High prevalence of erectile dysfunction in male patients with acute stroke was associated with age but not to modifiable cardiovascular risk factors

Christel Baagø Schjørring ^(a), ¹ Heidi Shil Eddelien, ^{1,2} Jawad Haider Butt ^(b), ^{1,3,4} Christina Kruuse ^(b), ^{1,2,5}

ABSTRACT

Background Erectile dysfunction (ED) and stroke share common risk factors, and symptoms of ED often precede the development of clinical cardiovascular disease (CVD). However, little is known about how ED is associated with cardiovascular (CV) risk factors in patients who had a stroke and if concomitant ED is a marker of more severe CVD.

Aims We aimed to identify the prevalence of ED and CV risk factors in patients admitted with a stroke or transient ischaemic attack (TIA). Further, we wanted to test if self-reported ED associated with presence of CV risk factors, and if patients with ED had increased stroke severity compared with patients without ED.

Methods This was a post hoc analysis of data retrieved in a cross-sectional survey from two non-comprehensive stroke units in Denmark. Multiple logistic regression adjusted for covariates was performed to investigate the association between CV risk factors and self-reported ED. **Results** We included 287 male patients of which

116 (40.4%) had self-reported ED. Advanced age was significantly associated with self-reported ED (reference \leq 60 years: OR 3.93, 95% Cl 1.84 to 8.37 for men 71–80 years and OR 4.61, 95% Cl 1.92 to 11.08 for men >80 years). Self-reported ED was not significantly associated with CV risk factors or stroke severity.

Discussion Four in 10 men with acute stroke or TIA reported to have ED prior to their stroke, and this was associated with age rather than CV risk factors. Hence, self-reported ED was not restricted to the CVD load, nor was ED a risk marker for increased stroke severity. However, our population was of high age with well-established CVD, and the presence of ED may be a stroke risk marker in younger patients who had a stroke. Based on the prevalence, potential treatment of ED should be addressed in stroke recovery.

INTRODUCTION

Erectile dysfunction (ED) is an important preliminary marker for cardiovascular disease (CVD),¹ and symptoms of ED often precede the development of clinical CVD by 2-5 years.²³

Persistent ED is mainly vasculogenic and related to endothelial dysfunction and

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Erectile dysfunction (ED) shares risk factors with stroke and can be a preliminary marker for cardiovascular disease.

WHAT THIS STUDY ADDS

⇒ Four out of 10 male patients who had a stroke suffer from ED, and ED is associated with age, but not with modifiable cardiovascular risk factors or stroke severity.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study identifies a high prevalence of reported ED in patients who had a stroke which increases with age. Presence of ED and the use of specific ED medication should be addressed by the stroke physician, and potential interaction with secondary stroke prevention should be discussed if ED treatment is requested by the patient.

atherosclerosis.⁴ The shared risk factors between ED and CVD (eg, hypertension, diabetes, dyslipidaemia, obesity, physical inactivity and smoking)⁴ are the underlying pathology linking the two diseases (figure 1).

ED is defined as the inability to achieve or maintain an erection sufficient for satisfactory sexual performance.⁵ The presence of ED often affects the quality of life negatively, hence ED should be identified and addressed if requested by the patient.⁵ In stroke survivors, ED was reported to be present in almost half of the patients,⁶ which may be due to sequelae of stroke or the medication required to treat stroke risk factors. Little is known about the prevalence of ED in male patients immediately before a stroke. It also remains to be shown if reported ED is a prognostic marker of a more severe stroke due to a more elaborate vascular dysfunction.

The main objective of this study was to investigate the prior prevalence of ED in

To cite: Schjørring CB, Eddelien HS, Butt JH, *et al.* High prevalence of erectile dysfunction in male patients with acute stroke was associated with age but not to modifiable cardiovascular risk factors. *BMJ Neurology Open* 2024;**6**:e000795. doi:10.1136/ bmjno-2024-000795

Additional supplemental material is published online only. To view, please visit the journal online (https://doi.org/10.1136/ bmjno-2024-000795).

Received 08 June 2024 Accepted 15 July 2024

Check for updates

© Author(s) (or their employer(s)) 2024. Re-use permitted under CC BY-NC. No commercial re-use. See rights and permissions. Published by BMJ.

For numbered affiliations see end of article.

Correspondence to

Professor Christina Kruuse; ckruuse@dadInet.dk

\$1

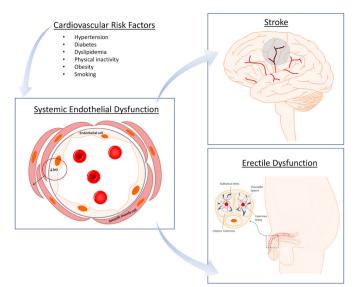


Figure 1 Pathophysiological association between stroke and erectile dysfunction. Cardiovascular risk factor can cause endothelial dysfunction, which is a systemic disorder that affects different vascular beds. The endothelium is important for regulation of the blood vessels locally. Nitric oxide released from the endothelial cells is impaired due to endothelial dysfunction. This may cause platelet aggregation, leucocyte aggregation, impaired smooth muscle relaxation and release of vasoconstrictors. The net result is impaired vasodilation and atherosclerosis. In the internal carotid arteries or intracerebral arteries this may lead to a stroke and in the penile arteries this may cause erectile dysfunction.

patients admitted with acute stroke or transient ischaemic attack (TIA). Second, we aimed to identify if ED was associated with a higher prevalence of cardiovascular (CV) risk factors, and if the presence of ED was associated with a more severe stroke compared with no reported ED.

PATIENTS AND METHODS Patients and data collection

In a cross-sectional survey, we acquired data from a stroke population by use of a structured questionnaire and performed a post hoc analysis to identify presence of ED.⁷ The inclusion periods were February to June 2018 and September 2018 to January 2019. During the study period, 1155 patients were admitted to the Herlev and Gentofte or North Zealand Hospital with symptoms of stroke (International Classification of Diseases 10th edition codes of non-traumatic intracerebral haemorrhage (I61), ischaemic stroke (I63) stroke not specified as haemorrhage or ischaemic (I64) or TIA (G45)). Patients were approached by a research assistant trained in administering the structured questionnaire during admission to the stroke unit. Patients responded verbally to the questionnaire that was performed in person. If a patient was not able to contribute with information for example, due to aphasia, a relative could contribute to information during the interview with consent from the patient. Medical charts and emergency medicine

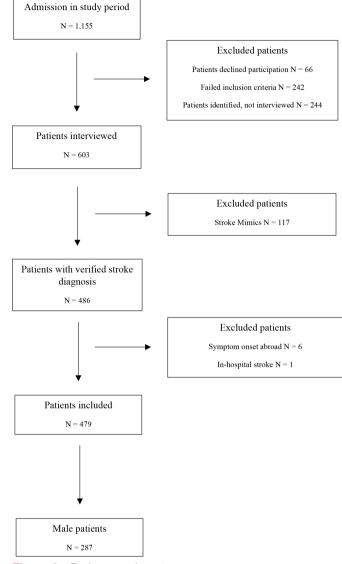


Figure 2 Patient enrolment.

services data were used to validate the patients' questionnaire responses. As the question on ED was considered a sensitive subject, the question was preferably asked at the patients' discretion without the presence of relatives, or by discreetly turning the paper on which the question was displayed so the patient could read the question and point to the right answer. Detailed design and methods were described previously.⁷ For this post hoc analysis, self-reported questions regarding ED were only included in the questionaries from the second inclusion period which comprised 287 male patients (figure 2). Data were recorded and managed electronically in a REDCap consortium, Vanderbilt University, USA, V.9.1.0 hosted at Region Hovedstaden.⁸

Study variables

Baseline characteristics were retrieved at time of interview (table 1). Risk factors were self-reported, by the patient or relatives and further verified by medical records. The following CV risk factors were included; hypertension,

Variables	Men without ED (n=134) n (%)	Men with ED (n=116) n (%)	P value
Age			
Median (25th–75th percentile)	68 (59–76)	75 (68–81)	< 0.001
≤60	39 (29.1)	14 (12.1)	<0.001
61–70	36 (26.9)	18 (15.5)	
71–80	40 (29.9)	54 (46.6)	
>80	19 (14.2)	30 (25.9)	
Hypertension	72 (53.7)	65 (56.0)	0.72
Diabetes	16 (11.9)	20 (17.2)	0.23
Hypercholesterolaemia	58 (43.3)	53 (45.7)	0.70
Cardiovascular disease	37 (27.6)	45 (38.8)	0.06
Prior myocardial infarction	10 (7.5)	13 (11.2)	0.31
Claudication	8 (6.0)	11 (9.5)	0.30
Carotid stenosis	8 (6.0)	7 (6.0)	0.98
Prior stroke	21 (15.7)	26 (22.4)	0.17
Sleep apnoea	11 (8.2)	16 (13.8)	0.16
Smoking			
Current	31 (23.1)	27 (23.3)	0.85
Former	62 (46.3)	50 (43.1)	
Never	41 (30.6)	39 (33.6)	
Excessive alcohol consumption*	95 (70.9)	77 (66.4)	0.44
Physical activity			
Vigorous	4 (3)	0 (0.0)	0.04
Moderate	38 (28.4)	21 (18.1)	
Mild	72 (53.7)	71 (61.2)	
None	20 (14.9)	24 (20.7)	
Medication for			
ED		46 (39.7)	
Hypertension	57 (42.5)	61 (52.6)	0.11
Diabetes	13 (9.7)	16 (13.8)	0.31
Hypercholesterolaemia	34 (25.4)	41 (35.3)	0.09
Antithrombotic	38 (28.4)	47 (40.5)	0.04
Sleep apnoea (CPAP)	1 (0.8)	7 (6.0)	0.03
ED duration			
0–1.9 years		16 (13.8)	
2–5.9 years		39 (33.6)	
6–10 years		31 (26.7)	
>10 years		30 (25.9)	
Education level			
Basic	48 (35.8)	44 (37.9)	0.83
Further	54 (40.3)	48 (41.4)	
Higher	32 (23.9)	24 (20.7)	

CPAP, Continuous Positive Airway Pressure.

Table 2International Classification of Disease 10th edition(ICD-10) codes and Scandinavian Stroke Scale (SSS) scorereported in the stroke study population according to erectiledysfunction (ED) status

Variables	Men without ED (n=134) n (%)	Men with ED (n=116) n (%)	P value	
ICD-10 code				
l61*	13 (9.7)	8 (6.9)	0.21	
163+164†	92 (68.7)	72 (62.1)		
G45‡	29 (21.6)	36 (31.0)		
SSS score				
Median (25th and 75th percentile)	55.5 (48–58)	56 (50–58)	0.68	
Mild	118 (88.1)	103 (88.8)	0.90	
Moderate	11 (8.2)	10 (8.6)		
Severe	5 (3.7)	3 (2.6)		

*Non-traumatic intracerebral haemorrhage.

†Ischaemic stroke and stroke without further specification.

±Transient ischaemic attack.

diabetes, hypercholesterolaemia, sleep apnoea and CVD (ie, prior myocardial infarction, prior stroke, claudication or carotid stenosis). Further to this, we included the lifestyle factors of smoking (current or former), excessive alcohol consumption (more than 14 units alcohol/ week)⁹ and physical inactivity (only sedentary activity)¹⁰ as risk factors. We also recorded prehospital medication (yes/no) for treatment of any risk factor and ED. Level of education was identified as being basic, further or higher. The Scandinavian Stroke Scale (SSS) score on admission, mandatory to report in Denmark,¹¹ was obtained from patients' electronic medical records and categorised into three-stroke severity groups: mild (43–58), moderate (26–42) and severe (0–25) (table 2).¹²

Outcome definitions

ED was self-reported and assessed through a single question: 'Have you experienced problems with obtaining erections?'. The question was chosen to reflect if the men had ED prior to their admission for stroke. ED was defined as present if patients responded 'yes' and absent if patients responded 'no'. Men who responded, 'do not know' were excluded from the main analysis. If ED was reported, patients were then asked about the duration of ED and if they had active or previous use of phosphodiesterase type 5 (PDE5) inhibitors (ie, sildenafil or tadalafil) for treatment of ED.

Statistical analysis

Baseline characteristics were compared in men with and without ED and summarised as frequencies with percentages or medians with 25th and 75th percentiles (table 1). Differences in baseline characteristics were identified using the χ^2 test or Fisher's exact test for categorical

variables and the Wilcoxon test for continuous variables. The associations between each stroke risk factor and selfreported ED were tested with univariate logistic regression adjusted for age. The association between stroke risk factors (including age) and prevalent ED was assessed in a multivariable logistic regression model, and ORs with 95% CIs were reported. The level of statistical significance was set at 5%. All analyses were done by using SAS statistical software V.9.4 (SAS Institute).

A sensitivity analysis was performed in which patients who reported 'do not know' were categorised as ED patients to investigate the possible impact of these patients on the strength of association between the risk factors and prevalent ED.

RESULTS

Baseline characteristics

In total, 287 of the admitted patients who had a stroke were men (figure 2). ED was reported by 116 (40.4%), while 134 (46.7%) reported no ED and 37 (12.9%) responded 'do not know'. The majority had ischaemic stroke (62.1% and 68.7%, in the ED and the non-ED group, respectively) (table 2). Median age was significantly higher in patients with ED compared with patients without ED (75 years vs 68 years; p<0.001) (table 1). Patients with ED were more physically inactive than those without ED (p=0.04). In patients with ED, one in three (33.6 %) had noticed ED symptoms within 2–5.9 years, and 46 (39.7%) men received medical treatment (PDE5 inhibitors) for ED. Preadmission use of antithrombotic medication and continuous positive airway pressure was significantly higher among patients with ED than those without (p=0.04 and p=0.03, respectively).

Risk factors

Increasing age was associated with an increased likelihood of prevalent ED. Men aged 71–80 had increased OR for ED (OR 3.93, 95% CI 1.84 to 8.37) compared with men 60 years or younger. The OR increased in men aged 80 years or older (OR 4.61, 95% CI 1.92 to 11.08) (figure 3). There was a non-significant trend towards an increased likelihood of prevalent ED with CVD as well as with other stroke risk factors: sleep apnoea, diabetes, hypercholesterolaemia and physical inactivity. We found no association between current or former smoking, excessive alcohol consumption or hypertension and an increased risk of ED (figure 3).

Stroke severity

The stroke severity on admission was mild, with a median SSS score of 56 and 55.5 (max score 58) in men with and without ED. 10 men (8.6%) with ED and 11 men (8.2%) without ED suffered from a moderate stroke (SSS score 26–42) and 3 men (2.6%), respectively, 5 men (3.7%) suffered a severe stroke (SSS score 0–25). There was no significant difference in stroke severity between the groups (p=0.90) (table 2).

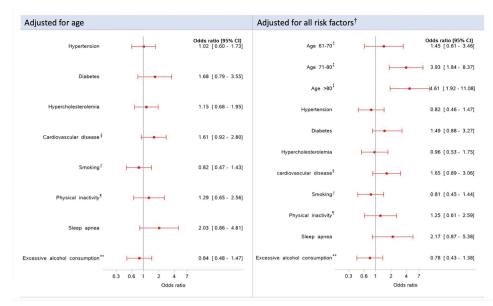


Figure 3 ORs with 95% CI of erectile dysfunction (ED) by stroke risk factors. †Adjusted for age, hypertension, diabetes, hypercholesterolaemia, cardiovascular disease, current or former smoking, physical inactivity, sleep apnoea and excessive alcohol consumption. ‡Reference was men ≤60 years. §Prior myocardial infarction, prior stroke, claudication or carotid stenosis. ||Current or former. ¶Only doing sedentary activity. **>14 units of alcohol/week.

Sensitivity analysis

Several patients (37 men, 12.9%) responded 'do not know' to the question on ED. This could reflect reluctancy in replying to the question, as ED may be stigmatising.⁴⁵ However, the sensitivity analysis, in which these patients were categorised as having ED, demonstrated similar findings to those in the main analysis (online supplemental tables S1–S4).

DISCUSSION

In this post hoc analysis, we found self-reported ED to be high before the stroke or TIA, with four in 10 males affected. Increase in age was the only risk factor significantly associated with ED in our population. Only a nonsignificant trend for association of prevalent ED with CV risk factors (CVD, sleep apnoea, diabetes, hypercholesterolaemia and physical inactivity) was found, likely reflecting a generally high load of CVD in our population.

The strength of our study is the inclusion of a large and diverse stroke patient population early after stroke, reducing the risk of recall bias in prestroke ED. Some limitations may, however, apply. Due to this study being a post hoc analysis, the assessment of prevalent ED was done by response to a single question. The use of a verified self-administered questionnaire, for example, the 5-item version of the International Index of Erectile Function (IIEF-5),¹³ would have improved the assessment of ED status and should be used for future studies. One in eight men reported not to know if they had problems with obtaining an erection. Such a lack of report may result in an underestimation of ED prevalence as the nonresponders were discarded from the primary analysis. The cognitive status of the patients following a stroke where only assessed in the regular neurological examination on admission in this study, and only if the patient was estimated to provide informed consent they were included. However, minor cognitive deficits may have been present which could have influenced the patient's response to the questionnaire. This study was a post hoc analysis in an observational study. The sample size was not high enough to detect and minor association between stroke, CV risk factors, and ED with a power of 0.8.

In our population, 4 in 10 men reported ED, which corresponds to previous studies on ED in patients who had a stroke.⁶¹⁴ The prevalence of ED is known to increase markedly with age.¹⁵ In a study of 2126 US male participants, the prevalence of ED ranged from 5.1% in men aged 20-39 years to 70.2% in men aged 70 years and older.¹⁵ The prevalence of CV risk factors in men with ED was similar to the findings in our study in regard to hypertension, hypercholesterolaemia and current smoking, but lower for diabetes, with 17.2% in the present and 30.9% in the US study. Previous CVD was higher in the present study (38.9% vs 12.9%).¹⁵ The high age of our study population with a median of 75 years for men with ED and 68 years for men without ED introduces a general high frequency of CV in both groups decreasing power to identify specific associations in either group. However, when investigating ED in a younger stroke population (mean age 56.1±9.8 years) including 605 male patients who had a stroke, CV risk factors (hypertension, hypercholesterolaemia, diabetes, current smoking and obesity) were significantly more prevalent in men with ED compared with men without ED.⁶ Increasing prevalence of risk factors with advancing age, rather than the ageing process itself, have been hypothesised to account for the strong association between age and ED.^{5 16} It remains to be resolved if age itself, or the accumulation of risk factors with age, contributes to the strong association between ED and advanced age. The most common aetiology for ED is vasculogenic related to impaired endothelial function but may also include neurogenic, psychogenic, hormonal, cavernosal or drug-related issues.^{15 17} The endothelial dysfunction is considered to be the underlying pathology linking CVD and ED together (figure 1).¹⁸ In our study, the non-significant association between ED and modifiable CV risk factors does not necessarily imply that ED is not vasculogenic as the CV risk factors are present. Other causes of ED factors may, however, also apply.

We could not detect an association between ED and higher stroke severity, and this result reflects those previously reported.¹⁴ In men with ED, there was a more frequent use of antithrombotic and lipid-lowing medication, which could account for a lower risk of severe stroke, as secondary preventions were initiated prior to the stroke. A possible selection bias in our data may be that patients with moderate and severe stroke were less likely to be included in a questionnaire study based on the nature of their symptoms (aphasia or altered consciousness).

Addressing ED in male patients who had a stroke should be considered on discharge, due to a potential impact on the quality of life, in particular considering the high prevalence of ED in an elderly stroke population.¹⁹ Suffering a stroke may worsen pre-existing ED, also, ED may be caused by the stroke,¹⁴ or the poststroke medication, such as beta-blockers or antidepressant.¹⁷ In a cross-sectional survey, only one in two men with ED addressed their problems to a health professional.³ In our study, only 40% of the patients with ED were treated medically for ED with PDE5 inhibitors. The low number treated could reflect that men refrain from discussing ED, perhaps reflecting a stigma experienced with ED.45 Health professionals often fail to discuss sexuality with patients who had a stroke.²⁰ The importance of clinicians to include questions on sexual function in patients who had a stroke, to optimise treatment by avoiding beta-blockers and antidepressants if ED is reported, needs to be emphasised. Also, the use of PDE5 inhibitors in ED treatment poststroke is challenged by recommended restrictions in the use of PDE5 inhibitors within the first 6 months from stroke, based on case reports of PDE5 inhibitors causing intracerebral haemorrhage.²¹ Conversely, PDE5 inhibitors have been hypothesised to be protective of CV outcomes,²² and the safety of these drugs should be further investigated in patients who had a stroke.

Addressing ED in patients who had a stroke is of great importance since it could help to improve the quality of life for the patients, help doctors to avoid ED provoking drugs and advise on relevant ED treatments. Future studies of ED among patients who had a stroke should be done using a verified self-administered questionnaire, for example, the 5-item version of the IIEF-5. It is also of interest to include a younger study population since the current results are not generalisable to this population. In conclusion, 4 in 10 men reported the presence of ED prior to their stroke or TIA. We only found an association with age, but not with modifiable CV risk factors as this was present in both patient groups. In our population, prevalent ED did not serve as a marker for more severe stroke. However, it remains to be detected if patients with ED poststoke need a more intense risk factor modification than patients without ED.

Author affiliations

¹Department of Neurology, Neurovascular Research Unit, Copenhagen University Hospital, Herlev and Gentofte, Copenhagen, Denmark

²Institute of Clinical Medicine, Copenhagen University, Copenhagen, Denmark ³Department of Cardiology, Copenhagen University Hospital, Rigshospitalet, Copenhagen, Denmark

⁴Department of Cardiology, Zealand University Hospital Roskilde, Roskilde, Denmark ⁵Department of Brain and Spinal Cord Injury, Neuroscience Center, Copenhagen University Hospital, Rigshospitalet, Copenhagen, Denmark

X Christina Kruuse @C_Kruuse_MD

Acknowledgements In memory of supervisor Anne Kjærgaard Danielsen, Department of Gastroenterology, Copenhagen University Hospital – Herlev and Gentofte, Copenhagen, Denmark & Dept of Clinical Medicine, University of Copenhagen, Denmark.

Contributors CK is the guarantor. CK and AKD conceived the main study and were responsible for main protocol development, gathering ethical approval and data permission. The post hoc study regarding erectile dysfunction was conceived by CK and HSE. HSE was involved in patient recruitment and data collection. JHB, CBS, HSE and CK in data analysis. CBS wrote the first draft of the manuscript. All authors made critical revisions to the manuscript and approved the final version before submission.

Funding This work was supported by TrygFonden, grant number 128669. Christina Kruuse was supported by Novo Nordisk Foundation Borregaard stipend, grant number NNF180C0031840.

Competing interests None declared.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and was approved by the Capital Region ethics committee (no. 2012-58-004) and the Danish Data Protection Agency (no. 2012-58-0004; internal reference: HGH-2017-110, I-Suite no. 06014). Participants gave oral and written informed consent before inclusion.

Provenance and peer review Not commissioned; internally peer reviewed.

Data availability statement Data are available on reasonable request. Data are available on reasonable request from the corresponding author (ckruuse@dadInet. dk) and if in adherence to Danish legislation.

Supplemental material This content has been supplied by the author(s). It has not been vetted by BMJ Publishing Group Limited (BMJ) and may not have been peer-reviewed. Any opinions or recommendations discussed are solely those of the author(s) and are not endorsed by BMJ. BMJ disclaims all liability and responsibility arising from any reliance placed on the content. Where the content includes any translated material, BMJ does not warrant the accuracy and reliability of the translations (including but not limited to local regulations, clinical guidelines, terminology, drug names and drug dosages), and is not responsible for any error and/or omissions arising from translation and adaptation or otherwise.

Open access This is an open access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited, appropriate credit is given, any changes made indicated, and the use is non-commercial. See: http://creativecommons.org/licenses/by-nc/4.0/.

ORCID iDs

Christel Baagø Schjørring http://orcid.org/0000-0001-9743-243X Jawad Haider Butt http://orcid.org/0000-0002-7380-4144 Christina Kruuse http://orcid.org/0000-0002-4210-0523

0

- REFERENCES
 - 1 Nehra A, Jackson G, Miner M, *et al.* The Princeton III consensus recommendations for the management of erectile dysfunction and cardiovascular disease. *Mayo Clin Proc* 2012;87:766–78.
 - 2 Montorsi P, Ravagnani PM, Galli S, *et al.* Association between erectile dysfunction and coronary artery disease. Role of coronary clinical presentation and extent of coronary vessels involvement: the COBRA trial. *Eur Heart J* 2006;27:2632–9.
 - 3 Hodges LD, Kirby M, Solanki J, et al. The temporal relationship between erectile dysfunction and cardiovascular disease. Int J Clin Pract 2007;61:2019–25.
 - 4 Billups KL. Erectile dysfunction as an early sign of cardiovascular disease. *Int J Impot Res* 2005;17 Suppl 1:S19–24.
 - 5 NIH Consensus Conference. Impotence. NIH consensus development panel on impotence. *JAMA* 1993;270:83–90.
 - 6 Bener A, Al-Hamaq AOAA, Kamran S, et al. Prevalence of erectile dysfunction in male stroke patients, and associated co-morbidities and risk factors. Int Urol Nephrol 2008;40:701–8.
 - 7 Eddelien HS, Butt JH, Amtoft AC, et al. Patient-reported factors associated with early arrival for stroke treatment. *Brain Behav* 2021;11:e2225.
 - 8 Harris PA, Taylor R, Thielke R, *et al.* Research electronic data capture (REDCap)--a metadata-driven methodology and workflow process for providing translational research informatics support. *J Biomed Inform* 2009;42:377–81.
 - 9 Sundhedsstyrelsen (Danish Health Authority). Danskernes Sundhed -Den Nationale Sundhedsprofil 2017 (The Danes Health - The National Healthprofile 2017).2018:134.
 - 10 Sundhedsstyrelsen (Danish Health Authority). Fysisk Aktivitet - Håndbog Om Forebyggelse Og Behandling (Physical Activity -Manual for Prevention and Treatment).2018:494.

- 11 Johnsen SP, Ingeman A, Hundborg HH, et al. The Danish stroke registry. Clin Epidemiol 2016;8:697–702.
- 12 Govan L, Langhorne P, Weir CJ. Categorizing stroke prognosis using different stroke scales. *Stroke* 2009;40:3396–9.
- 13 Rosen RC, Cappelleri JC, Smith MD, et al. Development and evaluation of an abridged, 5-item version of the international index of erectile function (IIEF-5) as a diagnostic tool for erectile dysfunction. Int J Impot Res 1999;11:319–26.
- 14 Koehn J, Crodel C, Deutsch M, et al. Erectile dysfunction (ED) after ischemic stroke: association between prevalence and site of lesion. *Clin Auton Res* 2015;25:357–65.
- 15 Selvin E, Burnett AL, Platz EA. Prevalence and risk factors for erectile dysfunction in the US. *Am J Med* 2007;120:151–7.
- 16 Solomon H, Man JW, Jackson G. Erectile dysfunction and the cardiovascular patient: endothelial dysfunction is the common denominator. *Heart* 2003;89:251–3.
- 17 Lue TF. Erectile dysfunction. *N Engl J Med* 2000;342:1802–13.
- 18 Billups KL, Bank AJ, Padma-Nathan H, et al. Erectile dysfunction is a marker for cardiovascular disease: results of the minority health institute expert advisory panel. J Sex Med 2005;2:40–50.
- 19 Krane RJ, Goldstein I, Saenz de Tejada I. Impotence. *N Engl J Med* 1989;321:1648–59.
- 20 McGrath M, Lever S, McCluskey A, et al. How is sexuality after stroke experienced by stroke survivors and partners of stroke survivors? A systematic review of qualitative studies. *Clin Rehabil* 2019;33:293–303.
- 21 Alpsan MH, Bebek N, Ciftci FD, et al. Intracerebral hemorrhage associated with sildenafil use: a case report. J Neurol 2008;255:932–3.
- 22 Ölmestig JNE, Marlet IR, Hainsworth AH, *et al.* Phosphodiesterase 5 inhibition as a therapeutic target for ischemic stroke: a systematic review of preclinical studies. *Cell Signal* 2017;38:39–48.