with more old infarcts than men, due to the high infarct prevalence's in young females caused by pregnancy and the use of oral contraceptives. The most common age group affected was 70–79 years, which correlates with literature.

Lacunar infarcts were more prevalent in men and only occurred in patients over 55 years, as stipulated in literature. Venous infarcts only presented in young females and were mainly thrombotic in origin. MID only affected patients aged 70–79 years of age, as was expected, since this disease occurs due to age-related changes. In our study however, MID was more prevalent in women than men.

The right hemisphere presented with mild infarct predominance with the right parietal lobe and right MCA presenting with the highest infarct prevalence's.

The uniqueness of this study is the in-depth evaluation of infarcts according to dominant brain lobe, hemisphere and structures affected, as well as the different infarct categories (old, new, other) and different infarct type prevalence's (i.e. lacunar, etc.) noted. No other studies have focused on these factors. This study therefore pinpointed the exact infarct locations found. This is in contrast to other studies which mainly focused on one infarct factor i.e. infarct territory, causative factor or preferred modality to be used. This is also one of few South African population-based studies performed concerning stroke prevalence's noted in South Africa (Gauteng). It is therefore recommended that other studies be performed in other regions on the South African population, focusing on population affinity and socio-economic determinants. This study can therefore be used as a baseline comparative study.

References

1. Ricci PE. Imaging of cerebral ischemia and infarction. *Stroke* 2000; **11(2).**
2. Osborn AG, Hedlund GL, Blaser SI, Salzman KL, Katzman GL, Provenzale J, Castillo M, Illner A, Hornsberger HR, Cooper JA, Jones BV, Hamilton BE. Diagnostic Brain Imaging. 1st ed. Amirsys Inc: Salt Lake City, 2004.
3. Williams PE, Bannister LH, Berry MM *et al*. Gray's Anatomy: The anatomical basis of medicine and surgery. 38th ed. Churchill Livingstone: Edinburgh, 1999.
4. Windle BCA. Arteries forming the Circle of Willis. *J Anat Thysiol* 1888; **22**:289-293.
5. Fawcett E, Blachford JV. The Circle of Willis: an examination of 700 specimens. *J Anat* 1906; **40:**63-70.
6. Riggs HE, Rupp C. Variation in form of circle of Willis. *Arch Neurol Psychiat* 1963; **8**:8-14.
7. Puchades-Orts A, Nambela-Gamez M, Ortuno-Pacheco G. Variation in form of the Circle of Willis. Some anatomical and embryological considerations. *Anat Rec* 1976; **185**:119-123.
8. Fields WS, Bruetman ME, Weibel J (eds.). *Collateral circulation of the brain*. Baltimore: Williams & Wilkins, 1965.
9. Van Overbeeke JJ, Hillen B, Bruetman ME, Tulleken CAF. A comparative study of the circle of Willis in fetal and adult life. The configuration of the posterior bifurcation of the posterior communicating artery. *J Anat* 1991; **176**:45-54.
10. McCormick WF. Vascular disorders of nervous tissue: anomalies; malformations and aneurysms. In: Bourne GH (ed). *Structure and function of nervous tissue*. New York: Academic Press. 1969: 537-596.

11. Zeal AA, Roton AL. Microsurgical anatomy of the post cerebral artery. *J Neurosurg* 1978; **48**:534-559.
12. Abbie AA. The blood supply of the geniculate body, with a note on the morphology of the choroidal arteries. *J Anat* 1933; **67**:491-521.
13. Williams DJ. Origin of posterior cerebral artery. *Brain* 1936; **59**:175-180.
14. Kaplan HA. Arteries of the brain; anatomical study. *Acta Radiol* 1956; **46:**364-470.
15. Sedzmir CB. An angiographic test of collateral circulation through the anterior segment of the circle of Willis. *J Neurol Neurosurg Psychiat* 1959; **22:**64-68.
16. Osborn AG. Diagnostic Cerebral Angiography. 2nd ed. Philadelphia: Lippincott Williams & Wilkins: 1999: 381-403.
17. Osborn AG. Stroke. Diagnostic Neuroradiology. St Louis: Mosby, 1994: 330–398.
18. Khan SN, Vohra EA. Risk factors for stroke: A hospital based study. *Pak J Med Sci: January – March* 2007; **23**:117-122.
19. Abrahamsen AF. Oral contraceptives and the incidence of thrombosis. *Tidsskrift for den Norske Laegeforening* 1970; **90**:403-408. (Abstract: Norwegian)
20. Prabhakaran S, Wright CB, Yoshita M, Delapaz R, Brown T, DeCorli C, Sacco RL. The prevalence and determinants of sub-clinical brain infarction. The Northern Manhattan Study. *Neurology* 2008; **70**:425-430.
21. Dishman RIC, Washburn RA, Heath G. Physical activity epidemiology. *Medical Human Kinetics* 2003, 115-117.
22. Williams GH, Braunwald E. In: Braunwald E, et al. Harrison's principles of Internal medicine. New York, McGraw Hill. 1987:1024.
23. Vermeer SE, Hollander M, van Dijk EJ, Hofman A, Koudstaal PJ, Breteler MMB. Silent brain infarcts and white matter lesions increase stroke risk in the general population. The Rotterdam Scan Study. *Stroke* 2003; **34**:1126-1129.

24. Albers GW, Barnett HJM et al. Guidelines for the management of transient ischemic attacks. From the Ad Hoc Committee on guidelines for the management of transient ischemic attacks of the Stroke Council of the American Heart Association. *Stroke* 1994; **25**:1320.
25. Launer LJ. Migraine in mid-life predicts brain infarcts in old age: A Longitudinal population-based MRI study. 132nd American Neurological Association Annual Meeting, October 9, 2007.
26. Ginsberg MD. The new language of cerebral ischemia. *AJNR* 1997; **18**:1435–1445.
27. Afifi AK, Bergman RA. The Basal Ganglia. Functional Neuro-anatomy: Text and Atlas. McGraw Hill, 1998: 275-285.
28. Agamandis DP. An illustrated interactive course for medical students' and residents. *Neuropathology*: Chapter 2: Cerebral ischaemia and stroke. North Eastern Ohio Universities' College of Medicine. September 2006.
29. Caplan LR. Hemorrhage into embolic brain infarcts. *Pharmacotherapy* 1999; **19(2):**125-127.
30. Anderson JR. Muir's Textbook of pathology. 11th ed. Edward Arnold, 1980: 743-744.
31. Bamford JM, Warlow CP. Evaluation and testing of the lacunar hypothesis. *Stroke* 1988; **19(9):**1074-1082.
32. Agranoff AB, Wong EH. Lacunar stroke. October 11, 2006. E-medicine.
33. Pullicino P, Caplan LR, Hammel M. Lacunar syndromes. In: Pullicino PM, Caplan LR, Hammel M, editors. Cerebral small artery disease. New York: Raven Press, 1993:141-160.
34. Beers MH, Berkew R. The Merck Manual of Diagnosis & Therapy. Whitehouse Station; New York: Merck Research Laboratories, 1999. www.minddisorders.com
35. Erkinjuntii MIP, Sulkova R. Diagnosis of MID. Alzheimer's disease. *Association Disorders* (Summer) 1991; **5(2):**112-121.

36. Meyer JS, McClintic KL, Rogers RL, Sims P, Mortel KF. Aetiologyal considerations and risk factors for MID*. J Neurol Neurosurg Pshyciatry* 1988 Dec **51(12):**1489-1497.
37. Chaves CJ, Silver B, Schlaug G, Dashe J, Caplan L, Warach S. Diffusion- and Perfusion-Weighted MRI Patterns in Borderzone infarcts. *Stroke* 2000; **31(5):** 1090.
38. Hendrikse J, Hartkamp MJ, Hillen B, Mali W, van der Grond J. Collateral ability of the Circle of Willis in patients with unilateral internal carotid artery occlusion: Border zone infarcts and clinical symptoms. *Stroke* 2001; **32**:2768.
39. Adams HP. Guidelines for early management of patients with ischemic stroke. *Stroke* 2003; **34(4):**1056-1083.
40. Davis SM, Hand PJ, Dannon GA. Tissue plasminogen activator for ischemic stroke: Highly affective, reasonably safe and grossly underused. *MJA* 2007; **187(10):**548-549.
41. National Institute of neurological Diseases and stroke. Rt-PA Stroke Study Group. TpA for acute ischaemic stroke. *N Engl J Med* 1995; **333**:1581-1587.
42. Merino JG, Lawrence L, Jason W *et al.* Lesion volume change after treatment with tPA can discriminate clinical responders from non-responders. *Stroke* 2007; **38(11):** 2919 2923.
43. Lindgren A, Lindoff C, Norrving BO *et al*. Tissue plasminogen activator & plasminogen activator inhibitor-1 in stroke patients. *Stroke* 1996; **27**:1066-1071.
44. Xavier AR, Qureshi AI, Kirmani JF, Yahia AM, Bakshi R. Neuroimaging of stroke: A review. *SouthMed J* 2003; **96(4):**367-379.
45. Wegener OH et al. Whole Body Computed Tomography. 2nd edition. Blackwell Scientific Publications: Boston; 1993.
46. Rowley HA, Roberts HC, Dillon WP, et al. Early CT signs in 177 angiographically proved MCA occlusions-PROACT II findings.

Proceedings of 38th Annual Meeting of the ASNR, April 3-8, 2000, Atlanta GA, page 47.

47. Bryan RN, Levy LM, Whitlow WD, Kilian JM, Preziosi TJ, Rosario JA. Diagnosis of acute cerebral infarction: comparison of CT and MR imaging. *AJNR* 1991; **12(4):** 611-620.
48. Brott T, Marler JR, Olinger CP, et al. Measurements of acute cerebral infarction: lesion size by computed tomography. *Stroke* 1989; **20:**871-875.
49. Hertanu JS, Demopoulos JT, Yang WL, *et al*. Stroke rehabilitation: correlation and prognostic value of computerized tomography and sequential functional assessments. *Arch Phys Med Rehabil* 1984; **65**: 505-508.
50. Bakshi R, Ketonen L. Brain MRI in clinical neurology, In: Joynt RJ, Griggs RC (eds): Baker's Clinical Neurology on CD-ROM, 2001 Edition. Philadelphia, Lippincott Williams & Wilkins, 2001.
51. Ricci PE, Burdette JH, Elster AD, Reboussin DM. A comparison of fast spin-echo, fluid-attenuated inversion-recovery, and diffusion-weighted MR imaging in the first 10 days after cerebral infarction. *AJNR* 1999; **20(8):**1535-1542.
52. LeBihan D, Breton E, Grenier P, cabanis E, Laval-Jeantet L. MR imaging of intravoxel incoherent motions: Application to diffusion and perfusion in neurological disorders. *Radiology*. 1986; **161**:401-407.
53. Caso V, Budak K, Georgiadis D, Schuknecht B, Baumbartner RW. Clinical significance of detection of multiple acute brain infarcts on diffusion weighted magnetic resonance imaging. *Journal of Neurology, Neurosurgery and Psychiatry* 2005; **76**:514-518.
54. Lansberg MG, Albers GW, Beaulieu C, Marks MP. Comparison of diffusion-weighted MRI and CT in acute stroke. *Neurology* 2000; **54(8):**1557-61.

55. Sorensen AG, Copen WA, Ostergaard L, et al. Hyper acute stroke: simultaneous measurement of relative cerebral blood volume, relative cerebral blood flow, and mean tissue transit time. *Radiology* 1999; **210(2):**519-527.
56. Noguchi K, Ogawa T, Inugami A et al. MRI of acute cerebral infarction: comparison of FLAIR & T2-weighted fast spin-echo imaging. *Neuroradiology* 1997; **39**:406-410.
57. Kamran S, Bates V, Bakshi R, Wright P, Kinkel W, Miletich R. Significance of hyperintense vessels on FLAIR MRI in acute stroke. *Neurology* 2000; **(55)**: 265-269.
58. Burgener FA, Tan RK, Meyers SP, Zaunbauer W, Raymond K (eds.). *Differential Diagnosis in Magnetic Resonance Imaging*. New York: Thieme Stuttgart, 2002.
59. Cotran RS, Kumar V, Robbins SL,: Robbins Pathologic Basis of Disease. 5th ed. Philadelphia*, W.B. Saunders* 1994: 1309-1311.
60. Van der Lugt A. Incidental MRI findings, including subclinical infarcts and tumors: A Rotterdam study. *N. Engl J Med* 2007; **357**:1821 – 1828.
61. Saunders DE, Clifton AG, Brown MM. Measurement of infarct size using MRI predicts prognosis in middle cerebral artery infarction. *Stroke* 1995; **26**:2272-2276.
62. Bilello M, Lao Z, Krejza J, Hillis AE, Herskovits EH. Statistical atlas of acute stroke from magnetic resonance diffusion-weighted-images of the brain. *Neuro-informatics* 2006, **4(3):** 235-42.
63. Dvalos A, Thrombolisis in acute ischemic stroke: Successes, failures and new hope in acute ischemic stroke. *Cerebrovas Pls* 2005; **20**:135-9.
64. Arboix A. Thrombolytic treatment in acute cerebral infarction. *Neurol India* 2007; **55**:8-9.
65. Ingelheim B. South African Launch of First Treatment for Acute Ischaemic Stroke. The Doctor's Newspaper: 41st yr. *Medline Chronicle*: January 2007. Circ 13500.

66. Unterberg A, Juettler E. The role of surgery in ischemic stroke: Decompressive surgery. *Critical Care* 2007; **13(2):**175-179.
67. Park J, Park D, Kim S *et al*. Surgical Treatment for Acute Severe Brain Infarction. *J Korean Neurosurg Soc* 2007; **42**: 326-330.
68. Martel AL, Allder SJ, Delay GS, Morgan PS, Moody AR. Measurement of infarct volume in stroke patients using adaptive segmentation of diffusion-weighted MR Images. In: *Medical Image Computing and Computer-Assisted Intervention (MICCAI). Lecture Notes in Computer Science* 1999; **1679**:22-31.
69. Canella KA. Another decision to evaluate: choice of standardized mean difference effect size estimator. *Syst Rev Evid Action Int Cochrane Colloq 6th 1998 Baltim Md* 1998; **6**:60.
70. Vohra EA, Ahmed WU, Ali M. Etiology and prognostic factors of patients admitted for stroke. *J Pak Med Assoc* 2000; **50(7):**234-236.
71. Murray CJL, Lopez AD. The Global Burden of disease: A comprehensive assessment of mortality and disability from diseases, injuries and risk. Factors in 1990 and projected to 2020. Harvard University Press: Boston 1996.
72. Neurological, Psychiatric and Developmental Disorders: Meeting the challenges in the developing world. Committee on Nervous System Disorders in Developing Countries. Board on Global Health: Institute of Medicine. National Academy Press, Washington 2001.
73. South African Medical Association. Neurological Association of South Africa. Stroke working group. Stroke therapy clinical guidelines. *South African Medical Journal* 2000; **(90):**292-306.
74. Wolf-Maier K, Cooper RS, Banegas JR, Giampaoli S, Hense H, Joffres M, Kastarinen M, Poulter N, Primatesta P, Rodriguez-Artalejo F, Stegmayr B, Thamm M, Tuomilehto J, Vanuzzo D, Vescio F. Hypertension prevalence and blood pressure levels in 6 European countries, Canada, and the United States. *JAMA* 2003; **289**: 2363–2369.

75. Giovannoni G, Fritz VU. Transient Ischemic Attacks in younger and older patients. A comparative study of 798 patients in South Africa. *Stroke* 1993; **24**:947-953.
76. Hoffmann M. Stroke in the Young South Africa – an analysis on 320 patients. *S Afr Med J* 2000; **90**:1226-237.
77. Connor MD, Thorogood M, Casserley B, Dobson C, Warlow CP. SASPI Project team. Prevalence of stroke survivors in rural South Africa, results from the Southern Africa Stroke Prevention Initiative (SASPI) Agincourt field site. *Stroke* 2004; **35**:627-632.
78. New Internationalist 2002, Issue 346.
79. AIDS Epidemic Update, UNAIDS, December 2001.
80. Crabb C. HIV link with vascular disease and stroke. *AIDS* 2004; **18(11).**
81. Perls TT, Frets RC. Why women live longer than men. *Scientific American Presents*1998; **9**:100-104.
82. Walters BNJ, Franzcog F, Eundem AD. Venous thrombosis and embolism in pregnancy. *O&G (Obstetrics & Gynaegology)* 2005 ; **7(4):**44-45.
83. Rosing J. Oral contraceptives, thrombosis and haemostasis*. European Journal of Obstetrics & Gynecology and Reproductive Biology;* **95(2):**193–197.
84. Kahn K, Tollman SM. Stroke in rural South Africa: Contributing to the little known about a big problem. *S Afr Med J* 1999; **(89):**63-65.
85. Ueda Y, Okada K, Ogasawara N, Oyabu J, Hirayama A, Kodama K. Acute myocardial infarction without disrupted yellow plaque in young patients below 50 yrs old. *J Interven Cardiol* 2007; **20(3):** 177-181.
86. Tollman SM, Herbst K, Garenne M. The Agincourt Demographic and Health Study: Phase I. Johannesburg South Africa: Department of Community Health, University of Witwatersrand, Health Systems Development Unit; 1995. (Monograph)

87. Connor M, Bryer A. Stroke in SA. In: Steyn K, Fourie J, Temple N (eds). Chronic Diseases of Lifestyle in South Africa. Technical Report. Cape Town. South African Medical Research Council. Chapter 14, 1995–2005.
88. Bejot Y, Catteau A, Caillier M, Rouaud O, Durier J, Marie C, DiCarlo A, Osseby GV, Moreau T, Giroud M. Trends in incidence, risk factors, and survival in symptomatic lacunar stroke in Dijan, France, from 1989 to 2006: A population-based study. *Stroke* 2008; **39(7):**1945-1951.
89. Terai S, Hori T, Miake S, Tamaki K, Saishoji A. Mechanism in progressive lacunar infarction. *Arch Neurol* 2000; **57(2):**255-258.
90. Baumgartner RW, Sidler C, Mosso M, Georgiadis D. Ischemic lacunar stroke in patients with and without potential mechanism other than small-artery disease. *Stroke* 2003; **34(3):**653.
91. Slater DI, Mallahan E, Wetzel P, Cifu DX, Corne W, Baron MS, Johns JS.. MCA Stroke. *Amer J Phys Rehab* 2006*; **47(7):**619-627.*
92. Daube JR, Reagan TJ, Sandok BA, Westmoreland BF**.** Medical Neurosciences: An Approach to Anatomy, Pathology, and Physiology by Systems and Levels. Boston/Toronto: Little, Brown and Company: New York; 1994.
93. Cloud GC, Markus HS. Diagnosis and management of vertebral artery stenosis. *Q J Med* 2003; **96**:27-54.
94. August P. Hypertension in men*. The Journal of Clinical Endocrinology and Metabolism*, **84(10):**3451-3454.

Appendix A: Abbreviations used

- **MID:** Multi infarct dementia
- **CSO:** Centrum semi ovale
- **MCA:** Middle cerebral artery
- **ACA:** Anterior cerebral artery
- **PCA:** Posterior cerebral artery
- **ICA:** Internal carotid artery
- **n:** Number
- **%:** Percentage
- **M:F:** Male to female ratio
- **(f):** Female patients
- **(m):** Male patients
- **L:** Left
- **R:** Right
- **TIA:** Transient ischaemic attack
- **LDL:** Low density lipo-protein
- **HDL:** High density lipo-protein
- **CBF:** Cerebral blood flow
- **tPA/ PLAT:** Tissue plasminogen activator
- **FDA:** Food & Drug Administration
- **CT:** Computed tomography
- **MRI:** Magnetic resonance imaging
- **HU:** Hounsfield units
- **DWI:** Diffusion-Weighted Imaging
- **FLAIR:** Fluid-attenuated inversion recovery
- **rCBF:** Relative cerebral blood flow
- **rCBV:** Relative cerebral blood volume
- **SD:** Standard Deviation
- **df:** Degrees of freedom
- **F:** Frequency

Appendix B: Monthly statistics regarding the prevalence of infarcts

Months	Total Brain MRI's	Not Found	Children	Sample Size	No Infarcts	Infarct %
Jan '06	210	14	35	161	27	16.77%
Feb '06	222	15	27	180	29	16.11%
Mrch '06	215	20	37	158	24	15.19%
Apr '06	195	15	37	143	25	17.48%
May '06	208	7	40	161	21	13.04%
Jun '06	247	27	38	182	27	14.84%
Jul '06	207	30	33	144	16	11.11%
Aug '06	231	34	46	151	26	17.22%
Sept '06	214	30	42	142	14	9.86%
Oct '06	252	34	38	180	21	11.67%
Nov '06	229	35	33	161	25	15.53%
Dec '06	136	12	16	108	20	18.52%
Jan '07	226	17	37	172	24	13.95%
Total Sample Size	**2 588**	**290**	**454**	**1 844**	**299**	**16.21%**

Appendix C: Summary of Brain lobe infarcts according to age and sex

a. **Prevalence according to infarct categories**

Lobe	Old	New	Total
Right Parietal	11.40%	6.56%	18.93%
Left Parietal	14.04%	19.79%	15.98%
Parietal Lobe	**25.44%**	**46.35%**	**34.91%**
Left Frontal	7.02%	19.27%	13.31%
Right Frontal	8.77%	17.19%	12.72%
Frontal Lobe	**15.79%**	**36.46%**	**26.03%**
Left Occipital	13.16%	10.42%	10.36%
Right Occipital	7.89%	10.94%	9.17%
Occipital Lobe	**21.05%**	**21.36%**	**19.53%**
Right Temporal	7.02%	7.29%	6.51%
Left Temporal	7.02%	4.17%	4.73%
Temporal Lobe	**14.04%**	**11.46%**	**11.24%**

b. **Brain Lobe infarcts according to sex**

LOBE	MALE	FEMALE		LOBE	MALE TOTALS	FEMALE TOTALS
R Parietal	31	33		**Parietal Lobe**	58	60
L Parietal	27	27				
L Frontal	22	23		**Frontal Lobe**	45	43
R Frontal	23	20				
L Occipital	21	14		**Occipital Lobe**	38	28
R Occipital	17	14				
R Temporal	6	16		**Temporal Lobe**	10	28
L Temporal	4	12				
Totals	**151**	**159**				

c. **Brain Lobes according to age**

LOBE	18–29	30–39	40–49	50–59	60–69	70–79	> 80
R Parietal	2	4	5	15	11	16	11
L Parietal	1	4	5	15	11	12	5
L Frontal	1	2	3	10	11	12	6
R Frontal	-	6	2	8	10	12	4
L Occipital	-	3	4	6	3	12	7
R Occipital	-	5	4	4	2	13	3
R Temporal	3	4	2	4	3	1	5
L Temporal	1	3	2	3	3	3	1
Totals	8	31	27	65	54	81	42

d. **Brain Lobe infarcts according to age & sex**

AGES	18–29		30–39		40–49		50–59		60-69		70-79		> 80	
LOBES	**M**	**F**	**M**	**F**	**M**	**F**	**M**	**F**	**M**	**F**	**M**	**F**	**M**	**F**
R Parietal	-	2	2	2	2	3	7	8	6	5	11	5	3	8
L Parietal	-	1	1	3	1	4	10	5	6	5	7	5	2	3
L Frontal	-	1	-	2	1	2	7	3	7	4	7	5	-	6
R Frontal	-	-	2	4	1	1	4	4	6	4	8	4	2	2
L Occipital	-	-	1	2	3	1	5	1	1	2	8	4	3	4
R Occipital	-	-	2	3	2	2	3	1	1	1	8	5	1	2
R Temporal	1	2	1	3	1	1	1	3	-	3	-	1	2	3
L Temporal	1	-	1	2	1	1	1	2	-	3	-	3	-	1
Totals	**2**	**6**	**10**	**21**	**12**	**15**	**38**	**27**	**27**	**27**	**49**	**32**	**13**	**29**

Appendix D: Anatomical structure infarct prevalence's

Anatomical structure	Infarct Prevalence No	%	Male Prevalence No	%	Female Prevalence No	%
	no	%	no	%	no	%
R MCA	39	11.54	16	41.03	23	58.97
L MCA	30	8.88	16	53.33	14	46.67
R Lentiform nucleus	7	2.07	3	42.86	4	57.14
L Lentiform nucleus	12	3.55	9	75	3	25
Internal Capsule	11	3.25	5	45.45	6	54.55
R PCA	8	2.37	2	25	6	75
L PCA	6	1.78	2	33.33	4	66.67
R CSO	23	6.80	9	39.13	14	60.87
L CSO	19	5.62	11	57.89	8	42.11
Subcortical white matter	8	2.37	6	75	2	25
Periventricular white matter	10	2.96	6	60	4	40
R Vertebral artery	-	-	-	-	-	-
L Vertebral artery	2	0.59	-	-	2	100
Medulla Oblongata	6	1.78	5	83.33	1	16.67
Pons	16	4.73	10	62.50	6	37.50
Lateral Ventricle	20	5.92	9	45	11	55
Sylvian Fissure	11	3.25	5	45.45	6	54.55
R ICA	8	2.37	4	50	4	50
L ICA	8	2.37	5	62.50	3	37.50
Cerebellum	13	3.85	7	53.85	6	46.15
Vermis	3	0.89	2	66.67	1	33.33
Brainstem	10	2.96	6	60	4	40
Globus Pallidus	2	0.59	2	100	-	-
Midbrain	4	1.18	2	50	2	50
Cerebral Peduncle	1	0.30	1	100	-	-
Basal Ganglia	13	3.85	7	53.85	6	46.15
Corona Radiations	7	2.07	4	57.14	3	42.86
Thalamus	7	2.07	5	71.43	2	28.57
L ACA	-	-	-	-	-	-
R ACA	4	1.18	3	75	1	25
L Caudate nucleus	8	2.37	5	62.50	3	37.50
Precentral Gyrus	2	0.59	2	100	-	-
R Caudate nucleus	4	1.18	2	50	2	50
Optic tract	1	0.30	1	100	-	-
External Capsule	3	0.89	2	66.67	1	33.33
Corpus Callosum	2	0.59	1	50	1	50
Putamen	3	0.89	3	100	-	-
Frontal Gyrus	1	0.30	-	-	1	100
Postcentral Sulcus	1	0.30	1	100	-	-

Appendix E: Comparing Infarct Quantities in Brain Lobes and Structures

a. **Comparing Left & Right Frontal Lobe Infarct Quantities**

Frontal Lobe	Infarcts Present	Chi^2	df	P-value
Right Frontal	43			
Left Frontal	45			
Total Infarcts Noted	*88*			
Symmetry (asymptotic)		0.09	1	*0.7681*
Marginal homogeneity (Stuart-Maxwell)		0.09	1	*0.7681*

b. **Comparing Left & Right Temporal Lobe Infarct Quantities**

Temporal Lobe	Infarcts Present	Chi^2	df	P-value
Right Temporal	22			
Left Temporal	16			
Total Infarcts Noted	*38*			
Symmetry (asymptotic)		1.00	1	*0.3173*
Marginal homogeneity (Stuart-Maxwell)		1.00	1	*0.3173*

c. **Comparing Left & Right Occipital Lobe Infarct Quantities**

Occipital Lobe	Infarcts Present	Chi^2	df	P-value
Right Occipital	31			
Left Occipital	35			
Total Infarcts Noted	*66*			
Symmetry (asymptotic)		0.36	1	*0.5465*
Marginal homogeneity (Stuart-Maxwell)		0.36	1	*0.5465*

d. **Comparing Left & Right Parietal Lobe Infarct Quantities**

Parietal Lobe	Infarcts Present	Chi^2	df	P-value
Right Parietal	64			
Left Parietal	54			
Total Infarcts Noted	*118*			
Symmetry (asymptotic)		1.35	1	*0.2450*
Marginal homogeneity (Stuart-Maxwell)		1.35	1	*0.2450*

e. **Comparing Left & Right Frontal Lobe Infarct quantities in Males**

Frontal Lobe	Infarcts Present	Chi^2	df	P-value
Right Frontal	23			
Left Frontal	22			
Total Infarcts Noted	*45*			
Symmetry (asymptotic)		0.05	1	*0.8273*
Marginal homogeneity (Stuart-Maxwell)		0.05	1	*0.8273*

f. **Comparing Left & Right Frontal Lobe Infarct quantities in Females**

Frontal Lobe	Infarcts Present	Chi^2	df	P-value
Right Frontal	20			
Left Frontal	23			
Total Infarcts Noted	*43*			
Symmetry (asymptotic)		0.36	1	*0.5485*
Marginal homogeneity (Stuart-Maxwell)		0.36	1	*0.5485*

g. **Comparing Left & Right Temporal Lobe Infarct quantities in Males**

Temporal Lobe	Infarcts Present	Chi^2	df	P-value
Right Temporal	6			
Left Temporal	4			
Total Infarcts Noted	*10*			
Symmetry (asymptotic)		0.40	1	*0.5271*
Marginal homogeneity (Stuart-Maxwell)		0.40	1	*0.5271*

h. **Comparing Left & Right Temporal Lobe Infarct quantities in Females**

Temporal Lobe	Infarcts Present	Chi^2	df	P-value
Right Temporal	16			
Left Temporal	12			
Total Infarcts Noted	*28*			
Symmetry (asymptotic)		0.62	1	*0.4328*
Marginal homogeneity (Stuart-Maxwell)		0.62	1	*0.4328*

i. **Comparing Left & Right Occipital Lobe Infarct quantities in Males**

Occipital Lobe	Infarcts Present	Chi^2	df	P-value
Right Occipital	17			
Left Occipital	21			
Total Infarcts Noted	*38*			
Symmetry (asymptotic)		0.67	1	*0.4142*
Marginal homogeneity (Stuart-Maxwell)		0.67	1	*0.4142*

j. **Comparing Left & Right Occipital Lobe Infarct quantities in Females**

Occipital Lobe	Infarcts Present	Chi^2	df	P-value
Right Occipital	14			
Left Occipital	14			
Total Infarcts Noted	*28*			
Symmetry (asymptotic)		0.00	1	*1.0000*
Marginal homogeneity (Stuart-Maxwell)		0.00	1	*1.0000*

k. **Comparing Left & Right Parietal Lobe Infarct quantities in Males**

Parietal Lobe	Infarcts Present	Chi^2	df	P-value
Right Parietal	31			
Left Parietal	27			
Total Infarcts Noted	*58*			
Symmetry (asymptotic)		0.47	1	*0.4927*
Marginal homogeneity (Stuart-Maxwell)		0.47	1	*0.4927*

l. **Comparing Left & Right Parietal Lobe Infarct quantities in Females**

Parietal Lobe	Infarcts Present	Chi^2	df	P-value
Right Parietal	33			
Left Parietal	27			
Total Infarcts Noted	*60*			
Symmetry (asymptotic)		0.90	1	*0.3428*
Marginal homogeneity (Stuart-Maxwell)		0.90	1	*0.3428*

m. **Comparing Left & Right Middle cerebral artery Infarct Quantities**

MCA	Infarcts Present	Chi^2	df	P-value
Right MCA	39			
Left MCA	30			
Total Infarcts Noted	*69*			
Symmetry (asymptotic)		1.29	1	*0.2568*
Marginal homogeneity (Stuart-Maxwell)		1.29	1	*0.2568*

n. **Comparing Left & Right Lentiform nucleus Infarct Quantities**

Lentiform nucleus	Infarcts Present	Chi^2	df	P-value
Right Lentiform nucleus	7			
Left Lentiform nucleus	12			
Total Infarcts Noted	*19*			
Symmetry (asymptotic)		1.92	1	*0.1655*
Marginal homogeneity (Stuart-Maxwell)		1.92	1	*0.1655*

o. **Comparing Left & Right Posterior cerebral artery Infarct Quantities**

PCA	Infarcts Present	Chi^2	df	P-value
Right PCA	8			
Left PCA	6			
Total Infarcts Noted	*14*			
Symmetry (asymptotic)		0.50	1	*0.4795*
Marginal homogeneity (Stuart-Maxwell)		0.50	1	*0.4795*

p. **Comparing Left & Right Centrum semi ovale Infarct Quantities**

CSO	Infarcts Present	Chi^2	df	P-value
Right CSO	23			
Left CSO	19			
Total Infarcts Noted	*42*			
Symmetry (asymptotic)		0.80	1	*0.3711*
Marginal homogeneity (Stuart-Maxwell)		0.80	1	*0.3711*

q. **Comparing Left & Right Vertebral artery Infarct Quantities**

Vertebral a.	Infarcts Present	Chi^2	df	P-value
Right Vertebral artery	0			
Left Vertebral artery	2			
Total Infarcts Noted	*2*			
Symmetry (asymptotic)		2.00	1	*0.1573*
Marginal homogeneity (Stuart-Maxwell)		2.00	1	*0.1573*

r. **Comparing Left & Right Internal carotid artery Infarct Quantities**

ICA	Infarcts Present	Chi^2	df	P-value
Right ICA	8			
Left ICA	8			
Total Infarcts Noted	*16*			
Symmetry (asymptotic)		0.00	1	*1.0000*
Marginal homogeneity (Stuart-Maxwell)		0.00	1	*1.0000*

s. **Comparing Left & Right Caudate nucleus Infarct Quantities**

Caudate nucleus	Infarcts Present	Chi^2	df	P-value
Right Caudate nucleus	4			
Left Caudate nucleus	8			
Total Infarcts Noted	*12*			
Symmetry (asymptotic)		2.67	1	*0.1025*
Marginal homogeneity (Stuart-Maxwell)		2.67	1	*0.1025*

Appendix F: Circumference of infarcts according to age and sex

Circumference: 2 (L+B) x 2 (L+B)
C

Watershed infarcts	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	-	-	-	2	76.14	16.27
50–69	-	-	-	-	-	-
70 >	-	-	-	-	-	-
Totals	-	-	-	2	76.14	16.27

Multi Infarct Dementia	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	0	-	-	4	88.48	28.42
70 >	8	120.70	46.37	14	93.13	55.84
Totals	8	120.70	46.37	18	92.10	50.31

Left frontal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	72.30	-	5	80.03	45.56
50–69	10	84.59	48.02	6	126.81	67.32
70 >	7	92.67	56.40	10	120.35	53.20
Totals	18	87.05	48.71	21	112.59	56.37

Right frontal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	2	62.02	1.66	5	99.16	84.91
50–69	7	98.99	69.70	6	107.17	58.01
70 >	9	113.74	69.19	6	90.07	53.00
Totals	18	102.26	65.05	17	98.78	61.54

Right temporal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	3	156.43	59.68	6	106.47	20.01
50–69	1	73.96	-	5	272.55	208.98
70 >	2	139.62	27.32	4	107.33	35.11
Totals	6	137.08	50.97	15	162.06	139.37

Left temporal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	3	147.33	30.31	3	97.87	46.57
50–69	1	99.24	-	5	86.75	31.05
70 >	0	-	-	5	129.97	63.00
Totals	4	135.31	34.50	13	105.94	49.15

Right occipital lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	3	144.87	7.15	6	94.15	44.26
50–69	4	127.15	79.24	3	116.32	38.96
70 >	7	106.94	50.59	7	113.82	57.76
Totals	14	120.84	53.74	16	106.91	47.91

Left occipital lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	4	128.41	42.59	3	77.18	19.55
50–69	6	106.04	19.41	3	135.78	61.81
70 >	10	116.50	51.02	7	114.20	56.34
Totals	20	115.74	41.02	13	110.64	52.28

Right parietal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	3	77.67	35.02	7	92.06	40.44
50–69	10	143.03	41.26	11	131.14	72.30
70 >	12	150.01	100.10	13	96.18	55.49
Totals	25	138.54	76.62	31	107.65	60.15

Left parietal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	2	129.91	12.24	7	597.06	1360.34
50–69	12	107.04	41.89	10	101.56	28.05
70 >	9	161.91	120.78	8	114.86	76.55
Totals	23	130.50	83.02	25	244.56	717.64

Right mca	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	6	196	79.94	5	105.94	60.86
50–69	5	133.95	81.16	5	144.11	62.90
70 >	5	343.20	172.97	8	215.77	94.48
Totals	16	222.61	140.06	18	165.36	88.57

Left mca	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	3	263.32	137.50	0	-	-
50–69	4	159.57	72.79	7	112.52	80.99
70 >	8	219.30	128.05	6	142.49	78.42
Totals	15	212.17	115.82	13	126.35	78.00

Right lentiform nucleus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	97.59	-
50–69	2	105.89	33.95	0	-	-
70 >	0	-	-	3	63.68	41.01
Totals	2	105.89	33.95	4	72.15	37.53

Left lentiform nucleus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	95.79	-	0	-	-
50–69	4	95.77	38.99	2	55.53	0.46
70 >	3	76.20	14.40	1	90.86	-
Totals	8	88.44	28.52	3	67.31	20.40

Internal capsule	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	72.17	-	0	-	-
50–69	2	72.35	1.63	2	84.32	13.18
70 >	2	79.21	59.37	3	85.38	20.39
Totals	5	75.06	29.94	5	84.96	15.86

Right pca	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	81.71	-	2	114.01	83.10
50–69	0	-	-	2	75.36	-
70 >	1	102.91	-	2	65.10	0.84
Totals	2	92.31	15.00	6	84.83	43.74

Left pca	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	2	116.50	32.92
50–69	0	-	-	2	109.85	0.00
70 >	1	71.38	-	0	-	-
Totals	1	71.38	-	4	113.17	19.39

Right cso	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	2	41.52	11.20	4	27.67	3.27
50–69	4	42.16	20.63	5	46.48	19.86
70 >	1	59.82	-	4	44.43	7.37
Totals	7	44.50	16.72	13	40.06	14.91

Left cso	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	3	37.81	16.61
50–69	6	41.22	7.24	3	47.25	10.21
70 >	4	50.82	14.38	2	47.74	8.59
Totals	10	45.06	11.08	8	43.83	12.00

Subcortical white matter	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	47.56	-	1	29.16	-
50–69	3	69.64	25.88	0	-	-
70 >	1	65.44	-	1	64.56	-
Totals	5	64.39	20.66	2	46.86	25.04

Periventricular white matter	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	92.35	-	1	35.25	-
50–69	4	57.29	6.93	1	53.40	-
70 >	1	85.51	-	1	115.59	-
Totals	6	67.84	17.34	3	68.42	42.70

Left vertebral artery	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	70.45	-
50–69	0	-	-	0	-	-
70 >	0	-	-	1	68.27	-
Totals	0	-	-	2	69.36	1.54

Medulla oblongata	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	2	133.63	27.84	0	-	-
50–69	0	-	-	0	-	-
70 >	2	145.87	68.71	1	68.27	-
Totals	4	139.75	43.38	1	68.27	-

Pons	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	71.05	-	0	-	-
50–69	3	61.88	20.15	4	84.48	27.76
70 >	5	85.80	69.41	2	47.22	22.69
Totals	9	76.19	51.46	6	72.06	30.58

Lateral ventricle	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	116.41	-	2	159.10	27.50
50–69	1	107.95	-	3	75.47	21.23
70 >	5	114.74	27.92	5	132.51	63.79
Totals	7	114.01	22.96	10	120.72	55.50

Sylvian fissure	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	122.24	-	1	87.16	-
50–69	2	107.97	23.44	0	-	-
70 >	2	150.58	17.44	4	126.87	44.68
Totals	5	127.87	26.20	5	118.93	42.57

Right ica	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	2	214.02	128.23	2	116.59	3.63
70 >	2	137.94	38.04	2	206.14	165.08
Totals	4	175.98	88.84	4	161.36	108.45

Left ica	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	82.25	-	0	-	-
50–69	4	173.16	86.57	2	156.52	85.25
70 >	0	-	-	0	-	-
Totals	5	154.97	85.28	2	156.52	85.25

Cerebellum	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	154.11	-	0	-	-
50–69	4	148.83	76.03	0	-	-
70 >	2	84.47	36.65	6	133.39	33.36
Totals	7	131.20	64.32	6	133.39	33.36

Vermis	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	74.99	-
50–69	0	-	-	0	-	-
70 >	2	77.81	36.11	0	-	-
Totals	2	77.81	36.11	1	74.99	-

Brainstem	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	1	218.07	-	2	130.07	15.44
70 >	4	78.93	26.82	2	83.81	25.68
Totals	5	106.76	66.42	4	106.94	31.82

Globus pallidus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49		73.40	-	0	-	-
50–69	1	45.69	-	0	-	-
70 >	0	-	-	0	-	-
Totals	2	59.54	19.59	0	-	-

Midbrain	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	52.72	-
50–69	0	-	-	0	-	-
70 >	1	61.74	-	1	34.14	-
Totals	1	61.74	-	2	43.43	13.13

Cerebral peduncle	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	0	-	-	0	-	-
70 >	1	50.91	-	0	-	-
Totals	1	50.91	-	0	-	-

Basal nuclei	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	0	-	-	3	127.26	64.39
50–69	5	73.27	11.29	3	92.93	43.40
70 >	2	138.18	43.47	1	94.20	-
Totals	7	91.81	37.46	7	107.82	48.38

Corona radiations	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	1	152.87	-	1	75.03	-
50–69	0	-	-	1	68.55	-
70 >	3	63.46	33.82	1	127.51	-
Totals	4	85.81	52.55	3	90.36	32.33

Thalamus	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	0	-	-	1	151.12	-
50–69	2	74.64	19.83	1	155.84	-
70 >	2	109.57	55.82	0	-	-
Totals	4	92.11	39.70	2	153.48	3.33

Right aca	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	1	112.22	-	0	-	-
50–69	2	114.79	39.20	0	-	-
70 >	0	-	-	1	61.34	-
Totals	3	113.94	27.76	1	61.34	-

Left caudate nucleus	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	0	-	-	0	-	-
50–69	3	89.42	15.46	0	-	-
70 >	2	94.75	52.54	2	52.95	25.53
Totals	5	91.55	28.60	2	52.95	25.53

Precentral gyrus	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	0	-	-	0	-	-
50–69	1	38.59	-	0	-	-
70 >	0	-	-	0	-	-
Totals	1	38.59	-	0	-	-

Right caudate nucleus	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	0	-	-	1	47.11	-
50–69	2	86.73	28.73	0	-	-
70 >	0	-	-	1	73.20	-
Totals	2	86.73	28.73	2	60.15	18.45

Optic tract	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	0	-	-	0	-	-
70 >	1	31.74	-	0	-	-
Totals	1	31.74	-	0	-	-

External capsule	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49		122.93	-	0	-	-
50–69	1	50.79	-	0	-	-
70 >	0	-	-	1	42.51	-
Totals	2	86.86	51.01	1	42.51	-

Corpus callosum	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49		-	-	1	46.80	-
50–69	0	-	-	0	-	-
70 >	1	168.62	-	0	-	-
Totals	1	168.62	-	1	46.80	-

Putamen	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	79.23	-	0	-	-
50–69	1	79.53	-	0	-	-
70 >	1	58.10	-	0	-	-
Totals	3	72.29	12.28	0	-	-

Postcentral sulcus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	0	-	-	0	-	-
70 >	1	426.17	-	0	-	-
Totals	1	426.17	-	0	-	-

Appendix G: ANOVA Circumference one-way analysis

Male and female age groups were grouped together to get bigger sample sizes and were coded as follow:

MALES	FEMALES
18–39: 1	18–39: 6
40–59: 2	40–59: 7
60–69: 3	60–69: 8
70–79: 4	70–79: 9
> 80: 5	> 80: 10

The results were:

Hemispheric measurement	Abbreviation
Right hemisphere: Length	RL
Right hemisphere: Width	RW
Right hemsiphere: Circumference	RC
Left hemisphere: Length	LL
Left hemsiphere: Width	LW
Left hemsiphere: Circumference	LC

		Sum of squares	df	Mean squares	F	Signifi cance
RL	**Between groups**	3 726.596	9	414.066	0.702	0.707
	Within groups	158 675.174	269	589.871		
	Total	162 401.770	278			
RW	**Between groups**	1 328.055	9	147.562	1.074	0.382
	Within groups	36 954.769	269	137.378		
	Total	38 282.824	278			
RC	**Between groups**	31 191.011	9	3 465.668	0.594	0.802
	Within groups	1 569 446.795	269	5 834.375		
	Total	1 600 637.806	278			
LL	**Between groups**	7 176.187	9	797.354	1.150	0.328
	Within groups	171 241.146	247	693.284		
	Total	178 417.333	256			
LW	**Between groups**	2 284.510	9	253.834	1.866	0.058
	Within groups	33 593.655	247	136.007		
	Total	35 878.165	256			
LC	**Between groups**	78 363.175	9	8 707.019	1.413	0.183
	Within groups	1 522 472.883	247	6 163.858		
	Total	1 600 836.058	256			

Dependant variable	Code	Code	Mean difference	Standard error	Significance
RW	4	6	6.56	2.92	**0.026**
RW	5	6	8.17	3.89	**0.037**
RW	6	4	-6.56	2.92	**0.026**
RW	6	5	-8.17	3.89	**0.037**
LL	2	6	16.64	7.22	**0.022**
LL	4	6	15.88	7.32	**0.031**
LL	6	2	-16.64	7.22	**0.022**
LL	6	4	-15.88	7.32	**0.031**
LW	1	3	13.28	6.42	**0.040**
LW	1	6	15.54	6.45	**0.017**
LW	1	7	12.08	6.17	**0.051**
LW	2	3	6.60	3.13	**0.036**
LW	2	6	8.87	3.20	**0.006**
LW	2	7	5.41	2.61	**0.039**
LW	3	1	-13.28	6.42	**0.040**
LW	3	2	-6.60	3.13	**0.036**
LW	3	4	-6.54	3.18	**0.041**
LW	4	3	6.54	3.18	**0.041**
LW	4	6	8.80	3.24	**0.007**
LW	4	7	5.35	2.66	**0.046**
LW	6	1	-15.54	6.45	**0.017**
LW	6	2	-8.87	3.20	**0.006**
LW	6	4	-8.80	3.24	**0.007**
LW	6	9	-7.09	3.40	**0.038**
LW	7	1	-12.08	6.17	**0.051**
LW	7	2	-5.41	2.61	**0.039**
LW	7	4	-5.35	2.66	**0.046**
LW	9	6	7.09	3.40	**0.038**
LC	2	3	41.94	21.10	**0.048**
LC	2	6	54.20	21.53	**0.012**
LC	3	2	-41.94	21.10	**0.048**
LC	3	4	-41.18	21.41	**0.056**
LC	4	3	41.18	21.41	**0.056**
LC	4	6	53.43	21.83	**0.015**
LC	6	2	-54.19	21.52	**0.012**
LC	6	4	-53.43	21.83	**0.015**

Appendix H: Surface Area of infarcts according to age and sex

Surface Area: $\frac{2 (L+W)}{C} \times (L \times W)$

Watershed infarcts	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	2	569.35	370.00
50–69	0	-	-	0	-	-
70 >	0	-	-	0	-	-
Totals	0	-	-	2	569.35	370.00

Multi Infarct Dementia	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	0	-	-	4	516.38	285.20
70 >	8	914.34	561.57	14	534.56	647.06
Totals	8	914.34	561.57	18	530.52	578.44

Right frontal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	2	215.27	94.84	5	788.09	1083.03
50–69	7	769.36	1104.66	6	705.90	792.99
70 >	9	1033.42	1041.89	6	615.20	608.35
Totals	18	839.83	1004.90	17	698.06	781.37

Left frontal lobe	Males			Females		
AGE GROUPS	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	197.57	-	5	451.39	414.77
50–69	10	520.99	513.80	6	1068.72	889.81
70 >	7	629.32	680.51	10	898.10	738.41
Totals	18	545.15	559.98	21	840.83	729.99

Right temporal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	3	1333.29	932.57	6	796.51	288.92
50–69	1	246.39	-	5	2246.11	1042.57
70 >	2	1310.99	422.75	4	877.92	522.32
Totals	6	1144.71	759.87	15	1301.42	937.05

Left temporal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	3	1423.08	674.87	3	624.17	469.88
50–69	1	543.92	-	5	443.06	287.84
70 >	0	-	-	5	1055.91	920.00
Totals	4	1203.29	704.89	13	720.57	654.07

Right occipital lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	3	1235.39	185.96	6	590.06	641.96
50–69	4	1204.78	1103.27	3	800.36	507.94
70 >	7	732.22	580.55	7	857.86	621.88
Totals	14	975.06	710.92	16	746.65	585.34

Left occipital lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	4	1035.37	550.10	3	335.18	219.78
50–69	6	663.94	313.98	3	1128.98	860.98
70 >	10	907.20	820.19	7	762.44	603.08
Totals	20	859.85	642.00	13	748.43	626.47

Right parietal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	3	342.52	251.55	7	562.39	482.75
50–69	10	1151.74	584.90	11	1079.84	1151.92
70 >	12	1579.46	1876.60	13	691.18	590.47
Totals	25	1259.94	1381.59	31	800.01	821.83

Left parietal lobe	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	2	825.36	140.09	7	5858.44	14211.15
50–69	12	790.89	596.30	10	604.93	346.81
70 >	9	1430.95	1307.69	8	983.73	881.29
Totals	23	1044.34	949.24	25	2197.13	7497.85

Right mca	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	6	2269.63	1670.05	5	791.82	797.57
50–69	5	1143.57	1223.98	5	1504.81	996.72
70 >	5	4126.43	3197.78	8	2828.08	2333.56
Totals	16	2497.99	2360.58	18	1894.88	1854.22

Left mca	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	3	4507.48	3321.64	0	-	-
50–69	4	1692.20	1135.20	7	960.87	1288.54
70 >	8	3403.29	3137.32	6	1512.63	1468.47
Totals	15	3167.84	2795.01	13	1215.53	1345.60

Right lentiform nucleus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	438.25	-
50–69	2	581.14	373.86	0	-	-
70 >	0	-	-	3	237.00	276.81
Totals	2	581.14	373.86	4	287.31	247.40

Left lentiform nucleus	Males			Females		
Age groups	N	Mean	SD	n	Mean	SD
18–49	1	454.43	-	0	-	-
50–69	4	460.28	294.61	2	152.34	8.13
70 >	3	345.46	141.02	1	591.66	-
Totals	8	416.49	215.27	3	298.78	253.71

Internal capsule	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	1	252.92	-	0	-	-
50–69	2	276.14	56.62	2	390.25	311.84
70 >	2	376.25	430.01	3	278.18	131.65
Totals	5	311.54	224.96	5	323.01	191.69

Right pca	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	1	401.63	-	2	943.94	998.48
50–69	0	-	-	2	294.77	-
70 >	1	457.65	-	2	282.80	46.18
Totals	2	429.64	39.61	6	507.17	560.63

Left pca	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	0	-	-	2	710.69	343.00
50–69	0	-	-	2	653.76	-
70 >	1	264.90	-	0	-	-
Totals	1	264.90	-	4	682.22	200.74

Right cso	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	2	87.48	57.77	4	38.97	10.08
50–69	4	93.27	78.94	5	130.50	126.41
70 >	1	161.50	-	4	99.77	30.95
Totals	7	101.36	66.20	13	92.88	84.65

Left cso	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	0	-	-	3	84.92	82.17
50–69	6	81.32	32.81	3	110.32	57.17
70 >	4	131.18	62.93	2	124.91	47.85
Totals	10	101.27	50.80	8	104.44	59.06

Subcortical white matter	Males			Females		
Age groups	n	Mean	SD	n	Mean	SD
18–49	1	94.89	-	1	39.61	-
50–69	3	232.41	150.42	0	-	-
70 >	1	153.55	-	1	202.84	-
Totals	5	189.13	123.51	2	121.23	115.42

Periventricular white matter	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	369.11	-	1	51.93	-
50–69	4	183.26	47.44	1	118.86	-
70 >	1	334.79	-	1	554.06	-
Totals	6	239.49	95.17	3	241.62	272.64

Left vertebral artery	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	239.98	-
50–69	0	-	-	0	-	-
70 >	0	-	-	1	177.74	-
Totals	0	-	-	2	208.86	44.01

Medulla oblongata	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	2	1060.48	758.07	0	-	-
50–69	0	-	-	0	-	-
70 >	2	1490.00	1338.95	1	177.74	-
Totals	4	1275.24	922.31	1	177.74	-

Pons	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	230.73	-	0	-	-
50–69	3	191.48	104.47	4	370.88	232.49
70 >	5	673.23	1157.40	2	145.78	124.61
Totals	9	463.48	857.05	6	295.84	221.47

Lateral ventricle	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	756.32	-	2	1411.87	170.23
50–69	1	578.61	-	3	335.19	149.14
70 >	5	660.74	270.41	5	1218.24	1264.29
Totals	7	662.66	226.69	10	992.05	964.36

Lateral fissure	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	762.72	-	1	301.87	-
50–69	2	711.77	312.44	0	-	-
70 >	2	1082.85	378.67	4	1052.76	1060.50
Totals	5	870.39	313.53	5	902.58	977.89

Right ica	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	2	2360.56	1660.91	2	825.75	228.03
70 >	2	971.86	549.99	2	2921.64	3624.53
Totals	4	1666.21	1289.65	4	1873.70	2420.88

Left ica	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	374.98	-	0	-	-
50–69	4	1921.27	1304.26	2	2560.38	3539.70
70 >	0	-	-	0	-	-
Totals	5	1612.01	1324.40	2	2560.38	3539.70

Cerebellum	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	1557.35	-	0	-	-
50–69	4	1481.41	1460.68	0	-	-
70 >	2	413.42	306.43	6	1043.06	540.11
Totals	7	1187.12	1167.29	6	1043.06	540.11

Vermis	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	313.12	-
50–69	0	-	-	0	-	-
70 >	2	337.26	284.89	0	-	-
Totals	2	337.26	284.89	1	313.12	-

Brainstem	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	1	2240.31	-	2	1052.81	93.07
70 >	4	354.14	238.21	2	369.55	248.38
Totals	5	731.37	868.38	4	711.18	423.16

Globus pallidus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	282.28	-	0	-	-
50–69	1	134.24	-	0	-	-
70 >	0	-	-	0	-	-
Totals	2	208.26	104.67	0	-	-

Midbrain	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	130.53	-
50–69	0	-	-	0	-	-
70 >	1	231.86	-	1	50.31	-
Totals	1	231.86	-	2	90.42	56.73

Cerebral peduncle	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	0	-	-	0	-	-
70 >	1	124.46	-	0	-	-
Totals	1	124.46	-	0	-	-

Basal nuclei	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	3	964.10	873.06
50–69	5	275.63	75.20	3	465.57	388.90
70 >	2	1057.88	612.99	1	572.08	-
Totals	7	499.13	460.53	7	694.44	607.90

Corona radiations	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	1433.62	-	1	294.75	-
50–69	0	-	-	1	227.41	-
70 >	3	198.39	137.07	1	1038.99	-
Totals	4	507.20	627.67	3	520.38	450.38

Thalamus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	1190.51	-
50–69	2	261.31	171.89	1	1203.13	-
70 >	2	768.23	704.76	0	-	-
Totals	4	514.77	510.95	2	1196.82	8.92

Right aca	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	567.47	-	0	-	-
50–69	2	841.98	440.07	0	-	-
70 >	0	-	-	1	178.62	-
Totals	3	750.47	349.21	1	178.62	-

Left caudate nucleus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	3	399.28	160.04	0	-	-
70 >	2	526.34	414.57	2	158.84	125.16
Totals	5	450.11	246.20	2	158.84	125.16

Precentral gyrus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	1	61.85	-	0	-	-
70 >	0	-	-	0	-	-
Totals	1	61.85	-	0	-	-

Right caudate nucleus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	136.30	-
50–69	2	397.52	260.12	0	-	-
70 >	0	-	-	1	265.05	-
Totals	2	397.52	260.12	2	200.68	91.04

Optic tract	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	0	-	-	0	-	-
70 >	1	47.62	-	0	-	-
Totals	1	47.62	-	0	-	-

External capsule	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	851.28	-	0	-	-
50–69	1	138.18	-	0	-	-
70 >	0	-	-	1	80.01	-
Totals	2	494.73	504.24	1	80.01	-

Corpus callosum	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	1	101.93	-
50–69	0	-	-	0	-	-
70 >	1	965.51	-	0	-	-
Totals	1	965.51	-	1	101.93	-

Putamen	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	1	312.44	-	0	-	-
50–69	1	369.81	-	0	-	-
70 >	1	166.20	-	0	-	-
Totals	3	282.81	104.99	0	-	-

Postcentral sulcus	Males			Females		
Age groups	**n**	**Mean**	**SD**	**n**	**Mean**	**SD**
18–49	0	-	-	0	-	-
50–69	0	-	-	0	-	-
70 >	1	426.17	-	0	-	-
Totals	1	426.17	-	0	-	-