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Original article

Decrease in post-stroke spasticity and shoulder pain prevalence over the last 15 years

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ABSTRACT

Objectives: The usual complications after recent stroke such as disabling spasticity and shoulder pain seemed less frequent in recent years. This study examined the frequency of spasticity and shoulder pain in recent post-stroke patients over time in our physical and rehabilitation medicine department.
Methods: This was a retrospective study of post-stroke inpatients over the last 15 years. Spasticity and shoulder pain prevalence were analyzed, as were demographic, clinical and stroke characteristics.
Results: We reviewed medical records for 786 patients (506 men); mean age 58.1 years (SD 13.2); 530 (68%) with ischemic stroke and 256 (32.36%) hemorrhagic stroke. After a first increase from 2000 to 2006, the prevalence of disabling spasticity decreased from 2006 to 2015 (31%–10%; $P < 0.001$). Shoulder pain at admission and during hospitalization also decreased (13% of patients in 2000 to 8% in 2015, $P < 0.001$). Disabling spasticity was associated with shoulder pain (26% of patients with disabling spasticity presented shoulder pain at admission vs 7% with hyperreflexia of the deep tendon reflexes, $P < 0.05$). Characteristics of stroke, time of admission after stroke and length of stay did not change over the years. We observed an increase in number of walking patients at admission and number with a functional paretic arm at admission and discharge ($P < 0.05$), which may explain the increase in functional independence measure scores at admission and discharge (both $P < 0.05$). Prevalence of cognitive disorders increased over the same period (24% in 2000 vs 63% in 2015, $P < 0.05$).
Conclusions: Disabling spasticity and shoulder pain frequency in recent post-stroke patients decreased over the last 15 years, and functional abilities both at admission and discharge improved. Confirmation of these results in a multicentric study may be important evidence of an improvement in stroke healthcare both in stroke and physical and rehabilitation medicine units in the last 10 years in France and could affect future estimations of the need for rehabilitation care after stroke.

1. Introduction

In France, stroke is the third leading cause of death and the foremost cause of severe acquired disability in adults (defined by the need for a caregiver for some daily activity) [1]. Healthcare provisions for stroke patients in France have increased considerably over the last 20 years: increased number of stroke units, new therapies such as thrombolysis and thrombectomy and the development of neurological specialized physical and rehabilitation medicine (PRM) departments.

Spasticity is a frequent consequence of brain lesions, with usually a negative impact on motor control. The main negative consequences are the increased risk of muscle contractures, inhibition of antagonistic control and co-contractions during voluntary movement. Spasticity can be (1) limited to hyperreflexia of the deep tendon reflexes; (2) marked by a stretch reflex during passive mobilization without a negative impact on passive or active range of motion (ROM) or (3) marked with a negative impact on passive ROM with hygienic or pain consequences and/or on active ROM, such as gait and spastic foot or stiff knee gait. In these last cases, spasticity is then considered disabling, and treatment can be expected to have a positive effect. The prevalence of post-stroke spasticity is variably reported, from 4% to 42%, and the prevalence of disabling spasticity ranges from 2% to 13% [2].

The clinical status of recent post-stroke patients seems to have changed over the past years, especially regarding spasticity and shoulder pain, which does not seem to require treatment as often.

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This study aimed to investigate whether spasticity and shoulder pain have indeed decreased over the last 15 years.

2. Materials and methods

2.1. Study population

Because this was an anonymous retrospective study, a statement from the Commission Nationale de l'informatique et des Libertés was obtained (no. 2040356). We included patients directly admitted in our PRM department from the acute care unit after a recent stroke, whatever the type, number, or localization of the stroke. Patients with other associated neurological disorders were excluded. We analyzed all hospitalization reports when patients left the department; hence, patients for 2002 were discharged in 2002, for example.

2.2. Design

We conducted a retrospective observational monocentric study (PRM unit, Lariboisière-Fernand Widal Hospital) by analysing medical records for patients included over 9 years, between 2000 and 2015. Evaluations were conducted every 2 years except for 2015 because of an in-progress recording in 2016 (2000, 2002, 2004, 2006, 2008, 2010, 2012, 2014 and 2015). We collected the following data: demographic characteristics (age, sex), hospitalization characteristics (initial unit of treatment [Lariboisière stroke unit, other stroke unit, other medical unit (mainly neurosurgery)]), time of admission after stroke and length of stay in our PRM department, discharge location (home or institution), stroke characteristics (ischemic or hemorrhagic, localization [right or left or bilateral hemispheric or posterior: cerebellar and brainstem stroke], unique or multiple, first stroke or recurrence), and specific treatment (thrombolysis, thrombectomy, craniotomy). Clinical characteristics were collected as functional independence in daily activities measured by the functional independence measure (FIM) (systematically used for all our patients at least at department entry and at discharge), walking ability (defined by a functional ambulation category [FAC] $\geq$level 4), functional paretic arm (defined as the ability of patients to use their hand for some daily activities), sensitivity (classified as normal, abnormal but preserved proprioception and altered proprioception), dysexecutive syndrome (when 2 symptoms were noted at least in planning, reasoning, inhibiting, or behavioral disorders), aphasia, gastrostomy, and tracheotomy. Spasticity was classified into 3 categories: (1) spasticity limited to hyperreflexia of the deep tendon reflexes; (2) marked spasticity, with a stretch reflex without any impact on passive or active ROM; and (3) disabling spasticity defined by marked spasticity with a negative impact on passive ROM resulting in hygienic or pain problems and/or on active ROM, such as gait and spastic foot or stiff knee gait (in these cases, treatment is required). Shoulder pain at admission and during hospitalization (defined by a required treatment, pharmacologic or physiotherapy) were specified.

These data are almost systematically present in our medical records. Several scales were used over time to assess spasticity, pain, or cognitive function. To homogenize the patient data and allow for comparison, most of the criteria were simplified by their presence or not.

3. Statistical analysis

Data are expressed as median (interquartile range [IQR] = Q3–Q1) or mean (SD). Frequencies are expressed as number (%). Time-series graphs were created to identify the time evolution of each variable. When a general pattern (increase or decrease over the years) was identified, chi-square test was used to analyze the evolution over time for categorical data and ANOVA for quantitative data. Associations between characteristics were analyzed by chi-square test. A secondary analysis was performed to compare patients with and without dysexecutive function. $P < 0.05$ was considered statistically significant. Statistical analysis involved use of R software Ri 386 3.2.5, R commander (R Development Core Team, http://www.R-project.org).

4. Results

We reviewed records for 786 patients (506 men) [65%]; mean age 58.1 years (SD 13.2) (IQR 17–93): 530 (68%) had ischemic stroke and 256 (32%) hemorrhagic stroke (Table 1). The frequency of thrombolysis and thrombectomy increased over the years ($P < 0.05$), with no change in hospitalization or stroke characteristics, or discharge location (Table 1).

The frequency of disabling spasticity significantly decreased over the years ($P < 0.05$). After a first increase until 2006 (18%–31% from 2000 to 2006), we observed a decrease, to 10%, in 2015 ($P < 0.05$) (Table 2; Fig. 1). Conversely, the frequency of hyperreflexia of the deep tendon reflexes increased from 2000 to 2015: 52%–80% ($P < 0.05$).

From 2000 to 2015, the frequency of shoulder pain significantly decreased, both at admission ($P < 0.05$) and during hospitalization ($P < 0.05$) (Fig. 2). Shoulder pain at admission concerned 13% of patients in 2000 and 8% in 2015 and during hospitalization concerned 29% in 2000 and 6% in 2015. Also, shoulder pain at admission first increased until 2006 (13%–30% in 2006), then decreased thereafter. Shoulder pain at admission and during hospitalization was more frequent in non-walking patients, those with altered proprioception and those with a non-functional upper limb ($P < 0.05$).

Disabling spasticity was associated with shoulder pain ($P < 0.05$): at admission, shoulder pain was present in 26% of patients with disabling spasticity versus 7% with hyperreflexia of the deep tendon reflexes; during hospitalization, shoulder pain was present for 37% of patients with disabling spasticity versus 9% with hyperreflexia of the deep tendon reflexes.

Disabling spasticity was frequently associated with a non-functional upper limb ($P < 0.05$): only 5% of patients with disabling spasticity had a functional upper limb versus 65% of patients with hyperreflexia of the deep tendon reflexes. It was frequently associated with a sensitivity disorder ($P < 0.05$): 65% of patients with disabling spasticity had altered proprioception versus 34% with hyperreflexia of the deep tendon reflexes.

Disabling spasticity was also associated with longer time to admission after stroke: (mean 26.6 days [SD 26.4] for hyperreflexia of the deep tendon reflexes, 31.7 days [SD 25.2] for marked spasticity and 48.7 days [SD 47.2] for disabling spasticity [$P < 0.05$]) as well as length of time spent in the PRM unit (mean 50.6 days [SD 59.6], 93.7 days [SD 93.9] and 169 days [SD 201], respectively [$P < 0.05$]).

We found no association between spasticity and revascularisation: 14% and 18% of patients with hyperreflexia of the deep tendon reflexes and disabling spasticity, respectively, underwent thrombolysis ($P = 0.06$), and 6% and 1% of patients with deep tendon reflexes and disabling spasticity underwent thrombectomy ($P = 0.19$).

We observed an increase over time in number of walking patients at admission and number of patients with a functional paretic arm at admission and discharge ($P < 0.05$) which may explain the increase in FIM scores at admission and discharge (both $P < 0.05$). We also observed an increase over time in number of cognitive disorders ($P < 0.05$). We noted no evolution in frequency of aphasia, sensitivity, gastrostomy, or tracheotomy (Table 2).

Table 1
Demographic, hospitalization, stroke and treatment characteristics of post-stroke patients hospitalized in a physical and rehabilitation medicine department, 2000–2015.

Characteristics		2000	2002	2004	2006	2008	2010	2012	2014	2015	Total	P
Demographics	No. of patients	97	80	61	64	99	70	107	94	114	786	
	Male (%)	65	61	57	63	65	66	68	67	64	65	–
	Age (years), mean (SD)	58.6 (12.1)	58.1 (12.9)	56.7 (16.4)	58.2 (13.7)	57.9 (12.6)	56.1 (17.2)	56.3 (12.3)	58.4 (12.5)	60.7 (11)	58.1 (13.2)	–
Hospitalization	Time of admission (days), median (IQR)	22 (21)	22 (19.25)	20 (24)	25.5 (27.5)	17 (17)	21 (26.75)	21 (23)	19.5 (19)	16 (16.75)	20.44 (23)	–
	Length of stay (days), median (IQR)	44 (77)	51.5 (78.75)	61 (95)	62 (96.75)	40 (61.5)	53 (86)	53 (82.5)	30 (64.5)	30.5 (53.25)	47.22 (77)	–
	Lariboisière stroke unit (%)	72	78	79	59	80	63	61	72	69	70	–
	Other stroke unit (%)	5	1	5	8	6	7	16	8	20	8	–
	Other medical unit (%)	23	21	16	33	14	30	23	20	11	22	–
Way of discharge	Home (%)	45	44	29	20	28	34	37	37	40	35	–
	Daily hospitalization (%)	32	30	49	44	44	30	37	49	53	41	–
	Institution (%)	23	25	22	36	28	36	26	14	7	24	–
	Death, no.	0	1	1	0	1	0	2	0	0	5	–
Stroke	Ischemic (%)	74	65	77	72	61	60	66	62	72	68	–
	Hemorrhagic (%)	26	35	23	28	39	40	34	38	28	32	–
	Right hemisphere (%)	37	42	32	48	35	33	33	37	27	36	–
	Left hemisphere (%)	46	44	52	39	46	46	47	48	38	45	–
	Posterior (%)	16	13	13	9	14	13	13	5	18	13	–
	Bilateral (%)	1	1	3	3	5	8	7	10	17	6	–
	Unique (%)	80	96	69	84	77	70	83	76	76	79	–
	Multiple (%)	20	4	31	16	23	30	17	24	24	21	–
	First (%)	81	88	89	89	90	90	85	84	87	87	–
	Recurrence (%)	19	12	11	11	10	10	15	16	13	13	–
Therapeutic	Thrombolysis (%)	0	3	2	6	5	11	12	16	15	8	*
	Thrombectomy (%)	0	0	0	2	1	4	6	5	10	3	*
	Craniotomy (%)	11	1	2	6	7	11	2	9	2	6	–

IQR, interquartile range (Q3–Q1).
* Significant ($P < 0.05$).

Table 2
Clinical characteristics of post-stroke patients hospitalized in a physical and rehabilitation medicine department, 2000–2015.

Clinical characteristics		2000	2002	2004	2006	2008	2010	2012	2014	2015	Total	P
Autonomy	Entry FIM/126, median (IQR)	76 (46.5)	76 (44.5)	75.5 (41)	65 (52)	76.5 (53.75)	76 (42)	84 (61.5)	93.5 (53.5)	96 (50.5)	81 (49)	*
	Leaving FIM/126, median (IQR)	110 (22)	108 (18.75)	109 (20)	105 (23)	112 (29)	110 (22)	116 (21)	119 (15.25)	117 (15.5)	112 (22)	*
Walking ability	Entry (%)	25	18	25	14	24	31	45	50	36	30	*
	Leaving (%)	86	80	84	73	74	69	91	87	89	81	–
Functional paretic arm	Entry (%)	36	38	34	31	45	54	45	59	60	45	*
	Leaving (%)	47	50	39	56	65	70	70	70	80	61	*
Spasticity	Hyperreflexia of the deep tendon reflexes (%)	52	48	49	42	60	70	78	71	80	61	*
	Marked spasticity (%)	30	31	26	27	22	19	16	16	10	22	*
	Disabling spasticity (%)	18	21	25	31	18	11	6	13	10	17	*
Shoulder pain	At admission (%)	13	13	18	30	18	4	10	7	8	14	*
	During hospitalization (%)	29	25	28	19	21	11	13	13	6	18	*
Sensitivity	Normal (%)	38	36	36	31	53	63	48	36	39	42	–
	Abnormal but preserved proprioception (%)	18	16	21	16	15	11	19	12	16	16	–
	Altered proprioception (%)	44	48	43	53	32	26	33	52	45	42	–
Aphasia	(%)	31	29	36	27	31	30	33	34	27	31	–
Dysexecutive syndrome	(%)	24	18	36	28	44	49	62	64	63	43	*
Tracheotomy	(%)	2	0	2	2	1	1	1	0	1	1	–
Gastrostomy	(%)	2	1	5	2	5	9	3	2	4	4	–

IQR: interquartile range (Q3–Q1); FIM: functional independence measure.
* Significant ($P < 0.05$).

We performed a specific analysis to check for absence of bias due to a possible increase in number of patients with only post-stroke cognitive disorders without motor deficit but found no difference between patients with or without dysexecutive disorders. The intensity of spasticity was not associated with presence or absence of dysexecutive syndrome ($P = 0.8$). Hence, dysexecutive patients also had physical disability. We observed the same evolution as for the first analysis ($P < 0.05$): decrease in disabling spasticity ($P < 0.05$) (22% and 13% in 2000 vs 2015), decrease in shoulder pain at department entry (17% and 7% in 2000 vs 2015), and decrease in shoulder pain during hospitalization (30% and 6% in 2000 vs 2015).

5. Discussion

After a first increase until 2006 in frequency of disabling spasticity among recent post-stroke patients in our PRM department, the frequency decreased progressively over the last 10 years.

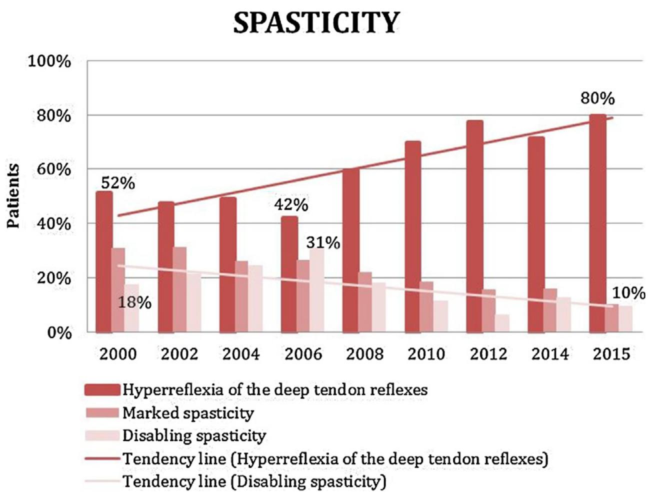

Fig. 1. Evolution of spasticity in post-stroke patients hospitalized in a physical and rehabilitation medicine department, 2000–2015.

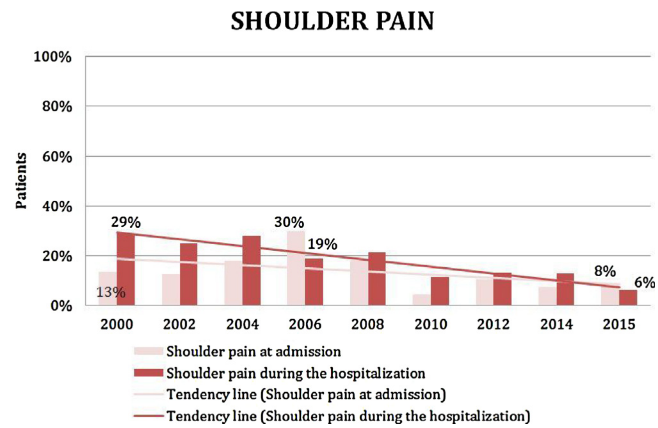

Fig. 2. Evolution of shoulder pain in post-stroke patients at entry and during hospitalization, 2000–2015.

To our knowledge, this is the first study suggesting that post-stroke spasticity could have changed over time, becoming less of a problem. At the same time, we observed a decreased frequency in shoulder pain.

The frequency of spasticity, and especially disabling spasticity, is difficult to estimate because of the different definitions in the literature. In our study, spasticity was assessed at admission (median time of admission after stroke 20.4 days [IQR 23; min 16.8; max 27.5]) but also during hospitalization (median hospitalization time in the PRM unit 47.22 days [IQR 77; min 53.3; max 96.8]). We did not distinguish spasticity at admission and during hospitalization. In total, 39% of all our patients had marked spasticity for the first 2 or 3 months after the stroke including 17% with disabling spasticity, which confirmed recent studies [2,3]. According to a systematic review including 7 studies published between 2002 and 2010, the reported prevalence of post-stroke spasticity ranged from 4% to 27% during the first 6 weeks after stroke, reached 19% at 3 months, and was from 21.7% to 42.6% at 4 to 6 months post-stroke and from 17% to 38% at 12 months. Severe or disabling spasticity ranged from 9.6% to 15.6% in the first 6 months. [4]. In a study of 140 patients at 1 year after a first stroke, Lundström et al. [5] estimated the prevalence of disabling spasticity, defined as spasticity having such an impact that patients should be offered an intervention, such as intensive physiotherapy, orthoses or pharmacological treatment. The prevalence of any spasticity (Modified Ashworth Scale score $\geq$1) was 17% and disabling spasticity 4%.

The negative impact of spasticity on the health-related quality of life of stroke survivors was reported in a longitudinal cohort study [6]. Three months after stroke, only 16% of stroke survivors with available spasticity data reported having spasticity. The low prevalence was explained by spasticity being patient-reported spasticity. In a previous chronic post-stroke population, we estimated that 41% of the patients had disabling lower-limb spasticity (i.e., spasticity that deserved to be treated, with good probability of improving the walk [7]). In the present study, disabling spasticity was defined as spasticity requiring treatment in addition to physical therapy. We found that the frequency decreased over the past 15 years in our department and represented less than 10% of patients hospitalized in 2015, which is less than the frequency described in the literature. Post-stroke spasticity is considered an important contributing factor to disability and could increase the prevalence of joint stiffness after stroke. For Schnitzler et al., 10.3% of 764,719 post-stroke patients reported stroke-related stiffness, which was associated with a high Modified Rankin Score (>2) (odds ratio 2.18, 95% confidence interval 1.29–3.67) and impaired climbing stairs, walking and grasping objects ($P < 0.01$ for each) [8].

The increase in spasticity due to nociceptive signals is well known, and the link between shoulder pain and spasticity after stroke is often described. Spasticity can be increased by a painful signal but can also be painful because of muscle contractures and tendinopathies. Van Ouwenaller et al. [9] studied 219 patients with hemiplegia for 1 year after stroke: 85% of those with spasticity experienced pain as compared with 18% with flaccid hemiplegia. Spasticity of the subscapularis muscle was identified as a potential cause of reduced ROM and pain in the hemiplegic shoulder [10]. A recent Cochrane systematic review of 6 randomized controlled trials including 164 post-stroke patients with shoulder pain caused by spastic hemiplegia or arthritis showed than an injection of botulinum toxin A seemed to reduce pain severity and improve shoulder function and ROM as compared with placebo [11]. However, other studies did not find any relation between spasticity and hemiplegic shoulder pain [12,13].

Post-stroke shoulder pain is also frequently linked to a complex regional pain syndrome called shoulder-hand syndrome (SHS). Several SHS risk factors identified include moderate spasticity (Ashworth scale 2–3), presence of subluxation, upper-extremity weakness, and visual-perceptual deficits [14]. According to Braus et al. [14], SHS is initiated by peripheral lesions, and preventive care in stroke patients to protect the affected limb against trauma could reduce the frequency of SHS. Repeated (minor) trauma may lead to microbleeding and aseptic inflammation of peripheral tissue, especially synovial tissue, causing traumatic arthritis. A systematic review of post-stroke hand edema and SHS confirmed that SHS usually coincides with increase arterial blood flow and that trauma causes aseptic inflammation in SHS [15].

Our results suggest a decrease in frequency of spasticity with a decrease in frequency of shoulder pain in our department. Indeed, the best treatment of shoulder pain is preventive, based on the education of all professionals involved in caring for hemiplegic patients: nurses, physical therapists, physicians and also relatives.

In our opinion, the decrease in frequency of disabling spasticity and shoulder pain is related at least in part to the improved quality of care over the last past 15 years, both in the acute stage in stroke units and in rehabilitation departments. Indeed, in our department, the occurrence of shoulder pain is considered a marker of "mis-care" (micro-trauma of the shoulder during nursing and transfers). Paramedical and medical teams are both used, with new care as a precaution for shoulders.

Between 2010 and 2014, a French national action plan for stroke led to the development of stroke care networks and prevention and health education. How we approach stroke rehabilitation has been completely altered from the end of the 20th century. Rehabilitation in stroke units has been started faster for a few years and since the publication of European and US recommendations promoting very early mobilization [16]. This change could explain in part the walking ability and improvement in admission FIM scores. The principles of stroke rehabilitation for patients with hemiplegia are now similar to those for motor learning [17]. To improve rehabilitation care for stroke patients, an algorithm has been developed, combining clinical, neurophysiological and neuroimaging measures at the sub-acute stage to predict the potential for recovery of upper-limb function [18]. Rehabilitation plans can then be tailored appropriately.

In the literature, we did not find any study dealing with the evolution of these aspects of the consequences of stroke over the years. To explain all these changes, we hypothesize that the professional teams in both stroke and PRM units are better used to handling patients and precautions for shoulders. As well, the development of stroke units has had an economic impact, explained by the impact on disability, showing that disability decreases with the presence of stroke units [19]. The great improvement in early care for stroke patients may explain this favorable effect on spasticity, including the whole care, from early thrombolysis and thrombectomy to very early mobilization, and the education of professionals. The link between revascularisation (thrombolysis and thrombectomy) and spasticity has not been proven, and our study did not show a significant association, but we cannot exclude a low statistical power to prove a hypothetical association.

This study has some limitations. First, it was a retrospective study with the usual limitations. Also, it was a monocentric study, with most patients from the same stroke unit. Our results may not be generalizable. The evolution in the typology of patients is not due to a change in our admission criteria but could be due to changes in the typology of patients at the acute stage. We found an evolution in patients admitted in our department (increasing number of walking patients at admission as well as patients with a functional paretic upper limb at admission and discharge) and patients seemed to be more disabled by cognitive disorders and slightly less by motor disorders. Information about stroke severity in the acute stage such as the US National Institutes of Health Stroke Scale (NIHSS) would have been valuable. The NIHSS and FIM scores are correlated (r^2 0.02–0.36) [20], but we cannot extrapolate on initial stroke severity and neurological evolution in the stroke unit. A specific study in the stroke department is needed.

Another explanation for the evolution in patients admitted in our department is the development of new post-stroke PRM units in the area, with a day hospital, which allowed for direct home discharge from the stroke unit for independent patients without cognitive disorders. A center effect secondary to competition with new stroke rehabilitation units cannot be eliminated, and our results must be confirmed in a multicentric study.

The increase in number of patients with executive disorders could have been a bias. The evaluation of dysexecutive syndrome could have changed over time to explain its increased prevalence. In the literature, the prevalence of dysexecutive syndrome in stroke patients seems to increase over time, but diagnostic criteria and time to evaluation were not the same (11%–20% in a study from 2000 to 2003 vs 56% in a study from 2011 to 2012) [21,22]. In our study, the frequency of spasticity and shoulder pain also decreased in dysexecutive patients, and we found no association between the intensity of spasticity and dysexecutive disorder, so even a possible recruitment bias could not explain our results completely.

6. Conclusions

The decreasing frequency of disabling spasticity and shoulder pain over the last 10 years in our PRM department could be a good marker of the improvement in quality of care after stroke. Our results should be considered with caution, but we hope they can be confirmed in a multicentric study. Such confirmation would be important evidence of the great improvement in stroke healthcare both in stroke and PRM units in the last 10 years in France and could affect future estimations for the need for rehabilitation care after stroke.

Disclosure of interest

The authors declare that they have no competing interest.

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