How well does the Oxfordshire Community Stroke Project classification predict the site and size of the infarct on brain imaging?

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Abstract

***Objectives*—The Oxfordshire Community Stroke Project (OCSP) classification is a simple clinical scheme for subdividing first ever acute stroke. Several small studies have shown that when an infarct is visible on CT or MRI, the classification predicts its site in about three quarters of patients. The aim was to further investigate this relation in a much larger cohort of patients in hospital with ischaemic stroke.**

***Methods*—Between 1994 and 1997, inpatients and outpatients with ischaemic stroke were assessed by one of several stroke physicians who noted the OCSP classification. A neuroradiologist classified the site and extent of recent infarction on CT or MRI.**

***Results*—Of 1012 patients with ischaemic stroke, 655 (65%) had recent visible infarcts. These radiological lesions were appropriate to the clinical classification in 69/87 (79%) patients with a total anterior circulation syndrome, 213/298 (71%) with a partial anterior circulation syndrome, 105/144 (73%) with a lacunar syndrome, and 105/126 (83%) with a posterior circulation syndrome. Overall, 75% of patients with visible infarcts were correctly classified clinically. If patients without a visible infarct did have an appropriate lesion in the brain (best case), the classification would have correctly predicted its site and size in 849/1012 (84%) patients, compared with only 492/1012 (49%) in the worst case scenario.**

***Conclusion*—The OCSP classification predicted the site of infarct in three quarters of patients. When an infarct is visible on brain imaging, the site of the infarct should guide the use of further investigations, but if an infarct is not seen, the OCSP classification could be used to predict its likely size and site.**

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Keywords: Oxfordshire Community Stroke Project; classification of stroke; computed tomography

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The Oxfordshire Community Stroke Project (OCSP) classification is a simple clinical scheme to subdivide acute strokes which was originally devised for patients with first ever in a lifetime stroke.[1] Patients are classified using clinical criteria only. Lacunar syndromes (LACS) include pure motor stroke, pure sensory stroke, sensorimotor stroke and ataxic hemiparesis. Patients with brain stem or cerebellar signs, and/or isolated homonymous hemianopia are classified as posterior circulation syndrome (POCS). Those with total anterior circulation syndromes, (TACS), by definition, present with the triad of hemiparesis (or hemisensory loss), dysphasia (or other new higher cortical dysfunction) and homonymous hemianopia. Patients with partial anterior circulation syndrome, by definition, present with only two of the features of TACS, or isolated dysphasia or parietal lobe signs. Patients are classified as "syndromes" (TACS, PACS, LACS, and POCS), unless brain imaging has excluded intracerebral haemorrhage, in which case the patients are reclassified as total or partial anterior circulation infarct (TACI or PACI), lacunar infarct (LACI), and posterior circulation infarct (POCI).

The classification is easy to communicate, has good interobserver reliability, is of some value in predicting recovery, disability, and patterns of recurrent stroke, and is a guide to aetiology.[1–3] Several studies which have investigated the validity of the classification found that it could predict the site and size of any cerebral infarct (if visible) on CT or MRI in about three quarters of patients.[4–8] However, one study included only a few patients (108) and is therefore rather imprecise[6]; one classified patients retrospectively[4]; and in another study, only about half of the CT reports (but no scans at all) were available.[7] Cerebral infarcts are not always visible on imaging, although there may still be an infarct which is appropriate to the syndrome, but only two studies considered the effect on accuracy of including patients without visible infarction.[6–8] The presence of pre-existing neurological signs can make it difficult to classify a patient correctly, but only three studies included patients with previous strokes.[6–8]

Our aim was to assess how well the OCSP classification could determine infarct site and size in a much larger group of patients with recurrent as well as first acute ischaemic stroke presenting as inpatients and outpatients in everyday practice, and to give a more precise estimate of the validity of the classification.

Methods

We prospectively identified consecutive patients with a stroke within the previous 3 months who either required inpatient care or were referred to our neurovascular outpatient clinics from November 1994 to December

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Table 1 Appearance of the infarct on the brain scan and the clinical syndromes considered appropriate to that appearance

CT or MRI appearance	*Clinical syndrome*	*Abbreviation*
Large cortical MCA infarct (whole of the cortex supplied by the MCA plus adjacent white matter and part or all of the ipsilateral basal ganglia) or more than half of the MCA territory plus ACA or PCA territory	Total anterior circulation infarction	TACI
Medium sized cortical infarct (about half the MCA territory)	Partial or total anterior circulation infarction	PACI or TACI
Small cortical infarct (less than a quarter of the MCA territory) or any of the ACA territory	Partial anterior circulation infarction	PACI
Border zone cortical infarct between ACA and MCA or PCA and MCA territories	Partial anterior circulation infarction	PACI
Large (>1.5 cm) subcortical infarct (striatocapsular)	Total or partial anterior circulation infarction	TACI or PACI
Small (<1.5 cm) subcortical infarct (lacunar) (including centrum semiovale infarcts)	Lacunar infarction	LACI
Cortical infarct in PCA territory	Posterior circulation infarction	POCI
Brainstem or cerebellar infarct (including small infarcts in the pons)	Posterior circulation infarction	POCI

MCA=middle cerebral artery; ACA=anterior cerebral artery; PCA=posterior cerebral artery.

1997, thereby excluding patients seen before this who were reported in a previous study.[6] A stroke physician (consultant or trainee) examined the patients as soon as possible after stroke onset and classified them as TACS, PACS, LACS, POCS, or "uncertain" stroke types.[1] The clinician assessed the patient before the brain imaging and was therefore blind to the results. The classification was reviewed at a weekly meeting also blind to the brain imaging. The 44 patients with uncertain stroke types were reclassified at the end of the study by a trainee (GEM) into the most likely OCSP subtype (blind to the imaging) using clinical information collected at the time of the event, as a previous study had demonstrated that the "best guess" OCSP subtype was usually correct.[2]

Brain imaging was reviewed by a consultant neuroradiologist (JMW), who after initial scan review to identify any possible infarct, was made aware of the clinical details so that recent infarcts could be classified as appropriate or inappropriate to the clinical syndrome. Patients with intracerebral haemorrhage were excluded. Patients not scanned within 4.5 months after stroke onset (allowing up to 3 months from stroke onset to hospital assessment and a further 6 weeks from assessment to scan for those referred as outpatients) were excluded from the analysis. We included patients scanned up to 4.5 months because this reflects clinical practice for patients seen in the outpatient department. An infarct was defined as an area of hypodensity (with or without mass effect) whose shape indicated vascular origin. "Old" infarcts were distinguished from recent infarcts by the their more marked hypodensity, lack of mass effect, and clear borders. Recent infarcts seen on brain imaging with their appropriate OCSP clinical syndromes were classified as in table 1. It should be noted that the size of cortical infarcts (large, medium, or small) was defined according to the proportion of the MCA territory affected rather than by infarct volume. Both medium sized cortical infarcts and large subcortical infarcts were considered to be compatible with a clinical diagnosis of either a TACI or a PACI, because both a PACI which is nearly severe enough to be a TACI and a true TACI indicate significant cortical and/or subcortical damage likely to be due to occlusion of the trunk of the MCA or a major branch.[6]

Table 2 CT or MRI appearance in relation to the clinical syndrome

CT/MRI	*TACI*	*PACI*	*LACI*	*POCI*
Large cortical	**42**	18	1	0
Medium cortical	**11**	**39**	1	1
Small cortical	5	**90**	17	6
Border zone cortical	4	**55**	10	7
Large subcortical	**16**	**29**	3	0
Small subcortical	1	38	**105**	7
PCA territory cortical	8	28	3	**58**
Brainstem or cerebellar	0	1	4	**47**
No recent infarct	7	143	121	86
Total	94	441	265	212

The case notes for one patient who was classified as "unknown" were missing, so this patient could not be classified retrospectively and is excluded from the analysis.
The bold numbers indicate infarcts which are appropriate to each of the syndromes.

Sensitivity, specificity, and positive and negative predictive values were calculated for the relation between the clinical syndrome and appropriate infarcts as defined in table 1.

We calculated the number of patients with appropriate infarcts for a theoretical worst case scenario (in which all the normal scans would have an infarct in a site other than predicted clinically), and for a theoretical best case scenario (in which all the patients with normal scans would have an infarct in the site predicted clinically).

Results

Between November 1994 and December 1997, 1187 consecutive patients with acute stroke were assessed. One hundred and seventy four were excluded from the analysis (89 with primary intracerebral haemorrhage, 17 who were assessed longer than 3 months after the stroke, 66 who did not have CT or MRI, and two who had CT more than 4.5 months after the stroke).

Therefore, 1013 patients with ischaemic stroke are the subject of this report, of whom 460 (45%) were female, 4 (<1%) were non-white, 411 (41%) had a history of hypertension, 123 (12%) were in atrial fibrillation before their stroke, and 120 (12%) had diabetes mellitus (on diet, oral hypoglycaemic agents, or insulin). Three hundred and twenty three (32%) were assessed clinically within 2 days of stroke onset, 615 (61%) within 7 days, and the remainder within 3 months. The median time between stroke onset and clinical

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Table 3 Proportion of patients (%) in each of the of OCSP syndromes who had appropriate infarcts; including the best case scenario where all normal scans might have had an infarct in the site predicted clinically, and the worst case scenario, where all patients might have had an infarct in a site other than predicted clinically

	TACI	*PACI*	*LACI*	*POCI*	*Total*
Excluding scans without recent infarct	69/87 (79)	213/298 (71)	105/144 (73)	105/126 (83)	492/655 (75)
Best possible scenario	76/94 (81)	356/441 (81)	226/265 (85)	191/212 (90)	849/1012 (84)
Worst case scenario	69/94 (73)	213/441 (48)	105/265 (40)	105/212 (50)	492/1012 (49)

classification was 5 days (interquartile range 2–15 days), and between stroke onset and CT (or MRI) 4 days (interquartile range 1–21.5 days). Four hundred and forty two (44%) were scanned within 2 days of stroke onset, 611 (61%) within 7 days, and the remainder within 131 days. Overall, 92% had CT and 8% had MRI. One of three consultants performed the initial assessment in 270 (27%) patients, and the remainder were initially seen by trainees and discussed later with the consultant. Seventy nine (8%) patients had had a previous disabling stroke and a further 117 (12%) a previous non-disabling stroke. The clinical and imaging results are shown in table 2.

Of the 655 patients with visible infarcts, these were appropriate (as defined in table 1) in 69/87 (79%) TACIs, 213/298 (71%) PACIs, 105/144 (73%) LACIs, and 105/126 (83%) POCIs (table 3). Overall, 75% of patients were classified correctly. The sensitivity, specificity, and positive and negative predictive values of the syndromes in relation to brain imaging are shown in table 4. When the analysis was restricted to patients without previous strokes, 403/532 (76%) patients were classified correctly. If any cortical infarct (irrespective of size), or a large subcortical infarct, was considered appropriate for TACIs or PACIs, then 78/87 (90%) TACIs and 231/298 (78%) PACIs had appropriate infarcts, and overall 519/655 (79%) patients with visible infarcts were classified correctly. However, if large subcortical infarcts were considered appropriate only for PACIs (rather than both TACIs and PACIs), only 53/87 (61%) TACIs had appropriate infarcts, and the overall accuracy fell slightly to 476/655 (73%). If the only appropriate infarct for TACIs was defined as large cortical, then only 42/87 (54%) of TACIs would have appropriate infarcts, but the overall accuracy of the classification would remain high at 71%.

If all CT or MRI without a visible infarct did in fact have an appropriate lesion (using the definitions in table 1), the classification would have correctly predicted its site and size in 84% of patients, whereas if all patients without visible infarction in fact had an inappropriate lesion, the classification would have correctly predicted its site and size in only 49% (table 3).

As clinical examination may be insensitive to subtle cortical signs in the non-dominant hemisphere, we investigated the influence of the side of the infarct on the relation between the clinical syndrome and site of infarct. For LACIs with a lacunar infarct, 51 had right sided infarcts, 51 had left sided, and three had both left and right sided infarcts. For LACIs with an "inappropriate" cortical infarct, 18 were right sided infarcts, and 14 were left sided. Hence 74% of right sided and 78% of left sided infarcts were correctly classified. For PACIs with small, medium, or border zone infarcts, 124 (86%) were left sided, 86 (83%) right sided, and three had both. For PACIs with an inappropriate lacunar infarct, 21 were left sided and 17 right sided.

For the patients undergoing CT or MRI within 48 hours of stroke onset the numbers with an appropriate infarct were 50/64 (78%) TACIs, 75/102 (74%) PACIs, 17/29 LACIs (59%), and 31/34 (91%) POCIs. For patients undergoing CT over 48 hours after stroke onset, the numbers with an appropriate infarct were 19/23 (83%) TACIs, 138/196 (70%) PACIs, 88/115 (77%) LACIs and 74/92 (80%) POCIs. Thus there was no obvious difference in accuracy between those scanned sooner rather than later after the stroke, but too few patients were seen within the first few hours of stroke to determine the accuracy of the classification very early on.

Table 4 Sensitivity, specificity, positive predictive values, and negative predictive values (%) and 95% CI for each of the syndromes compared with brain imaging (including patients where a visible infarct is seen)

	TACI	*PACI*	*LACI*	*POCI*
Sensitivity	43 (35-53)	72 (65-78)	70 (62-77)	70 (65-78)
Specificity	96 (93-98)	76 (71-91)	93 (90-94)	96 (94-97)
Positive predictive value	79 (68-87)	71 (66-75)	73 (63-80)	83 (75-89)
Negative predictive value	84 (80-90)	77 (71-81)	91 (88-93)	92 (90-94)

Discussion

About one third of our patients did not have visible infarction on CT or MRI, which is similar to the results of previous studies.[4 5] When a recent infarct was present on brain imaging, the OCSP clinical classification correctly predicted its site and size in about three quarters of patients, irrespective of whether the patient had had a previous stroke, and whether scanned within 48 hours or not. This is similar to the results of smaller studies, despite the differences in methodology, particularly in the way that cortical infarcts were classified. This previous literature is summarised in table 5, and includes additional unpublished data from Lindgren *et al*[5] (and personal communication). The overall accuracy of two studies which considered any cortical infarct (irrespective of size) as being appropriate for TACIs or PACIs was 70% and 82% respectively.[4 7] The other three studies which took into account the extent of cortical infarction found that the overall accuracies were 74%, 88%, and 95% respectively (personal communication 1998).[5 6 8]

The overall accuracy is partly dependent on the definition of "appropriate infarcts". In the study of Lindgren *et al* if large cortical or large subcortical infarcts were considered appropriate for TACIs, and small cortical or large subcortical infarcts for PACIs (rather than

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Table 5 Summary of studies investigating the relation between the OCSP clinical classification of ischaemic stroke and the site of any lesion on CT or MRI

Author	Number of ischaemic strokes	Recent infarcts on CT or MRI (%)	Proportion of infarcts appropriate to each of the syndromes(%)					Comments
			TAC1	PAC1	LAC1	POC1	Overall	
Anderson *et al* 1994[4]	248	162 (65)	46/58 (79)	25/44 (57)	30/48 (62)	12/20 (60)	113/162 (70)	Community first ever strokes, retrospectively classified
Lindgren *et al* 1994[5] (and personal communication)	179	110 (61)	35/39 (90)	21/37 (57)	13/22 (59)	12/12 (100)	81/110 (74)	First ever stroke. Previously unpublished data
Wardlaw *et al* 1996[6]	108	91(84)	30/33 (91)	30/36 (83)	12/14 (86)	8/8 (100)	80/91 (88)	Hospital series. Included previous non-disabling strokes
Mead *et al* 1996[7]	195	158 (81)	41/46 (89)	48/57 (84)	25/37 (68)	16/18 (89)	130/158 (82)	Hospital series. Only half of CT reports of 378 patients with ischaemic stroke were available
Al-Buhairi *et al* 1998[8]	378	239 (63)	40/49 (82)	79/82 (94)	65/66 (98)	32/32 (100)	216/228 (95)	Hospitals series of acute ischaemic strokes. Included previous strokes
Current study	1012	655 (65)	69/87 (79)	213/298 (71)	104/144 (73)	105/126 (83)	492/655 (76)	Validity similar for those with and without previous strokes

"Appropriate infarcts" are defined in different ways.
For Anderson *et al*, Mead *et al*, and Lindgren *et al*,[4 5 7] any cortical infarct or large subcortical infarct is defined as "appropriate" for both TACIs and PACIs, small subcortical for LACIs and posterior circulation for POCIs.
Wardlaw *et al*[6] and the current study used stricter definitions of "appropriate infarcts": large cortical, medium cortical, and large subcortical were appropriate for TACIs; and medium cortical, small cortical, and large subcortical were appropriate for PACIs.
Al-Buhairi *et al*[8] classified complete MCA territory infarction, ACA territory infarction as appropriate for TACIs, partial MCA infarcts, and subcortical infarcts as appropriate for PACIs.

considering any cortical and large subcortical infarcts appropriate for either TACIs or PACIs), the number of infarcts appropriate to the syndrome falls from 81/110 (74%) to 68/110 (62%) (personal communication 1998).[5] In the current study, the accuracy rises slightly from 75% to 79% if any cortical infarct or large subcortical infarct is considered appropriate for either PACIs or TACIs. However, the overall accuracy falls to 71% if the only infarct appropriate to TACIs was defined as large cortical.

Patients were assessed up to 3 months after the stroke onset. The presence or absence of a visible infarct may be related to the time between stroke onset and brain imaging, but in the study by Lindgren *et al*, there were no significant differences in the mean volume of brain infarct in patients scanned at 0–2 days, then 3–15 days, and then 16–180 days.[5] We found that the validity of the classification was similar for those who had brain imaging less than 2 days after stroke and those having brain imaging later.

In the very acute stage of stroke, the clinical signs may evolve over time; for example, initially a patient might present with weakness of the right arm and be classified as a PACI, but an hour later have weakness of the ipsilateral leg and face so changing the classification to an LACI; and then becomes dysphasic with hemianopia so changing the classification to a TACI. Ideally, the maximal neurological deficit should be used to classify the patient,[2] but this is obviously inappropriate very early on, when treatment might be particularly effective. In the current study, few patients were assessed so early after stroke onset and there have been no other studies of the accuracy of the classification within the first few hours of stroke. It would be useful to validate the OCSP in the hyperacute setting.

There are probably several reasons why the site and size of infarct was not appropriate to the clinical syndrome in a quarter of the patients in the current study. The maximum deficit should be used to categorise patients, but because we assessed patients up to 3 months after stroke onset, some neurological signs may have resolved, and an accurate history of the acute event may have been difficult to obtain at a later stage. Hence, a TACI could be misclassified as a PACI if a homonymous hemianopia, dysphasia, or inattention had resolved, and a PACI could be misclassified as an LACI due to resolution of cortical signs. From a history alone, dysarthria may be difficult to distinguish from minor degrees of dysphasia, and hence difficulty may arise in distinguishing a PACI from a LACI or from a POCI. Patients with hemianopia or neglect may be unaware of their deficits. A few PACIs were misclassified as LACIs which may be due to the insensitivity of clinical examination to subtle cortical signs, although there was little difference in the accuracy of the classification for dominant or non-dominant hemisphere infarcts. Some infarcts may actually have been old, and the patient may have been scanned too early to see the new infarct. A posterior cerebral artery (PCA) infarct could be misclassified as a PACI or TACI because proximal PCA artery occlusion may cause infarction of part of the temporal lobe and lead to hemiparesis (usually mild) and language disturbance in addition to visual field loss.[9] This could be recognised clinically as a "TACI who is able to walk". In patients with a dominant posterior communicating artery, occipital infarcts can be caused by interruption of the carotid circulation.

The neuroradiologist was not completely blind to the clinical syndrome. This was a deliberate policy to ensure that infarcts could be correctly classified as appropriate or inappropriate to the clinical syndrome. This is a potential source of bias which would tend to improve the validity of the classification. In

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practice, the age of small infarcts is more difficult to determine than for larger infarcts, so knowledge of the OCSP classification was important to determine appropriateness of the lesion in those scanned more than 3 weeks after the stroke. Although our neuroradiologist was not blind to the syndrome, the validity of the OCSP in our study was no better than previous studies in which the scans were assessed blind to the clinical details.[4–6 8]

We considered large subcortical infarcts to be appropriate for TACIs or PACIs. In trials of neuroprotection it may be important to distinguish between subcortical and cortical infarcts given the potentially different effects of neuroprotective agents. In this case, appropriate calculations could be done using the data in table 2.

Our data support the use of the OCSP classification in clinical practice. The OCSP classification correctly predicts the site and size of infarct in about three quarters of patients. Knowledge of the likely site and extent of infarct is useful when decisions need to be made about further investigations, particularly when any brain imaging is normal, or not available. For example, an embolic aetiology (for example, carotid stenosis or atrial fibrillation) is more common in cortical than lacunar infarcts, implying that an embolic aetiology would be more common in TACIs and PACIs than in LACIs.[10–12] This is important when planning carotid Doppler ultrasound or echocardiography, (including transoesophageal echocardiography), particularly when a visible infarct is not seen on CT. LACIs are likely to have small subcortical infarcts, suggesting that the aetiology is probably small rather than large vessel disease, so carotid Doppler ultrasound is less likely to identify a clinically significant carotid lesion.[10–12]

The OCSP classification may be useful in stratifying patients in clinical trials, particularly in the very acute phase of stroke when imaging may still be normal, although of course classification can be more difficult when symptomatic signs are still evolving and this hypothesis still needs to be tested. It might be postulated that a new acute treatment would be more effective in cortical than lacunar infarcts, or in large cortical than small cortical infarcts, but patients cannot be stratified according to the site and size of infarction if the brain imaging is normal. In the best case scenario, 84% of patients would have an infarct in a site predicted clinically, whereas in the worst case scenario, 49% would have an infarct in a site predicted clinically. Patients could be stratified according to the clinical classification as a quite reasonable guide to the likely site and size of any cerebral infarction.

Although a quarter of patients were misclassified, the OCSP classification is particularly useful if a visible infarct is not seen on CT and in epidemiological studies where access to brain imaging may be limited.

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