Increased Prevalence of Posttraumatic Stress Disorder in Patients After Transient Ischemic Attack

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Background and Purpose—A transient ischemic attack (TIA) involves temporary neurological symptoms but leaves a patient symptom-free. Patients are faced with an increased risk for future stroke, and the manifestation of the TIA itself might be experienced as traumatizing. We aimed to investigate the prevalence of posttraumatic stress disorder (PTSD) after TIA and its relation to patients’ psychosocial outcome.

Methods—Patients with TIA were prospectively studied, and 3 months after the diagnosis, PTSD, anxiety, depression, quality of life, coping strategies, and medical knowledge were assessed via self-rating instruments.

Results—Of 211 patients with TIA, data of 108 patients were complete and only those are reported. Thirty-two (29.6%) patients were classified as having PTSD. This rate is 10x as high as in the general German population. Patients with TIA with PTSD were more likely to show signs of anxiety and depression. PTSD was associated with the use of maladaptive coping strategies, subjectively rated high stroke risk, as well as with younger age. Finally, PTSD and anxiety were associated with decreased mental quality of life.

Conclusions—The experience of TIA increases the risk for PTSD and associated anxiety, depression, and reduced mental quality of life. Because a maladaptive coping style and a subjectively overestimated stroke risk seem to play a crucial role in this adverse progression, the training of adaptive coping strategies and cautious briefing about the realistic stroke risk associated with TIA might be a promising approach. Despite the great loss of patients to follow-up, the results indicate that PTSD after TIA requires increased attention. (Stroke. 2014;45:3360-3366.)

Key Words: anxiety ■ depression ■ ischemic attack, transient ■ quality of life ■ stress disorders, posttraumatic

See related article, p 3182.

With an estimated lifetime prevalence of 5 per 1000 people, transient ischemic attack (TIA) is one of the most common neurological conditions.1 TIAs have been defined as brief episodes of neurological dysfunction resulting from focal cerebral ischemia not associated with permanent cerebral infarction.2 Symptoms may include the sudden onset of numbness, weakness or paralysis, slurred speech, aphasia, blurred vision, confusion, and severe headache. With =15% of all ischemic strokes being preceded by a TIA,3 TIAs are considered a warning event for a stroke.4,5 Therefore, guidelines recommend that suspected TIA patients are immediately referred to rapid access clinics so secondary prevention treatments can be commenced (eg, prescription of antiplatelet and cholesterol-lowering medication) and those at highest risk of stroke be identified.6

It is well known that even a mild stroke can cause clinical levels of depression, anxiety, and posttraumatic stress disorder (PTSD).7 In fact, in the case of subarachnoid hemorrhage, it has been found that such psychosocial sequelae have a bigger impact on survivors’ quality of life (QoL) than neurological or neuropsychological disorders.8,9 Despite the often dramatic and sudden onset of TIAs, their aftermath, and requisite treatments, little is known about the psychosocial impact of experiencing a TIA, including the information patients are given about their health. Can such an experience cause chronic psychosocial problems? Can the diagnosis of a TIA, with the associated knowledge that the patient is of increased risk to have a stroke in the future, reduce QoL, cause anxiety, depression, or PTSD? Having answers to these questions is important so that appropriate support can be offered to patients and families, not least because disorders such as PTSD are associated with low adherence to medication,10,11 which might in turn elevate a TIA patient’s risk of experiencing further health events.

A recent systematic review found no study that focused exclusively on the prevalence of PTSD after TIA.12 Instead, studies have mixed stroke and TIA patients together.13-16 However, with a mixed sample it is not clear what the critical factor for developing...
PTSD is. Is it the residual impairment (in the case of stroke) or the traumatic experience of neurological symptoms during the ictus (in the case of both stroke and TIA)? This can only be determined in a TIA-only sample. Thus, the primary aim of our study was to investigate how TIA on its own influences the prevalence of PTSD. Secondary aims were to explore the link between PTSD, anxiety, depression, and QoL, and to identify potential risk factors for PTSD, such as demographic variables, dispositional coping style, and knowledge about the disorder. Therefore, we prospectively investigated patients who were diagnosed with a TIA and assessed their anxiety, depression levels, diagnosis of PTSD, QoL, and coping style with standardized questionnaires. Additionally, the patients’ clinical status was documented and their knowledge about the disorder was assessed.

Methods

Patients

The charts of patients who were admitted between October 2010 and December 2011 to the stroke unit of the Department of Neurology of the University Hospital Erlangen because of a TIA were screened. To ensure that we only included patients with reliable signs of a TIA, we determined the ABCD2 score (a score that takes into account age, blood pressure, clinical features, duration of symptoms, and diabetes mellitus), which indicates the risk to suffer a stroke <2 days after a TIA. We included patients with an ABCD2 score >3 (meaning a 4% risk of having a future stroke) or patients with an ABCD2 score ≤3, who also reported the experience of a hemiparesis, aphasia, or amaurosis fugax during the ictus. To exclude patients with mild stroke, all patients received a brain scan, and only those patients without any sign of brain lesion were included. Furthermore, only those patients were included where the discharge letter from the responsible neurologist in our clinic stated that all symptoms related to the ictus had resolved.

During the study period, 243 patients were admitted to our clinic because of suspected TIA, and 211 patients met our inclusion criteria and received our questionnaires 3 months after the ictus via mail. In the patient information form, it was stated that the purpose of the study was to investigate whether psychological problems may follow the experience of transient neurological symptoms. The protocol was conducted in accordance with the Declaration of Helsinki II and was approved by the ethics committee of the Friedrich-Alexander University Erlangen-Nuremberg. All participants gave their written informed consent before the investigation.

One hundred twenty-eight patients sent back the questionnaires. A further exclusion criterion was applied here. In separate letters patients were sent forms of the modified Rankin Scale (indicating the degree of disability or dependence in daily activities). On the first form they were asked to estimate the degree of disability before the TIA, on the second form they had to indicate their current status of disability. Only patients with modified Rankin Scale scores between 0 and 2 on both forms were included. There was a minor worsening of symptoms only in 3 patients (ie, score 0, pre-TIA; score 1, 3 months post-TIA; see Table). Thus, we can exclude that our patients had developed disabilities because of TIA.

Of the 211 included patients, data of 108 patients were complete and included in the analyses (Table), meaning a loss of 103 patients for analysis. However, sex, age, and atrial fibrillation did not significantly differ between patients whose data were included and patients who did not respond or had incomplete data (all \( P > 0.05 \)).

Self-Rating Instruments

Patients were asked to fill in the following questionnaires.

Posttraumatic Stress Diagnostic Scale

The German version (Ehlers A et al, unpublished data) of the well-established posttraumatic stress diagnostic scale\(^{17} \) was used to test if a participant warranted a diagnosis of full PTSD in relation to their TIA according to criteria of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. The posttraumatic stress diagnostic scale asks participants to rate their experience of the 17 PTSD symptoms in the past month and allows a symptom severity score, ranging from 0 to 51, to be calculated. To be classified as having a diagnosis of PTSD, a participant’s answers needed to show that they experienced intense fear, helplessness, or horror at time of the event (criterion A); had persistently re-experienced the event (criterion B intrusion); persistently avoided stimuli associated with their TIA and showed a numbing of responsiveness (criterion C); and had persistent symptoms of increased arousal (criterion D). This symptom picture must have been present for ≥1 month (criterion E) and caused them significant distress or impairment in social, occupational, or other important areas of functioning (criterion F). Psychometric evidence shows that the posttraumatic stress diagnostic scale performs well in relation to standardized diagnostic interview techniques. It has adequate diagnostic agreement compared with interview (82%) and good sensitivity (0.89) and specificity levels (0.75).\(^{11} \)

Hospital Anxiety and Depression Scale

The degree of depression and anxiety during the preceding week was assessed with the German version\(^{20} \) of the hospital anxiety and depression scale.\(^{20} \) Separate anxiety and depression subscale scores can be computed. When compared with diagnostic interview, an optimal balance between sensitivity (0.80) and specificity (0.80) is achieved most frequently when caseness is defined by a score of 28 on either of its subscales.\(^{21} \) A score of 8 to 10 is been taken as indicating mild to moderate distress, whereas a score ≥11 as indicating moderate to severe distress.\(^{21} \)

Short Form 36

Physical QoL and mental QoL were measured with the Short Form-36 self-rating questionnaire and compared with the normative sample described in the test manual.\(^{23} \)

Brief COPE Inventory

The German adaptation\(^{24} \) of the brief COPE Inventory\(^{25} \) was applied to assess patient’s disposition for using maladaptive coping strategies. Studies having used this German adaptation showed its usefulness. For example, higher scores in scales measuring burnout, depression, and anxiety were correlated with higher scores in maladaptive coping in German students.\(^{26} \) The occurrence of depression and PTSD after spinal cord injury was mediated by maladaptive coping style.\(^{27} \) The sum of maladaptive coping strategy scores (denial, substance use, behavioural disengagement, self-distraction, self-blame) was computed as an indicator of maladaptive coping. The higher the maladaptive coping test score, the more patients habitually use maladaptive coping strategies.

Medical Knowledge About TIAs

To measure the patients’ medical knowledge about TIAs, they had to answer questions (see the online-only Data Supplement) dealing with the risk to suffer a stroke in the future, the impact of lifestyle habits and medication on this risk, and indicate whether they considered themselves to be well informed on TIAs or not. They were also asked to rate their personal risk for a further attack in percentage terms on a visual scale. A score between 1 and 4, with 1 meaning no medical knowledge about TIAs and 4 meaning very good medical knowledge about TIAs, was computed.

Statistical Analyses

Descriptive statistical analyses were performed, and the prevalence rates of PTSD and reduced QoL were determined and compared with German normative samples.\(^{28,29} \) \( \chi^2 \) and Mann–Whitney \( U \) tests were used to look for differences in the assessed variables between patients...
with and without PTSD. Nonparametric tests were chosen for this purpose because of the different group sizes (n=33 versus 77). Stepwise multiple regressions with backward elimination methods were computed when the outcome variable was continuous, and logistic regression with backward elimination methods (likelihood ratio test) was applied when the outcome variable was dichotomous. Nonparametric tests were chosen for this purpose because of the different group sizes (n=33 versus 77).

### Results

#### Prevalence of PTSD

Thirty-two of 108 patients (29.6%) were diagnosed with PTSD according to the posttraumatic stress diagnostic scale. The median symptom severity score of this group was 17.5 (interquartile range, 12). The prevalence found in our sample was 10x higher than in the general German population, where a prevalence of 2.9% was found with the same measure. This difference is statistically significant, as indicated by the 95% confidence interval of 12.27% to 20.36% of the prevalence of our sample for mental QoL and 5.77% to 8.22% of physical QoL, lying clearly above the 5% cutoff of the general German population for mental QoL and marginally for physical QoL.

#### Comparison Between Patients With Versus Without PTSD

Patients diagnosed with PTSD and patients without PTSD did not significantly differ according to age and sex (see Table). Patients with a PTSD diagnosis scored significantly higher in the depression, anxiety, and negative coping scales, estimated their risk to suffer a stroke as higher, and showed significantly reduced mental and physical QoL compared with patients without PTSD. Note that the median anxiety score of the PTSD group is almost identical with the cutoff point of 10.

#### Variables Associated With PTSD

We used a logistic regression model with the dichotomous version of the PTSD variable (present/absent) as our dependent variable and age, sex, maladaptive coping, and medical knowledge as independent variables. We did not include depression and anxiety as independent variables because of the significant content overlap between items for depression/anxiety and PTSD. The full model was significant ($\chi^2(4)=17.66; P=0.001$), indicating that the model distinguishes successfully between patients with and without PTSD. The model explained between 15.1% (Cox and Snell $R^2$) and 21.4%...
(Nagelkerke $R^2$) in the variance of PTSD occurrence and correctly classified 75% of cases. Only maladaptive coping strategies and age significantly contributed to the model with maladaptive coping strategies as the strongest associated variable (odds ratio, 1.2; 95% confidence interval, 1.07–1.32; B [SE]=0.173 [0.054]; Wald[1]=10.297; $P=0.001$). This means that the risk to develop PTSD is 1.2× higher for patients with TIA using maladaptive coping strategies. In contrast, age seems to act as a protective factor. With every additional year, patients are 0.96× less likely to develop PTSD (odds ratio, 0.96; 95% confidence interval, 0.924–0.999; B [SE]=−0.04 [0.02]; Wald[1]=4.08; $P=0.043$). Figure 1 shows the box plot for maladaptive coping scores of patients with/without PTSD.

We repeated the above-mentioned logistic regression but replaced the independent variable medical knowledge (total score of the medical knowledge scale) with the independent variable perceived risk of a future stroke (subscore of the corresponding item in the medical knowledge scale). The full model was again significant ($\chi^2[4]=35.59; P<0.001$), explaining between 28.1% (Cox and Snell $R^2$) and 39.9% (Nagelkerke $R^2$) in the variance of PTSD occurrence, and correctly classified 79.6% of cases. Maladaptive coping strategies, subjectively rated stroke risk, and age significantly contributed to the model with subjectively rated stroke risk as a significantly associated variable (odds ratio, 1.06; 95% confidence interval, 1.03–1.09; B [SE]=0.057 [0.015]; Wald[1]=13.61; $P<0.001$). This means that patients who estimate their risk to have a stroke as high are more likely to develop PTSD. Again, maladaptive coping strategies (odds ratio, 1.2; 95% confidence interval, 1.07–1.34; B [SE]=0.18 [0.06]; Wald[1]=9.16; $P=0.002$) and age (odds ratio, 0.95; 95% confidence interval, 0.91–0.99; B [SE]=−0.055 [0.022]; Wald[1]=6.168; $P=0.013$) were also significantly associated with PTSD occurrence. This link between perceived stroke risk and PTSD is also confirmed by an additional analysis where we split the sample of patients into 2 groups according to whether their stroke risk estimate was unrealistically high (risk >20%; n=61) or correct or too low (risk ≤20%; n=47). Only 14.9% of the correct/low-risk patients were diagnosed with PTSD in contrast with a much higher 40.9% in the high-risk group. This difference was significant ($\chi^2[1]=8.67; P=0.003$).

### Variables Associated With QoL

Thirty-eight percent of variance in mental QoL was accounted for ($R^2=0.385; F_{1,107}=16.141; P<0.001$) in the stepwise multiple regression with age, sex, PTSD severity score, and anxiety as independent variables. We have not included the depression score as independent variables in the model because mental QoL includes depression as a component dimension. PTSD symptom severity score (squared semipartial correlation $sr^2=0.027$; standardized $\beta=0.261; P=0.032$) and anxiety score ($sr^2=0.054$; standardized $\beta=0.364; P=0.003$) were significantly associated variables. Age and sex were not predictive for mental QoL. Figure 2 shows the box plot for PTSD severity scores of patients with/without reduced mental QoL. Sixteen percent of variance of physical QoL was accounted for ($R^2=0.165; F_{4,107}=4.02; P=0.002$) in the linear regression with age, sex, PTSD severity score, anxiety, and depression as independent variables. Age emerged as the only variable significantly associated with physical QoL ($sr^2=0.109$; standardized $\beta=0.346; P<0.001$). This means the older the patients the lower the physical QoL. Sex, anxiety, depression, and PTSD severity were not predictive of physical QoL.

### Discussion

To our knowledge, this is the first study investigating the prevalence of PTSD in a TIA-only sample, which is essential for disentangling the contribution of residual impairment after stroke and other factors such as the traumatic experience of the ictus for developing PTSD. Given that patients with TIA experience only temporary neurological symptoms, which completely recover without leaving brain lesions, the finding of an increased prevalence of PTSD and poor psychosocial outcome is remarkable and deserves increased attention and prevention efforts. The fact that patients with TIA can also develop PTSD supports the assumption that neither brain damage nor physical or mental disability on its own explain the association of neurological disorders with increased rates...
of PTSD. Elevated prevalence of PTSD has previously been found after cerebrovascular diseases, including subarachnoid hemorrhage, spontaneous cervical artery dissection, or stroke. In line with those studies, we found a 10× higher prevalence of PTSD in patients having experienced a TIA in comparison with the general German population. Some studies have investigated the occurrence of PTSD in stroke/TIA patients before. Kronish et al and Goldfinger et al reported a PTSD prevalence of 18% in patients having experienced a stroke or TIA in the previous 5 years. Patients with PTSD had higher modified Rankin Scale scores, were younger, and were more likely to be women and to show depressive symptoms compared with patients without PTSD. In a study by Favrole et al, a PTSD prevalence of 25% (Impact of Events Scale) or 10% (Interview) was observed 1 to 6 months after the ictus in a mixed stroke and TIA sample. Furthermore, 40% of the patients with PTSD were depressed. Sembé et al found a prevalence between 7% (Penn Inventory of PTSD) and 21% (Impact of Events Scale) 1 to 18 months after a first-ever stroke or TIA using self-report measures.

Although TIA causes no brain lesion, metabolic changes such as alterations of lactate/N-acetylaspartate ratio can occur up to 3 days after TIA. Changes in N-acetylaspartate have also been observed in patients with PTSD. Thus, we cannot exclude that such metabolic processes play a role in the development of PTSD after TIA. However, we assessed PTSD 3 months after TIA and metabolic changes were only assessed <3 days after TIA. Furthermore, those studies only show correlational associations between metabolic changes and TIA and metabolic changes and PTSD respectively; thus, the direction of the associations is not clear.

Which other factors might be responsible for the development of PTSD after TIA? It seems likely that the sudden experience of the neurological symptoms itself is a major factor in triggering PTSD in patients with TIA. In this vein, Favrole et al showed that the risk to develop PTSD was higher for stroke/TIA patients reporting strong peritraumatic reactions. Furthermore, the fear that the experienced neurological symptoms might be precursors of a stroke leading to more permanent deficits might also contribute to the development of PTSD. This fear is partly justified, but it is possible that patients who overestimate this risk might be also at higher risk of developing PTSD. Our findings provide support for this hypothesis. We found that the perceived risk of a future stroke is significantly linked to PTSD. On the contrary, knowledge about TIA and the associated stroke risk might be a protective factor. However, we found no correlation between the patient’s medical knowledge on TIAIs and the likelihood of developing PTSD. But, because we used a nonvalidated measure for the assessment of the patients’ knowledge, further research is needed to clarify this relationship. Furthermore, the range of the factor medical knowledge score was rather narrow, a fact that could be in part responsible for its insignificant association with our outcome measures.

Another potential factor for the development of PTSD after TIA might be the experience of acute pain, such as severe headache, which can occur during the attack. Acute pain was shown before to predict PTSD symptoms. Pain may be a sufficient but probably not necessary condition for the development of PTSD. There are certainly cases of PTSD where pain is involved (eg, subarachnoid hemorrhage), but there are also cases where physical pain does not play a role (eg, PTSD in relatives and friends of subarachnoid hemorrhage patients). In the context of our study, pain probably also plays a role, but only a minor one because only 3 patients mentioned headache as one of the TIA symptoms. Instead, PTSD was predicted by a maladaptive coping style, a factor that has been shown to be predictive for PTSD in patients with other cerebrovascular diseases. This provides a possible starting point for the prevention of PTSD in patients with TIA. The training of adaptive coping strategies and cautious briefing about the realistic stroke risk associated with TIA might reduce the risk for developing PTSD after TIA. Such a prevention approach may be of particular importance to younger patients who are more likely to have PTSD (see the findings from this study, but also Kronish et al for patients with TIA) and Noble et al for patients with subarachnoid hemorrhage). To ensure that patients who are at increased risk of developing PTSD are identified, collaboration with psychiatrists is important.

Interestingly, the presence of PTSD together with anxiety was associated with reduced mental QoL. A relation between anxiety and QoL has been reported before in stroke patients and between both anxiety and depression in a mixed stroke and TIA sample. Moreover, a correlation between PTSD and mental QoL was previously found in subarachnoid haemorrhage and spontaneous cervical artery dissection. Furthermore, Goldfinger et al observed decreased physical and mental health in stroke/TIA patients with PTSD. Franzén-Dahlin and Laska found a higher decrease in QoL in female compared with male patients between 6 and 8 weeks after a TIA. No such relation was found in a mixed TIA and stroke sample, but when the delay of 15 years between ictus and assessment in the study by van Wijk et al can be assumed that adaptation had probably taken place.

Patients with TIA with PTSD had a lower mental QoL, were more likely to have depression and anxiety, thereby corroborating findings from previous studies in TIA/stroke patients. The adverse psychosocial outcome in patients with TIA with PTSD underlines once more that early prevention, for example via briefing and coping training, is advisable. With respect to physical QoL, a different pattern emerged. Age rather than PTSD or depression was found to be significantly associated with physical QoL in our sample. Older patients had a more reduced physical QoL score. This finding is in line with age differences reported in the test manual of the German version of the Short Form 36.

We acknowledge the following potential limitations to our study: First, because of the cross-sectional design, we were only able to detect a correlation between coping style and PTSD. However, in a prospective study, it has been shown that habitual use of maladaptive coping strategies predicted the occurrence of PTSD, indicating that maladaptive coping style may be a cause rather than a consequence of PTSD. The same may be true in our group of patients, but to confirm the causal link between coping style and PTSD, an interventional study is needed. It is also possible that coping style is an
enduring personality trait that is not amenable to coping training. Positive findings of studies on coping training in other patient groups do, however, suggest otherwise. The cross-sectional nature of our design also affects the interpretation of the observed correlations between perceived stroke risk, PTSD, depression, and anxiety. In all of these cases, it remains unclear whether these variables act as causes, mediators, or reflect consequences.

Second, with respect to the correlation between perceived stroke risk and PTSD, a further limitation needs to be acknowledged. The perceived stroke risk is a subjective estimate by the patients. We do not know to what extent this estimate is informed by objective clinical information and reflects variations in objective stroke risk. Although it would have been interesting to parcel out the objective stroke risk and calculate the extent to which patients overestimate the risk of a future stroke, such a calculation is fraught with difficulties. The available figures relate to the risk of patients with TIA left untreated. But all the patients in our sample received treatment that is known to reduce the risk on average by 80%. Furthermore, the timescale for the objective stroke risk (typically the risk for a stroke within the next 90 days) differs from the timescale for the subjective estimate (patients were asked to predict their risk for their remaining lifetime). As a consequence, all we can say at the moment is that the perceived risk of a future stroke is related to an increased risk of developing PTSD; we cannot say to which extent these concerns of having a stroke in future are warranted or justified.

Third, given the relative short time period between ictus and PTSD assessment, we currently do not know whether the high rate of PTSD in our sample will persist. However, studies in patients with other neurological disorders such as stroke or subarachnoid hemorrhage have found an increased prevalence of PTSD 1 year after the ictus. No significant change was found in a study with patients with subarachnoid hemorrhage when PTSD rates at 3 and 13 months post ictus were compared.

Fourth, given the high number of patients who failed to return or complete all forms, there is the possibility that our sample is not fully representative of the eligible population of patients with TIA. However, the reader should note that even if we assumed the most extreme case, namely that PTSD did not occur at all in the drop-out group, we still had a prevalence of 15.16% in the entire population, which amounts to a 5-fold increase compared with the general population. Conversely, it is also possible that patients with PTSD tended to avoid to fill in questionnaires dealing with the traumatic experience, and thus the prevalence of PTSD might in fact be even higher. Under both assumptions, PTSD is significantly elevated in patients after a TIA. Furthermore, the investigated sample was on average 3 years younger than the patients who were excluded. As we found age to be a protective factor, we would expect the prevalence of PTSD to be lower in the excluded population, but we would not expect that this reduction is substantial because the likelihood to develop PTSD is reduced merely by 0.96x per additional year. Therefore, we think that the central message of our study is still valid.

Conclusions

The experience of a TIA, despite leaving patients symptom-free, increases the risk of developing a PTSD and associated depression, anxiety, and reduced QoL. Especially, patients using maladaptive coping strategies and patients overestimating their stroke risk are more likely to have PTSD after a TIA, a cycle that may be broken by offering patients better risk counseling and more adaptive strategies of coping with their experience of a TIA. The great loss of patients to follow-up in our study does not change the important conclusion that PTSD after TIA and the associated psychosocial outcome is a serious concern.

Disclosures

None.

References


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Stroke. 2014;45:3360-3366; originally published online October 2, 2014;
doi: 10.1161/STROKEAHA.113.004459

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/45/11/3360

Data Supplement (unedited) at:
http://stroke.ahajournals.org/content/suppl/2014/10/02/STROKEAHA.113.004459.DC1
Dear patient,

Three months ago, you suffered from a transient ischemic attack. With this questionnaire we are trying to assess your knowledge about this disease. The questions refer solely to the disease that led to the admission to the stroke unit of the neurological clinic.

First of all, please indicate whether you feel well informed about your disease (Question 1). Afterwards, please specify for each question, which of the listed statements is correct (Questions 2-4). Finally, we ask you to estimate the risk to suffer a stroke (Question 5)?

1. How well informed do you feel about your disease (transient ischemic attack)?
   
   □ good  □ bad

2. Only one answer is correct. Please tick the correct answer.
   
   □ (a) I have an increased risk that I might suffer a stroke
   □ (b) There is only a small risk that I might suffer a stroke

3. Only one answer is correct. Please tick the correct answer.
   
   □ (a) The medication that I received during my stay in this clinic (ASS, Aggrenox, Plavix, Iscover or Marcumar) will give me full protection against a stroke.
   □ (b) The medication that I received during my stay in this clinic (ASS, Aggrenox, Plavix, Iscover or Marcumar) will lower the risk of a stroke.
   □ (c) The medication that I received during my stay in this clinic (ASS, Aggrenox, Plavix, Iscover or Marcumar) will not change the risk of a stroke.
4. Only one answer is correct. Please tick the correct answer.

☐ (a) By modifying my lifestyle (low-fat diet, regular exercise, avoidance of nicotine) I can reduce the risk of suffering a stroke.

☐ (b) By modifying my lifestyle (low-fat diet, regular exercise, avoidance of nicotine) I cannot reduce the risk of suffering a stroke.

5. Please make a cross on the line below to indicate your estimated risk of suffering a stroke. (0=I will definitely not suffer a stroke; 100=I will definitely suffer a stroke)

Thank you for your cooperation!