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Twelve-month experience of acute stroke thrombolysis in Christchurch, New Zealand: emergency department screening and acute stroke service treatment

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Abstract

Aims To determine the safety and efficiency of an acute stroke thrombolysis service in a New Zealand public hospital setting.

Methods A 12-month audit of patients referred to the Christchurch Hospital Stroke Thrombolysis Service (STS) between 1 April 2002 and 31 March 2003 was undertaken.

Results Sixty-one patients were referred to the STS during the study period, of whom 16 were treated with tissue plasminogen activator (t-PA). For treated patients, the median time from stroke onset to hospital presentation was 60 minutes, 'door-to-CT' time was 60 minutes, and the 'door-to-needle' time was 99 minutes. Minor protocol violations were recorded in two patients, but did not influence outcome. No patient was treated after 3 hours of stroke onset. Intracerebral haemorrhage occurred in two patients: one patient was significantly improved compared with pre-treatment status; a minor temporary deterioration occurred in the other patient. Eight of 16 patients had improved by 4 or more points on the NIH Stroke Scale Score at 24 hours.

Conclusions Acute stroke thrombolysis can be delivered safely and in accordance with internationally accepted guidelines using the Christchurch Hospital STS model of emergency department screening and acute stroke service treatment. Further improvements in performance of the STS remain possible.

Following publication of the National Institute of Neurological Disorders and Stroke (NINDS) t-PA Stroke Study Group trial in 1995,[1] thrombolytic treatment for acute ischaemic stroke within 3 hours of stroke onset (with intravenous tissue plasminogen activator [t-PA]) was approved by the United States Food and Drug Administration in 1996.

Since then, widespread adoption of this treatment outside of the USA has been slow; important factors being concerns regarding the safety of this treatment outside of a clinical trial setting[2] and logistical difficulties inherent in the rapid delivery of this acute stroke treatment. While review of randomised evidence shows that treatment with intravenous t-PA is associated with a substantial reduction in the outcome of death and disability for carefully selected patients within 3 hours of stroke onset,[3] deviation from accepted treatment protocols is known to be associated with increased mortality.[4]

Acute stroke thrombolysis was introduced to Christchurch Hospital in April 2002. An audit of the first 12 months' experience of this service was planned to establish whether the treatment could be delivered safely and in accordance with established guidelines for this therapy.

Methods

All referrals to the acute stroke thrombolysis service (STS) between 1 April 2002 and 31 March 2003 were recorded in a prospective database. Data including time of stroke onset; hospital arrival; emergency department (ED) screening; computed tomography (CT) scanning; stroke service assessment; and administration of thrombolytic treatment, inclusion, and exclusion criteria for thrombolysis and neurological examination findings at presentation were recorded prospectively on ED screening and STS proforma specifically developed to enhance comprehensive but rapid assessment of these patients (Appendix 1).

Times of ambulance call-out, dispatch, patient location, scene departure, and hospital arrival were obtained from ambulance service patient reports. All patients who received t-PA were admitted to a special care unit with established protocols for post-thrombolysis care—including frequent nursing observations for 24 hours; rigorous blood pressure control; protocols for avoidance, recording, and management of bleeding complications; and neurological deterioration.[5]

Full neurological examination at 24 hours, and repeat CT scanning within 48 hours, were part of the standard clinical protocol for patients who received t-PA. National Institutes of Health Stroke Scale (NIHSS) scores were calculated retrospectively according to a validated algorithm.[6] The clinical records of patients treated with t-PA were examined to determine final discharge destination, total length of hospital stay (including rehabilitation facilities), and level of function at discharge, recorded using the modified Rankin scale (mRS).[7]

Formal radiology reports of pre-treatment CT scans were compared with the acute CT assessment of contraindications to t-PA treatment. All follow-up brain imaging was examined for signs of haemorrhage. The total number of ischaemic stroke patients admitted to Christchurch Hospital over the same 12-month period was determined by search of the hospital discharge coding database.

Statistical analyses (using Student *t* test for parametric data, Wilcoxon rank sum test for non-parametric data, and Fisher exact test for 2x2 tables with low cell count) were performed for data comparing patients assessed by the STS and excluded from treatment versus those patients who were prescribed t-PA,.

Results

Sixty-one patients were referred to the STS over the 12-month audit period. This represented 11.8% of the 519 patients with stroke admitted to Christchurch Hospital (as identified from discharge coding data over the same period).

Of the 61 patients referred, 15 were excluded as candidates for thrombolysis following their telephone consultation, and 46 proceeded under the acute stroke protocol for STS review. Sixteen patients were treated with t-PA—comprising 26% of those referred from ED, 35% of those reviewed by the STS; and 3.1% of the total number of stroke admissions.

Among patients reviewed by the STS, there was no age or gender difference between patients treated with t-PA and those not treated, however stroke severity was significantly greater at baseline in the treated group (Table 1).

Pre-hospital transport—Forty-four of 61 patients referred to the STS arrived by ambulance, 8 by car, and 7 had in-hospital stroke. For the 44 patients seen by the ambulance service, median time from receipt of call to ambulance departure was 2 minutes (range 0–12 minutes), median time at the scene was 13 minutes (range 2–31 minutes) and total travel time was 20 minutes (range 12–101 minutes).

Table 1. Comparison of tissue plasminogen activator (t-PA)-treated and untreated patients

Variable	Patients treated with t-PA	STS screening but no t-PA	P value	NINDS trial [1]
Baseline patient characteristics				
Number of patients	16	45		312
Mean age, years	64.9 [33-84]	64.7 [40-83]	0.94	68
Gender	M8, F8	M27, F18	0.56	M58%, F42%
Median NIHSS score	16 [5-23]	4.5 [0-22]	<0.001	14 [1-37]
STS time performance measures				
Median onset time*	0955 [0720-1530]	1222 [0700-2130]	0.14	
Onset to door†‡	59 [41-128]	68 [16-656]	0.87	
Door to CT†	60 [30-106]	87 [16-611]	0.01	
CT to CT reading†	5 [2-10]			
Door to bolus†	99 [47-2:40]			100-110
Onset to bolus†	150 [60-177]			
Bolus to infusion†	16 [5-30]			

Figures in square brackets denote the range. 'Door' refers to time of presentation to the emergency department, as recorded by the ED admission clerk, or time of stroke onset for in-hospital strokes. *24 hour clock; †times expressed as medians, in minutes; ‡excludes 7 patients who were in hospital at the time of stroke onset (3 t-PA, 4 no t-PA).

In-hospital time performance—The key time performance measures of the STS service, including time from hospital arrival to CT ('door to CT') and 'door to bolus' time are shown in Table 1. For patients presenting to ED, the median time from hospital arrival to ED medical assessment was 12 minutes (range <1 minute to 89 minutes, mean 14 minutes). Accurate time of STS assessment was recorded in 37 of 46 patients accepted by the STS. Mean door to STS assessment time was 70 minutes (range 25–160), while mean door to CT time was 82 minutes in these patients. The STS assessment preceded or was within 10 minutes of CT time in all but 8 patients. Median bolus-infusion time was 22.5 minutes for the first 8 patients, and 12.5 minutes for the last 8 patients treated (p=0.21).

Exclusion from t-PA—The reasons for exclusion from thrombolysis of 45 patients reviewed by the STS were: minor or resolving deficits (12 patients); time >3 hours (12 patients); intracerebral haemorrhage (ICH) on CT (7 patients); INR >1.3 (4 patients); non-stroke diagnosis (5 patients); and other (5 patients)—(comorbidities, recent myocardial infarction [MI], past history of sub arachnoid haemorrhage, early CT signs, logistical).

Of the 12 patients excluded because of 'minor' deficits (NIHSS 0–3), 10 patients were discharged home from Christchurch Hospital, 1 patient was transferred to a provincial hospital, and the records were lost for the remaining patient. Median length of Christchurch Hospital stay for these patients was 4 days (range 1–17 days); and at discharge, 6 had mRS score of 0–1, 4 patients had mRS=2, and 1 patient had mRS=3 (no data for 1 patient).

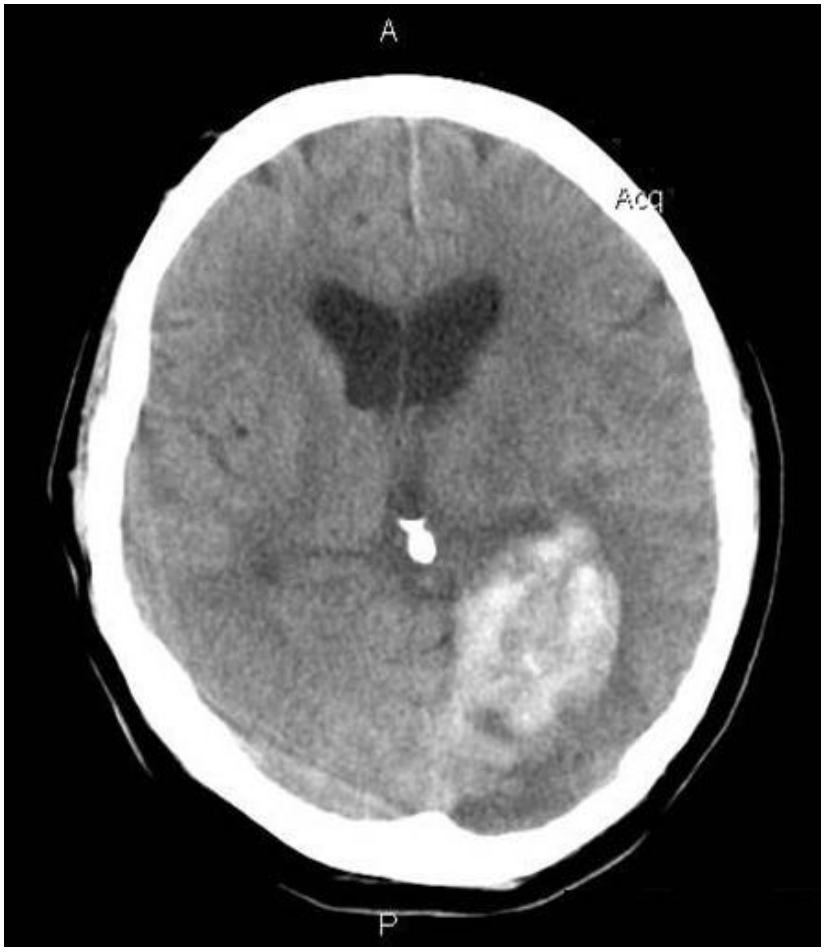
Protocol violations—Two patients treated with t-PA had documented protocol violations (12.5%): one patient had received subcutaneous low molecular weight heparin on the same day prior to stroke onset (no bleeding complications), and one patient had no documented blood glucose result prior to t-PA administration.

No patient had treatment initiation after 3 hours. No patient was treated with t-PA with BP >180/100. Two patients were treated with IV labetalol to reduce blood-pressure prior to t-PA.

Acute CT assessment—The acute CT assessment (performed by a radiology consultant, radiology registrar, or neurology consultant) was concordant with the formal radiology report for exclusion criteria for t-PA treatment in all patients. The CT scans of 13 of the patients treated with t-PA were reported as showing no signs of acute cerebral infarction, the "insula ribbon" sign[8] was seen in 1 patient, subtle reduction in grey-white differentiation was seen in 1 patient, and subtle swelling with reduced sulcation was seen in 1 patients.

Haemorrhagic complications—No patient required cessation of t-PA treatment or administration of blood products due to haemorrhage. Two patients had intracerebral haemorrhage following t-PA. One patient had minor haemorrhagic transformation of a large left MCA territory stroke associated with a transient deterioration in deficits which subsequently returned to baseline. The second patient presented with aphasia and right hemiparesis, consistent with a left MCA territory stroke. The following day, he was alert and his hemiparesis and dysphasia had substantially resolved, but a new right homonymous hemianopia was noted, due to ICH (Figure 1).

Figure 1. CT head image taken 24 hours after t-PA administration in a patient presenting with left MCA stroke. An obvious new left occipital lobe ICH is present, with little evidence of infarction in left MCA territory.



The NIHSS score had improved from 12 at initial assessment to 4. Three patients required treatment for extracerebral bleeding: cauterisation of epistaxis was required in one patient, suturing of a scalp laceration (due to a fall at the time of stroke) was required in one patient, and empiric treatment with ranitidine was required in one patient who had a single episode of suspected 'coffee-ground' vomitus within 24 hours of t-PA but normal cardiovascular status and negative gastroscopy.

Outcome after t-PA—For the 16 patients treated with t-PA, the median NIHSS score was 16 (range 5–26) at presentation, and 8 (range 3–26) the following day. Eight of 16 patients improved by 4 or more points on the NIHSS, while 7 patients had either no change or improvement by 1 point; 1 patient had worsened (by 3 points).

Improvement of >10 NIHSS points within 24 hours was seen in 3 patients, including 1 patient with dramatic recovery during t-PA infusion from reduced consciousness, quadriplegia, and impending respiratory failure due to acute basilar artery occlusion.

At final discharge from hospital or rehabilitation facility, five patients had recovered to mRS score of 0–2 (indicating full independence), seven patients had mRS=3, three patients had mRS=4, and one patient had died. The death occurred 12 days after t-PA at a time of slow neurological improvement; no definite cause was determined, but pulmonary embolism was suspected. Median length of acute plus rehabilitation hospital stay was 49 days.

Discussion

This audit of 12 months' experience of an acute stroke thrombolysis service in Christchurch shows that we are able to provide this acute stroke treatment safely and in accordance with established treatment guidelines. Protocol violations occurred in two patients, were recognised by the treating physician prior to the decision to continue with treatment, and did not influence patient outcome.

Direct comparison of our cohort with that of the NINDS trial is not possible. The baseline characteristics appear similar, except that the range of baseline stroke severity is narrower in our cohort (due to exclusion of patients with very mild or very severe stroke from thrombolysis in Christchurch). This protocol difference also likely accounts for the lower death rate recorded in Christchurch (6% vs 17% in NINDS t-PA group).[1]

The outcome for patients excluded because of minor deficits at presentation was generally favourable in our series, although others have found that as many as a third of patients excluded for this reason were dead or dependent at follow-up.[9] Intracerebral bleeding occurred in two of our patients: it was clinically insignificant in one of the patients, while the other patient appeared to have a net benefit from treatment with t-PA, despite the bleed. Extracerebral bleeding was more common, but generally minor with no lasting adverse sequelae in any patient.

ED triage and acute assessment of acute stroke patients was a critical first step in the STS pathway. Close to 10% of the total number of stroke admissions were referred to the STS, with a median time from arrival to referral of 12 minutes. However, it is possible that this figure overestimates the efficiency of the ED assessment process—as patients for whom delayed ED assessment resulted in failure to meet the 3-hour time-window will not have been captured in the audit.

A European community stroke study recorded that 25% of patients admitted to hospital with stroke had presented within 3 hours,[10] and an American community stroke study recorded 20–28% of patients presenting within 2 hours of stroke onset.[11]

Referral practices seemed to vary over the course of the audit period, suggesting the need for improvement in the continuing education of resident medical officers rotating through the ED and Neurology service regarding acute stroke thrombolysis.

The response times of the ambulance service to acute stroke calls appeared excellent. However, we cannot exclude the possibility that higher ambulance service prioritisation for acute stroke call-outs may have enabled a larger number of patients to be considered for referral to the STS. There is also evidence that education programmes for paramedics regarding acute stroke recognition may allow more efficient notification and mobilisation of hospital services, thus reducing in-hospital delays.[12,13]

In general, the performance of the STS appeared satisfactory. Time to CT scan was more frequently a rate-limiting factor, with a longer door-to-CT time in patients who were ultimately declined t-PA treatment than in those who received it. However, there were eight recorded cases where the STS review was delayed more than 10 minutes after the CT scan, indicating that STS performance can also be improved.

After-hours cover appeared as a significant issue. Our protocol allows assessment of patients up to 10pm, and although patients with stroke onset as late as 9.30pm were referred to the STS, no patient with stroke onset later than 3.30pm was actually treated with t-PA during the study period. Although after-hours performance would be expected to improve with time (indeed, several patients have subsequently been treated at later time points), no specific additional resources have been allocated to STS staffing.

Although no specific data are available, our impression was that delays to CT scanning were not due to waiting time after patient arrival in the radiology department, but instead resulted from delays in earlier steps of the protocol (including transport of the patient from ED or the hospital wards to CT scanning). CT reporting was not a significant delay in t-PA-treated patients. Most patients' scans were read off the CT scanner console while the patient was being taken from the scanning room.

ICH is an absolute contraindication to thrombolysis and easily recognised on CT by trained observers. The Christchurch Hospital Thrombolysis Protocol also considers early oedema (defined as hypodensity less than white matter intensity) or mass effect (significant effacement of cerebral sulci, sylvian fissure, or other basal cisterns; or compression of the ventricular system) as relative contraindications to thrombolysis. These signs are known to be associated with increased haemorrhage rate, although patients with such signs treated with t-PA in the NINDS trial still had a more favourable outcome than placebo-treated patients.[14]

Acute STS assessment and t-PA bolus administration were generally performed in the CT scanning annex. Transfer to the SCU and initiation of t-PA infusion was a source of further delay in treatment, but appeared to improve over the course of the audit period.

Prior to the establishment of the safety and effectiveness of this service, no specific attempts were made to publicise the acute stroke thrombolysis service to the general

public during the audit period. Although public education campaigns through mass media have been shown to increase public knowledge about stroke symptoms and risk factors,[15,16] they may not result in more rapid presentation to hospital.[11] Education campaigns that have included professional education to paramedic and emergency personnel have been associated with an increase in acute stoke thrombolysis.[11,17]

Intravenous t-PA is now licensed treatment for acute stroke in the USA, Canada, the European Union, Australia and New Zealand. Ongoing safety monitoring of treatment is a condition for approval of t-PA in the European Union.[18] Meta-analysis of community studies confirms that comparable outcomes to those of randomised trials can be achieved, especially when established treatment guidelines are followed,[4] and an Australian incremental cost-effectiveness analysis suggests that t-PA for stroke is cost-saving due to reduction in nursing home and rehabilitation costs.[19]

The New Zealand Stroke Guidelines (published in November 2003) recommend the use of thrombolytic treatment with intravenous t-PA following the NINDS protocol,[1,5] with the caveat that 'thrombolytic treatment should be administered only in specialist centres by physicians with expertise in the assessment and management of people with acute stroke and where protocols for use of thrombolysis are in place.'[20]

To our knowledge, Christchurch, Auckland, and Middlemore Hospitals are the only centres in New Zealand where acute stroke thrombolysis is currently offered as a standard treatment with established local protocols for eligible patients, although the treatment has been used occasionally elsewhere.[21] The main prerequisite for adoption of this treatment is identification a stroke physician or neurologist with the necessary expertise and motivation to develop and maintain a robust stroke thrombolysis protocol. Detailed published guidelines for use of thrombolytic therapy and management of patients post-thrombolysis are available[5] and should be incorporated into hospital nursing and medical standing orders with appropriate education for staff who will be involved prior to the use of thrombolysis.

Other important prerequisites include the need for close cooperation with the hospital emergency department physicians and triage nurses, front-line medical staff with sufficient expertise to distinguish stroke from 'stroke mimics', rapid access to CT imaging and interpretation, a immediate access to t-PA (preferably without the need to involve pharmacy staff), and a 'Special Care' nursing unit or equivalent where the close cardiovascular and neurological observations needed may be performed (cardiac monitoring is not essential).

Clinical audit following establishment of an acute stroke thrombolysis protocol should also be considered a prerequisite. These requirements do not necessarily limit this therapy only to major teaching hospitals. In Christchurch, however, pre-existing infrastructure (such as the presence of two dedicated neurology registrars, the neurology/neurosurgery special care unit, and previous local experience with acute stroke clinical trials) greatly facilitated the establishment of the acute stroke thrombolysis care pathway.

While introduction of acute stroke thrombolysis may not currently be feasible in all New Zealand centres, the establishment of organised acute and rehabilitation stroke services that are available to all patients presenting with stroke has been identified as an important health priority in New Zealand,[21,22] and is considered to be the single

most effective change needed in the management of stroke.[20] In most larger centres, this is likely to take the form of a geographical stroke unit.[20–22]

Although the benefit of stroke units is not dependent on the provision of stroke thrombolysis,[23] the establishment of stroke units and the development of improved pathways for (and expertise in) stroke management may allow a larger number of New Zealand centres to offer acute stroke thrombolysis in the future.

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References:

1. Tissue plasminogen activator for acute ischemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. N Engl J Med. 1995;333:1581–7.
2. Katzan IL, Furlan AJ, Lloyd LE, et al. Use of tissue-type plasminogen activator for acute ischemic stroke: the Cleveland area experience. JAMA. 2000;283:1151–8.
3. Wardlaw JM, Zoppo G, Yamaguchi T, Berge E. Thrombolysis for acute ischaemic stroke. Cochrane Database Syst Rev. 2003:CD000213.
4. Graham GD. Tissue plasminogen activator for acute ischemic stroke in clinical practice: a meta-analysis of safety data. Stroke. 2003;34:2847–50.
5. Adams HP, Jr., Brott TG, Furlan AJ, et al. Guidelines for thrombolytic therapy for acute stroke: a supplement to the guidelines for the management of patients with acute ischemic stroke. A statement for healthcare professionals from a Special Writing Group of the Stroke Council, American Heart Association. Circulation. 1996;94:1167–74.
6. Williams LS, Yilmaz EY, Lopez-Yunez AM. Retrospective assessment of initial stroke severity with the NIH Stroke Scale. Stroke. 2000;31:858–62.
7. van Swieten JC, Koudstaal PJ, Visser MC, et al. Interobserver agreement for the assessment of handicap in stroke patients. Stroke. 1988;19:604–7.
8. Truwit CL, Barkovich AJ, Gean-Marton A, et al. Loss of the insular ribbon: another early CT sign of acute middle cerebral artery infarction. Radiology. 1990;176:801–6.
9. Barber PA, Zhang J, Demchuk AM. Why are stroke patients excluded from TPA therapy? An analysis of patient eligibility. Neurology. 2001;56:1015–20.
10. Heuschmann PU, Berger K, Misselwitz B, et al. Frequency of thrombolytic therapy in patients with acute ischemic stroke and the risk of in-hospital mortality: the German Stroke Registers Study Group. Stroke. 2003;34:1106–13.
11. Morgenstern LB, Staub L, Chan W, et al. Improving delivery of acute stroke therapy: The TLL Temple Foundation Stroke Project. Stroke. 2002;33:160–6.
12. Harbison J, Hossain O, Jenkinson D, et al. Diagnostic accuracy of stroke referrals from primary care, emergency room physicians, and ambulance staff using the face arm speech test. Stroke. 2003;34:71–6.
13. Kidwell CS, Starkman S, Eckstein M, et al. Identifying stroke in the field. Prospective validation of the Los Angeles prehospital stroke screen (LAPSS). Stroke. 2000;31:71–6.
14. Intracerebral hemorrhage after intravenous t-PA therapy for ischemic stroke. The NINDS t-PA Stroke Study Group. Stroke. 1997;28:2109–18.

15. Stern EB, Berman M, Thomas JJ, Klassen AC. Community education for stroke awareness: An efficacy study. Stroke. 1999;30:720–3.

16. Becker K, Fruin M, Gooding T, et al. Community-based education improves stroke knowledge. Cerebrovasc Dis. 2001;11:34–43.

17. Kwan J, Hand P, Sandercock P. Improving the efficiency of delivery of thrombolysis for acute stroke: a systematic review. QJM. 2004;97:273–9.

18. Hack W, Kaste M, Bogousslavsky J, et al. European Stroke Initiative Recommendations for Stroke Management-update 2003. Cerebrovasc Dis. 2003;16:311–37.

19. Moodie ML, Carter R, Mihalopoulos C, et al. Trial application of a Model of Resource Utilization, Costs, and Outcomes for Stroke (MORUCOS) to assist priority setting in stroke. Stroke. 2004;35:1041–6.

20. McNaughton H, Baskett J. Life after stroke: New Zealand guideline for management of stroke. Best practice evidence-based guideline. Wellington: Stroke Foundation of New Zealand; 2003, p84. Available online. URL: http://www.nzgg.org.nz/guidelines/dsp_guideline_popup.cfm?&guidelineID=37 Accessed April 2005.

21. Barber PA, Anderson NE, Bennett P, Gommans J. Acute stroke services in New Zealand. N Z Med J. 2002;115:3–6.

22. Gommans J, Barber A, McNaughton H, et al. Stroke rehabilitation services in New Zealand. N Z Med J 2003;116(1174). URL: http://www.nzma.org.nz/journal/116-1174/435

23. Langhorne P, Pollock A. What are the components of effective stroke unit care? Age Ageing. 2002;31:365–71.

Appendix 1: Christchurch Hospital Stroke Thrombolysis Proforma

CPH STS t-PA Proforma Page 1

EMERGENCY DEPARTMENT

STROKE ONSET TIME ____:____ ____/____/____ Onset not known / on waking

ARRIVAL TIME ____:____ ____/____/____ Arrival by ambulance Other

E.D. ASSESSMENT TIME ___:___ ___/___/___

- **BLOOD PRESSURE** on arrival: _____/_____
- **THROMBOLYSIS INCLUSION CRITERIA MET:**

Age 18-85, Previously independent

Rapid-onset FOCAL neurological deficit - likely due to stroke

<4 hours from symptom onset

Note: >3h may be eligible for ***EPITHET*** trial of t-PA (using CT+MRI), or SAINT neuroprotectant trial, hence acute stroke call up to 4h from stroke onset.

EXCLUSION CRITERIA: proceed with acute stroke call if all 5 criteria are negative. If uncertain, CALL.

Yes	No	Comatose/severely obtunded with fixed eye deviation and complete hemiplegia
Yes	No	On warfarin with INR >1.3 [if on warfarin: **URGENT** INR required, arrange CT / stroke call while results pending]
Yes	No	SBP =185 or DBP =110
Yes	No	Recent stroke/head trauma [3months], major surgery [30d], GI/GU bleed [21d]
Yes	No	PHx intracranial haemorrhage, aneurysm, AVM

ACTIVATE ACUTE STROKE CALL [0600-2200hrs]

URGENT FBC & coag. sent. Finger-stick glucose:_____mmol/l. 18G cannula placed. ECG performed

Call **ACUTE NEUROLOGY REGISTRAR:**
- **working hours = pager 111** Time called ___:___
- **a/hours = via hospital operator**

Call CT : "ACUTE STROKE SCAN" Time CT requested ____:____

"Stroke pack" obtained from ED drug increment cupboard (**10mg vial t-PA**, 10ml water, syringe, needles) [Transport with patient to CT]

URGENT transport of patient to CT. Nurse escort required. Time transported to CT ____:____
Admit patient to **Team Neurology, Acute Stroke Unit, Ward 31** prior to departing ED.

CT SCANNING

Time of CT ____:____ Time CT read ____:____ Read by: rad reg / consultant

CT Result: Haemorrhage No Yes Hypodensity* No Yes

Mass effect No Yes (* less than white-matter density)

STROKE TEAM ASSESSMENT

Time of assessment ____:____ Initials:_____

Confirm clinical diagnosis of ischaemic stroke				**EPITHET candidate?**		
Onset <3h, Age 18-85y	No	Yes		Presumed septic embolus	No	Yes
CT exclusion	No	Yes		Stroke/head trauma < 3 months	No	Yes
SBP>185, DBP>110	No	Yes		Major surgery <30 days	No	Yes
(or aggressive antiHT Rx required)				Organ biopsy / MI <30 days	No	Yes
Trivial (non-disabling) deficits	No	Yes		GI or GU haemorrhage <21 days	No	Yes
Coma & complete hemiplegia	No	Yes		Non-compressible puncture <21d	No	Yes
Seizure at onset*	No	Yes		Pregnancy, parturition, lact <30 d	No	Yes
Presentation suggestive of SAH	No	Yes		PHx ICH, aneurysm, AVM	No	Yes
On warfarin:	No	Yes		PHx other serious neurol d.	No	Yes
- if yes, is INR >1.3? (=_____)	No	Yes	N/A	PHx bleeding diathesis	No	Yes
Glucose <2.8 or >22.0 (=_____)*	No	Yes		Other serious/terminal illness	No	Yes
Platelets <100 or HCT <0.25	No	Yes		RISKS/BENEFITS discussed		
*warnings, not absolute contraindications				& documented in chart	No	Yes

NEUROLOGICAL EXAMINATION AT ACUTE ASSESSMENT:

Level of consciousness:	Alert	Arousable	Hard to arouse	Coma
Orientation:	Full	Partial	Disoriented	
Obeys simple commands/gesture:	Yes	Partial	No	
Dysphasia:	No	Mild/Mod	Severe	Total
Dysarthria:	No	Mild/Mod	Severe	
Gaze palsy:	No	Partial	Total forced deviation	
Hemianopia:	No	Partial	Complete	Bilateral/blind
Facial weakness:	No	Minor	Marked lower	Complete upper & lower
Power [proximal]	**R arm**: ____/5		**R leg**: ____/5	
	L arm: ____/5		**L leg**: ____/5	
Incoordination (?weak)	No	one limb	two limbs	
Hemisensory deficit:	No	Mild/Mod	Severe	
Extinction / inattention:	No	extinction bilat simultaneous	profound inattention/neglect	

NIHSS Score

BLOOD PRESSURE: _____/_____ (<185/110) **Temperature**: _____ ^{o}C

Stroke Neurologist called Time ____:____ Time from stroke onset ____:____

PATIENT WEIGHT: _____kg

Total dose t-PA (0.9 mg/kg, max90mg):____mg Bolus 10% ____mg Infusion 90%____mg

IV t-PA bolus given Time ____:____ Time from stroke onset ____:____

IV t-PA infusion commenced Time ____:____

Acute Stroke Unit transfer Time ____:____ If not ASU, where?_____

ANY PREVENTABLE DELAYS? Details: _____

PROTOCOL VIOLATION? Details: _____

CONSENT FOR ACUTE STROKE TREATMENT WITH t-PA

Treatment Information

Your doctors have determined that you have had a stroke, and that you might benefit from treatment with t-PA (tissue plasminogen activator).

Your stroke has been caused by a blockage to a blood vessel in the brain. The blockage to the blood vessel is caused by a blood clot. t-PA is a drug which causes blood clots to dissolve [thrombolysis] and may help restore blood flow to your brain. The treatment is given intravenously (through a 'drip') over one hour. If it is to be used, this treatment must be given within 3 hours of the first sign of stroke.

Thrombolysis with t-PA under strictly controlled conditions is a recommended treatment for acute stroke according to the New Zealand Guideline Group New Zealand Stroke Guidelines (2003). This treatment is estimated to result in one more patient recovering full independence after a stroke for every 8 patients treated. Other patients may get smaller benefits. But thrombolysis with t-PA can also cause bleeding into the brain and worsening of the stroke. This occurs to around one in twenty patients treated with t-PA. On occasion, this bleeding may cause death.

Your consent for treatment with this medication is voluntary (your choice). If you do not wish to receive this treatment, you will receive full standard hospital treatment for stroke, which may include aspirin or other anticoagulants.

Consent for treatment with intravenous t-PA

I have read and understood the treatment information regarding the use of t-PA for acute stroke. I understand that t-PA does not benefit all patients with stroke and can cause worsening of stroke in some people.

I have had a chance to have my questions answered about this treatment.

I consent to treatment of myself / my relative with intravenous t-PA for stroke.

Signed: _____ Print: _____

Date: ____/____/____ Capacity: patient / next-of-kin / other (state): _____

Witness: _____ Print: _____

Date: ____/____/____

Verbal Consent Only: from:_____ reason:_____

Treatment without formal consent:

reason:_____

second witness or authorising consultant: _____ Date: ____/____/____

Drug administration

- Reconstitution: dilute with sterile water to 1mg/1ml
  - Swirl gently if necessary to mix, DO NOT SHAKE
  - Small amount of frothing is normal. Allow to settle.

- Dose: 0.9 mg/kg (max 90mg):
  - 10% total dose given as bolus over 1 minute
  - remaining 90% as infusion over 60 minutes: given undiluted via burette.
  - **Estimated** weight may be used if measured weight would result in significant delay in treatment

- No other anticoagulant or antiplatelet agents are to be given within 24 hrs of t-PA administration.

		Vol of 1mg/1ml t-PA					**Vol of 1mg/1ml t-PA**	
Patient Weight (kg)	Total dose (mg) [0.9mg/kg]	10% Bolus (ml)	90% 1hr infusion (ml)		**Patient Weight (kg)**	Total dose (mg) [0.9mg/kg]	10% Bolus (mL)	90% 1hr infusion (ml)
40	36	3.6	32.4		**70**	63	6.3	56.7
41	36.9	3.7	33.2		**71**	63.9	6.4	57.5
42	37.8	3.8	34.0		**72**	64.8	6.5	58.3
43	38.7	3.9	34.8		**73**	65.7	6.6	59.1
44	39.6	4.0	35.6		**74**	66.6	6.7	59.9
45	40.5	4.1	36.4		**75**	67.5	6.8	60.7
46	41.4	4.1	37.3		**76**	68.4	6.8	61.6
47	42.3	4.2	38.1		**77**	69.3	6.9	62.4
48	43.2	4.3	38.9		**78**	70.2	7.0	63.2
49	44.1	4.4	39.7		**79**	71.1	7.1	64.0
50	45.0	4.5	40.5		**80**	72.0	7.2	64.8
51	45.9	4.6	41.3		**81**	72.9	7.3	65.6
52	46.8	4.7	42.1		**82**	73.8	7.4	66.4
53	47.7	4.8	42.9		**83**	74.7	7.5	67.2
54	48.6	4.9	43.7		**84**	75.6	7.6	68.0
55	49.5	5.0	44.5		**85**	76.5	7.7	68.8
56	50.4	5.0	45.4		**86**	77.4	7.7	69.7
57	51.3	5.1	46.2		**87**	78.3	7.8	70.5
58	52.2	5.2	47.0		**88**	79.2	7.9	71.3
59	53.1	5.3	47.8		**89**	80.1	8.0	72.1
60	54.0	5.4	48.6		**90**	81.0	8.1	72.9
61	54.9	5.5	49.4		**91**	81.9	8.2	73.7
62	55.8	5.6	50.2		**92**	82.8	8.3	74.5
63	56.7	5.7	51.0		**93**	83.7	8.4	75.2
64	57.6	5.8	51.8		**94**	84.6	8.5	76.1
65	58.5	5.9	52.6		**95**	85.5	8.6	76.9
66	59.4	5.9	53.5		**96**	86.4	8.6	77.8
67	60.3	6.0	54.3		**97**	87.3	8.7	78.6
68	61.2	6.1	55.1		**98**	88.2	8.8	79.4
69	62.1	6.2	56.0		**99**	89.1	8.9	80.2
					100kg	90.0	9.0	81.0