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GUIDELINES

Rehabilitation of patients with stroke: summary of SIGN guidance

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Stroke is the third most common cause of death and the most frequent cause of severe adult disability in Scotland[1] Despite considerable advances in organised stroke care over recent years, improvements are still needed,[2] because patients have been reported to spend up to 50% of their time in bed[3] and only 20% of their time in treatment.[4] Since publication of the previous Scottish Intercollegiate Guidelines Network (SIGN) guideline on rehabilitation after stroke (SIGN 64), several small studies have shown the effectiveness of new therapeutic techniques and technologies.[5] This guideline supersedes the earlier guideline and summarises the most recent recommendations from SIGN on rehabilitation after stroke.[6] It also complements SIGN guidelines 119 and 108 on other aspects of the management of stroke.[7][8]

Recommendations

SIGN recommendations are based on systematic reviews of best available evidence. The strength of the evidence is graded as A, B, C, or D (figure), but the grading does not reflect the clinical importance of the recommendations. Recommended best practice ("good practice points"), based on the clinical experience of the guideline development group, is also indicated (as GPP).

Arranging appropriate care

- Admit stroke patients who require admission to hospital to a stroke unit staffed by a coordinated multidisciplinary team with a special interest in stroke care (A).
- In exceptional circumstances, when admission to a stroke unit is not possible, provide rehabilitation in a generic rehabilitation ward on an individual basis (B).
- The core multidisciplinary team should include appropriate levels of nursing, medical, physiotherapy, occupational therapy, speech and language therapy, and social work staff (B).
- Stroke inpatients should be treated 24 hours a day by nurses who specialise in stroke and are based in a stroke unit (B).
- Actively involve patients and carers early in the rehabilitation process (A) and routinely provide them with information using active information strategies, which include a mixture of education and counselling techniques (A).

Mobility and activities of daily living

- Mobilise patients as early as possible after stroke (B).
- Physiotherapists should not limit their practice to one "approach" but should select interventions according to the patient's individual needs (B).
- Where the aim of treatment is the immediate improvement of walking speed, walking efficiency, gait pattern, or weight bearing during stance, an appropriately qualified health professional should assess the patient's suitability for ankle foot orthoses (A).
- Consider treadmill training to improve gait speed in people who are walking independently at the start of treatment (B).
- When the goal of treatment is to improve functional ambulation, offer gait oriented physical fitness training to all patients assessed as medically stable and functionally safe to participate (A).
- When the aim of treatment is to improve gait speed, walking distance, functional ambulation, or "sit to stand to sit," include repetitive task training, where it is assessed to be safe and acceptable to the patient (B).
- Where considered safe, pursue every opportunity to increase the intensity of treatment for improving gait (B).
- Occupational therapists should include training in personal activities of daily living as part of an inpatient stroke rehabilitation programme (B).
- Splinting is not recommended for improving upper limb function because it does not prevent the development of contractures or improve muscle extensibility (B).

Assessment of nutritional status and continence

- Include ongoing monitoring of nutritional status with the following parameters (D):
  - Swallowing status
  - Nutritional intake
  - Feeding assessment and dependence
  - Unintentional weight loss
  - Biochemical measures (low prealbumin, impaired glucose metabolism).
- Every service caring for patients with stroke should develop and adhere to local urinary and faecal continence guidelines, including advice on appropriate referral (GPP).

The grade of recommendation relates to the strength of the supporting evidence on which the evidence is based. It does not reflect the clinical importance of the recommendation

A
- At least one high quality meta-analysis, systematic review of randomised controlled trials, or randomised controlled trial with a very low risk of bias and directly applicable to the target population; or
- A body of evidence consisting principally of well conducted meta-analyses, systematic reviews of randomised controlled trials, or randomised controlled trials with a low risk of bias directly applicable to the target population, and demonstrating overall consistency of results

B
- A body of evidence including studies rated as high quality systematic reviews of case-control or cohort studies, and high quality case-control or cohort studies with a very low risk of confounding or bias and a high probability that the relation is causal and that are directly applicable to the target population, and with overall consistency of results; or
- Extrapolated evidence from studies described in A

C
- A body of evidence including well conducted case-control or cohort studies with a low risk of confounding or bias and a moderate probability that the relation is causal and that are directly applicable to the target population and with overall consistency of results; or
- Extrapolated evidence from studies described in B

D
- Non-analytic studies, such as case reports, case series, expert opinion; or
- Extrapolated evidence from studies described in C

Good Practice Points (GPP)

☑ Recommended best practice based on the clinical experience of the guideline development group

Explanation of SIGN grades of recommendations

Cognitive and emotional assessment
- Fully assess for cognitive strengths and weaknesses when undergoing rehabilitation or when returning to cognitively demanding activities such as driving or work (GPP).
- Occupational therapists with expertise in neurological care may carry out cognitive assessment, although some patients with more complex needs should be referred for specialist neuropsychological expertise (GPP).
- Screen all stroke patients for visual problems and refer appropriately (C).
- Consider appropriate referral to health and clinical psychology services for patients and carers to promote good recovery and adaptation and to prevent and treat abnormal adaptation to the consequences of stroke (GPP).
- Screen all stroke patients for mood disturbance as early as possible using a validated tool, such as the stroke aphasic depression questionnaire (SAD-Q)[9] or general health questionnaire of 12 items (GHQ-12)[10] (GGP).
- Screen patients with post-stroke fatigue for depression (GPP).
- Consider patients with post-stroke depression for treatment with antidepressants (A).[11]
- Choose the appropriate antidepressant on an individual basis (GPP).

Pain assessment
- Ask patients about pain and assess the severity of pain (using a validated pain assessment tool such as the visual analogue scale or numerical rating scale)[12] and treat appropriately as soon as possible (GPP).
- In patients with central post-stroke pain that does not respond to standard treatment, and where clinician and patient are aware of potential side effects, consider the use of amitriptyline (titrated to a dose of 75 mg). If amitriptyline is ineffective or contraindicated, lamotrigine or carbamazepine are alternatives, although they have a high incidence of side effects (B).[13]
- Given the complexity of post-stroke shoulder pain, consider the use of algorithms or an integrated care pathway for its diagnosis and management (GPP).

Shoulder subluxation
- Consider electrical stimulation to the supraspinatus and deltoid as soon as possible after stroke in patients at risk of developing shoulder subluxation from having little or no activity in shoulder muscles (A).

Transfer from hospital to home
- Patients with mild to moderate stroke should be able to access stroke specialist early supported discharge services in addition to conventional organised stroke inpatient services (A).
- Health boards should consider providing a specific local expert therapist to advise rehabilitation teams on subjects such as providing information on relevant statutory services such as disability employment advisers at job centres, organisations that provide opportunities for people with disabilities (such as Momentum), or voluntary services that can provide help and support (such as Chest Heart and Stroke Scotland (CHSS) and the Stroke Association (GPP)).

Living in the community
- Ask patients about vocational activities, initiate liaison with employers early in the rehabilitation pathway, and assess the patient's ability to meet the needs of current or potential employment (GPP).
- Advise patients that they must not drive for at least one month after their stroke (GPP).
- If there is doubt about a patient's ability to drive, refer the patient to the local Disabled Drivers' Assessment Centre (www.dft.gov.uk/dvla) (D).

Overcoming barriers
Specialist rehabilitation is central to successful recovery after stroke and specific standards and targets to drive improvements must be implemented. The evidence base for rehabilitation interventions is expanding, and it challenges traditional patterns of care in many areas. Stroke rehabilitation teams need to adopt these evidence based changes and translate them into routine clinical practice. Patients should receive treatment seven days a week, and increasing the use of generic therapy assistants across rehabilitation disciplines may help achieve this. Moving rehabilitation from hospital to home at an earlier stage improves outcomes but will need further development

of early supported discharge teams with specialist stroke skills.[14] Recovery often continues at a slower rate for many years after stroke. It is important to maintain access to rehabilitation services throughout this time, and the use of local leisure centres to facilitate ongoing exercise programmes should be encouraged.[15] Finally, despite the best medical care, around 20% of stroke patients die within the first 30 days.[16] The provision of high quality palliative care by stroke teams to these patients and their families is an area that requires further development.

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LESSON OF THE WEEK
Delayed diagnosis of primary hyperaldosteronism

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The effects of dihydropyridine calcium antagonists may delay the diagnosis of primary hyperaldosteronism

About 5-13% of patients with hypertension have primary hyperaldosteronism,[1-4] defined as autonomous production of aldosterone, which leads to excessive retention of sodium plus water and resistant hypertension. It is important to identify such patients because they have a higher morbidity and mortality from cardiovascular disease than age and sex matched patients with essential hypertension.[2] In addition, surgery can significantly improve long term outcomes in such patients.[5]

With improved management of hypertension and greater awareness of primary hyperaldosteronism, more investigations are being performed in patients taking antihypertensive agents. Dihydropyridine calcium antagonists increase renin production and reduce aldosterone, thereby reducing the aldosterone:renin ratio—the initial test for primary hyperaldosteronism.[6] However, these effects are underappreciated,[7] with recent publications reporting insignificant effects on the aldosterone:renin ratio.[8-12] We describe three patients who were initially misclassified as having essential hypertension because of repeatedly unremarkable aldosterone:renin ratios while taking dihydropyridine calcium antagonists.

Case reports

Between 2003 and 2008, three patients were diagnosed with biochemically confirmed primary hyperaldosteronism caused by autonomous aldosterone producing adenomata. All three had initially been misclassified because of repeatedly unremarkable aldosterone:renin ratios while taking dihydropyridine calcium antagonists. They had otherwise been appropriately investigated—other interfering antihypertensive drugs had been withdrawn and plasma potassium had been carefully corrected. Diagnosis was finally confirmed by repeatedly raised ambulatory aldosterone:renin ratios after calcium antagonist withdrawal. All three patients underwent adrenal vein sampling, which confirmed lateralisation of aldosterone secretion, and two patients proceeded to laparoscopic adrenalectomy.

Case 1

A 56 year old man presented with a 15 year history of resistant hypertension; he had initially been treated with amlodipine 10 mg daily with subsequent addition of lisinopril 10 mg

Table 1 | Concentrations of aldosterone and renin and aldosterone:renin ratios before, 2 weeks, and 4 weeks after patients stopped taking dihydropyridine calcium antagonists

Scenario	Renin (mU/l)*	Aldosterone (nmol/l)†	Ratio‡
Case 1			
On calcium antagonist	23.7	0.310	0.013
2 weeks after stopping	1.4	0.129	0.092
4 weeks after stopping	3	0.627	0.209
Postoperatively	23.7	0.310	0.013
	7.2	0.110	0.015
Case 2			
On calcium antagonist	10.7	0.497	0.046
2 weeks after stopping	7.4	0.547	0.074
4 weeks after stopping	3	0.643	0.214
Case 3			
On calcium antagonist	12.2	0.333	0.027
2 weeks after stopping	8.5	0.642	0.076
4 weeks after stopping	7.9	1.051	0.133
Postoperatively	17	0.126	0.007

*Renin ambulatory reference interval 7 to 50 mU/l; 1 pg/ml=1.67 mIU/l.
†Aldosterone ambulatory reference interval 0.08 to 0.80 nmol/l.
‡Reference interval <0.05.

daily. He developed severe hypokalaemia, requiring up to 4.8 g of oral potassium daily to maintain normokalaemia. Spot urinary potassium was 44 mmol/l after withholding supplements for 24 hours, indicating severe, inappropriate urinary potassium wasting. Multiple aldosterone:renin ratios obtained while taking the above antihypertensive agents were interpreted as inconsistent with primary hyperaldosteronism. Lisinopril was stopped because of side effects. After stopping amlodipine and starting prazosin 1 mg twice daily, aldosterone:renin ratios were repeatedly above the limit for a diagnosis of primary hyperaldosteronism (table 1). Lateralisation of aldosterone production to the right adrenal gland was confirmed by adrenal vein sampling (table 2), which showed a raised unilateral aldosterone:cortisol ratio and suppressed contralateral ratio.[13] Elective right adrenalectomy was performed and histology showed multiple adrenocortical micronodules. Postoperatively, his aldosterone:renin ratio normalised, hypokalaemia resolved without potassium supplements, and blood pressure improved.

Case 2

A 58 year old woman developed hypertension in her mid-30s, after having intermittent episodes of mild hypokalae-

Table 2 | Results of adrenal venous sampling

			Adrenal vein sampling		
Case	Adenoma size (cm)	Sample site*	Adrenal aldosterone (nmol/l)	Adrenal cortisol (nmol/l)	Adrenal aldosterone:cortisol ratio
1	Right multiple adrenocortical micronodules†	RAV	213	14 900	0.014
		LAV	3.46	4230	0.0008
		IVC	1.46	670	0.0022
2	Right 9mm nodule‡	RAV	10.7	1860	0.0057
		LAV	1.23	884	0.0014
		IVC	0.478	104	0.0046
3	Right 24 mm nodule‡	RAV	8.43	610	0.0138
		LAV	0.967	1900	0.0005
		IVC	0.414	630	0.0007

*IVC=inferior vena cava, LAV=left adrenal vein, RAV=right adrenal vein.
†On histology.
‡On computed tomography.

mia requiring transient administration of oral potassium. She had been started on amlodipine 5 mg daily. She had also been surgically treated for primary hyperparathyroidism after resection of a solitary chief cell adenoma in 2004. Aldosterone:renin ratios measured over 10 years were all below the diagnostic limit for primary hyperaldosteronism, but after amlodipine was withdrawn and prazosin started at 1 mg twice daily, ratios increased above this limit (table 1). Lateralisation of aldosterone secretion to the right adrenal gland was confirmed by adrenal vein sampling (table 2) and computed tomography of the abdomen showed a 9 mm dominant right adrenal nodule. After careful consideration of the options, the patient declined surgery. She was restarted on amlodipine 5 mg daily and advised on lifestyle modification. Recent blood pressure measurement was 125/80 mm Hg on treatment.

Case 3

A 44 year old woman developed hypertension 10 months before referral, with episodes of severe recurrent hypokalaemia needing large oral doses, up to 6.6 g, of potassium; she was treated with felodipine 15 mg daily. She had no relevant medical history. Multiple aldosterone:renin ratios had been performed over the preceding 10 months, with all results below the decision limit for primary hyperaldosteronism. After felodipine was withdrawn and prazosin started at 1 mg twice daily, aldosterone:renin ratios increased above the limit consistent with primary hyperaldosteronism (table 1). Lateralisation of aldosterone secretion to the right adrenal gland was confirmed by adrenal vein sampling (table 2), and computed tomography of the abdomen showed a 2.5 cm right adrenal nodule. Elective laparoscopic right adrenalectomy and histology confirmed a solitary adenoma. She remains normotensive and normokalaemic without the need for antihypertensive drugs or potassium supplements.

Discussion

All three patients with biochemically proven and adrenal venous sampling confirmed unilateral aldosterone production were initially diagnosed with essential hypertension. After withdrawal of dihydropyridine calcium antagonists and repeated measurement of the aldosterone:renin ratio over four weeks, the diagnosis of primary hyperaldosteronism was confirmed. Current recommended first line drugs for essential hypertension include calcium antagonists, singly or in combination.[5 14 15] Around 17% of treated patients with hypertension receive a calcium antagonist,[7] so patients may often be investigated for secondary hypertension while taking dihydropyridine calcium antagonists.

Ambulatory assessment of the aldosterone:renin ratio is the most reliable and accessible way to screen for primary hyperaldosteronism. Many studies have shown that this ratio is superior to isolated potassium or aldosterone measurements (both have lower sensitivity) or a single renin measurement (which is less specific).[16] It is prudent to take note of individual renin and aldosterone results, however, because a low or suppressed renin may be caused by primary hyperaldosteronism or low renin hypertension even when the aldosterone:renin ratio is unremarkable. But in our three patients, renin was not suppressed initially, and individual aldosterone and renin values were within appropriate

ambulatory reference intervals. Two of the three patients had severe, inappropriate renal potassium wasting, however, so in the setting of hypertension the index of suspicion for primary hyperaldosteronism was high. The other patient was normokalaemic, but a revised diagnosis was pursued because of coexistant endocrinopathy. Other patients who are normokalaemic, with no other pre-existing endocrinopathies, and who are taking dihydropyridine calcium antagonists could consequently be misdiagnosed.

Dihydropyridine calcium antagonists alter the aldosterone:renin ratio by competitively blocking the L type calcium channel, inhibiting calcium influx, and lowering aldosterone secretion.[17 18] Aldosterone suppression results in higher plasma renin concentrations, compounding the effect of these antagonists on interpretation of the aldosterone:renin ratio. Within two weeks of withdrawal of calcium channel antagonists, serum aldosterone increased, plasma renin decreased, and the aldosterone:renin ratio became diagnostic of primary hyperaldosteronism in all three cases.

It is recommended that confounding drugs are withdrawn for one to two weeks before testing.[8 16 19] Although the aldosterone:renin ratio continued to rise after two weeks, all our patients had a positive diagnostic ratio within two weeks of stopping treatment.

Adrenal vein sampling was performed in all three cases after carefully counselling the patients about the risks and limitations of the procedure and ascertaining their willingness to have surgery in the event of lateralised aldosterone production. The current treatment for a lateralising single aldosterone producing tumour is laparoscopic adrenalectomy, which is considered curative. Bilateral aldosterone secretion can be successfully treated by the addition of aldosterone blocking agents, such as spironolactone.[10 20]

We describe a series of patients with unilateral, adrenal venous sampling confirmed, primary hyperaldosteronism, whose diagnosis was delayed by the affects of dihydropyridine calcium antagonists on initial aldosterone:renin ratio assessments. Doctors should be aware of this phenomenon and consider repeat assessment after drug withdrawal even in patients who are normokalaemic at presentation.

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I can't really talk right now

One may remember a time when mobile phones were not ubiquitous, and the only people who felt it necessary to use them regularly were stockbrokers and drug dealers. With rose tinted spectacles, we recall a time when you would call an acquaintance and if he or she did not answer you would leave a message or call back later. It seems that the ubiquity of the mobile phone has been accompanied by an irrepressible need to be contactable at all times.

While we were operating on a man under a regional block to his arm a mobile phone started ringing in the operating theatre. To our astonishment, the patient retrieved his mobile phone from his undergarments and, while brandishing it with his mobile hand, asked whether he would be able to take the call.

We wonder what other essential or important activities the modern man or woman would be willing to interrupt to take a call on their mobile phone? A funeral, their own wedding, child birth?

"Sorry darling, can't talk right now I'm just burying my father/tying the knot/trying to deliver a placenta."

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