

# Article review on critical analysis

[Health & Medicine](#), [Disease](#)



## **Part 5: Final Draft**

This paper has several sections. First, it gives an analysis of a psychological report which was published in popular media. The second part documents the original research cited in the psychological report in section one. The third step gives a comparison between the first and second report.

### **Analysis of a Psychological Report**

The title of the report was Silent Cerebral Emboli May Hasten the Progression of Dementia (Journal Watch, 2012). The author notes that the findings of the report were drawn from two groups: patients with dementia (loss of cognitive ability particularly, memory, attention, and language) and patients with Alzheimer disease. The author begins by notifying readers that vascular changes (changes in the flow of blood) in the brain are a common phenomenon in Alzheimer patients. The author cites a UK study in which 60 subjects of dementia, as well as 84 subjects of Alzheimer were investigated to elucidate the effect of cerebral emboli (blockage in the blood veins of the brain) on their cognitive functions (Journal Watch, 2012). The author of the report indicates that a technique known as transcranial doppler ultrasonography (technique used to evaluate vascular changes) was employed in the said UK study. Procedures involving this technique were conducted for 18 months at intervals of 6 months in an endeavor to collect data from the participants under investigation. Additionally, the author indicates that changes in behavior and cognition in the subjects under study were also evaluated for a period of two years.

According to the author, 43 percent of participants with Alzheimer disease

were found to have spontaneous cerebral emboli (cerebral emboli that is uncontrollable) (as cited in Purandare N. et al., 2012). On the other hand, only 45 percent of their counterparts with dementia had the same result: spontaneous cerebral emboli (as cited in Purandare N. et al., 2012).

According to the study findings, the author notifies the readers that patients with cerebral emboli registered a notable deterioration in their cognitive functions when compared to their counterparts without cerebral emboli.

Furthermore, the level of deterioration was almost similar in participants with both vascular dementia and Alzheimer disease. The author gives quantitative statistics of the UK study's findings. The author indicates that on a scale of 30 points, with respect to mental stability, dementia subjects with cerebral emboli registered 6.9 points while 3.4 points were registered by their counterparts without cerebral emboli (as cited in Purandare N. et al., 2012).

On the other hand, a 70-points scale assessing the level of Alzheimer disease was used to evaluate the progression of the Alzheimer disease in subjects with and without cerebral emboli (as cited in Purandare N. et al., 2012). The author notifies the reader that subjects with both cerebral emboli and Alzheimer disease recorded 15.4 points (as cited in Purandare N. et al., 2012). Their counterparts without cerebral emboli registered 6.0 points (as cited in Purandare N. et al., 2012).

However, the researchers of the UK study leave two questions unanswered. First, they do not highlight the mechanism through which cerebral emboli leads to the progression of dementia and consequently Alzheimer disease. From the Journal Watch report, the reader is unaware of the mechanism through which cerebral emboli contributes to the progression of Alzheimer

disease. The reader only knows that the progression of this disease is greater in patients with cerebral; emboli than those without a vascular disease. Therefore, the original article can be essential in explaining this link. The original article/study will be essential in substantiating how cerebral emboli contributes to loss of memory, language control and attention, which are notable symptoms of Alzheimer disease. Secondly, the author does not highlight the source of cerebral emboli in the subjects under investigation. The primary research will also be of essential help in elucidating the sources of cerebral emboli. In order to understand the findings reported by the author of the Journal Watch article, an initiative to find the primary research that was cited by the author was taken.

In the beginning of Journal Watch report, the author indicated that the findings were drawn from a UK study. The author then gave full bibliographic information of the study at the end of the report. The author noted that the UK study was conducted by Purandare N. et al. (2012); the article was published in the American Journal of Psychiatry on March 1st 2012. The volume and issue number of the study were also provided (volume 169, no. 3). Upon conducting several web searches, the cited journal article was identified. The information given in the Journal Watch report and the original article were compared and they were found to have 100 percent match. For instance, the author us of the article (Purandare N. et al. (2012); the article was published in the American Journal of Psychiatry on March 1st 2012; volume and issue number of the study were volume 169 and no. 3 respectively).

## **Analysis of the Original Research/Article**

In the article authored by Nathan Purandare et al (2012), the authors evaluated the impact of cerebral emboli on the cognitive function in not only patients with Alzheimer's disease, but also those with dementia. In the examination of Alzheimer's disease, it is extremely helpful to find out how cognitive functions are affected by various factors such as embolism.

Purandare et al (2012), counted cerebral emboli present in the middle brain arteries of the participants. The measurements were taken at baseline and after every six months; the whole study took 18 months. Collectively, the participants were 144 in number (vascular dementia: n= 60; Alzheimer dementia n= 80; dementia patients n= 4). Participants were drawn from communal and outpatient geriatric clinics in Manchester, UK. The researchers did not highlight the age or gender profile of the participants. On the other hand, deterioration in cognition (dementia levels) was measured for two years at an interval of six months. Thereafter, Purandare et al (2012) analyzed the relationship between the deterioration of cognitive function and occurrence of cerebral emboli.

The results posted by Purandare et al (2012) showed that the levels of spontaneous dementia in dementia, Alzheimer's disease, vascular dementia patients were as follows 44 per cent, 43 percent, and 23 percent respectively. These quantitative statistics substantiated the conclusion results posted by Purandare et al (2012). Patients who were found to have cerebral emboli registered a notable decrease in their cognitive function over the two year period when the study was conducted. The confirmation of a reduction in cognitive function (increase in dementia levels) was conducted

using two analytical techniques: neuropsychiatry inventory scores and daily interviews to check for dementia in the participants. Both techniques revealed that participants had increased dementia levels (a significant reduction in their cognitive function).

The study proved that spontaneous emboli have a significant influence on the cognitive function of both dementia patients and patients with Alzheimer's disease (Purandare et al, 2012). There is a notable decrease in cognitive function (heightened dementia levels) in these patients as a result of cerebral emboli (Purandare et al, 2012).

This study had significant findings. The study findings shade more light on the relationship between cerebral emboli, and cognitive function and this is exceptionally essential in the management of patients affected with this disorder. The other significant aspect of this study is that the researchers discussed at length the possible sources of cerebral emboli. In their discussion, Purandare et al (2012) noted that cerebral arteries, aorta, and carotid arteries are major sources of cerebral emboli. In addition, the researchers highlighted that cerebral emboli may stem from the heart or the venous circulation of individuals with a patent foramen ovale (Purandare et al., 2012). Furthermore, the scientists noted that the presence of atherosclerosis of the carotid arteries heightens the development and progression of dementia (Purandare et al, 2012). Moreover, inflammation resulting from cardiopulmonary bypass or a major surgery may trigger the development of an emboli and subsequent deterioration in cognitive functions (Purandare et al., 2012).

In their study, Purandare et al (2012) endeavored to prove whether cerebral

emboli cause a decrease in the cognitive function in patients with dementia and Alzheimer's disease. The researchers managed to prove their hypothesis; spontaneous cerebral emboli lead to deterioration in the cognitive functions of patients with dementia and Alzheimer's disease. As indicated earlier, the researchers have provided quantitative statistics to support their conclusion (the levels of spontaneous dementia in dementia, Alzheimer's disease, vascular dementia patients were as follows 44 per cent, 43 percent, and 23 percent respectively). The researchers employed a correlational study technique in the evaluation of their participants.

The study conducted by Purandare et al (2012) was well organized. First, the researchers recruited patients from geriatric clinics after the doctors' confirmation that the patients actually had either dementia or Alzheimer's disease, or both. Thereafter, they monitored the cognitive function and cerebral emboli levels of the participants for two years. They then analyzed the relationship between cognitive function and levels of cerebral emboli.

This approach formed the basis of their study; these formed the strengths of the study. However, the development of dementia is a multifaceted affair, and it is important to have a large sample size in order to make general conclusions. There could be a potential bias during the measurement of emboli in the participants' brain region. As such, some patients may have been wrongly found to have a cerebral embolus. In future, scientists should have advanced methods of detecting and measuring cerebral embolism. In addition, large samples should be used in futures studies.

## **Comparison of the two reports**

The first article was a psychological report from Journal Watch. The psychological report was on cerebral emboli and the progression of dementia is analyzed; the report was retrieved from Journal Watch, and it was published on 5 April 2012. The report's findings were drawn from a study conducted by Purandare et al (2012).

In the Journal Watch report, the author notifies the readers that the findings of the report were drawn from two groups: patients with dementia (loss of cognitive ability particularly, memory, attention, and language) and patients with Alzheimer disease. The author also notifies the readers that vascular changes (changes in the flow of blood) in the brain are a common phenomenon in Alzheimer patients. The author cites Purandare et al.'s study and he indicates that 60 subjects of dementia, as well as 84 subjects of Alzheimer were investigated to elucidate the effect of cerebral emboli. The author of the report further indicates that a technique known as transcranial doppler ultrasonography (technique used to evaluate vascular changes) was employed in the said UK study. Procedures involving this technique were conducted for 18 months at intervals of 6 months in an endeavor to collect data from the participants under investigation. Additionally, the author indicates that changes in behavior and cognition in the subjects under study were also evaluated for a period of two years. The author of the Journal Watch report indicated that results from the UK study indicate that vascular disease (cerebral emboli) plays a significant role in the progression of Alzheimer disease (Journal Watch, 2012).

In the original article, the authors evaluated the impact of cerebral emboli on



the cognitive function in not only patients with Alzheimer's disease, but also those with dementia. In the examination of Alzheimer's disease, it is extremely helpful to find out how cognitive functions are affected by various factors such as embolism.

When the two articles are compared, several similarities can be noted. Both articles documented the number of subjects who took part in the study, and how the study was conducted. In the original article, the authors report that the measurements were taken at baseline and after every six months; the whole study took 18 months. Collectively, the participants were 144 in number (vascular dementia:  $n= 60$ ; Alzheimer dementia  $n= 80$ ; dementia patients  $n= 4$ ). Participants were drawn from communal and outpatient geriatric clinics in Manchester, UK. In the Journal Watch report, the author indicated that 60 subjects of dementia, as well as 84 subjects of Alzheimer were investigated to elucidate the effect of cerebral emboli. Secondly, they give quantitative statistics which substantiate their conclusions. In the first report, the author reported that 43 percent of participants with Alzheimer disease were found to have spontaneous cerebral emboli (cerebral emboli that is uncontrollable) (as cited in Purandare N. et al., 2012). On the other hand, only 45 percent of their counterparts with dementia had the same result: spontaneous cerebral emboli (as cited in Purandare N. et al., 2012). In the original report/article, the results posted by Purandare et al (2012) showed that the levels of spontaneous dementia in dementia, Alzheimer's disease, vascular dementia patients were as follows 44 per cent, 43 percent, and 23 percent respectively. According to the study findings, the author notifies the readers that patients with cerebral emboli registered a notable

deterioration in their cognitive functions when compared to their counterparts without cerebral emboli. However, the original article is more detailed than the report published in the Journal Watch report. For instance, the authors give the origins of cerebral emboli, which were not mentioned in the Journal Watch report. In their discussion, Purandare et al (2012) noted that cerebral arteries, aorta, and carotid arteries are major sources of cerebral emboli. In addition, the researchers highlighted that cerebral emboli may stem from the heart or the venous circulation of individuals with a patent foramen ovale (Purandare et al., 2012). Furthermore, the scientists noted that the presence of atherosclerosis of the carotid arteries heightens the development and progression of dementia (Purandare et al, 2012). Moreover, inflammation resulting from cardiopulmonary bypass or a major surgery may trigger the development of an emboli and subsequent deterioration in cognitive functions (Purandare et al., 2012). On top of that, the second article (the original article) highlights the limitations of the study. They indicate that future studies should use a larger sample so as to enhance the generalization of the findings. They should also use advanced techniques to measure vascular changes in the subjects. Both reports highlight the same conclusion: spontaneous cerebral emboli lead to deterioration in the cognitive functions of patients with dementia and Alzheimer's disease.

The report from Journal Watch (1st report), documents to the reader what was reported by Purandare et al. The author manages to highlight key aspects of the study such as the sample size, research process, findings and conclusions. The second report (the original article) gives a detailed account

of the study. The article reports how the subjects were recruited, sample size, research design and process, research findings, and limitations of the study.

## **References**

Purandare, N. et al. (2012). Association of Cerebral Emboli with Accelerated Cognitive Function Deterioration In Alzheimer’s Disease and Vascular Dementia. *The American Journal of Psychiatry*, 169 (3), 300-308. Retrieved on 20 June 2013 from <http://ajp.psychiatryonline.org/article.aspx?articleid=1028559>