lesions and 10% had left hemispheric lesions. This finding confirms Gainotti's view that denial of depressed mood is common in right-sided lesions. On further analysis of the data, it was found that only 6 among 21 depressed patients with right hemispheric lesions present with denial of depressed mood. Although the reasons for rarity of this symptom remain largely undetermined, this could be explained in the context of intrahemispheric lesions and its correlations with depression and denial of illness in right-sided lesions. The literature suggests that right posterior lesions are correlated with depression, whereas right frontal lesions are correlated with a reaction of indifference that has been associated with denial of illness. Based on this evidence, one may assume that denial of illness is less common among depressed patients with right hemispheric lesions, as the sites of lesions producing depression and those producing denial of illness vary significantly.

The second issue is related to anosognosia and its association with poststroke depression. Psychological theories indicate that anosognosia protects the organism from realizing the full extent of deficits and thereby prevents depression. Based on psychological theory, Gainotti suggested that depression and anosognosia may not coexist. However, evidence against the psychological hypothesis of anosognosia came from a study that demonstrated that depression was equally frequent among patients with and without anosognosia. Hence, studying the phenomenon of anosognosia in poststroke depression highlighted many facts that include anosognosia or denial of illness is not only concerned to physical disability but also to depression. Second, the reactive theory of poststroke depression has been challenged with the finding that anosognosia and depression coexist, which suggests that denial of physical disability does not protect patients from developing poststroke depression. Third, denial of depression may be associated with right anterior frontal lesions, whereas poststroke depression with depressed mood may be associated with right posterior lesions. Fourth, identification of depression in the absence of depressed mood may be a difficult task in clinical practice. However, the presence of cognitive and vegetative symptoms that fulfill the criteria for depression is sufficient to make the diagnosis of depression even in the absence of depressed mood.

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References

Response
We appreciate the chance to respond to the comment of Dr Ramasubbu that we did not discuss poststroke depression and denial of illness. As they rightly point out, our article focused on physical disability, with particular emphasis on denial of hemiplegia. The literature concerning the investigation of mood disorders after stroke is extensive and, indeed, in itself deserving of a comprehensive review. We therefore welcome a letter that highlights this aspect of denial.

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Leukocytes in Acute Stroke
Wang and coworkers have described a useful method for imaging leukocyte infiltration in acute ischemic stroke. In reviewing the literature concerning the role of leukocytes in acute stroke, they failed to mention studies linking leukocytosis with reduced survival after stroke. Among 283 subjects with acute stroke, elevated leukocyte count proved to be a significant univariate predictor of short- and long-term mortality. Among 492 elderly subjects with acute stroke followed up for a mean of 18 months, elevated leukocyte count was a strong univariate predictor of mortality; it remained a significant predictor of mortality in multivariate models containing initial level of consciousness, age, serum creatinine, and Mini-Mental State score. Further studies are required to determine the role of leukocytes in acute stroke.

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References

Response
Dr Friedman's study showed that an elevated leukocyte count was the third-best univariate predictor of mortality and the fifth-best multivariate predictor of mortality in acute stroke patients. Our study focused on the dynamics of leukocyte infiltration in acute stroke. A previous study with the same methodology did show poor prognosis with high leukocyte infiltration in acute stroke patients. The postulated effects of leukocytes to the pathogenesis of cerebral stroke include (1) limitation of cerebral blood flow by vessel plugging or vasoconstrictive mediator release; (2) exacerbation of blood-brain barrier or parenchymal injury through hydrolytic enzyme release, lipid mediator production, or oxygen radical production; and (3) initiation of thrombosis.

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