

Cardiac Pathology and Brain Changes

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Received Date: August 03, 2017; Accepted Date: October 04, 2017; Published Date: October 20, 2017

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Citation: Perna R (2017) Cardiac Pathology and Brain Changes. J Heart Cardiovasc Res. Vol. 1 No.3: e102

Editorial

Because the brains continuous need for oxygen, cardiac output and functioning is central to optimal brain functioning and neurological health. Heart functioning and brain functioning are highly intertwined with cardiac output and brain perfusion highly coupled. Common cardiac conditions that are typically very manageable, such as hypertension and cardiac dysrhythmia often places a person at risk for brain dysfunction and injury. Hypertension is thought to double the risk for stroke and atrial fibrillation may quadruple the risk of stroke. Hypertension is also one of the significant causes of white matter lesions in the brain due to changes in cerebral autoregulation and subsequent small vessel damage. The white matter lesions are very common after the age of 55 in both normotensive and hypertensive individuals with an incidence of 25% and 50%, respectively [1]. The white matter lesions are usually silent until a critical mass is reached. As a result, someone could be headed toward subsequent progressive decline without any awareness of this risk. Atrial fibrillations not only affect cardiac efficiency as a pump, but the coagulation potential is significant in that 15 to 20% of strokes in the US are thought to be secondary to it. Many stroke patients when asked will say that they had not known they had atrial fibrillation before they had their embolic stroke. Additionally, some research suggests that most "cryptogenic" strokes have an embolic and possibly cardioaortic origin related to PFOs and other anomalies. A significant portion of people with hypertension and/or atrial fibrillation will go on to suffer from lacunar infarcts and also possibly small artery disease. Some research suggests that up to 18% of lacunar

infarcts may be from cardioembolisms [2]. Small subcortical infarcts are often considered to be synonymous with lacunar infarction caused by small artery disease. Though there is some published literature on typical clinical presentations for lacunar infarcts, people with small artery disease and lacunar infarcts may not experience an abrupt decline in functioning and may actually have multiple cumulative infarcts and perhaps an insidious cognitive decline before anyone suspects any neurological issues. Reported rates of hypertension are very high in patients with lacunar infarcts, but vary widely, ranging from less than 50% to 97%. The though is that diabetes and some other powerful variables also explain a significant amount of risk for lacunar infarcts. Thus, hypertension and diabetes appear equally common in lacunar and non-lacunar ischemic stroke, but lacunar stroke is less likely to be caused by embolism from the heart or proximal arteries.

Based on several bodies of evidence it appears apparent that a subgroup of individuals with certain cardiac issues should be screened or tracked to reduce the risks of both stroke and cumulative insidious decline related to cerebrovascular pathology that is related to cardiac issues.

References

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