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ABSTRACT

Background: Motor dysfunction in the contralateral hand has been well characterised after stroke. The ipsilateral hand has received less attention, yet may provide valuable insights into the structure of the motor system and the nature of the recovery process. By tracking motor function of both hands beginning in the acute stroke period in patients with cortical versus subcortical lesions, we sought to understand the functional anatomy of the ipsilateral deficit.

Methods: We examined 30 patients with first-ever unilateral hemiparetic stroke, 23 with subcortical lesions affecting the corticospinal tract, seven with cortical involvement. Patients performed hand dynamometry and the 9-Hole Peg Test (9HPT) with each hand at 24–48 h, 1 week, 3 months and 1 year after stroke. Linear regression was used to compare the two different motor tasks in each hand. Repeated measures ANOVA was used to compare recovery rates of the two tasks in the first 3 months.

Results: Ipsilateral 9HPT scores averaged $z = -7.1$, -3.6, -2.5 and -2.3 at the four time points whereas grip strength was unaffected. The initial degree of impairment of grip strength in the contralateral hand did not correlate with the degree of impairment of 9HPT in either the contralateral or ipsilateral hand ($r = 0.001$, $p = 0.98$), whereas the initial degree of impairment of 9HPT in the contralateral hand correlated with the degree of impairment of 9HPT in the ipsilateral hand ($r = 0.79$, $p = 0.035$). The rate of recovery also differed for the two tasks ($p = 0.005$).

Conclusion: Ipsilateral motor deficits are demonstrable immediately after stroke and extend into the subacute and chronic recovery period. Dissociation between grip strength and dexterity support the notion that dexterity and grip strength operate as anatomically and functionally distinct entities. Our findings in patients with subcortical lesions suggest that the model of white matter tract injury needs to be refined to reflect the influence of a subcortical lesion on bi-hemispheral cortical networks, rather than as a simple "severed cable" model of disruption of corticofugal fibres. Our data have implications for both stroke clinical trials and the development of new strategies for therapeutic intervention in stroke recovery.

Unilateral stroke causing hemiparesis is commonly thought of as affecting only the contralateral hand. What has been much less completely addressed is the effect of stroke on the hand ipsilateral to the lesioned hemisphere. More than 30 years ago, Norwegian neuroanatomist Brodal observed that his right side handwriting was impaired after he suffered a right hemisphere stroke causing left hemiparesis.[1] Other investigators have subsequently reported impairment of function of the ipsilateral hand after hemiparetic stroke.[2–10] Much of that work, however, was cross sectional rather than longitudinal, which prevented the inclusion of the time course in the characterisation of dysfunction and recovery. Only three studies have tracked ipsilateral motor dysfunction longitudinally, and those studies started with the subacute stroke period[11–13] eliminating early time point assessment when ipsilateral deficits may be more prominent . In addition, small numbers in many of these studies precluded statistical assessment of the effects of different anatomical locations or comparisons between different aspects of motor dysfunction.

In this study, we sought to address ipsilateral hand performance after hemiparetic stroke, starting in the early post-stroke period. Using serial measurements of power (maximum grip strength) and dexterity (9-hole peg test (9HPT)), we tracked motor performance of the ipsilateral and contralateral hands at several time points from 24–48 h to 1 year after stroke. We were particularly interested in determining how ipsilateral control of motor function differed from contralateral control, and whether subcortical lesions which have no evident commissural connection to the opposite hemisphere could produce ipsilateral dysfunction. Our hypotheses were (1) that the ipsilateral hand could be shown to be functionally impaired throughout the early time period of recovery, (2) that the impairment of dexterity would dissociate from impairment of grip strength, both in the acute phase and in the rate of recovery, which would suggest divergent mechanisms of dysfunction for the two motor functions and (3) that the ipsilateral dysfunction would be present subcortical lesions, which, if hypothesis (2) were true, would necessitate a re-examination of how subcortical lesions can influence cortical function.

METHODS

Patients were enrolled between 24 and 48 h after stroke onset if they had first-ever clinical ischaemic stroke causing contralateral weakness and had a positive diffusion weighted image lesion. Subjects were allowed prior, asymptomatic diffuse white matter disease or small strokes on fluid inversion recovery or T2 scans, but could not have had prior symptomatic subcortical or asymptomatic cortical strokes, particularly strokes potentially affecting the motor tracts. Aphasia or hemineglect were

allowed as long as there was sufficient comprehension to give informed consent. Excluded were patients with global inattentiveness, dementia, psychiatric illness or other medical or neurological conditions, aside from current stroke, precluding task performance (eg, severe arthritis, neuropathy, blindness, etc); patients with seizure at stroke onset were excluded.

The prospective study design, part of the NIH sponsored Columbia Specialised Program of Translational Research in Acute Stroke (SPOTRIAS), involved examination of motor function at 24–48 h (baseline), 1 week and 3 months after stroke onset. An additional examination at 1 year was also performed in some patients. All study subjects signed informed consent for the research study which was approved by the Columbia University Institutional Review Board.

Two motor functions were assessed. As a measure of strength, hand dynamometry was assessed in the contralateral and ipsilateral hand at all time points. An average of three attempts was calculated, using z scores to correct for gender, age and hand dominance.[14] For patients too weak to generate any force on the dynamometer, a z score of −6.6 was assigned, representing a score just below the score of the weakest non-plegic patient. As a measure of dexterity, patients performed the 9HPT, which is a timed, standardised, quantitative test requiring coordinated reaching and precision grip. Specifically, the patient is presented with a plastic block containing nine empty holes and a small, shallow container holding nine pegs. On a start command, the patient picks up the nine pegs one at a time with one hand as quickly as possible, puts them in the nine holes and, once they are in the holes, removes them again as quickly as possible one at a time, replacing them into the shallow container. The total time to complete the task is recorded. Normal times are 19–22 s depending on age and hand dominance. Standard performance norms were used to calculate individual z scores which corrected for age and hand dominance.[15] The National Institutes of Health Stroke Scale (NIHSS) and the Medical Research Council (MRC) Motor Strength Scale for wrist extensors, flexors, biceps, triceps and deltoid muscles were obtained at each of the four time points. Ideomotor apraxia was assessed by the ability to correctly pantomime use of scissors. The Zung self-rating depression scale[16] was used to assess for depression.

Table 1 Demographic, lesioned hemisphere side and dominance, baseline NIHSS, contralateral hand dynamometry and a lesion group assignment for the 30 patients

Patient No	Sex	Age (y)	Lesioned hemisphere side (dominance)	Baseline NIHSS	Affected hand dynamometry (kg)	Cort/high SC (CS) vs low SC/ brainstem (S)	Lesion location
1	F	56	R (ND)	14	0.0	S	Post internal capsule, corona radiata
2	F	51	L (ND)	3	18.7	C/S	Putamen, corona radiata, internal capsule
3	F	59	R (ND)	3	10.7	S	Paramedian pons
4	M	70	R (ND)	4	33.0	S	Paramedian pons
5	M	48	R (ND)	3	26.7	S	Putamen, corona radiata, caudate
6	M	47	L (D)	4	1.3	S	Paramedian pons
7	M	61	R (ND)	4	0.0	C/S	Occipito-parietal, motor, and premotor cortex
8	M	63	L (D)	6	0.0	S	Post internal capsule
9	F	65	L (D)	5	14.7	S	Paramedian pons, internal capsule
10	F	59	L (D)	8	0.0	S	Corona radiata, posterior internal capsule
11	F	65	R (ND)	5	5.0	S	Corona radiata
12	M	62	L (D)	5	23.0	S	Post internal capsule, corona radiata
13	M	77	R (ND)	5	0.0	C/S	Motor and sensory cortex
14	F	67	R (ND)	9	0.0	S	Post internal capsule, thalamus
15	M	56	R (ND)	12	0.0	C/S	Putamen, corona radiata
16	F	82	R (ND)	6	0.0	S	Basal ganglia
17	M	59	L (D)	11	0.0	S	Paramedian pons
18	F	79	L (D)	6	0.0	C/S	Motor and premotor cortex, corona radiata
19	M	53	R (ND)	8	0.0	S	Corona radiata, posterior internal capsule
20	M	49	R (ND)	12	0.0	C/S	Prefrontal cortex, frontal operculum, corona radiata
21	M	69	R (ND)	11	0.0	S	Paramedian pons
22	F	42	L (D)	7	26.0	C/S	Subinsula, corona radiata
23	F	64	R (ND)	8	13.0	C/S	Frontal operculum
24	F	86	R (ND)	6	8.0	C/S	Premotor cortex
25	M	69	L (D)	5	0.0	S	Midbrain peduncle
26	M	56	R (ND)	5	2.0	C/S	Corona radiata
27	M	78	R (ND)	9	0.0	C/S	Premotor cortex, corona radiata, insula, occipital cortex
28	M	43	R (ND)	6	30.0	S	Paramedian pons
29	M	62	L (D)	6	0.0	C/S	Corona radiata, posterior internal capsule, putamen
30	M	48	L (D)	6	21.0	S	Corona radiata

Cort, cortical; L, left; ND, non-dominant; NIHSS, National Institutes of Health Stroke Scale; R, right; SC, subcortical.

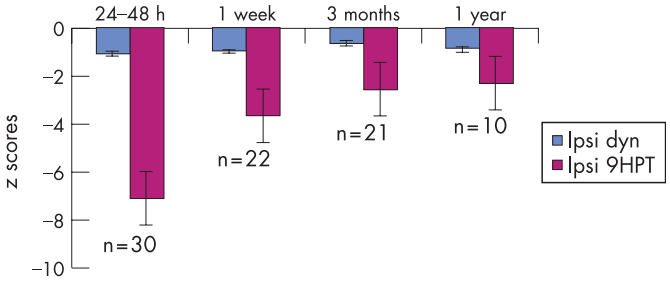

Figure 1 Ipsilateral hand dynamometry (Ipsi dyn) and 9-Hole Peg Test (Ipsi 9HPT) z scores among all patients at the four time points.

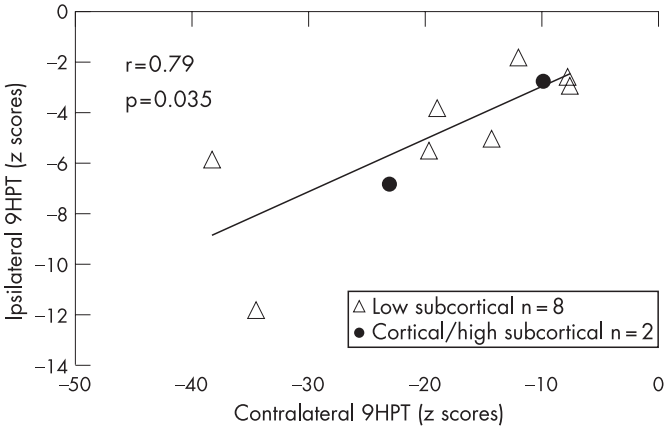

Figure 2 Contralateral 9-Hole Peg Test (9HPT) versus ipsilateral 9HPT in the non-plegic group, only showing a significant correlation between hands.

Statistical analysis

Z scores of the two tests were used in all comparisons. Spearman correlation was used to assess the relationship between grip strength and dexterity in each hand, and between the contralateral and ipsilateral hand for each motor function. The p value chosen for significance was 0.05. Bonferroni correction for multiple comparisons was performed. Repeated measures ANOVA was used to compare rate of recovery between 24–48 h and 3 months for the two different motor functions. All statistical calculations were performed using SPSS V.12.0.

RESULTS

Table 1 shows the demographics, lesion location, dominance, baseline NIHSS and baseline grip strength for the 30 stroke patients. Included were 12 women and 18 men, mean age 62 (11) years. Twenty-three patients had subcortical infarcts only; seven had cortical involvement. In five patients with subcortical strokes the lesions extended above the superior margin of the lateral ventricle, thus potentially involving commissural fibres connecting frontal motor areas through the corpus callosum. We designated these patients as "high subcortical" and classified them along with the patients with only cortical lesions as a separate group (n = 12). The "low subcortical" group (n = 18) and brainstem patients comprised a second group. This nomenclature is consistent with depictions of the commissural fibres of the motor cortex as traversing the body of the corpus callosum above the roof of the lateral ventricles.[17–19]

Our subjects had variability in stroke severity at baseline, with NIHSS scores ranging from 3 to 14 (mean = 7), and upper extremity function ranging from total plegia to mild pronator drift. At baseline, 16 of the 30 patients were plegic or severely paretic enough in the contralateral hand so as to not be able to generate any force on hand dynamometry. Eighteen patients were unable to perform 9HPT with their contralateral hand, at baseline. None of the patients had apraxia. Two patients had a recurrent stroke in the first 3 months and, therefore, the follow-up data were not used. One patient was unable to follow-up because of dementia that developed after the baseline examination. Twenty-two of the remaining 27 patients had 3 month follow-up data (two lost to follow-up and three had not reached 3 months yet). Ten of the 22 patients had data available at 1 year (one had a recurrent stroke between 3 months and 1 year, and 11 had not reached 1 year). There was no difference in age, gender, dominance of lesioned hemisphere or baseline 9HPT in either hand between those for whom we had 1 year data and those for whom we did not. There was no difference in ipsilateral 9HPT performance between those with left versus right hemisphere stroke either at 24–48 h (mean z scores L = −6.1 (6.2), R = −7.7 (8.7); p = 0.58) or at 3 months (mean z score L = −2.1 (2.5), R = −2.8 (2.0); p = 0.44). The two patients in our cohort who had small asymptomatic subcortical strokes had no worse ipsilateral 9HPT performance than those without such lesions.

Among the non-plegic patients, average contralateral hand 9HPT z scores were −18.2, −9.6, −8.1 and −3.8 at 24–48 h, 1 week, 3 months and 1 year, respectively. Among all patients, the ipsilateral hand averaged z = −7.1, −3.6, −2.5 and −2.3 at the same points, despite normal NIHSS motor scores and normal MRC scores for that hand. Average hand dynamometry z scores were −3.1, −2.6, −1.7 and −1.3 for the contralateral hand in non-plegic patients and −1.1, −1.0, −0.9 and −1.0 for the ipsilateral hand among all patients at the four time points. Patients who were initially unable to perform the test were included at later time points. Inclusion of recovering patients did not alter our results. Figure 1 shows ipsilateral motor performance for the two tasks among all patients. 9HPT was significantly worse than hand dynamometry in the ipsilateral hand in the first 3 months ($p<0.001$ at 24–48 h, 1 week, 3 months; p = 0.144 at 1 year).

Although 9HPT appeared to dissociate from grip strength in the ipsilateral hand, we considered the possibility that our grip strength measurement was not sensitive enough to identify a subtle weakness, and thus we could not show a global motor dysfunction (ie, both power and dexterity) in the ipsilateral hand. We therefore looked for a correlation between severity of hemiparesis (as measured by grip strength in the contralateral hand) and impairment on 9HPT in the ipsilateral hand, which would suggest a unitary mechanism of motor dysfunction. We found that contralateral grip strength did not correlate with ipsilateral 9HPT performance (Spearman correlation coefficient (r) = −0.033, p = 0.93). In other words, greater weakness on hand dynamometry was not associated with worse performance on 9HPT in the ipsilateral hand, further suggesting the two functions were dissociable. Plegic patients could not be used in this correlation analysis due to a variance of 0, but we found that patients with greatest contralateral weakness or plegia did not cluster at the low end of the ipsilateral 9HPT scores. Tested as a group, the ipsilateral performance of the plegic patients on 9HPT was not significantly different from that of the non-plegic patients (p = 0.09). Of note, ipsilateral 9HPT performance was also no worse in those with cortical or high subcortical lesions than in those with low subcortical lesions (fig 2).

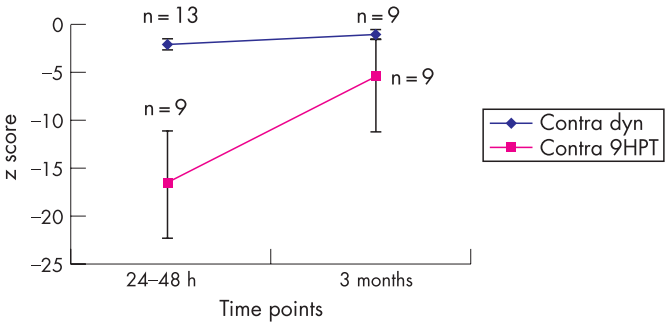

Figure 3 Pattern of recovery in the contralateral hand dynamometry (Contra dyn) and 9-Hole Peg Test (9HPT) over 3 months.

Having established a dissociation of 9HPT from dynamometry in the ipsilateral hand and from the contralateral to the ipsilateral hand, we next examined the relationship between the two tests in the contralateral hand alone. An absence of correlation was found ($r = -0.139$, $p = 0.74$). We then assessed the correlation between the two hands for each of the tasks. Whereas no correlation was found between the ipsilateral and contralateral hand on hand dynamometry ($r = 0.001$, $p = 0.98$), a significant correlation was found between the two hands on 9HPT performance ($r = 0.79$, $p = 0.035$) (fig 2). This correlation was performed on the group of patients who fell into the "low subcortical" category, specifically those whose lesions, subcortical and brainstem, should have had the least or no contribution to the commissural crossing fibres. As shown in fig 2, the two patients with higher lesions fell close to the correlation line.

Finally, in addition to a dissociation of performance at baseline, the course of recovery differed by task (fig 3). Ipsilateral hand 9HPT performance improved over time whereas hand dynamometry remained minimally affected throughout the recovery period, as illustrated in fig 1. Figure 3 shows that in the contralateral hand, the recovery rate was greater for 9HPT than for grip strength (repeated measures ANOVA: $F = 26.7$, $p<0.001$ for time×task interaction). Only non-plegic patients were again used because variance was 0 among patients with dynamometry = 0.

DISCUSSION

We found that the ipsilateral hand was affected by stroke throughout the first year of recovery. To our knowledge, this is the first study to show the ipsilateral effect beginning in the early acute period and the first to show dissociation by task type in patients with subcortical lesions alone. Specifically, we found that as early as 24 h post hemiparetic stroke we could identify impaired performance in the ipsilateral hand on the 9HPT but no significant impairment on hand dynamometry; this effect remained significant up to 3 months after the stroke. Further examination of the ipsilateral deficit revealed that there was no correlation between the degree of ipsilateral dexterity dysfunction and degree of weakness, yet the degree of dexterity appeared to correlate in the two hands in the early period. Finally, the rate of recovery also differed between the two functions. Taken together, our findings suggest that in both the ipsilateral and contralateral hands, dexterity exists as an anatomically and functionally distinct entity from motor power, with separate vulnerability to dysfunction, and a distinct pattern of recovery.

Our behavioural findings allowed us to consider the anatomical substrate for the ipsilateral dysfunction, in particular for subcortical infarcts which have not been described previously on the ipsilateral effects of stroke. While it is true that other post-stroke factors may have an impact on motor dexterity (eg, attentional deficits, depression scores or the effects of focused rehabilitative therapy) we did not find this in our population. We considered it unlikely that the ipsilateral motor dysfunction was due to general attentional deficits or cognitive slowing as a result of the acute brain injury as none of our patients had poor performance on tests of cortical function impairment or verbal fluency which can be used as a non-motor measure of cognitive processing speed. Furthermore, the ipsilateral motor dysfunction continued through the subacute and chronic phases, by which time any acute stroke effects would have dissipated.

A parsimonious explanation for our finding of bilateral motor dysfunction from subcortical stroke would be a model of a "severed cable" of white matter tracts with two outflow paths—the more prominent crossed corticospinal tract affecting the contralateral hand, and uncrossed motor pathways affecting the ipsilateral hand to a lesser degree. For the contralateral hand, there is evidence from diffusion tensor magnetic resonance imaging and transcranial magnetic stimulation (TMS) that the degree of hemiparesis and potential for recovery is proportional to the integrity of the corticospinal tract.[20 21] Whether the ipsilateral corticospinal tract plays a role in hemiparesis or recovery is more controversial. Although ipsilateral monosynaptic pathways[22 23] and multisynaptic, double crossed[24 25] motor projections are known to exist, disruptive TMS stimulation of the lesioned hemisphere has not been shown to result in a decrement in motor performance in the ipsilateral hand.[26]

Although the "severed cable" model could theoretically cause impairment in ipsilateral dexterity out of proportion to strength, our data suggest otherwise. A lesion affecting corticofugal fibres rostral to the medulla would be expected to produce a proportional decrement in motor performance for both hands as the information would be travelling through a common path. In contrast, we showed an absence of correlation of motor dysfunction between the two tasks in both the ipsilateral and contralateral hands. In particular, one would have expected more impaired ipsilateral 9HPT performance with more severe contralateral hemiparesis, but we did not find this correlation to hold true. Likewise, if the "severed cable" model were the complete explanation, recovery rate should be proportional also, with recovery of white matter tracts at the site of the lesion restoring function proportionally to all descending pathways distal to the lesion. Our data, however, showed that the rate of recovery also differed between grip strength and 9HPT.

An alternative explanation to simple disruption of crossed and uncrossed pathways is that different aspects of motor function require different degrees of bilateral cortical involvement. Functional imaging studies show that complex motor tasks requiring motor planning, integration of sensorimotor information and attention to sequencing are associated with bihemispheral activity.[27–30] In general, more complex unilateral motor tasks such as the 9HPT induce greater bilateral task related activity than do simple tasks[31–33] and, indeed, with the exception of an older study by Colebatch and Gandevia which identified a reduction in strength in the ipsilateral hand in 14 subacute-to-chronic stroke patients,[4] most other behavioural studies have identified post-stroke ipsilateral deficits in complex functions—target directed movements of the hand and foot,[5] abnormal anticipated grip forces when lifting novel test objects,[34] step tracking deficits in the wrist[8] and slowing on

the 9HPT[7] and grooved pegboard test.[35] Among the studies that measured ipsilateral hand function over time, Jones *et al*, who assessed eight patients with unilateral stroke at six time points starting from day 11 and ending at 12 months post stroke,[11] found persistently impaired reaction time, impairment of steadiness and tracking on visuomotor tasks in the ipsilateral hand but no ipsilateral weakness. Two other studies showed impairment and subsequent improvement of the ipsilateral hand on several complex motor tasks in patients beginning in the subacute period and recovering over 3–4 months.[12 13]

However, none of the above studies showed the ipsilateral effects in subcortical lesions alone as there was no differentiation between cortical and subcortical lesions. Furthermore, an explanation of how the ipsilateral effect occurs in unilateral cortical lesions has relied predominantly on the notion of disruption of interhemispheral interactions via transcallosal pathways.[10 35] Following cortical stroke, TMS and magnetoencephalography have shown that a lesion in one hemisphere can alter excitability in the opposite hemisphere.[36–41] Among our subcortical patients, we tried to exclude those whose effect on the opposite hemisphere was likely to be explained by a direct, monsynaptic, transcallosal disruption by grouping them with our cortical lesion patients. We found that the ipsilateral impairment in dexterity was present regardless of whether or not the lesions were likely to involve the callosal fibres. Furthermore, even if a minor transcortical effect from any supratentorial lesion exists, it still leaves unexplained the ipsilesional effect we observed in the brainstem lesions. Our findings raise the interesting possibility that the motor network of the ipsilateral hand is being influenced by the effect of the deep white matter lesion on overlying cortex, either as a direct feedback effect of deafferentation or via cortico-subcortical circuits such as those connecting the motor cortex with the cerebellum and basal ganglia.[42 43] Ipsilesional effects on cortical excitability have been shown by TMS[39] as well as functional imaging studies in an acute period after subcortical strokes.[44 45]

In summary, we demonstrated a decrement in ipsilateral hand function during the early course of recovery and dissociation from proportionality between strength and dexterity. Based on our findings, we posit that the ipsilateral motor dysfunction demonstrated in this study cannot be explained simply by severing of corticofugal motor pathways, nor by an effect on monosynaptic transcallosal pathways. We propose instead that subcortical injury disrupts complex motor function by altering activity in the overlying cortex, thus impairing performance of tasks that require bi-hemispheral involvement for normal function. Recent work with motor learning in subcortical lesions shows that unilateral injury to white matter tracts may influence sensory–motor calibration networks in both hemispheres.[46] The impact of particular subcortical lesion locations on ipsilateral motor function may be addressed in the future with greater numbers of patients. Our findings also suggest that in clinical studies of stroke recovery, the use of the so-called ''unaffected'' hand as a control may underestimate both the degree of impairment and the degree and rate of recovery. Further elucidation of recovery mechanisms may be achieved with prospective functional imaging studies that correlate task induced motor activity in the contralateral and ipsilateral hand with different aspects of clinical motor recovery.

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Neurological picture

"Hand Knob" infarction

A 67-year-old man with diabetes and hypertension developed left-hand weakness. Examination showed plegia of the left hand, including wrist extension and flexion (fig 1, panel D). The sensory examination was normal. Non-contrast head CT showed grey–white blurring of the right "hand knob" motor representation (fig 1, arrow, panel A). CT perfusion showed focal decreased perfusion in the same area (fig 1, arrow, panel B), and diffusion-weighed MRI confirmed local infarction (fig 1, panel C). CT angiogram of the neck showed a right internal carotid artery origin plaque with associated focal stenosis of >70% (fig 1, arrowheads, panel E and inset). Right internal carotid stenting was performed without complications.

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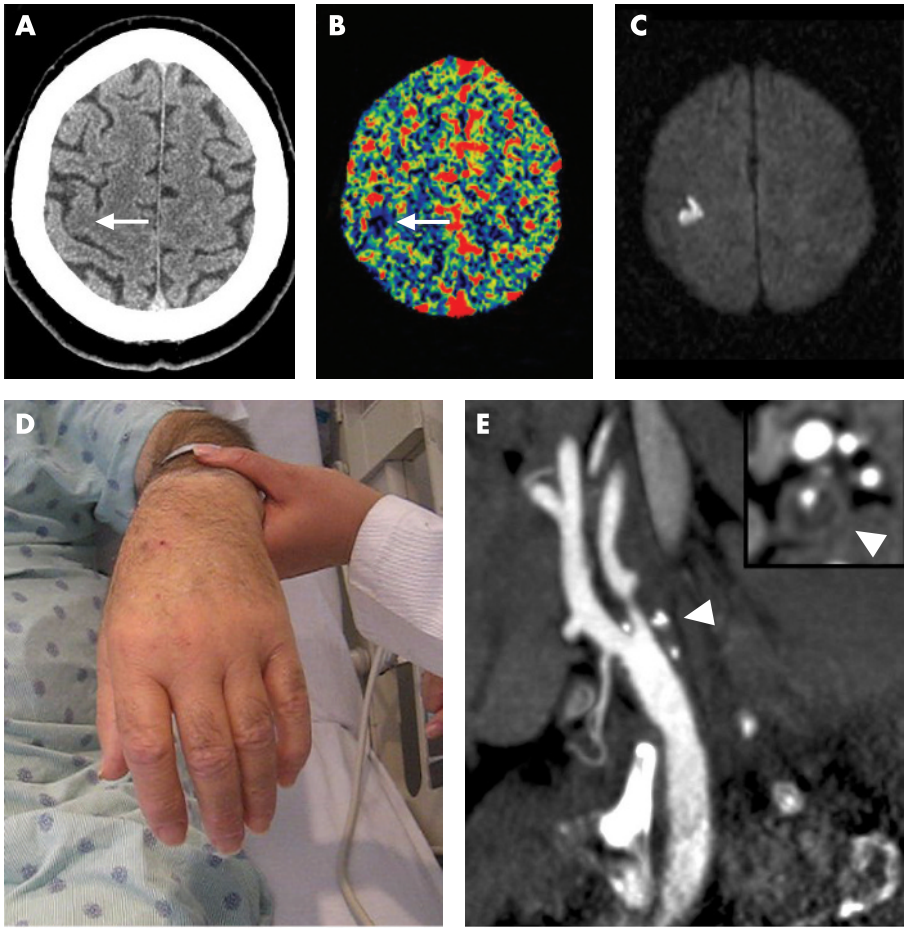

Figure 1 (A) Non-contrast head CT showing focal grey–white blurring. (B) CT perfusion showing focal hypoperfusion. (C) Diffusion-weighted MRI showing infarct of the "hand knob". (D) Patient image demonstrating plegia of the left hand and wrist. (E) CT angiogram of the neck, showing right internal carotid plaque with stenosis in longitudinal and cross-sectional (inset) views.