Introduction

More than 2 million cardiac catheterization procedures are performed annually in the United States. Rates of serious complications, including stroke, myocardial infarction, and death, are less than 1% for most catheterization procedures. However, because of high numbers of patients are performed, thousands of patients experience strokes after cardiac catheterization (SCCs) each year (Kopp, 2003).

Several lines of evidence suggest that silent ischemic strokes are embolic, either from dislodgement of a clot or athermatous debris off the aortic arch or from thrombus formation at the tip of a guide catheter (Hamon et al., 2006).

At present, diffusion-weighted (DW) magnetic resonance (MR) imaging is the most sensitive tool for early detection of cerebral infarction. DW MR imaging provides image contrast based on random translational motion of water molecules, which is substantially altered by acute cerebral ischemia (Omran et al., 2003).

The addition of this method to conventional brain MR imaging sequences makes it possible to detect very small and hyperacute infarction at almost any anatomic location within the brain hemispheres, the brainstem, and the cerebellum. DWMR imaging has been used to detect structural damage to the brain caused by silent embolism during cerebral and coronary angiography as well as during surgical and endovascular revascularization procedures in the carotid artery (Hamon et al., 2007).

The use of Transcranial Doppler (TCD) sonography allows the recording of microemboli entering the middle cerebral artery during various endovascular interventions, including cardiac catheterization (Leclercq et al., 2001).

During cardiac catheterization, cerebral microembolism that

being detected by TCD has frequently observed, but that's being clinically relevant remains unknown. However, recent studies have suggested that silent cerebral embolisms responsible for acute ischemic brain injury, as documented by DW-MRI could be related to some of these microemboli (Bladin et al., 1998).

Aim of the work

Detection silent and early cerebral ischemic insult during left ventriculography and diagnostic cardiac catheterization.

Stroke

Stroke is the rapid development of a focal neurologic deficit caused by a disruption of blood supply to the corresponding area of brain. Transient ischemic attack (TIA), by convention, is a focal neurologic deficit lasting less than 24 hours. Recent definitions of TIA describe focal symptoms that last less than 1 hour and do not reveal evidence of infarction (Albers et al., 2002).

The relevant concept is that TIA is a predictor of stroke (Johnston et al., 2000). The risk for stroke is greatest in the first 90 days after TIA (between 8%–10%), with practically half of those occurring within the first 7 days. Within 5 years, studies report that nearly 30% of people who had TIAs suffer a stroke (Rothwell and Warlow, 2005).

Classification of stroke

Strokes can either be ischemic (an occlusion of a blood vessel) or hemorrhagic (a rupture of a blood vessel). Hemorrhagic strokes include intracerebral hemorrhage (ICH, bleeding within the brain) and subarachnoid hemorrhage (SAH, bleeding between the inner and outer layers of tissue covering the brain within the subarachnoid space). Most strokes in the United States, approximately 87%, are ischemic (Rosamond et al., 2008).

Ischemic strokes have been further categorized into subtypes according to the mechanism of injury. These subtypes include large-artery atherosclerosis, cardiogenic embolism, small vessel occlusive disease, stroke of other determined cause, and stroke of undetermined cause. The majority, approximately 60%, of all new ischemic strokes are classified as large-artery atherosclerosis, cardioembolic, or small vessel diseases. ICH and SAH account for approximately 10% and 3% of all strokes, respectively (Rosamond et al., 2008).

About 36% to 69% of ICH is deep in location, 15% to 32% is

lobar, 7% to 11% is cerebellar, and 4% to 9% is in the brain stem (Flaherty et al., 2005).

Incidence, prevalence, and recurrence of stroke

In Egypt:

Khedr et al. (2013) carried a three phase trial to estimate epidemiology of stroke in Assiut and reported that, 65 participants were identified as positive on the survey questionnaire, but only 57 patients were found to have stroke, giving a crude prevalence rate of 963/100,000 inhabitants with an age-adjusted local prevalence rate of 699.2/100,000 and an age-adjusted prevalence relative to the standard world population of 980.9/100,000. The prevalence among males was higher than females (1174/100,000 vs. 736/100,000) with a ratio 1.7:1. There was a significantly higher prevalence of ischemic (895/100,000) than hemorrhagic (68/100,000) stroke. Stroke prevalence was the same in rural and urban areas and in males and females. There was, however, a significantly higher prevalence in illiterate (2413/100,000) than literate participants (357/100,000).

In a door to door survey to of major neurological disorders (project) in Al Quseir City, Red Sea Governorate, Egypt, Tallawy et al. (2013) reported a prevalence of stroke (6.2/1000 for those aged > 20 years).

Kandil et al. (1997) reported that, Prevalence rate of CVS in Upper Egypt was estimated to be 5.08/1000 and TIA was 0.36/1000, where the incidence of CVS was 1.8:1000 and TIA was 0.20000. Both prevalence and incidence were significantly higher in suburban and rural than urban communities. The prevalence of male/female was 1.05/1 while the incidence of male/female was l/0.85. Age specific prevalence and incidence rates showed rapid increase in thrombotic and hemorrhagic stroke as well as TIA after the age of 40 years, while the peak age for embolic stroke was from 20-40 years.

Worldwide

Stroke incidence ranges from 240 per 100,000 in Dijon, France (standardized to the European population aged 45–84 years), to about 600 per 100,000 in Novosibirsk, Russia (Donnan et al., 2008).

Data from the Framingham Heart Study (FHS) indicate that the age-adjusted incidence of clinical stroke and atherothrombotic brain infarction per 1000 person-years in 1950 to 1977, 1978 to 1989, and 1990 to 2004 was 7.6, 6.2, and 5.3 in men and 6.2, 5.8, and 5.1 in women, respectively. Clinical stroke in FHS excludes TIA, silent cerebral infarcts, or hemorrhage detected solely by imaging (Carandang et al., 2006).

The risk for recurrent ischemic stroke is 2% at 7 days, 4% at 30 days, 12% at 1 year, and 29% at 5 years after initial cerebral ischemia. The risk for recurrent stroke at 30 days on the basis of stroke subtype is 18.5% for large-artery cervical or intracranial atherosclerosis with stenosis, 5.3% for cardioembolism, 1.4% for lacunar infarction, and 3.3% for infarct of uncertain cause (Petty et al., 2000).

The risk for recurrence of cerebral hemorrhage is low, partly because of high 30-day mortality following the event. In 19 studies from 15 countries the crude cumulative recurrence rate of ICH varied from 0 to 24% (Hanger et al., 2007).

Case fatality and mortality

The overall mortality for stroke in the United States in 2004 was 50 per 100,000. About 70% to 80% of all stroke deaths are ischemic. Hemorrhagic strokes are less prevalent but more likely to be fatal. The proportion of hemorrhagic stroke deaths varies among race/ethnic group (Casper et al., 2003). Globally, the average 30-day case fatality following first ischemic stroke is about 22.9% with the exception of Japan (17%) and Italy (33%) (Feigin et al., 2003).

The 30-day case fatality among people 45 to 64 years of age in

the Atherosclerosis Risk in Communities study (ARIC) was 8% to 12% for ischemic stroke and 37% to 38% for hemorrhagic stroke. According to the Rochester Epidemiologic Project, the risk for death after first ischemic stroke was 7% at 7 days, 14% at 30 days, 27% at 1 year, and 53% at 5 years. The most common causes of death after ischemic stroke in the United States are cardiovascular events (22%), respiratory infection (21%), and initial stroke complications (14%). Long-term mortality following ischemic stroke varies according to stroke subtype. The 5-year mortality following each type of ischemic stroke in the Rochester Epidemiologic Project was: large-artery cervical or intracranial atherosclerosis with stenosis, 32.2%; cardioembolism, 80.4%; lacunar infarction, 35.1%; and infarct of uncertain cause, 48.6% (Petty et al., 2000).

Risk factors for ischemic stroke

Epidemiologic studies have established myriad stroke risk factors. Some of these are not modifiable, such as hereditary factors, but are pivotal in correctly identifying those at high risk. Factors relating to lifestyle and environment may typically be modified or controlled by proven strategies based on randomized clinical trials (Grysiewicz et al., 2008).

Non- modifiable risk factors

Age

For each consecutive decade after 55 years of age, the risk for stroke approximately doubles. Atherosclerosis increases with age, subsequently increasing the risk for ischemic stroke and myocardial infarction. The prevalence of stroke for individuals older than 80 years of age is approximately 27%, compared with 13% for individuals 60 to 79 years of age (Rosamond et al., 2008).

Race

The annual incidence of age-adjusted initial ischemic strokes per 100,000 in people 20 years of age or older was 88 in whites, 191 in blacks, and 149 in Hispanics. According to ARIC study data, the age-adjusted incidence of stroke per 100,000 populations in people 45 to 84 years of age is 360 in white males, 230 in white females, 660 in black males, and 490 in black females (White et al., 2005).

In 2004, the stroke death rate per 100,000 was 48.1 for white males, 74.9 for black males, 47.2 for white females, and 65.5 for black females (Rosamond et al., 2008).

Sex

In general, stroke is more prevalent in men than in women]. The incidence of stroke in the young (aged 35–44) is highest in women, however. The increased risk associated with pregnancy is most significant postpartum. Women accounted for 61% of stroke deaths in 2004, which is likely because of their greater longevity than men (Goldstein et al., 2006).

Family history

Parental history of stroke, TIA, or myocardial infarction is associated with 1.4 to 3.3 fold increased risk for stroke [29]. The increased prevalence of stroke between monozygotic and dizygotic twins is almost fivefold. Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), a rare genetic disorder, has been reported to cause recurrent strokes with typical onset between the ages of 30 to 50 years (Kalimo et al., 1999).

Modifiable risk factors

Hypertension

There is a well-established relationship between blood pressure

and the risk for developing stroke. The relationship is continuous, consistent, and independent of other risk factors. Data from observational studies indicate that risk for death from both ischemic heart disease and stroke increases steadily beginning at systolic blood pressure as low as 115 mm Hg. The mortality of heart disease and stroke double with each increment of 20 mm Hg systolic blood pressure. This risk is increased because hypertension accelerates the development of atherosclerosis, ultimately leading to an increased number of atherothrombotic events (Sacco et al., 2006).

Longitudinal studies indicate that individuals who have high-normal blood pressure (130–139 mm Hg systolic, 85–89 mm Hg diastolic, or both) have a twofold increased risk for developing heart disease and stroke than those who have blood pressure less than 120/80 mm Hg (Vasan et al., 2001).

Diabetes

Individuals who have known diabetes and elevated glucose are at increased risk for thromboembolic stroke independent of other cardiovascular risk factors. Several epidemiologic studies have indicated an independent association between diabetes and ischemic stroke with a twofold to six-fold increased risk (Goldstein et al., 2006).

It is estimated that nearly 40% of all ischemic strokes can be attributed to the effects of diabetes either alone or in combination with hypertension (Kissela et al., 2005).

This risk may be due to both the accelerated development of atherosclerosis over time and to the increased prevalence of other risk factors, including central obesity, elevated cholesterol, and hypertension associated with diabetes (Goldstein et al., 2006).

A longitudinal study of 13,999 people who had coronary heart disease suggests impaired glucose tolerance, defined as glucose levels between 140 and 199 mg/dL after a 2-hour glucose tolerance test, is associated with increased stroke risk in patients who have heart

disease (Tanne et al., 2004).

Another clinical study has identified impaired glucose tolerance as an independent risk factor for recurrent stroke in patients who have TIA or minor ischemic stroke (Vermeer et al., 2006).

Smoking

Smoking has been identified as an independent risk factor for stroke in a plethora of studies over the years. The relative risk for stroke ascribed to cigarette smoking is 1.5. Relative risk varies among stroke subtypes with ischemic stroke having a relative risk of 1.9. Smokers younger than 55 years of age have a relative risk of 2.9, which is considerably higher than smokers older than 55 years; relative risk for smokers aged 55 to 74 years is 1.8, and relative risk is 1.1 for smokers older than 70 years. Ex-smokers continue to have an increased risk for stroke despite cessation. Exposure to environmental tobacco smoke also increases the risk for stroke, with some studies reporting a nearly twofold increment of risk (Bonita et al., 1999).

Dyslipidemia

Elevated serum cholesterol and stroke is not a well-established risk factor for stroke. Previous studies were confounded by the inverse association of total cholesterol and cerebral hemorrhage, but more recent trials are specific to ischemic stroke. The Asia Pacific Cohort Studies Collaboration suggests a 25% increased risk for ischemic stroke with each 1-mmol/L (38.7-mg/dL) increase in total cholesterol (Zhang et al., 2003).

The Women's Pooling Project of 24,343 women reported a 25% increase in fatal stroke with each 1-mmol/L increase in total cholesterol in women aged 30 to 54 years (Horenstein et al., 2002).

The 10-year follow-up of the Copenhagen Stoke Study revealed than an increase of 1 mmol/L in total serum cholesterol resulted in an increase in the Scandinavian Stroke Scale score. Specifically, high

cholesterol levels were primarily associated with minor strokes and lower mortality (Olsen et al., 2007).

Cardiac disease

Multiple cardiac conditions, in addition to atrial fibrillation, are associated with increased stroke risk. There is a considerable risk for ischemic stroke in the first 5 years after a myocardial infarction, which is estimated to be 8.1% over 5 years. The increased risk is related to the extent of left ventricular dysfunction with an 18% increase of stroke risk with every 5% decrease of ejection fraction. Also, there is a reported risk for cardioembolic stroke with valvular disease, left ventricular thrombi, and congenital defects, such as patent foramen ovale (PFO) and atrial septal aneurysm (ASA). PFO can develop when this small hole in the atrial septum fails to close properly at birth, and leads to a right-to-left shunt. ASA is characterized by excessive atrial septal motion. The causal relationship between PFO/ASA and stroke is highly debated. The most commonly accepted pathophysiology is paradoxical embolism, but other possibilities may include an increased association with thrombus formation or atrial arrhythmias (Messe et al., 2004).

Despite these presumed mechanisms, some studies have failed to show an association between increased risk for initial stroke in individuals who have PFO or PFO in combination with ASA (Di Tullio et al., 2007).

Sickle cell disease

Sickle cell disease (SCD) is an autosomal recessive disorder that causes an alteration in the hemoglobin b chain. The red blood cells have decreased oxygen carrying capacity and have a tendency to adhere to blood vessel walls (Goldstein et al., 2006).

By 20 years of age, 11% of children who have homozygous SCD suffer a stroke, with greatest risk in early childhood. More than 22% of children who have homozygous SCD have evidence of stroke, either

clinical or subclinical, on MRI. Children who have high cerebral blood flow velocities determined by transcranial Doppler ultrasound have approximately a 10% risk for stroke per year, which is significantly reduced by up to 90% with frequent blood transfusions (Adams et al., 2004).

Diet

Studies have shown that there is a protective relationship between fruit and vegetable consumption and ischemic stroke risk. This relationship is especially evident with consumption of cruciferous vegetables, green leafy vegetables, and citrus fruit. There is a 6% reduction in risk for ischemic stroke with each one-serving increase of fruits and vegetables daily (Sauvaget et al., 2003).

In overweight individuals, higher sodium intake is associated with approximately 89% increased risk for stroke mortality. Analyses also reveal that a 10 mmol increase in daily potassium is associated with 40% reduction in stroke mortality independent of other cardiovascular risk factors. Potassium supplementation has shown a decrease in mean systolic and diastolic blood pressures. The association between alterations in sodium and potassium intake and the reduction in stroke mortality is hypothesized to be achieved primarily by blood pressure lowering (Nagata et al., 2004).

Physical inactivity

Moderate to high levels of physical activity have proved protective against stroke in middle-aged men. The relative risk for stroke is 1.82 in women aged 65 to 74 years who have low physical activity. Increased leisure time physical activity was protective against stroke across race, sex, and age. This decreased risk was related to level of intensity and duration (Sacco et al., 1998).

Obesity

Obesity is a risk factor for ischemic stroke in women and men.

After adjustment for cardiovascular risk factors, women who had a body mass index (BMI) greater than 27 had significantly increased risk for ischemic stroke. Relative risk for BMI 27 to 28.9 was 1.75, for BMI 29 to 31.9 was 1.9, and for BMI greater than 32 was 2.37. Also, the risk for stroke was associated with the amount of weight gained after 18 years of age with relative risk of 1.69 for a gain of 11 to 19.9 kg and relative risk of 2.52 for a gain of 20 kg or greater (<001). Men who have BMI greater than 30 have a 1.95 relative risk for ischemic stroke, and with each unit increase in BMI there is a 6% increase in adjusted relative risk (Kurth et al., 2002).

In addition to the relationship between BMI and stroke risk, studies have examined the relationship between abdominal adiposity and stroke risk. Abdominal adiposity may be defined as the highest quartile of waist circumference or hip/waist ratio, with obesity defined as greater than 102 cm in men and greater than 88 cm in women. Abdominal obesity is only found to be a risk factor for ischemic stroke in men (Hu et al., 2007).

Alcohol use

Studies have shown an increased risk for stroke with ‘‘irregular’’ drinking, including heavy and binge drinking. Acute consumption of an intoxicating amount of alcohol is suggested to be an independent risk for stroke with a relative risk of 1.82. Consumption of 151 g to greater than 300 g of alcohol is significantly associated with increased risk for cardioembolic and cryptogenic stroke. Drinking more than 40 g of alcohol may trigger a cardiogenic embolism in those who have a high-risk source. Moderate drinking (more than one drink in the past month to two drinks per day) is associated with decreased risk for ischemic stroke compared with individuals who had no drinks in the past year after adjusting for other risk factors (Elkind et al., 2006).

Hormone replacement therapy

Hormone replacement therapy was previously hypothesized to reduce stroke risk, but recent clinical trials have failed to show benefit

of postmenopausal hormone replacement therapy in reduction of stroke or severity. In a clinical trial, women who received estrogen replacement therapy had a 2.9 relative risk for fatal strokes, and the nonfatal strokes that occurred were associated with increased functional deficits (Viscoli et al., 2001).

Hyperhomocysteinemia

Elevated serum total homocysteine level (>12.1 mmol/L) is independently associated with risk for nonfatal stroke. The Homocysteine Studies Collaboration calculated that homocysteine levels reduced by 25% (about 3 mmol/L) are associated with a 19% reduction in stroke risk. Higher homocysteine levels are found with increasing age. Men, especially at younger ages, have higher homocysteine concentrations than women (Homocysteine Studies Collaboration, 2002).

Hypercoagulability

Inherited thrombophilias, specifically factor V Leiden mutation, are associated with increased risk for venous thrombosis. Inherited thrombophilias, including protein C deficiency, protein S deficiency, antithrombin III deficiency, factor V Leiden mutation, and prothrombin 20,210A mutation, have not been associated with increased risk for ischemic stroke on a consistent basis, however. The presence of an antiphospholipid antibody, which includes anticardiolipin and lupus anticoagulant antibodies, is typically an acquired thrombophilia, but can also be inherited (Juul et al., 2002).

The Antiphospholipid Antibodies and Stroke Study (APASS) did not show an association between antiphospholipid antibodies and recurrent ischemic strokes (Levine et al., 2004).

Inflammation

Atherosclerosis is a chronic inflammatory process that is initiated by endothelial dysfunction. C-reactive protein (CRP) is an acute phase

reactant that serves as a marker of systemic inflammation (Folsom et al., 2002) [98]. Studies show that CRP is a strong predictor of cardiovascular events (Hage and Szalai, 2007).

High-sensitivity C-reactive protein (Hs-CRP) levels have been shown to increase with severity of stroke (Elkind et al., 2006).

The CD40/CD40 ligand (CD40L) dyad is a powerful immune mediator and an inflammatory marker in the serum. CD40L is implicated in multiple stages of atherogenesis. Studies have shown that interruption of CD40 signaling is associated with plaque stabilization (Schonbeck and Libby, 2001).

Infection

Infection has long been implicated as a cause of atherosclerosis. Studies have shown an association between acute bacterial or viral infections and increased risk for ischemic stroke, especially among infections occurring within the week before the stroke. There is an association between chronic infections, specifically bronchitis and periodontal disease, and increased ischemic stroke risk. Infectious mechanisms for stroke pathogenesis remains unclear, but proposed mechanisms include increased cytokine expression and procoagulant effects (Lindsberg and Grau, 2003).

Pathophysiology of ischemic stroke

When a cerebral artery is occluded, a core of brain tissue dies rapidly. Surrounding this infarct core is an area of brain that is hypoperfused, but still viable because of collateral blood flow. This area of at-risk, but potentially salvageable, tissue is called the ischemic penumbra (Leiva-Salinas and Wintermark, 2010).

Studies in primates and positron emission tomography studies in humans have shown that brain parenchyma can compensate for