Sub-classifying stroke of undetermined etiology – The Nor-COAST study

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Abstract

Background and purpose

Etiological classification of ischemic stroke plays an important role in the choice of secondary prevention and further follow-up of patients. Novel diagnostic methods have resulted in an increase of strokes labeled with "undetermined etiology" because of competing risk factors and incomplete investigation. The aim of this study was to sub-classify strokes of undetermined etiology in the fifth group of The Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification, and compare vascular risk factors between the strokes labeled with undetermined etiology and strokes of the corresponding determined etiology classified into TOAST group 1-3. Further we wanted to look at prevalence of vascular risk factors in "strokes of undetermined etiology" compared to the other four groups of determined etiologies in the TOAST classification system.

Materials and methods

Participants included in the Norwegian Cognitive Impairment after Stroke (Nor-COAST) study, who had suffered ischemic stroke were classified according to TOAST criteria into five groups labeled TOAST group 1-5. Available information from diagnostic investigation including imaging of the cerebral, carotid and vertebral arteries, echocardiography and heart monitoring were used for further classification. Information on vascular risk factors and medication were used for comparisons between groups. The Ideal Cardiovascular Health Index was used to score the participants' vascular risk profile.

Results

Of the 709 included participants, 293 were classified with" strokes of undetermined etiology", labeled TOAST group 5. Within his group, multiple etiologies were found in 38 participants. Stroke of known etiology but not fulfilling classification criteria was found in eight participants, all of those with large artery disease as possible etiology, incomplete investigation was the case in 242 participants, where 103 participants had findings of large artery disease, 29 were

suspected having suffered embolic stroke with cardiac origin, small vessel disease was found in eight participants, and no etiology was found in 102 participants. Embolic stroke of undetermined origin was set as classification for five participants. The group with incomplete investigation and no detected possible etiology was significantly younger than the participants in TOAST-group 5 given a possible etiology (p-value=0.049). We detected no significant differences in vascular risk factors between etiologies found in TOAST group 5 and their respective TOAST group 1, 2 and 3.

Conclusion

Stroke of undetermined etiology form a heterogeneous group which is possible to further classify. Etiologies identified in TOAST group 5 do not differ from their respective TOAST groups of equal etiologies in regards to vascular risk factors. Novel classification systems in line with modern diagnostic evaluations are needed for use both in research and clinical setting in order to reduce the prevalence of stroke of undetermined etiology and give appropriate secondary prevention to all strokes.

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Selected abbreviations

AF	Atrial fibrillation
ASCO	Atherosclerosis-Small vessel disease-Cardiac source-Other
CSS	Causative Classification System
CT	Computer Tomography
CVH	Cardiovascular health
DALY	Disability-adjusted life-years
DOAC	Direct Oral Anticoagulant
ECG	Electrocardiogram
ESUS	Embolic stroke of undetermined source
LAD	Large artery disease
MI	Myocardial infarction
MRI	Magnetic Resonance Imaging
mRS	modified Rankin Scale
NIHSS	National Institute of Health Stroke Scale
Nor-COAST	Norwegian Cognitive Impairment After Stoke Study
PFO	Patent foramen ovale
SVD	Small vessel disease
TOAST	Trial of Org 10172 in Acute Stroke Treatment

1. Introduction

1.1 The burden of stroke

According to the Global Burden of Diseases, Injuries and Risk Factors Study (GBD 2010), stroke is the second most common cause of death (1) and third most common cause of disability-adjusted life years (DALYs) (2) worldwide. Even though mortality and DALY rates are declining, the absolute number of people either dying from stroke or becoming disabled has increased during the last twenty years (3). Especially in developing countries there is an increasing incidence of known risk factors for stroke like diabetes mellitus, hypertension, dyslipidemia, smoking, obesity, poor diet and physical inactivity (4). Globally, an increasing and aging population, reduced stroke case-fatality and increase in prevalence of stroke risk factors, are causing an increase in the number of incident strokes, stroke survivors and DALYs lost (5).

In Norway, approximately 12 000 cases of stroke are registered each year (6). The incidence has kept stable or slightly decreasing over the last couple of decades (7), but in line with global trends, a rise in prevalence of stroke survivors is expected (8). It is estimated that 25-74% of stroke survivors experience stroke sequelae resulting in need of assistance in activities of daily living (ADL) (9) affecting lives of the patients and relatives, in addition to representing a substantial burden on the health care system and the public health economy. Further knowledge about stroke treatment is therefore of great importance to the patients themselves, their families and network and the society.

1.2 Stroke etiology

The etiology of stroke is of relevance both in the individual patient care and in research, giving crucial information for treatment in the acute phase, secondary prevention and the prognosis of the patients (10). Stroke is defined as either ischemic or hemorrhagic, of which 85% are of ischemic origin (11).

Ischemic strokes are caused by occlusion of cerebral vessels, and are generally thought to be caused by emboli from the heart, artery-to-artery embolism or in situ small vessel occlusion. Atrial fibrillation (AF) is the most common cause of cardiac emboli, causing approximately

25% of all strokes (12). Other structural changes in the heart, like recent myocardial infarction, valve defects, patent foramen ovale and left ventricular thrombus are also thought to be other, less frequent, sources of cardiac emboli (13, 14). Large artery atherosclerosis, in both intra- and extracranial arteries, may result in platelet clots occluding the artery and unstable plaque with following embolism. Small vessel disease, which is associated with lacunar infarction, lead to in situ occlusion of intracerebral perforating arteries. A small proportion of ischemic stroke is caused by non-atherosclerotic etiology like vasculitis, dissection and hypercoaguable states, or hematologic disorders. When the etiology is not determined after standard evaluation, the stroke is labeled cryptogenic (15).

1.3 Classification of stroke

Classification of strokes was first applied in research, and later implemented in clinical practice. The purpose of these systems in research was primarily to describe and categorize the patients for prognostic analyses (16). However, in clinical practice, there is a need for more valid classification in order to individually predict prognosis and decide upon which type of secondary prevention is needed.

Several classification systems based on etiology have been proposed, categorizing ischemic strokes based on most likely cause. The Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification has been the most widely used etiologic classification system for ischemic stroke in both clinical and research setting the last two decades (17).

1.3.1 TOAST classification criteria

The TOAST classification divides into five categories: 1) large-artery atherosclerosis, 2) cardioembolism, 3) small-artery occlusion 4) stroke of other determined etiology, and 5) stroke of undetermined etiology (18). Criteria for the large-artery atherosclerosis category is clinical symptoms of cortical dysfunction (aphasia, agnosia, neglect, apraxia, hemianopsia) or cerebellar dysfunction (vertigo, nystagmus, gait ataxia, limb incoordination), and brain imaging findings of either significant (> 50%) stenosis or occlusion of a major cerebral artery or cortical branch of an artery. Cortical or cerebellar lesion and brain stem or subcortical hemispheric infarcts of > 1.5 cm in diameter on CT or MRI are considered to have large-artery atherosclerotic etiology. If duplex or arteriographic studies are normal or show only minimal changes, the stroke cannot be classified as secondary to large-artery atherosclerosis. Patients

with arterial occlusions that presumably are caused by an embolus from the heart, are classified with stroke secondary to cardioembolism. At least one cardiac source for an embolus most be identified, and potential large-artery atherosclerotic sources of thrombosis or embolism have to be eliminated. Small-artery occlusion includes strokes that are often labeled lacunar infarcts in other classifications. To be classified into this group, the patient should have findings of clinical lacunar syndromes, the most frequent being pure motor hemiparesis, pure sensory hemiparesis, ataxic hemiparesis, sensorimotor stroke, and dysarthria-clumsy hand syndrome (19). Evidence of cerebral cortical dysfunction should be absent. CT or MRI examinations should be normal, and if a brain stem or subcortical hemispheric lesion is present, this should have a diameter < 1.5 cm. Stenosis of > 50% on the ipsilateral extracranial arteries and potential cardiac sources for embolism should be absent. Stroke of other determined etiology includes patients with rare causes of stroke, such as dissection of cerebral or cervical arteries, hypercoaguable states, hematologic disorders or non-atherosclerotic vasculopathies. Relevant diagnostic evaluation such as blood tests or arteriography should verify the diagnosis.

A stroke is classified with undetermined etiology when 1) No etiology fulfilling the strict TOAST criteria is present despite extensive vascular, cardiac and biochemical evaluation, 2) No cause identified but the evaluation is incomplete, or 3) Two or more competing causes of stroke are identified.

The increasing availability of new diagnostic methods over the last years, has led to a large number of explanatory factors that may be the cause of stroke, making the TOAST system insufficient (20). Because of the spectrum of monitoring techniques and imaging data, some patients receive incomplete follow-up, most often because of limited utility of extensive investigations, but also based on cost-benefit considerations. Therefore, the physician cannot conclude on one single origin, and the stroke is classified with “undetermined etiology”, either due to competing risk factors or incomplete information. As a consequence, strokes with likely atherosclerotic etiology are classified together with strokes having no vascular risk factors identified. Thus, the TOAST classification makes a false high proportion of strokes of undetermined etiology, which justifies further sub-grouping.

According to Adams et al. (18), a “probable” etiologic classification can be done if clinical findings and diagnostic investigations are consistent with one subtype and other etiologies have been excluded. When clinical findings and neuroimaging data suggest an etiologic subtype, but no other investigations have been done, a “possible” classification is done. However, in many cases, stroke patients have completed a limited number of diagnostic evaluations, making it

difficult to exclude other etiologies, and strokes with "possible" etiology end up being classified with "undetermined etiology".

1.4 TOAST classification; stroke of undetermined etiology or cryptogenic stroke

There is no formal definition of the term "cryptogenic stroke", but it is widely agreed upon to include all ischemic strokes where a defined etiology is not identified (21). All strokes classified into the fifth TOAST group having no determined etiology, can be called cryptogenic. Studies have shown that rates of cryptogenic strokes in populations vary between 17-39% (15), depending on classification system, inclusion age and diagnostic evaluation. The cryptogenic stroke group in the TOAST classification includes strokes with multiple possible etiologies, strokes that are under-classified or under-measured (21), and strokes with no clear etiology despite extensive diagnostic evaluation.

1.4.1 Stroke of undetermined etiology with multiple possible etiologies

One of the main limitations of the TOAST classification is that the category "stroke of undetermined etiology" also includes patients with two or more potential causes of stroke present (18). This would be the case in a patient with atrial fibrillation as risk for cardioembolism and ipsilateral carotid stenosis greater than 50%, making large-artery atherosclerosis a possible etiology. The clinician cannot say which of those factors caused the stroke, and the patient is classified with undetermined etiology. In cases where none of the present risk factors fulfill classification criteria, the etiology would also be deemed undetermined.

1.4.2 Under-classified stroke of undetermined etiology

When the diagnostic investigations are completed and there are findings which do not fulfill the criteria for the etiologically specified TOAST classifications, the strokes are categorized as under-classified. E.g. cases where there is evident atherosclerosis in the ipsilateral external carotid, but with a stenosis degree of < 50%, the stroke cannot be classified as secondary to large-artery atherosclerosis because it doesn't meet the criteria for this TOAST group, and is thus classified as stroke of undetermined etiology.

1.4.3 Under-measured stroke of undetermined etiology

Patients diagnosed with strokes of undetermined etiology, who for various reasons have not received extensive diagnostic evaluation, are classified with under-measured stroke of undetermined etiology. An example of this scenario could be an older person with a high level of comorbidity having suffered from stroke, where any findings on extensive investigation would not lead to significant changes in treatment, and as such not be beneficial neither from an individual nor a health economic perspective.

1.4.4 Embolic stroke of undetermined source (ESUS)

Cryptogenic stroke has been established as the term for strokes of unknown cause. However, recent research has made use of the term embolic stroke of undetermined source (ESUS), as the majority of cryptogenic stroke is thought to be thromboembolic (15). To be defined as ESUS, imaging must show a non-lacunar brain infarct, there must be open arteries (< 50% stenosis) proximal to the infarct and no major-risk cardioembolic source should be detected. Of diagnostic investigations, brain CT or MRI showing non-lacunar infarct, precordial echocardiogram, cardiac monitoring for ≥ 24 hours, echocardiography, and imaging of the extracranial and intracranial arteries supplying the area of the brain infarct should be done.

In a systematic review from 2017, Hart et al. found that 17% of all stroke meet the criteria for ESUS (22). The high portion of ESUS in cryptogenic stroke is thought to be due to missing out other possible causes, despite extensive investigations, e.g. cardiac emboli sources may be intermittent and difficult to detect. For example paroxysmal AF is at times difficult to detect without lengthy cardiac monitoring.

There is a large focus on the treatment of ESUS patients nowadays (23-26), and a number of randomized controlled trials (RCTs) (27-29) have been conducted to investigate the utility of various medication strategies. Direct Oral Anticoagulants (DOACs) has been hypothesized to be effective, but so far no significant differences between conventional antiplatelet therapy and DOAC-treatment (26, 29) in ESUS patients have been documented.

1.5 Cardiovascular risk factors in stroke

Hypertension is the most common vascular risk factor in stroke patients (11, 30), in addition to being the most important factor in the development of AF and a contributor in the complication

of atherosclerosis (31). However, other risk factors are also thought to play a significant role in the development of stroke. In a large multinational study by O'Donnell et al. (32), five risk factors were identified as accounting for more than 80% of the global risk of all stroke (both ischemic and hemorrhagic): hypertension, current smoking, abdominal obesity, diet and physical activity. Hankey et al. (33) suggested that 60-80% of all ischemic strokes are caused by risk factors for atherosclerotic ischemic strokes (hypertension, hypercholesterolemia, cigarette smoking, carotid stenosis and diabetes mellitus), and risk factors for cardiogenic ischemic stroke (atrial fibrillation and valvular heart disease).

The high frequency of modifiable risk factors in stroke underline the importance of risk factor identification in stroke patients. Prior studies have shown that stroke of different etiologies have different risk factor burden (34, 35), and that cryptogenic strokes differ from strokes with a known etiology, both in age, with fewer atherosclerotic markers and a smaller burden of cardiovascular diseases (36). Therefore, further investigation of these associations would be of great interest.

1.6 The Nor-COAST study

This was a study based on data from the Norwegian Cognitive Impairment after Stroke (Nor-COAST) study; a large multicenter prospective observational study recruiting participants from five hospitals in Norway (37). The participating hospitals were St. Olav hospital, Bærum Hospital, Haukeland University Hospital, Oslo University Hospital Ullevål and Ålesund Hospital.

One of the main outcomes of the Nor-COAST study was to investigate the development of post stroke cognitive impairment and its association with different stroke etiologies. To achieve this, the majority of the patients should have their strokes classified into a defined homogenous subgroup. Preliminary analyses have shown that approximately 40% of the participants have been classified into the group "undetermined etiology" according to the TOAST system, which weakens further analyses, as this sample constitutes a heterogeneous group of stroke patients with different risk profiles. Information about their risk factors of stroke gave us the opportunity to describe and classify them into narrower and more homogenous subgroups.

1.7 Objectives

The TOAST classification categorizes strokes based on etiology, which is also hypothesized to be associated with cognitive impairment in stroke patients (38, 39). Associations between pathophysiological mechanisms of stroke and cognitive function post stroke, will give us valuable information about stroke outcomes. The aim of this study was to classify the participants in the TOAST category undetermined etiology into suitable subgroups, and compare prevalence of vascular risk factors in this group to the other TOAST groups.

Research questions:

I) Is it possible to classify the group of strokes of "undetermined etiology" further into suitable subgroups?

II) Are participants in TOAST group 5 with detected possible etiologies comparable to the other TOAST groups with corresponding etiology in regards to vascular risk factors?

2. Materials and Methods

2.1 Study design

This was a cross-sectional study using data from the Nor-COAST study. Data was collected between May 2015 and March 2017 at the five participating hospitals, representing three of four health regions in Norway. Experienced research nurses screened the participants for eligibility. Baseline data, which are used in this study, were retrieved from either the participants themselves, caregivers or medical records.

2.2 Study participants

Participants were included in the Nor-COAST study if they 1) were diagnosed with stroke according to the established WHO criteria (40) or with MRI findings compatible with acute infarction or intracerebral hemorrhage, 2) were admitted to one of the five participating hospitals and were living in the catchment area of this hospital, 3) had symptom onset within one week prior to inclusion, 4) was able to communicate in Norwegian, and 4) had a caregiver who was able to give supplementary information about cognition. Patients with a life expectancy of less than 3 months were excluded. In the present study, only Nor-COAST-participants given a TOAST classification were included.

2.3 Diagnostic evaluations

Experienced physicians classified the participants according to the TOAST classification. In addition, the physicians could register the probable cause if the stroke etiology was classified as "other determined etiology" or "stroke of undetermined etiology" according to the TOAST classification: dissection, protrombotic condition, pregnancy, atrial fibrillation, endocarditis, patent foramen ovale (PFO), myocardial infarction, valve defect, large artery disease and small vessel disease. In cases where the etiology was uncertain, the clinicians also had the option of leaving comments stating the most likely cause based on their clinical assessments.

In participants with no further classification than undetermined etiology, available information about diagnostic investigation were used. This included findings on electrocardiogram (ECG), 24h cardiac monitoring, CT and MRI of the brain, CT or MRI angiography (CTa/MRa) of intra- and extra cranial arteries, ultrasound carotid and vertebral arteries, echocardiography and blood

samples. The diagnostic investigation was regarded as complete if the participant had undergone all the aforementioned evaluations, and incomplete if missing one or more of them.

2.4 Classification of stroke

Classification of cryptogenic stroke, represented by TOAST group 5 in this study, was based on suggestions in *Stroke: Pathophysiology, Diagnosis and Management* (21): Multiple possible etiologies, under-classified, under-measured, and truly cryptogenic/embolic stroke of undetermined source (ESUS). Strokes with known etiology but not fulfilling criteria (e.g. < 50% carotid stenosis) were classified as under-classified, cases where diagnostic evaluation is incomplete were labeled under-measured, and strokes with multiple possible etiologies were categorized as such. ESUS was defined as a case with thorough diagnostic evaluation without pathologic findings (as described under 1.4.4). The participants classified with under-classified and under-measured strokes, were further divided into the subgroups possible large artery disease, possible cardiac emboli, possible small vessel disease and undetermined etiology.

Large artery atherosclerosis was set as possible etiology in cases with findings of stenosis of any degree, plaque or occlusion on any of the large-artery imagining. Cardiac embolus was set as possible etiology if there had been recorded periods of atrial fibrillation (AF) on ECG or 24h cardiac monitoring, or if the clinician had a high suspicion of paroxysmal AF. In addition, participants with recordings of patent foramen ovale (PFO) or myocardial infarction (MI) as possible cause of embolus, were put in this group. If the classifying clinician suspected small vessel disease, the stroke was classified as such.

Compared to the definition of "possible" etiology in the TOAST criteria (18), the participants in this study needed to have more certain findings on diagnostic evaluations to be classified with possible etiology. This puts the definition of "possible" etiology applied in this study somewhere between the "possible" and "probable" classifications from the TOAST criteria.

2.5 Vascular risk factors

Information on prestroke hypertension and hypercholesterolemia, diabetes mellitus, atrial fibrillation, smoking, and previous cerebrovascular and ischemic heart disease, in addition to medications such as antihypertensives, statins, antidiabetics, anticoagulants and antiplatelet therapy, were recorded and used for comparing TOAST group 5 to TOAST group 1-4.

2.5.1 The Ideal Cardiovascular Health Index

To evaluate the vascular risk profile, the Ideal Cardiovascular Health (CVH) Index from the American Heart Association (41) was applied. This is a score based on seven dichotomous variables; current self-reported non-smoker, body mass index (BMI) between 18.5 and 25 kg/m^2, adequate physical activity, a healthy diet, untreated total cholesterol < 5.2 mmol/L and not being treated with anti-cholesterol medication, untreated resting blood pressure <120/80 mmHg and not being treated with antihypertensive, and fasting glucose < 5.6 mmol/L or being treated with antidiabetic medication. Fulfilling 0 variables reflects poor outcome and fulfilling 7 indicates ideal CVH status.

To calculate the Ideal CVH Index, information on height, weight, blood pressure, blood samples on glucose and cholesterol, and medication for high blood pressure, high cholesterol and diabetes mellitus. All measurements were collected at index stay. The participants were scored with a healthy diet if they had reported eating vegetables every day and fish at least two times a week. Physical activity was scored as satisfactory if the participant had reported 30 minutes or more of daily activity. This index is used to evaluate risk in primary prevention settings, but in the present study the context is secondary prevention risk assessment. As all blood pressure measurements were taken in the acute phase of stroke, the majority would be elevated, and we chose to only include hypertension as risk factor in participants had recorded hypertension or were treated with antihypertensive prior to inclusion.

2.6 Statistical analysis

Descriptive statistical methods were used for baseline characteristics, classification of stroke etiology and calculation of vascular risk score. Pearson's chi-square test was used for comparison between dichotomous variables. Continuous variables were tested for normality, and non-parametric tests were applied due to skewed distributions. P-values < 0.05 were considered statistical significant. All analyses were carried out using IBM SPSS v.25.

2.7 Ethical considerations

The Nor-COAST study was approved by the Regional Committee for Medical and Health Research Ethics (REC) (REC no 2015/171 REK Nord). Informed, written consent was obtained

from all participants. In cases were the patients were unable to consent for themselves, their next of kin gave consent for them.

3. Results

3.1 Participant characteristics

Of the 734 participants recruited to the Nor-COAST study with ischemic strokes, 709 were classified according to the TOAST classification, with 416 classified within TOAST group 1-4 and 293 classified into TOAST group 5. A flow chart of the participants is shown in Figure 1.

In TOAST group 1-4, mean age of the 416 participants was 73.8 ±11.6 years, 43% were women, and 68% had suffered mild strokes with a score of 4 points or less on the National Institute of Health Stroke Scale (NIHSS) (42). On the modified Rankin Scale (mRS) (43), 90% scored two or less points prestroke, and 58% had a mRS score of two points or less at discharge. On the Charlson Comorbidity Index (44), 16% scored 1-2 point, 39% 2-3 points and 45% scored ≥ 5 points.

Among the 293 participants in TOAST group 5, mean age was 74.9 ±11.5, 47% were women, 68% had suffered mild strokes with a score of 4 points or less on the NIHSS and 84% had a prestroke mRS score of 2 or less, which had decreased to 55% at discharge. On the Charlson Comorbidity Index, 17% scored 1-2 point, 41% 2-3 points and 42% scored ≥ 5 points.

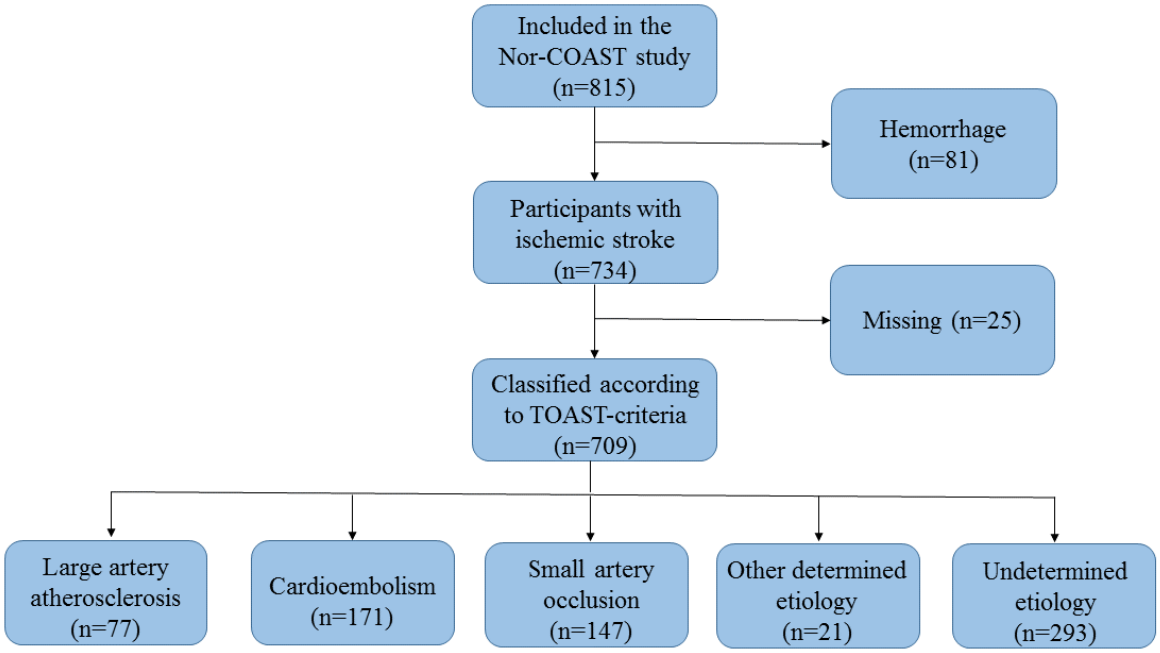

Figure 1. Flow chart on inclusion.

Table 1. Clinical and demographic characteristics[a]

	TOAST group 1-4 (N=416)[b]	**TOAST group 5 (N=293)[b]**	**P-value[c]**
Demographics			
Age, mean ±SD[d]	73.8 ±11.6	75.0 ±11.5	0.234
Females	177 (42.5)	137 (46.8)	0.266
Living alone	154 (37.0)	111 (37.9)	0.815
Education ≥ 9 years	333 (80.0)	231 (78.8)	0.694
Days admitted to hospital, mean ±SD	6.9 (5.0)[e]	6.6 ±5.0[f]	0.388
NIHSS[g] score at admittance, median (IQR)	3.0 (4.0)[h]	3.0 (5.0)[i]	0.232
Mild stroke (0-4)	288 (71.5)[h]	193 (68.0)[i]	
Moderate stroke (5-15)	85 (21.1)[h]	76 (26.8)[i]	
Moderate to severe stroke (16-20)	18 (4.5)[h]	9 (3.2)[i]	
Severe stroke (> 20)	12 (3.0)[h]	6 (2.1)[i]	
Modified Rankin Scale score, premorbid, mean ±SD	0.9 ±1.1[j]	1.1 ±1.2[k]	0.143
Modified Rankin Scale score, discharge, mean ±SD	2.3 ±1.5[l]	2.3 ±1.4[m]	0.740
Charlson comorbidity index score, mean ±SD	4.2 ±2.1	4.3 ±2.1	0.776
Vascular risk factors			
Hypertension prestroke	285 (68.5)	182 (62.1)	0.077
Hypercholesterolemia prestroke	175 (42.1)	111 (37.9)	0.264
Diabetes mellitus	138 (33.2)	50 (17.1)	0.993
Atrial Fibrillation	172 (41.3)	38 (13.0)	< 0.001
Previous cerebrovascular disease	79 (19.0)	107 (36.6)[m]	0.195
Previous ischemic heart disease	285 (68.5)	58 (19.8)	0.789

[a]Values presented as n (%) if not otherwise specified, [b]Unless otherwise specified, [c]Mann-Whitney Test for continuous variables and Pearson chi-square test for dichotomous variables, [c]Standard deviation, [e]N=400, [f]N=291, [g]National Institute of Health Stroke Scale, [h]N=403, [i]N=284, [j]N=414, [k]N=291, [m]N=415, [m]N=292

3.2 Diagnostic investigations

Findings on diagnostic evaluations were used as basis for the classification of strokes in TOAST group 5. Proportions of participants having undergone the different diagnostic investigations are displayed in Table 2. Atrial fibrillation was detected on ECG in 17 of the participants and on ≥ 24h cardiac monitoring in eleven participants. Ultrasound of extracranial carotids, showed

arterial occlusion in six participants, 13 had > 50% stenosis, 13 had < 50% stenosis, and plaque was found in 99 participants. Of the 126 participants having undergone CT or MRI angiography (CTa/MRa), arterial occlusion was found in 18 participants, > 50% stenosis in six of the participants, eight had < 50% stenosis, six had stenosis of non-specified severity, and plaque was found in 18 participants. On the CTa/MRa of the internal carotids, 28 had occlusion, one had > 50% stenosis, 18 has < 50% stenosis, ten had stenosis of non-specified severity, and 15 had findings of plaque.

Table 2: Diagnostic investigations in TOAST group 5, N=293 (n (%))	
CT[a*]	275 (93.9)
MRI[b*]	252 (86.0)
ECG[c]	286 (97.6)
≥ 24h cardiac monitoring	200 (68.3)
Ultrasound extracranial carotids	182 (62.1)
CTa[d]/MRa[e] extracranial carotids	126 (43.0)
CTa[d]/MRa[e] intracranial carotids	185 (53.9)
Echocardiography (n=80)	80 27.3)

[*]291 participants had recorded either CT or MRI investigation, [a]Computed tomography, [b]Magnetic resonance imaging, [c]Electrocardiogam, [d]Computed tomography angiography, [e]Magnetic resonance angiography

3.3 Sub-classification of strokes with undetermined etiology

We carried out further classification of the 293 participants in TOAST group 5 into one of the four following main categories: multiple possible etiologies, under-classified, under-measured, and ESUS (Figure 2).

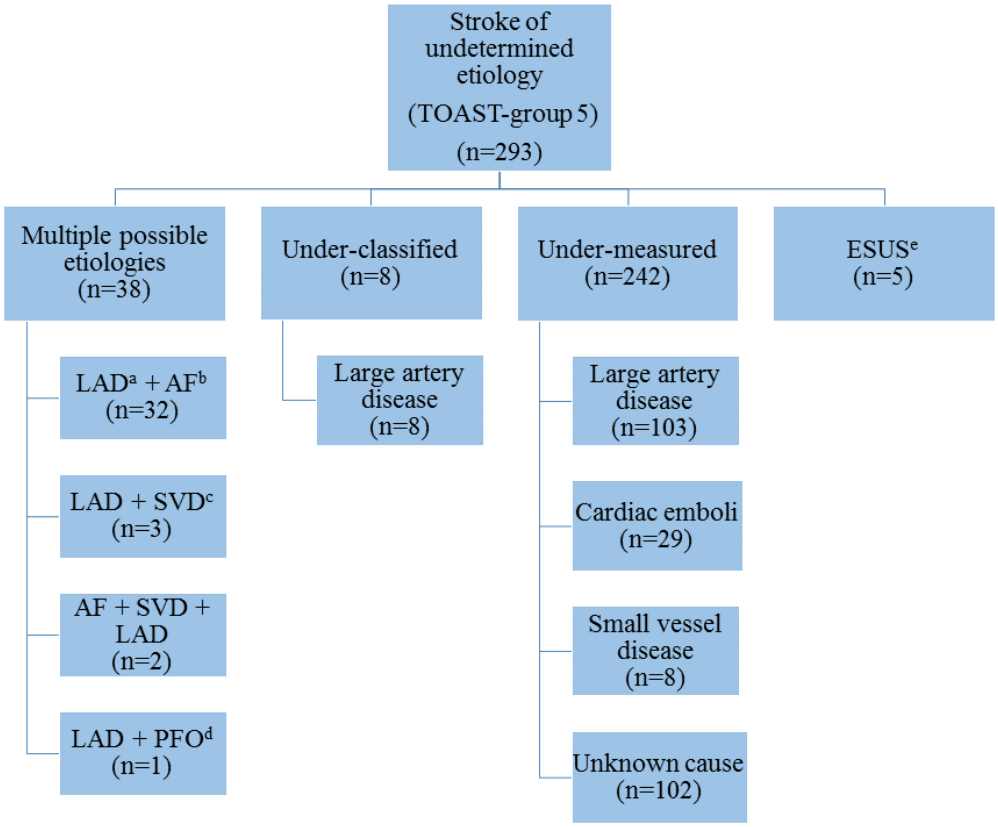

Figure 2. Classification of strokes in TOAST group 5
[a] Large artery disease, [b] Atrial fibrillation, [c] Small vessel disease, [d] Patent foramen ovale, [e]Embolic stroke of undetermined source

3.3.1 Multiple possible etiologies

Of the 293 participants in TOAST group 5, 38 had risk factors for multiple possible etiologies. Large artery disease (LAD) and atrial fibrillation (AF) were suspected etiologies in 32 participants, three had findings of SVD and LAD, and two had findings of LAD and small vessel disease (SVD) in addition to suspected AF, while one had findings of LAD and patent foramen ovale (PFO). Of the participants with multiple possible etiologies, four had received complete diagnostic investigation.

3.3.2 Under-classified

Participants with complete investigation and etiology present which did not meet the classification criteria of TOAST group 1-4 could be classified with under-classified stroke of undetermined etiology. This was the case for eight participants who had findings of LAD.

3.3.3 Under-measured

Of the participants in TOAST group 5, 242 individuals had received incomplete investigation and were classified into the category under-measured. Of those, 103 participants had findings of large artery disease of any degree, 29 were strongly suspected having AF, and eight most likely suffered from small vessel disease according to the classifying clinician. Participants with incomplete diagnostic work-up and no findings on the diagnostic evaluation they had undergone were labeled "unknown cause" (n=102).

3.3.3.1 Under-measured stroke with no possible etiology detected

The 102 participants classified into the group "unknown cause" had a mean age of 73.6 ±12.5 and were significantly younger than the 186 participants in TOAST group 5 with findings of possible etiology, who had a mean age 76.3 ±10.3 (p-value=0.049). The participants classified into the group "unknown cause" had undergone diagnostic investigations of varying degrees. ECG, 24h cardiac monitoring, CT and MRI were the only investigations carried out in 22 of the 102 cases. Two participants lacked only echocardiography, two participants lacked echocardiography and ultrasound of the external carotids, but had completed CT angiography or MR angiography (CTa/MRa) of the same arteries, and 13 participants were not evaluated with echocardiography and ultrasound of extracranial carotids.

3.3.4 Embolic stroke of undetermined source (ESUS)

Five participants had completed all diagnostic work-up without any findings, and were classified with ESUS.

3.4 Etiologies in TOAST group 5

We were able to further assign 148 of the 293 participants in TOAST group 5 with etiologies corresponding to TOAST group 1, 2 and 3. Of the remaining 145 participants, 38 had multiple possible etiologies present, 102 were under-measured with no findings on diagnostic investigations, and five were classified as ESUS. Distribution of etiology is shown in Figure 3.

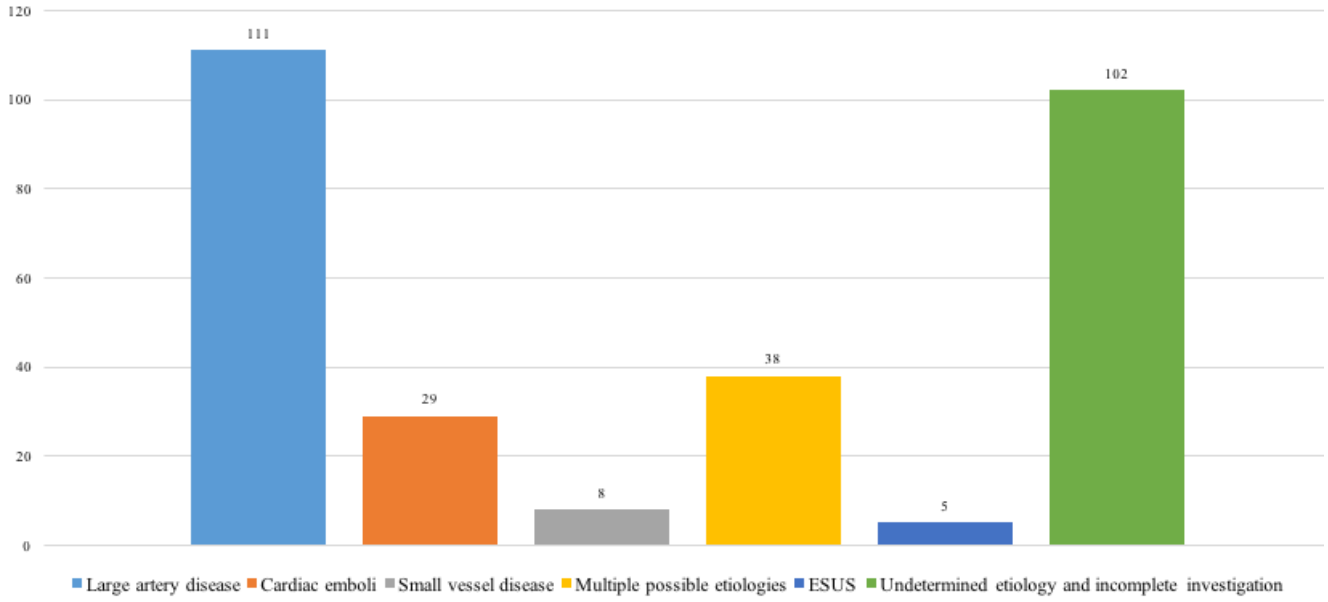

Figure 3. Etiologies in TOAST group 5.

3.5 Vascular risk factors across TOAST groups

We compared distribution of vascular risk factors in TOAST group 1, 2 and 3 with their corresponding etiologies in TOAST group 5. Distribution of vascular risk factors and use of anticoagulation at discharge is illustrated in Table 4 and Figure 4. There were no significant differences in neither vascular risk factors nor use of medication between groups with equal etiology present (Table 4).

Table 4. Vascular risk factors according to etiology in TOAST group 1,2 and 3 compared to corresponding etiologies in TOAST group 5, n (%)

	Large artery disease			**Cardiac emboli**			**Small vessel disease**		
	TOAST 1 (N=77)	LAD[a] in TOAST 5 (N=111)	P-value*	TOAST 2 (N=171)	CE[b] in TOAST 5 (N=29)	P-value*	TOAST 3 (N=147)	SVD[c] in TOAST 5 (N=8)	P-value*
Hypertension prestroke	52 (67.5)	74 (66.7)	0.901	126 (73.7)	23 (79.3)	0.520	92 (62.6)	3 (37.5)	0.156
Hypercholesterolemia prestroke	34 (44.2)	48 (43.2)	0.901	78 (45.6)	12 (41.4)	0.672	56 (38.1)	2 (25.0)	0.456
Atrial Fibrillation	5 (6.5)	0 (0.0)	0.007	125 (73.1)	16 (55.2)	0.050	5 (3.4)	0 (0.0)	0.596
Diabetes Mellitus	16 (20.8)	21 (18.9)	0.752	26 (15.2)	7 (24.1)	0.231	28 (19.0)	3 (37.5)	0.204
Previous cerebrovascular disease	31 (40.3)	50 (45.0)	0.515	75 (43.9)	10 (34.5)	0.345	62 (42.2)	3 (37.5)	0.749
Previous ischemic heart disease	18 (23.4)	31 (27.9)	0.485	39 (22.8)	5 (17.2)	0.503	18 (12.2)	1 (12.5)	0.983
Smoking	19 (25.3)	16 (14.4)	0.062	20 (11.7)	3 (10.3)	0.833	36 (24.5)	4 (50.0)	0.108
Anticoagulation at discharge	10 (13.0)	21 (18.9)	0.281	147 (86.0)	13 (44.8)	< 0.001	7 (4.8)	0 (0.0)	0.528

*Pearson's chi-square test, [a]Large artery disease, [b]Cardiac emboli, [c]Small vessel disease

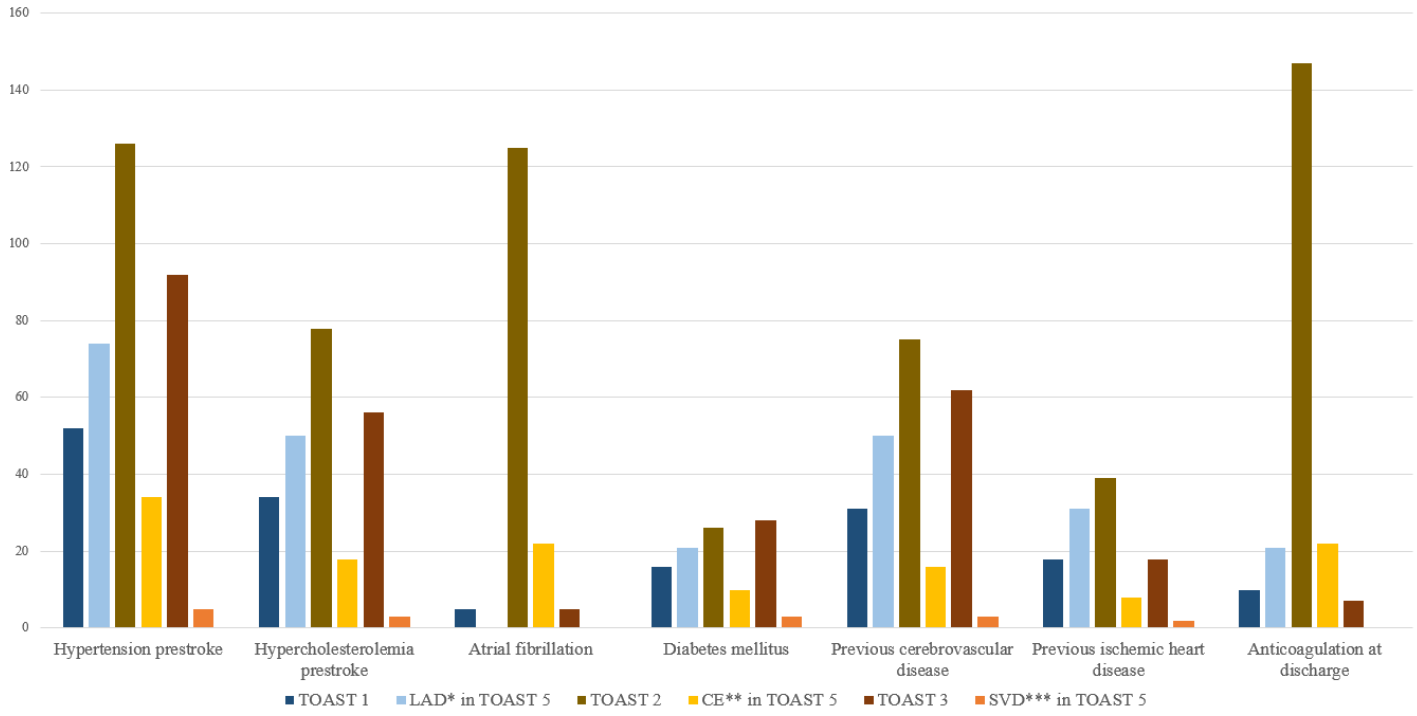

Figure 4. Risk factors and medication across etiologies.
*Large artery disease, **Cardiac emboli, ***Small vessel disease

3.5.1 The Ideal Cardiovascular Health Index

Of the 709 included participants, 474 had available information on all variables of the Ideal CVH Index. The participants were scored with one point for each of the vascular risk factors, where one point indicated having obtained the ideal values for the specific risk factor, making a score of 7 points the best possible outcome. Distribution of score across the five TOAST groups is shown in Table 5. Figure 5 illustrates the scores on the Ideal CVH Index in the subgroups of TOAST group 5.

Table 5. Score on Ideal CVH Index across TOAST-groups, n (%)

	TOAST 1 (N=46)	TOAST 2 (N=111)	TOAST 3 (N=99)	TOAST 4 (N=12)	TOAST 5 (N=206)
0-1 points	3(6.5)	6 (5.4)	6 (6.1)	0 (0.0)	8 (3.9)
2-3	22 (47.8)	49 (44.1)	36 (36.4)	4 (33.3)	97 (47.1)
≥ 4 points	21 (45.7)	56 (40.5)	57 (57.6)	8 (66.7)	101 (49.0)

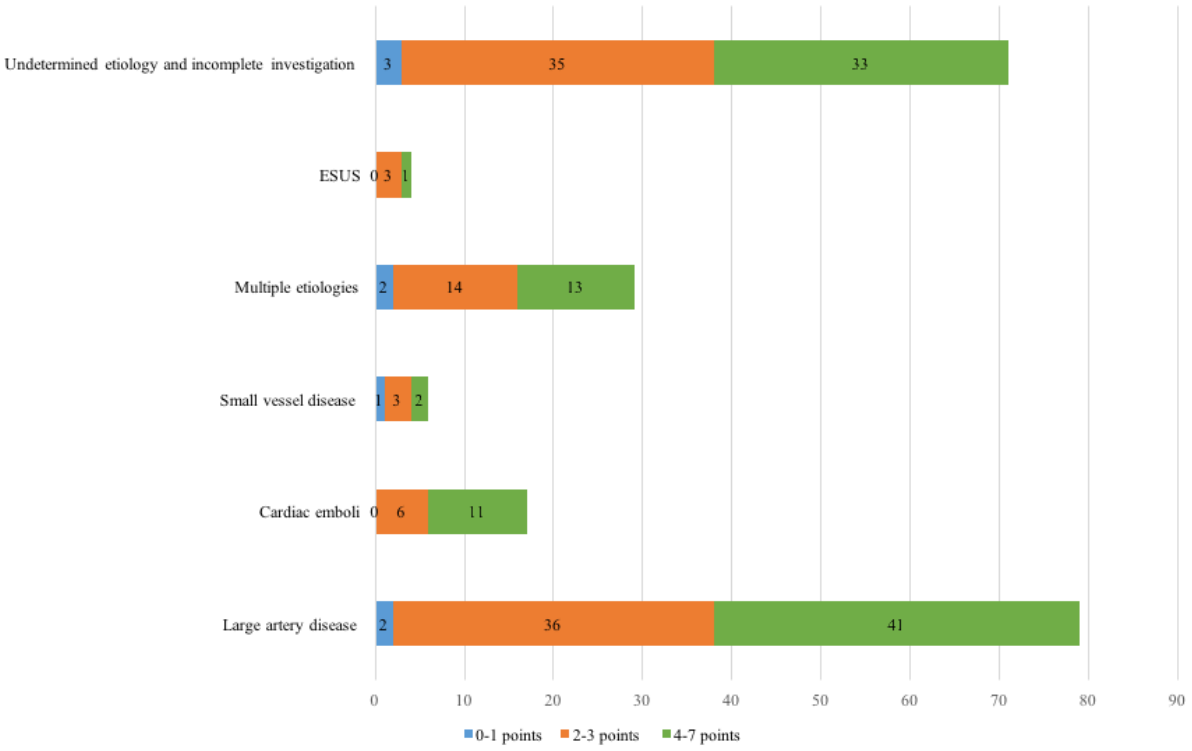

Figure 5. Ideal CVH Index in TOAST group 5.

4. Discussion

In this cross-sectional study of stroke patients classified with stroke of undetermined etiology, 148 of the 293 participants were given a possible etiology and 38 participants had two or three possible etiologies present, signifying that some type of etiology was identified in 63% of the participants. The cardiovascular risk profile did not differ between the TOAST groups with defined etiology and the participants with the corresponding possible etiology in TOAST group 5.

4.1 Discussion of results

4.1.1 Cryptogenic strokes and classification systems

As Fonseca et al. (45) pointed out, the proportion of strokes of undetermined etiology needs to be reduced, as this makes up a highly heterogeneous group of stroke patients. One important limitation of the TOAST system is that patients with more than one etiology are classified with stroke of undetermined origin. While intending to increase the accuracy of the categorization, the possibilities for extensive diagnostic investigations have been steadily increasing the last decades, and in today's practice, multiple competing etiologies are frequently detected (10).

The TOAST system may be well suited for RCTs because of its strict objective classification criteria, however, in clinical setting, valuable information on competing risk factors not recognized in the classification system, would be of interest. The strict cut offs for the TOAST classification is, in addition to the managing of the group multiple possible etiologies, a large contributor to the high percentage of strokes being classified with undetermined etiology.

The ASCO criteria (46) (A for atherosclerosis, S for small vessel disease, C for cardiac source, O for other cause (later upgraded to ASCOD with D for dissection (47))) and the Causative Classification System (CCS) (48) are newer systems for classification of stroke etiology. With CCS, the problem with multiple competing etiologies was solved by saying that the patients should be classified within the most likely cause (48), whereas the ASCO-system introduced grading of the different risk factors (46), taking a more phenotypic approach, with grading of evidence for each etiology and the possibility to mention more than one etiology. This takes into account valuable information about competing risk factors which are not included in the TOAST classification (49). Both classification systems have shown lower frequencies of

undetermined cases than the TOAST system (50-52), but the prevalence of cryptogenic strokes remains too high.

4.1.2 Large artery disease in stroke of undetermined etiology

Large artery disease was identified as possible etiology in 38% of the participants in TOAST group 5. When classifying based on possible etiology, all participants with any findings on vascular imaging, including plaque, stenosis of any degree and occlusion, were put into this group. We did not record information regarding localization of the findings; if they were ipsi- or contralateral to the stroke lesion, and because of this, we could not determine if participants with > 50% stenosis and occlusions would have fit the TOAST criteria for large artery atherosclerosis or not. As a result, participants both with occlusions and minor plaques are present in this group, making it heterogeneous. In an ideal classification system, the different degrees of large artery pathology should be given its own subgroup, similar to the proposal in the ASCOD classification (47).

Measuring stenosis degree in percent has traditionally been used to assess the risk of atherosclerotic origin in stroke. However, recent research implies that there should be more focus on carotid artery plaques which do not cause significant stenosis as a possible source of emboli (53). Carotid plaque with a stenosis degree < 50% has been found more prevalent ipsilateral to the stroke site in ESUS (54), suggesting an association between large but non-stenotic carotid plaque and cryptogenic stroke. Bogiatzi et al. (55) proposed a new classification system incorporating plaque burden into the large-artery atherosclerosis group. Doing this, they found that fewer strokes were classified with undetermined etiology.

4.1.3 Atrial fibrillation in stroke of undetermined etiology

Atrial fibrillation (AF), especially paroxysmal, has been thought to be a large contributor to cryptogenic stroke (56). AF of any length of time is considered an important finding, as even a short period predicts subsequent episodes, and is an independent risk factor of recurrent stroke (57). Cardiac monitoring for 24h is recommended in the current guideline, and in cases where there is a strong suspicion of paroxysmal AF, extensive monitoring is recommended by some guidelines (58).

Recent research recommend an even more thorough cardiac monitoring with an insertable cardiac monitor for up to one year of monitoring (59). In the present study, 12% were classified

with atrial fibrillation as possible etiology. Holter recording for 24 hours detects paroxysmal AF in 2-4% of all strokes of undetermined etiology, but increasing the monitoring period to 24-72 hours increased the incidence of AF to 18% (60). Detecting AF has significant clinical implications, as it is of importance in the choice of secondary prevention treatment, as oral anticoagulation is a better option than antiplatelet for the reduction of stroke recurrence in patients with AF (60).

4.1.4 Underestimation of ESUS prevalence

The percentage of strokes of undetermined etiology classified as ESUS was found to be 28% in a Polish cohort (61). In this study, only 1.7% of the participants could be classified as ESUS, and there is reason to believe that the ESUS prevalence is underestimated. 102 of the participants had incomplete investigation with no findings on the imaging that had been done. Most of them lacked echocardiography, and some of the participants in this group may have been classified with ESUS if all investigations had been completed without pathology on echocardiography. However, according to Norwegian guidelines (58), echocardiography should only be done in cases where there is strong suspicion of cardioembolic stroke. This is probably contributing to why only a small number of participants had received this investigation, which led to few participants being available for ESUS diagnosis.

ESUS patients on average are younger than the overall stroke population (22), and in this study, the group with incomplete investigation and unknown etiology was significantly younger than the participants in TOAST-group 5 who were given a possible etiology. However, a substantial amount of the participants had completed few diagnostic evaluations, and as such, assumptions about ESUS prevalence should be carefully made. As a result of this, it is reasonable to assume that the group with unknown etiology consists of a heterogeneous population.

Patients with intra or extracranial stenosis < 50% and complete investigation with no other findings, could, according to ESUS criteria (15), be classified with ESUS. This means that there is some overlap between ESUS and under-classified large artery disease. Seeing as we in the present study, were more interested in classifying according to possible etiology, we chose to place the eight participants with large artery disease with stenosis < 50% in the group for under-classified large artery disease. However, we recognize that this might has been dealt with differently in other studies.

Since the TOAST classification was introduced, the possibilities for extensive diagnostic investigations have been steadily increasing, and it has become more of a choice of whom to follow up further. Globally, there is a trend of lesser diagnostic testing being done on older individuals (62), and this seems to been reflected in the present study. In older, comorbid patients, it is important to consider the potential harm the diagnostic evaluations could create and what the patient would gain from the findings, which is a probable cause of this difference.

4.1.5 Vascular risk factors in cryptogenic stroke

Compared to the other TOAST groups, strokes of undetermined etiology have fewer makers of atherosclerotic disease (36). In the present study, however, we found no differences in vascular risk factors between the TOAST groups 1-4 and TOAST group 5. This might be due to the heterogeneous population categorized with stroke of undetermined etiology. In studies on cryptogenic stroke, the population is often younger, hence the fewer vascular risk factors, whereas the population of the present study does not differ much from the general stroke population in age (11).

All strokes which remained with undetermined etiology and lacked one or more diagnostic investigation, were classified into the same group, most likely mixing up younger patients referred to outpatient echocardiography and extended cardiac monitoring who are candidates for ESUS diagnosis with older, comorbid patients who will not gain much from extensive diagnostic investigations. Because of this, our results might have been different if we had available information from participants' visits to outpatient clinic and general practitioners in the months following discharge.

4.1.6 Importance of stroke etiology in choice of secondary preventive strategies

Risk of stroke recurrence has kept steadily at 25% (63, 64), and secondary prevention is thus continuing to be of importance in stroke care (65). In order to give the correct preventative treatment, the etiology needs to be known (66), as different etiology calls for different treatment strategies. A stroke patient with a cardiac embolus will have little to no effect of antiplatelet treatment, whereas another patient with large artery stenosis in the ipsilateral carotid will have a superior benefit of platelet treatment compared to anticoagulants.

So far, no difference in stroke recurrence has been detected between ESUS patients treated with anticoagulation and antiplatelet therapy (28), and there is still no consensus in secondary prevention in ESUS patients.

4.2 Methodological aspects

4.2.1 Study design

This study has some limitations. Due to the cross-sectional design, no conclusions can be drawn regarding the prognosis and follow-up of the study participants. Only information from index stay was available, and some of the participants may have been referred for further investigations at outpatient clinics after hospital discharge. Further, we did not have information side localization of the large artery findings, and we could not draw any conclusions regarding the causation between the findings on ultrasound or angiography and stroke lesion. Some of the etiologic subgroups were quite small samples, and in a few cases the comparisons were done between groups which varied in size. Lastly, this study was conducted in Norway, and incidence of stroke etiology may differ from other parts of the world.

There ae also some strengths. This was a large multicenter study including participants across different health regions in Norway, and we had extensive information on imaging and cardiac monitoring, in addition to information on previous disease and comments from the classifying clinicians, to utilize in the classification of etiology in TOAST group 5.

4.2.2 Diagnostic evaluation of the participants

There may be some differences between the hospitals regarding referral to diagnostic evaluations. As it is not always justifiable in a cost-benefit perspective referring nursing home residents to extensive diagnostic evaluations. This could contribute to the incomplete investigation of 94% of the participants with undetermined etiology. Only a small portion of TOAST group 5 was available for the ESUS classification, as most of the study participants with no findings of etiology on the available diagnostic work-up, were lacking two or more investigations.

Doing research in clinical practice, there is not always an agreement between what the best clinical practice is and what will serve research the best. As a result, the participants have been followed up with investigations relevant for their diagnosis and further treatment, not for research purposes. It is also a question of cost-benefit in health care services, as there is little to

gain in doing extensive diagnostic investigations when it will have no consequences for the patient treatment.

4.2.3 Measuring vascular risk factors

Taking into account other measurements than just the traditional risk factors, such as arterial stiffness and endothelial function, to compute a vascular risk score, may be a better measure of the individual risk than risk charts as the Ideal Cardiovascular Health (CVH) Index (67). However, the main focus in this study was to identify differences on a group level, and by evaluating all participants with the same criteria, we found this one-time measuring adequate. More extensive risk factor calculators like the Framingham Risk Score (68) with more variables and a more sophisticated scoring algorithm would most likely have given a more in-depth insight into each participant's vascular risk.

It is important to take into account that the scoring in the Ideal CVH Index is only a risk assessment, and not equivalent to diagnosis. E.g. zero point on the blood glucose variable, does not signify that the participant necessarily has diabetes mellitus, only that they have values indicating increased risk of cerebrovascular disease. Pase et al. (69) found that score on the Ideal CVH Index was associated with both cerebrovascular disease and dementia, underlining the utility of a simple yet informative score in system as the Ideal CVH Index.

The only blood pressure measurements available were recorded in the acute phase of stroke and were evaluated to not be representable enough to be included in the risk calculation. Blood glucose, which is prone rise in acute illness, was measured in the acute phase, which might have given an overestimation of prevalence.

A limitation is that all measurements were recorded in the acute phase of the stroke, making some of the measurements unsuitable for calculation of the Ideal CVH Index, and we had to modify the score to suit our available measurements. Physical activity and diet were self-reported, which is also a weakness, as this does not always reflect the actual situation.

In addition, only 67% of the included participants had available measures for all the variables needed to calculate the risk factor score, resulting in a substantial portion of missing values in the analyses.

4.3 Conclusion

The TOAST group of undetermined etiology makes up a heterogeneous population with various etiologies present. Especially large artery disease and cardiac emboli seems to be prevalent in this group. We found no significant differences in vascular risk factors when comparing the etiologies in TOAST group 1, 2 and 3 with the corresponding etiologies in TOAST group 5. Novel classification systems in line with modern diagnostic evaluations are needed for use both in research and clinical setting in order to reduce the prevalence of stroke of undetermined etiology.

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Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: ☐☐☐☐2☐☐☐☐

Tester: ☐☐☐

Pasient-ID: ☐☐☐☐☐☐

Metode for innhenting av data:

☐ Intervju av pasient ☐ Intervju av pårørende ☐ Intervju av helspersonell ☐ Sykejournal ☐ Telefonintervju

Opplysninger ikke tilgjengelig:

BAKGRUNNSINFORMASJON

Tidspunkt for symptomdebut:
dag/måned/år/timer/minutter

☐☐☐☐2☐☐☐☐ ☐☐☐☐

Oppvåkningsslag

☐ Ja ☐ Nei ☐ Ukjent

Tidspunkt for innleggelse:
dag/måned/år/timer/minutter

☐☐☐☐2☐☐☐☐ ☐☐☐☐

Utskrivingsdato: *dag/måned/år*

☐☐☐☐2☐☐☐☐

Boligforhold før slaget:
dag/måned/år

☐ Egen bolig uten hjemmesykepleie
☐ Egen bolig med hjemmesykepleie
☐ Omsorgsbolig
☐ Sykehjem
☐ Ukjent

Bosituasjon:

☐ Alene
☐ Sammen med noen (f.eks ektefelle/samboer, søsken, barn)
☐ Institusjon
☐ Ukjent

Sivil status:

☐ Gift eller samboer
☐ Enslig
☐ Enke eller enkemann
☐ Ukjent

Kjønn:

☐ Kvinne
☐ Mann

Dominant hånd:

☐ Høyre
☐ Venstre
☐ Ingen dominant side
☐ Ukjent

Etnisitet:

☐ Kaukasisk ☐ Afrikansk ☐ Asiatisk ☐ Latinamerikansk

Fødeland: _____

Utdanningsnivå:
(Kurs og internopplæring teller ikke som utdanning)

Formell skolegang (antall år) ☐☐

Utdanning

☐ Ufaglært
☐ Fagbrev
☐ Høyskole/universitet

Yrke/tidligere yrke: _____

Har noen i familien symptomer som kan tyde på demens?

	Ja	Nei	Ukjent
a) Førstegangsslektning *(foreldre, søsken, barn)*	☐	☐	☐
b) Andregradsslektning *(besteforeldre, foreldres søsken, søskenbarn, halvsøsken)*	☐	☐	☐

Hva lever du av?

☐ Arbeidsinntekt ☐ Uførepensjon
☐ Sykepenger ☐ Ektefelles inntekt
☐ Alderspensjon ☐ Annet

LEVEVANER FØR SLAGET

1) Røykestatus:

☐ Aldri ☐ Røyker ☐ Eksrøyker (røykfri > 1 mnd.) ☐ Ukjent

2) Alkoholforbruk:

1 alkoholenhet = En flaske (33 cl) pils på 4,5 vol % = Et lite glass vin (12,5 cl) på 12 vol % = Et enda mindre glass sterkvin (7,5 cl) 20 vol % = Et svært lite glass brennevin (4 cl) 40 vol %

Glassene rommer ofte mer. Skal du telle antall alkoholenheter, så vurder også størrelsene på glassene: En halvliter øl = 1,5 enhet alkohol, et stort glass vin (17,5 cl) = 1,5 enhet alkohol

Hvor mange enheter øl, vin eller brennevin drikker du vanligvis i løpet av 2 uker? (Regn ikke med lettøl) (Sett 0 hvis du ikke drikker alkohol)

	øl	vin	brennevin
Antall enheter	☐☐	☐☐	☐☐

Hvor ofte drikker du 5 enheter eller mer av øl, vin eller brennevin ved samme anledning?

☐ Aldri ☐ Månedlig ☐ Ukentlig ☐ Daglig

3) Kosthold før slaget

Tar du omega-3 eller tran?

☐ Ja

☐ Nei

	Aldri	1-2 ganger pr uke	3-4 ganger pr uke	5-6 ganger pr uke	Daglig
Hvor ofte har du spist fisk de siste 6 måneder?	☐	☐	☐	☐	☐
Hvor ofte har du spist grønnsaker de siste 6 måneder?	☐	☐	☐	☐	☐

4) Fysisk aktivitet før slaget

Hvor ofte drev du mosjon/fysisk aktivitet før slaget?
Med mosjon mener vi at du f. eks. går tur, går på ski, svømmer eller driver med trening/idrett
(Ta et gjennomsnitt av de siste 6 måneder)

☐ Aldri
☐ Sjeldnere enn en gang i uka
☐ En gang i uka
☐ 2-3 ganger i uka
☐ Omtrent hver dag

Dersom du driver slik mosjon, så ofte som en eller flere ganger i uka; hvor hardt mosjonerer du? (Ta et gjennomsnitt)

☐ Tar det rolig uten å bli andpusten eller svett
☐ Tar det så hardt at jeg blir andpusten og svett
☐ Tar meg nesten helt ut

Hvor lenge holder du på hver gang?
(Ta et gjennomsnitt)

☐ Mindre enn 15 min ☐ 30 min - 1 time
☐ 15-29 min ☐ Mer enn 1 time

Har du vanligvis minst 30 minutter fysisk aktivitet daglig på arbeid og/eller i fritida?

☐ Ja ☐ Nei

FUNKSJON FØR SLAGET

Kunne du gå 200 meter før slaget?

☐ Ja ☐ Nei

Kunne du gå opp/ned trapp en etasje

☐ Ja ☐ Nei

Har du i løpet av de siste 12 månedene hatt noen fall, inkludert om du har snublet eller glidd, slik at du har mistet balansen og havnet i bakken eller på gulvet uten å ville det?
Uavhengig av årsak eller om du har skadet deg.

☐ Ja ☐ Nei

Hvis ja, hvor mange fall? ☐ Ett fall ☐ To fall ☐ Flere fall

Skader ved fall ☐ Ja ☐ Nei

Hvis ja, evt.kommentar:

Fatigue før slaget

Var du plaget med utmattelse før slaget?

☐ Ja ☐ Nei

Hvor lenge var du plaget med utmattelse?

☐ < 1 uke ☐ < 3-6 måneder ☐ Vet ikke
☐ < 3 måneder ☐ > 6 måneder

Har du hatt ufrivillig vekttap de siste 6 måneder før slaget? ☐ Ja ☐ Nei Hvis ja: antall kilogram ☐☐

Har du hatt urinlekkasje eller problemer med å tømme blæra før slaget?

☐ ingen problemer
☐ lekkasje
☐ problemer med å tømme blæra
☐ annet, f. eks. RIK eller permanent kateter

Hvis annet, spesifiser:

Hvor alvorlig har vannlatingsproblemet vært?

☐ Mildt
☐ Moderat
☐ Alvorlig

TIDLIGERE SYKDOMMER

Tidligere cerebrovaskulær sykdom

☐ Ingen tidligere cerebrovaskulær sykdom
☐ Hjerneinfarkt
☐ TIA
☐ Hjerneblødning
☐ Hatt cerebrovaskulær sykdom, men ukjent om blødning, infarkt eller TIA
☐ Usikker

Alder for første hjerneslag: (infarkt/blødning) ☐☐

Tidligere TIA

☐ Ingen tidligere TIA
☐ TIA i løpet av siste uke
☐ TIA 1-4 uker før slaget
☐ TIA 4-12 uker før slaget
☐ TIA over 12 uker før slaget
☐ Usikkert om pasienten har hatt TIA

Hjerte-karsykdom

☐ Ingen tidligere hjerte-karsykdom
☐ Hjerteinfarkt
☐ Angina pectoris
☐ Atrieflimmer bekreftet med EKG nå eller tidligere (også paroksystisk atrieflimmer)
☐ Hjertesvikt
☐ Gjennomgått karkirurgi (halskar, aorta, arterier i underekstremiteter)
☐ Hypertensjon før debut av slag
☐ Claudicatio intermittens (perifer vaskulær sykdom)
☐ Usikker

Hvis hjerteinfarkt eller angina: gjennomgått kardiologisk intervensjon?

☐ PCI (innsettelse av stent)
☐ CABG (koronar bypass)
☐ Nei
☐ Usikker

Hvis PCI eller CABG; måned og år for (første) kardiologiske intervensjon

☐☐☐☐☐☐

Hvis gjennomgått karkirurgi; måned og år for karkirurgisk intervensjon

☐☐☐☐☐☐

Thyroideasykdom ☐ Ja ☐ Nei

Vitamin B12-mangel, folatmangel ☐ Ja ☐ Nei

Hyperkolesterolemi før debut av slag (behandlet med kolesterolsenkende eller påvist totalkolesterol ≥ 6 mmol/l) ☐ Ja ☐ Nei

Diabetes mellitus (nyoppdaget eller tidligere diagnostisert) ☐ Ja ☐ Nei

Hvis diabetes mellitus: kjente komplikasjoner

☐ Ingen kjente komplikasjoner ☐ Øyne ☐ Nyrer ☐ Nevropati

Pasienten har betydelig nedsatt:

- ☐ Syn
- ☐ Hørsel
- ☐ Både syn og hørsel
- ☐ Ingen
- ☐ Usikker

Psykiatrisk lidelse

- ☐ Ingen behandlingstrengende psykiatrisk lidelse
- ☐ Tidligere behandlingstrengende depresjon
- ☐ Pågående behandlingstrengede depresjon
- ☐ Demens (også ikke behandlingstrengende)
- ☐ Annen behandlingstrengende psykisk sykdom
- ☐ Usikker

Andre sykdommer

- ☐ Ingen andre sykdommer
- ☐ Alvorlig kronisk lungesykdom
- ☐ Nyresykdom (er det påvist nedsatt nyrefunksjon?)
- ☐ Systemsykdom (revmatiske sykdommer, betennelsestilstander)
- ☐ Leversykdom
- ☐ Ulcus pepticum
- ☐ Paraplegi
- ☐ HIV
- ☐ Kreftsykdom med eller uten spredning
- ☐ Tidligere anmerket alkoholmisbruk i pasientjournal
- ☐ Tidligere opplysninger om narkotikabruk i journal
- ☐ Annen sykdom av betydning for funksjonsnivå
- ☐ Usikker

Hvis leversykdom, alvorlig? (cirrhose)

☐ Ja ☐ Nei

Hvis kreftsykdom, spredning?

☐ Ja ☐ Nei

Hvis narkotikabruk, hvilket/hvilke?

_____

Andre opplysninger

_____

STATUS I AKUTTFASEN

Sidelokalisasjon av symptomer

☐ Høyre ☐ Venstre ☐ Bilateralt

☐ Ikke relevant ☐ Ukjent

Trombolytisk behandling ☐ Ja ☐ Nei

Starttidspunkt for trombolyse (dag/måned/år/timer/minutter)

☐☐☐☐ 2 0 ☐☐ ☐☐☐☐

Klassifisering av slaget

Oxfordshire klassifikasjon

☐ TACI ☐ PACI ☐ LACI

☐ POCI ☐ Blødning ☐ Uklassifiserbar

Trombektomi ☐ Ja ☐ Nei

Starttidspunkt for trombolyse (dag/måned/år/timer/minutter)

☐☐☐☐ 2 0 ☐☐ ☐☐☐☐

TOAST klassifikasjon, hvis hjerneinfarkt

☐ Aterosklerose (storkarsykdom) ☐ Kardial emboli ☐ Småkar-sykdom

☐ Annen årsak ☐ Ukjent årsak/ flere mulige årsaker

Annen sannsynlig årsak (tillegg til TOAST – velg én)

☐ Disseksjon ☐ Protrombotisk tilstand ☐ Graviditet

☐ Atrieflimmer ☐ PFO

☐ Hjerteinfarkt ☐ Endokarditt ☐ Storkarsykdom

☐ Småkarsykdom ☐ Klaffefeil

UNDER SYKEHUSOPPHOLDET

Blodtrykk og puls ved innkomst ☐☐☐/☐☐☐ ☐☐☐

Blodtrykk og puls dag 1 ☐☐☐/☐☐☐ ☐☐☐

Oksygenmetning i % i løpet av første 24 t. ☐☐☐

Blodtrykk og puls dag 7/utreisedag ved utreise før dag 7. ☐☐☐/☐☐☐ ☐☐☐

Aktivitetsbrikke under oppholdet

☐ Ja ☐ Nei

Årsak hvis nei:

_____

Vekt i kg ☐☐☐

Høyde i cm ☐☐☐

Midjeomkrets i cm ☐☐☐

Hofteomkrets i cm ☐☐☐

Hvis det ikke er mulig å veie/måle, angi årsak:

_____

Komplikasjoner

Kramper

☐ Ja ☐ Nei ☐ Usikker

Nevrologisk progresjon

☐ Ja ☐ Nei ☐ Usikker

Infeksjon behandlet med antibiotika

☐ Ja ☐ Nei ☐ Usikker

Hvis ja: ☐ UVI ☐ Luftveisinfeksjon ☐ Annet

Fall

☐ Ja ☐ Nei ☐ Usikker

Har pasienten kliniske tegn på neglekt?

☐ Ja ☐ Nei ☐ Usikker

Fremstår pasienten som skrøpelig?

☐ Ja ☐ Nei ☐ Usikker

BLODPRØVER

Elektrolytter

Natrium (Na) ☐☐☐

Kalium (K) ☐,☐

Kalsium (Ca) ☐,☐☐

Hematologi

Hemoglobin (Hb) ☐☐,☐

Leukocytter (Leuk) ☐☐,☐

Trombocytter (Tromb) ☐☐☐

Lipider

Total kolesterol ☐☐,☐

LDL ☐,☐

HDL ☐,☐

Triglycerider ☐,☐

Annet

Glucose ☐☐,☐

TSH ☐☐,☐

Fritt T4 ☐☐,☐

HbA1c ☐☐,☐

INR ☐,☐

Kreatinin ☐☐☐

CRP ☐☐☐

Høy-sensitiv CRP ☐,☐☐

Troponin T ☐☐☐☐

Vitamin B12 ☐☐☐

Folat ☐☐

Homocystein ☐☐,☐

Blodprøver tatt til biobank?

☐ Ja ☐ Nei

Hvis ja, løpenummer i biobank:

☐

BILDEDIAGNOSTIKK OG ANDRE MEDISINSKE UNDERSØKELSER

EKG ☐ Ja ☐ Nei

Rytme:

Sinusrytme ☐ Ja ☐ Nei

Atrieflimmer/flutter ☐ Ja ☐ Nei

Ventrikkeltachykardi ☐ Ja ☐ Nei

EKG-kompleks:

Normalt ☐ Ja ☐ Nei

Tidligere hjerteinfarkt ☐ Ja ☐ Nei

Akutt infarkt ☐ Ja ☐ Nei

Venstre-ventrikkel hypertrofi ☐ Ja ☐ Nei

ST-depresjon eller T-inversjon i minst 2 tilgrensende avledninger ☐ Ja ☐ Nei

Telemetri ☐ Ja ☐ Nei

Hvis ja:

Sinus ☐ Ja ☐ Nei

Atrieflimmer/flutter ☐ Ja ☐ Nei

Ventrikkeltachykardi ☐ Ja ☐ Nei

CT utført ☐ Ja ☐ Nei

Hvis ja:

Ferskt infarkt ☐

Gamle infarktforandringer ☐

Kronisk iskemi ☐

Blødning ☐

Tumor ☐

Negativt ☐

MR utført ☐ Ja ☐ Nei

Hvis ja:

Ferskt infarkt ☐

Gamle infarktforandringer ☐

Kronisk iskemi ☐

Blødning ☐

Tumor ☐

Negativt ☐

☐ Evt. Fazekas grad

Ultralyd ekstrakranielle kar ☐ Ja ☐ Nei

Hvis ja:

Stenose ☐ Ja ☐ Nei

Stenosegrad: _____ %

Okklusjon ☐ Ja ☐ Nei

Disseksjon ☐ Ja ☐ Nei

Plakk ☐ Ja ☐ Nei

Hvis plakk: ☐ harde ☐ bløte ☐ begge deler ☐ uspesifisert

Ekstrakraniell CT/MR angio utført? ☐ Ja ☐ Nei

Hvis ja:

Stenose ☐ Ja ☐ Nei

Stenosegrad: _____ %

Okklusjon ☐ Ja ☐ Nei

Disseksjon ☐ Ja ☐ Nei

Plakk ☐ Ja ☐ Nei

Annet (evt. funn, f.eks. aneurismer):

_____

Intracerebral CT/MR angio utført? ☐ Ja ☐ Nei

Hvis ja:

Stenose ☐ Ja ☐ Nei

Stenosegrad: _____ %

Okklusjon ☐ Ja ☐ Nei

Disseksjon ☐ Ja ☐ Nei

Plakk ☐ Ja ☐ Nei

Annet (evt. funn, f.eks. aneurismer, karmalformasjoner):

_____

Bildediagnostikk hjerte ☐ Ja ☐ Nei

Evt. hvilke patologiske funn:

_____

☐ Transthorakal ekkokardiografi

☐ Transøsofagal ekkokardiografi

Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

☐ Intervju av pasient ☐ Intervju av pårørende ☐ Intervju av helsepersonell ☐ Sykejournal ☐ Telefonintervju

Opplysninger mangler:

Hvordan håndteres legemidlene? ☐ selvhjulpen ☐ vha. pårørende ☐ hjemmesykepleie ☐ ingen faste medikamenter

MEDIKAMENTLISTE

FASTE MEDIKAMENTER preparatnavn		Dose ved innkomst	ATC-KODE							
1										
2										
3										
4										
5										
6										
7										
8										
9										
10										
11										
12										
13										
14										

MEDIKAMENTER VED UTREISE preparatnavn		Dose	ATC-KODE							
1										
2										
3										
4										
5										
6										
7										
8										
9										
10										
11										
12										
13										
14										

Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

☐ Intervju av pasient ☐ Intervju av pårørende ☐ Intervju av helsepersonell ☐ Sykejournal ☐ Telefonintervju

Opplysninger ikke tilgjengelig:

MODIFIED RANKIN SCALE

	Før hjerneslaget	**Dag 7** / Utreise hvis utskriving før dag 7
Dato		
Skår		

0. **Ingen symptomer i det hele tatt.**
Pasienten skal ikke ha noen begrensninger eller symptomer.

1. **Ingen betydningsfull funksjonssvikt til tross for symptomer; klarer å utføre alle oppgaver og aktiviteter som før.**
Pasienten har noen symptomer, enten fysiske eller kognitive, f. eks affeksjon av språk/tale, evne til å lese/skrive, fysisk mobilitet, sensibilitet, syn, svelg, humør, men kan fortsette å ta del i alt tidligere arbeid, sosial- eller fritidsaktiviteter. Det avgjørende spørsmålet for å skille mellom 1 og 2 kan være: Klarer pasienten alle aktiviteter som han før gjorde mer enn månedlig?

2. **Lett funksjonssvikt; klarer ikke å utføre alle aktiviteter som før, men klarer sine daglige gjøremål.**
Pasienten kan ikke lenger gjøre en del av de aktivitetene som han/hun tidligere vanligvis har gjort. (F. eks kjøre bil, danse, lese, arbeide), men klarer fortsatt å ta vare på seg selv uten hjelp fra andre fra dag til dag. Pasienten kan klare påkledning, forflytning, matlaging/spisesituasjon, toalettbesøk, lage enkle måltider, handle og reise i lokalmiljøet uten å måtte motta hjelp eller tilsyn fra andre. Pasienten skal kunne være overlatt til seg selv alene hjemme i en uke eller mer uten bekymring.

3. **Moderat funksjonssvikt; trenger noe hjelp, men går uten hjelp.**
Pasiebten trenger ikke hjelp til forflytning/gange (selvstendig forflytning med og uten hjelpemiddel som stokk, rullator). Klarer påkledning, toalettbesøk og å spise etc, men trenger hjelp til mer komplekse aktiviteter. Noen andre må handle, lage mat, vaske – og må besøke pasienten oftere enn ukentlig for å sørge for at disse aktivitetene er gjennomført. Assistansen kan være fysisk eller rådgivende, f. eks pasienten trenger tilsyn eller motivering for å klare finansielle gjøremål.

4. **Alvorlig funksjonssvikt; klarer ikke å gå uten hjelp og klarer ikke å ivareta sine grunnleggende behov uten hjelp.**
Pasienten må ha hjelp fra andre til noen daglige aktiviteter, f. eks gange, påkledning, toalett, spise. Pasienten blir besøkt minst en og vanligvis to eller flere ganger daglig, eller må bo i nærheten av en hjelper. For å skille 4 fra grad 5 – ta stilling til om pasienten kan bli latt alene for moderate perioder i løpet av dagen.

5. **Svært alvorlig funksjonssvikt; sengeliggende og trenger konstant tilsyn og hjelp.**
Noen andre må alltid være tilgjengelig på dagtid og noen ganger i løpet av natten – denne trenger ikke være en sykepleier.

6. **Død**.

Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

- ☐ Intervju av pasient
- ☐ Intervju av pårørende
- ☐ Intervju av helsepersonell
- ☐ Sykejournal
- ☐ Telefonintervju

☐ Testbar ☐ Ikke testbar

Årsak til ikke testbar:

☐ Afasi ☐ Redusert bevissthet ☐ Medisinsk ustabil

☐ Forstår ikke norsk ☐ Redusert kognitiv funksjon ☐ Demens

Annen årsak: _____

BARTHEL INDEX

	Dag 7 / utreise hvis utskriving før dag 7
DATO	
SPISING **10.** Helt selvhjulpen. Kan bruke nødvendige hjelpemidler og spiser innen rimelig tid **5.** Må ha hjelp til enkelte funksjoner, f. eks å skjære **0.** Totalt avhengig av andre for å kunne spise	
BADING/DUSJ **5.** Helt selvhjulpen **0.** Trenger hjelp	
PERSONLIG HYGIENE **5.** Selvhjulpen. Klarer å vaske ansikt, kamme hår, børste tenner og barbering **0.** Trenger hjelp til en eller flere funksjoner	
PÅKLEDNING **10.** Selvhjulpen. Klarer å knyte sko, kneppe knapper **5.** Trenger hjelp, men klarer halvparten innen rimelig tid **0.** Trenger hjelp til mer enn halvparten	
TARMKONTROLL **10.** Kontinent. Klarer selv eventuelt å sette stikkpille/klyx **5.** Nedsatt kontroll og enkelte "uhell". Trenger hjelp til eventuelt å sette stikkpille/klyx **0.** Helt inkontinent eller hyppige "uhell"	
BLÆREKONTROLL **10.** Kontinent. Selvhjulpen og holder seg tørr ved bruk av uridom **5.** Nedsatt kontroll og enkelte "uhell" og holder seg tørr med uridom o.l. men trenger hjelp til å bruke dette **0.** Helt inkontinent eller trenger permanent kateter	
TOALETTBESØK **10.** Selvhjulpen på toalett/dostol eller bekken. Ordner klær, tørker seg, spyler toalettet eller tømmer bekken **5.** Trenger hjelp til klær, papir etc. **0.** Kan ikke bruke toalett/dostol	
STOL/SENG - FORFLYTNING **15.** Selvhjulpen. Klarer også å låse rullestol og bevege forstøtte **10.** Klarer forflytning med litt hjelp eller tilsyn **5.** Kan sitte men må ha mye hjelp ved forflytning **0.** Kan ikke sitte. Sengeliggende eller må løftes	
MOBILITET **15.** Klarer å gå 50 meter. Kan bruke stokk eller krykke, men ikke rullator **10.** Kan gå 50 meter med rullator og støtte/tilsyn av en person **5.** Kan ikke gå, men kan kjøre rullestol uten hjelp/tilsyn i 50 meter **0.** Kan ikke kjøre rullestol uten hjelp	
TRAPPEGANG **10.** Selvhjulpen med eller uten bruk av hjelpemidler **5.** Trenger hjelp/tilsyn av en person **0.** Kan ikke gå i trapp	
SUM: (totalt 100 poeng)	

Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

☐ Intervju av pasient ☐ Intervju av pårørende ☐ Telefonintervju

Opplysninger mangler:

NOTTINGHAM I-ADL

SE NØKKEL FØR UTFYLLING

Få fram det personen faktisk gjør, og har gjort de siste to ukene (ikke hva vedkommende tror eller ønsker han/hun kan gjøre).

SKÅRSKALA

Nei	0
Med hjelp	1
Alene med vansker	2
Alene	3

	Nei	Med hjelp	Alene med vansker	Alene
MOBILITET				
1. Går du omkring utendørs?	☐	☐	☐	☐
2. Går du i trapper?	☐	☐	☐	☐
3. Kommer du deg inn og ut av bilen?	☐	☐	☐	☐
4. Går du på ujevnt underlag?	☐	☐	☐	☐
5. Krysser du veier?	☐	☐	☐	☐
6. Reiser du med offentlig transport?	☐	☐	☐	☐
7. Spiser du selv?	☐	☐	☐	☐
8. Lager du varm drikke?	☐	☐	☐	☐
9. Tar du med varme drikker fra ett rom til et annet?	☐	☐	☐	☐
10. Tar du oppvasken?	☐	☐	☐	☐
11. Lager du et enkelt varmt måltid til deg selv?	☐	☐	☐	☐
12. Håndterer du egne penger når du er ute?	☐	☐	☐	☐
13. Tar du småvask/håndvask?	☐	☐	☐	☐
14. Gjør du husarbeidet selv?	☐	☐	☐	☐
15. Gjør du innkjøpene dine selv?	☐	☐	☐	☐
16. Tar du en hel klesvask?	☐	☐	☐	☐
17. Leser du aviser eller bøker?	☐	☐	☐	☐
18. Bruker du telefonen?	☐	☐	☐	☐
19. Skriver du brev?	☐	☐	☐	☐
20. Går du ut for sosialt samvær?	☐	☐	☐	☐
21. Steller du din egen hage?	☐	☐	☐	☐
22. Kjører du bil?	☐	☐	☐	☐

Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

- ☐ Klinisk us.
- ☐ Sykejournal
- ☐ Testbar
- ☐ Ikke testbar

Årsak til ikke testbar:

- ☐ Afasi
- ☐ Redusert bevissthet
- ☐ Medisinsk ustabil
- ☐ Forstår ikke norsk
- ☐ Redusert kognitiv funksjon
- ☐ Demens

Annen årsak: _____

NIH STROKE SCALE (NIHSS)

		Dato / Tid	Ankomst	Dag 1	Dag 7/utreise (hvis utreise før dag 7)
1a	**Bevissthetsnivå** 0 = Våken 1 = Døsig, reagerer adekvat ved lett stimulering 2 = Døsig, reagerer først ved kraftigere/gjentatt stimulering 3 = Reagerer ikke, eller bare med ikke-målrettet bevegelse				
1b	**Orientering** (spør om måned + alder) 0 = Svarer riktig på to spørsmål 1 = Svarer riktig på ett spørsmål (eller ved alvorlig dysartri) 2 = Svarer **ikke** riktig på noe spørsmål				
1c	**Respons på kommando** (lukke øyne + knytte hånd) 0 =Utfører begge kommandoer korrekt 1 = Utfører en kommando korrekt 2 = Utfører ingen korrekt				
2	**Blikkbevegelse** (horisontal bevegelse til begge sider) 0 = Normal 1 = Delvis blikkparese (eller ved øyenmuskelparese) 2 = Fiksert blikkretning til siden eller total blikkparese				
3	**Synsfelt** (bevege fingre/fingertelling i laterale synsfelt) 0 = Normalt 1 = Delvis hemianopsi 2 = Total hemianopsi 3 = Bilateral hemianopsi/blindhet/koma				
4	**Ansikt** (vise tenner, knipe igjen øynene, løfte øyenbryn) 0 = Normal 1 = Utvisket nasolabialfure, asymmetri ved smil 2 = Betydelig lammelse i nedre ansiktshalvdel 3 = Total lammelse i halve ansiktet (eller ved koma)				
5	**Kraft i armen** (holde armen utstrakt 45° i 10 sekunder) 0 = Normal (også ved "ikke testbar") 1 = Drifter til lavere posisjon 2 = Noe bevegelse mot tyngdekraften, drifter til sengen 3 = Kun små muskelbevegelser, faller til sengen 4 = Ingen bevegelse/koma	ve hø			
6	**Kraft i benet** (holde benet utstrakt 30° i 5 sekunder) 0 = Normal (også ved ikke testbar) 1 = Drifter til lavere posisjon 2 = Noe bevegelse mot tyngdekraften, drifter til sengen 3 = Ingen bevegelse mot tyngdekraften, faller til sengen 4 = Ingen bevegelse	ve hø			
7	**Koordinasjon/ataksi** (finger-nese-prøve/hæl-kne-prøve) 0 = Normal (også ved ikke testbar eller koma) 1 = Ataksi i arm eller ben 2 = Ataksi i arm og ben				
8	**Hudfølelse** (sensibilitet for stikk) 0 = Normal 1 = Lettere sensibilitetsnedsettelse 2 = Markert sensibilitetsnedsettelse (også ved koma, tetraparese)				
9	**Språk/afasi** (spontan tale, taleforståelse, leseforståelse, benevning) 0 = Normal 1 = Moderat afasi, samtale mulig 2 = Markert afasi, samtale svært vanskelig eller umulig 3 = Ikke språk/koma				
10	**Tale/dysartri** (spontan tale) 0 = Normal 1 = Mild – moderat dysartri 2 = Nær uforståelig tale eller anartri/koma				
11	**Neglekt** (bilateral simultan stimulering av syn og hudsensibilitet) 0 = Normal (også ved hemianopsi med normal sensibilitet) 1 = Neglekt i en sansemodalitet 2 = Neglekt i begge sansemodaliteter/koma				
	Total NIHSS-Score				

Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: ☐☐☐☐ 2 0 ☐☐

Tester: ☐☐☐

Pasient-ID: ☐☐☐☐☐☐

Metode for innhenting av data:

☐ Klinisk undersøkelse

☐ Testbar

☐ Ikke testbar → **Årsak til ikke testbar:**

☐ Afasi ☐ Redusert bevissthet ☐ Medisinsk ustabil

☐ Forstår ikke norsk ☐ Redusert kognitiv funksjon ☐ Demens

Annen årsak: _____

MONTREAL COGNITIVE ASSESSMENT (MOCA) norsk versjon 7.1.

	POENG
1., 2. og 3. VISUOKONSTRUKTIV/EKSEKUTIV (utføres på eget ark) ☐ **1A2B** ☐ **Kopier kube** **Tegn en klokke** (ti over elleve) ☐ Kontur ☐ Tall ☐ Visere	/5
4. BENEVNING ☐ Løve ☐ Neshorn ☐ Kamel eller dromedar	/3
5. HUKOMMELSE Les ordene, forsøksperson må gjenta dem. Gjør to forsøk, selv om første forsøk gjennomføres helt riktig. Gjør gjenkalling etter 5 minutter. **ANSIKT** **FLØYEL** **KIRKE** **TUSENFRYD** **RØD** 1. forsøk ☐ ☐ ☐ ☐ ☐ 2. forsøk ☐ ☐ ☐ ☐ ☐	ingen poeng
6. OPPMERKSOMHET Les rekken med tall (1 tall/sekund) Forsøksperson skal gjenta i samme rekkefølge ☐ **2 1 8 5 4** Forsøksperson skal gjenta i baklengs rekkefølge ☐ **7 4 2**	/2
Les listen med bokstaver. På hver bokstav A skal forsøkspersonen banke på bordet med hånden sin. Ingen poeng ved 2 feil ☐ **F B A C M N A A J K L B A F A K D E A A A J A M O F A A B**	/1
Seriell subtraksjon med 7, begynnende med **100** **93** ☐ **86** ☐ **79** ☐ **72** ☐ **65** ☐ 4 eller 5 riktig: 3 png 2 eller 3 riktig: 2 png 1 riktig: 1png 0 riktig: 0 png	/3
7. SETNINGSREPETISJON **Gjenta etter meg:** Jeg vet kun at det er Jon som skal hjelpe i dag ☐ Katten gjemte seg alltid under sofaen når det var hunder i rommet. ☐	/2
8. ORDFLYT Si så mange ord du kan komme på som begynner med **F** innenfor ett minutt **Antall ord:** ☐ ☐ (N ≥ 11 ord)	/1
9. LIKHETER **Likhet mellom for eksempel en banan og en appelsin=frukt** ☐ tog-sykkel ☐ klokke-linjal	/2
10. UTSATT GJENKALLING **ANSIKT** **FLØYEL** **KIRKE** **TUSENFRYD** **RØD** Kun poeng for gjenkalling uten stikkord. ☐ ☐ ☐ ☐ ☐ Frivillig: Kategori-stikkord ☐ ☐ ☐ ☐ ☐ Frivillig: Multiple-choice stikkord ☐ ☐ ☐ ☐ ☐	/5
11. ORIENTERING ☐ **Dato** ☐ **Måned** ☐ **År** ☐ **Ukedag** ☐ **Sted** ☐ **By**	/6
Total skår Normal ≥26/30 Legg til 1 poeng dersom ≤ 12 år utdanning	/30

Kommentar:

Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

☐ Klinisk undersøkelse

☐ Testbar

☐ Ikke testbar

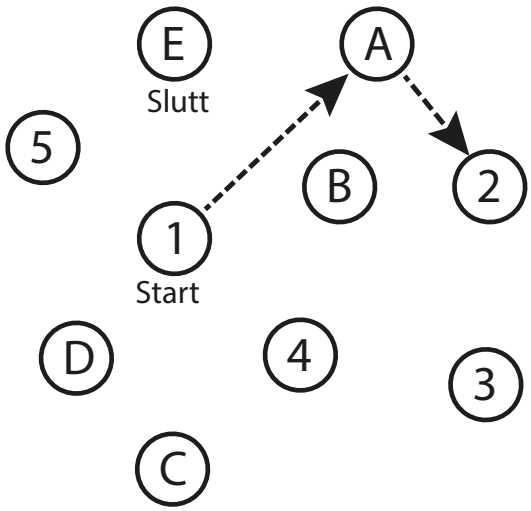
Årsak til ikke testbar:

☐ Afasi ☐ Redusert bevissthet ☐ Medisinsk ustabil

☐ Forstår ikke norsk ☐ Redusert kognitiv funksjon ☐ Demens

Annen årsak: _____

MOCA trailmaking, kube og klokke



Nor-COAST
Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

☐ Intervju av pasient ☐ Intervju av pårørende ☐ Telefonintervju

Opplysninger mangler:

AD8 DEMENS SCREENING INTERVJU

Husk, «Ja, en endring» indikerer at det har vært en endring i det siste året forårsaket av kognitive (tenkning og hukommelse) problemer.		JA, en endring	NEI, ingen endring	Vet ikke
1.	Problemer med å bedømme (for eksempel problemer med å ta beslutninger, dårlige økonomiske beslutninger, problemer med å tenke)			
2.	Mindre interessert i hobbyer/aktiviteter			
3.	Gjentar de samme tingene om og om igjen (spørsmål, historier eller uttalelser)			
4.	Problemer med å lære hvordan man bruker et verktøy, utstyr eller ulike tekniske enheter (eks. videospiller, data, mikrobølgeovn, fjernkontroll)			
5.	Glemmer korrekt måned eller år			
6.	Problemer med å håndtere kompliserte økonomiske/finansielle forhold (for eksempel bruk av nettbank, betale skatt og regninger)			
7.	Problemer med å huske avtaler			
8.	**Daglige** problemer med tenking og/eller hukommelse			
	TOTAL AD8 SKÅR			

Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: ☐☐☐☐2☐☐☐

Tester: ☐☐☐

Pasient-ID: ☐☐☐☐☐☐

Metode for innhenting av data:

☐ Klinisk us.

☐ Testbar

☐ Ikke testbar

Årsak til ikke testbar:

☐ Afasi ☐ Redusert bevissthet ☐ Medisinsk ustabil

☐ Forstår ikke norsk ☐ Redusert kognitiv funksjon ☐ Demens

Annen årsak: _____

NEGLEKT - test (del linje på midten)

Avstand fra linjens start til merket: ☐,☐ cm, mm

TRAIL-MAKING-test A

Tid (m:ss): ☐:☐☐

Klarer ikke gjennomføre ☐

Gjennomfører med hjelp ☐

TRAIL-MAKING-test B

Tid (m:ss): ☐:☐☐

Klarer ikke gjennomføre ☐

Antall feil ☐

Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

☐ Klinisk us.

☐ Testbar

☐ Ikke testbar

Årsak til ikke testbar:

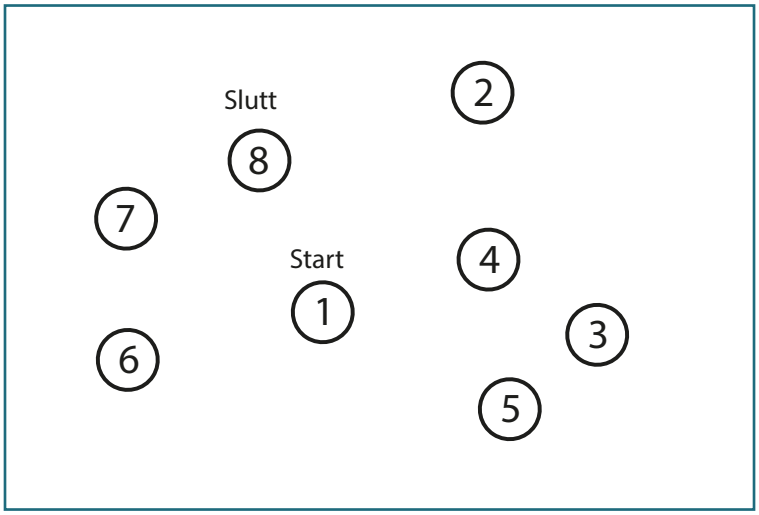
☐ Afasi ☐ Redusert bevissthet ☐ Medisinsk ustabil

☐ Forstår ikke norsk ☐ Redusert kognitiv funksjon ☐ Demens

Annen årsak: _____

Trailmaking A forsøk

EKSEMPEL



Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

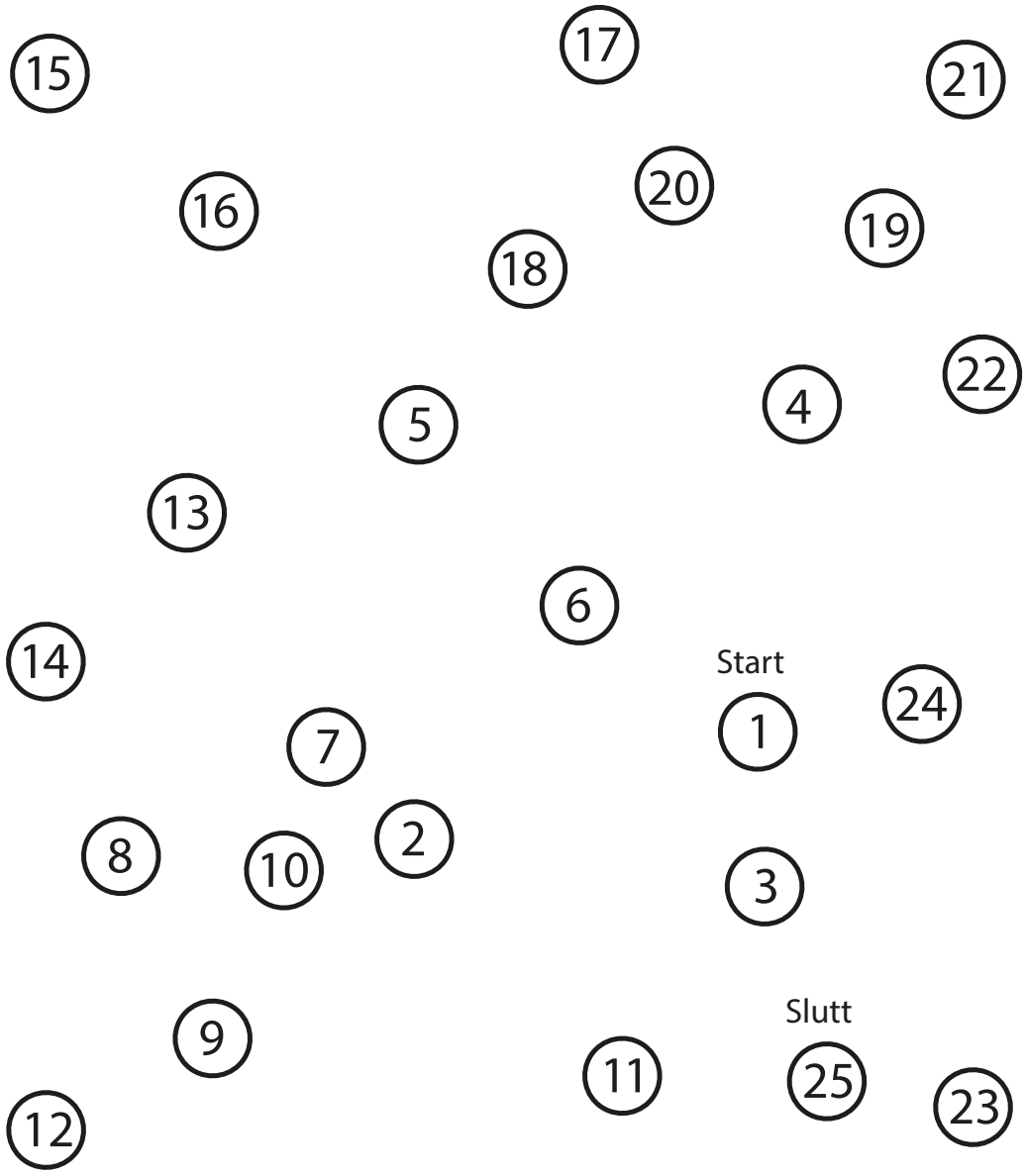
- ☐ Klinisk us.
- ☐ Testbar
- ☐ Ikke testbar

Årsak til ikke testbar:

- ☐ Afasi
- ☐ Redusert bevissthet
- ☐ Medisinsk ustabil
- ☐ Forstår ikke norsk
- ☐ Redusert kognitiv funksjon
- ☐ Demens

Annen årsak: _____

Trailmaking test A



Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

☐ Klinisk us.

☐ Testbar

☐ Ikke testbar

Årsak til ikke testbar:

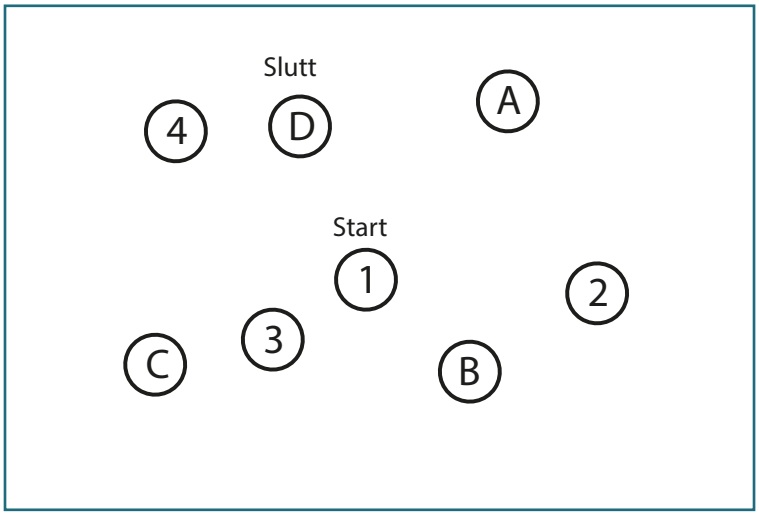
☐ Afasi ☐ Redusert bevissthet ☐ Medisinsk ustabil

☐ Forstår ikke norsk ☐ Redusert kognitiv funksjon ☐ Demens

Annen årsak: _____

Trailmaking B forsøk

EKSEMPEL



Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

- ☐ Klinisk us.

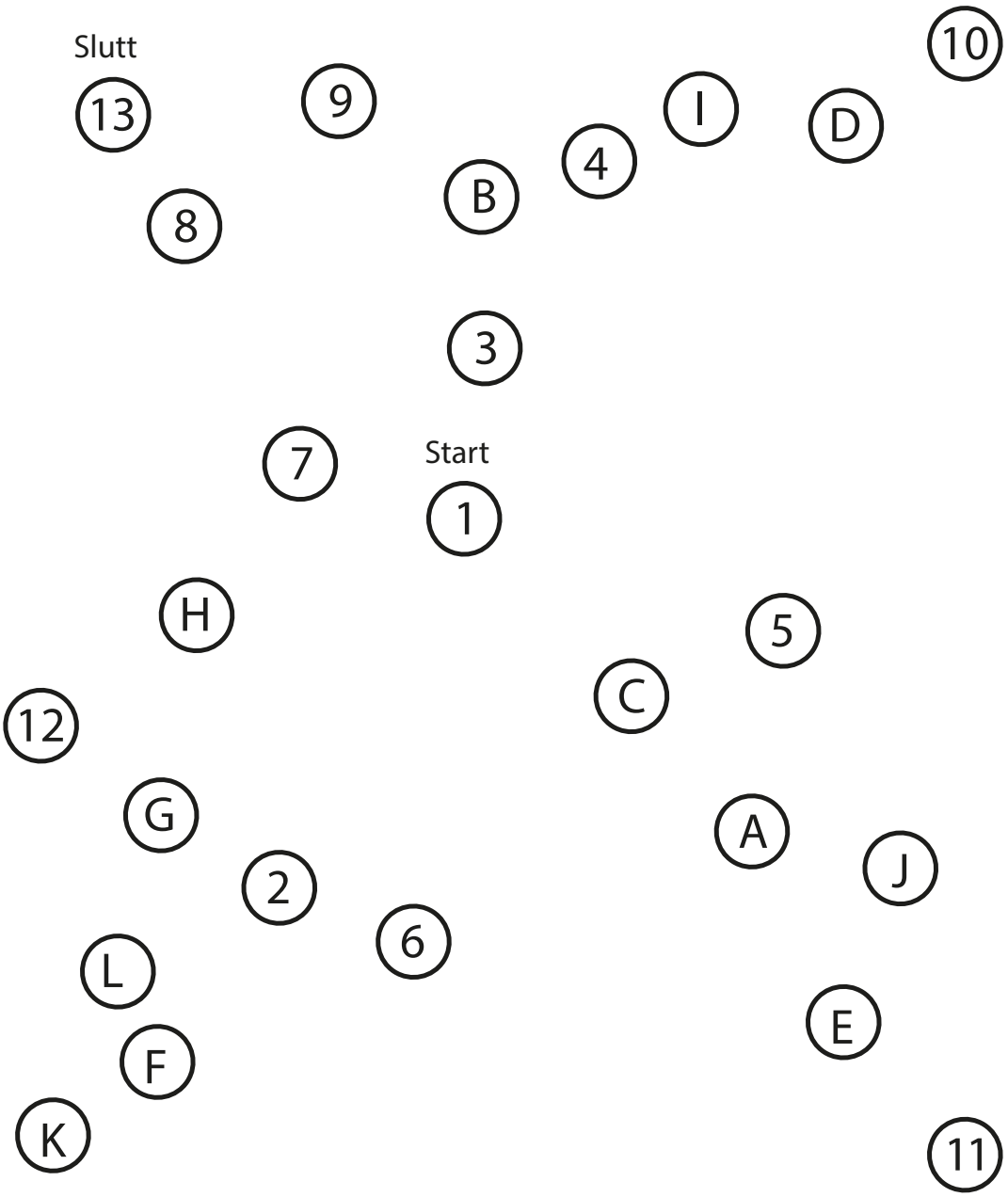
- ☐ Testbar
- ☐ Ikke testbar

Årsak til ikke testbar:

- ☐ Afasi
- ☐ Redusert bevissthet
- ☐ Medisinsk ustabil
- ☐ Forstår ikke norsk
- ☐ Redusert kognitiv funksjon
- ☐ Demens

Annen årsak: _____

Trailmaking test B



Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: | | | | | 2 | 0 | | |

Tester: | | | |

Pasient-ID: | | | | | | |

Metode for innhenting av data:

☐ Intervju av pasient ☐ Intervju av pårørende

Opplysninger mangler:

GLOBAL DETERIORATION SCALE (sett ring rundt eller strek under mest passende skår-nivå før slaget)

Skår- Nivå	Svikt i kognisjon og funksjon	Omsorgsbehov
1. Ingen kognitiv svikt		Uavhengig
2. Svært mild kognitiv svikt	Subjektiv opplevelse av mildt hukommelsestap. Ingen objektive tegn på kognitiv svikt ved intervju, arbeid eller sosial funksjon. Normal ved testing. Ingen funksjonssvikt.	Uavhengig
3. Mild kognitiv svikt (MCI)	Tidligste tydelige svikt, se fotnote. Normalt funksjonsnivå, men kolleger kan ha lagt merke til sviktende funksjon i arbeidssituasjon. Objektiv svikt ved testing. Benekting kan være til stede.	Uavhengig
4. Mild demens	Tydelig svikt ved grundig klinisk intervju, se fotnote. Vanskeligheter med å håndtere komplekse oppgaver, som økonomi, reiseaktivitet. Benektning er vanlig. Trekker seg tilbake fra utfordrende situasjoner.	Kan bo alene – trolig med hjelp fra familie eller omsorgsgiver.
5. Moderat demens	Kan ikke lenger leve uten en viss form for assistanse. Ikke i stand til å huske viktige deler av sin aktuelle livssituasjon, for eksempel adresse, telefonnummer som vedkommende har hatt i flere år, navn på barnebarn osv. En viss grad av desorientering for dato, ukedag, årstid, eller for sted. Trenger ikke assistanse ved toalettbesøk, spising, påkledning, men kan ha behov for hjelp til å velge passende påkledning.	Kan bo hjemme med familie. Kan bo i omsorgsbolig med hjemmehjelp. Det kan være nødvendig med bokollektiv, særlig hvis det er uttalte atferdssymptomer eller fysisk funksjonssvikt.
6. Moderat – alvorlig demens	Kan av og til glemme navnet til ektefellen. Mangler stort sett oversikt over nylige opplevelser og hendelser i deres liv. Trenger hjelp ved personlig ADL. Kan være inkontinent for urin. Atferdsmessige og psykologiske symptomer ved demens (APSD) er vanlig, f.eks. vrangforestillinger, repetitive atferd, agitasjon, angst etc.	Vanligvis sykehjem
7. Alvorlig demens	Personen mister språkfunksjonen. Inkontinens. Trenger mye hjelp i personlig ADL. Mister gangfunksjon, motoriske symptomer.	Sykehjem

3.symptomer på mild kognitiv svikt kan være at pasienten har mistet veien til ukjent sted, får problemer med ord/navn som merkes av pårørende, husker lite av det han leser, navn på nye personer, forlegger eller mister ting. Pasientens nærmeste merker sviktende funksjon

4.symptomer på mild demens: pasienten kan ha nedsatt kunnskap om nåværende og nylige hendelser, problemer med å redegjøre for eget livsløp, problem med hoderegning, håndtere økonomien sin, reise alene

5.symptomer på moderat demens: pasienten husker ikke sin adresse eller telefonnummer gjennom mange år, navn på familiemedlemmer (barnebarn for eksempel), hvilke skoler, arbeidsplasser etc. de har vært på, problemer med tidsorientering

Nor-COAST

Kartlegging av mental funksjon etter hjerneslag

PRIMÆROPPHOLD

Dato: ☐☐☐☐2☐☐☐

Tester: ☐☐☐

Pasient-ID: ☐☐☐☐☐☐

Metode for innhenting av data:

☐ Klinisk us.

☐ Testbar

☐ Ikke testbar

Årsak til ikke testbar:

☐ Afasi ☐ Redusert bevissthet ☐ Medisinsk ustabil

☐ Forstår ikke norsk ☐ Redusert kognitiv funksjon ☐ Demens

Annen årsak: _____

FYSISKE TESTER

1. Balansetest

☐ Gjennomført: ☐ Ikke i stand ☐ missing

1.Stå uten støtte
10 sekunder
↓
2.Samlede føtter
10 sekunder
↓
3.Semi-Tandem
10 sekunder
↓
4.Tandem
10 sekunder
↓
5.Ett ben stående inntil
20 sekunder

1. ☐☐.☐☐ sek.
2. ☐☐.☐☐ sek.
3. ☐☐.☐☐ sek.
4. ☐☐.☐☐ sek.
5. ☐☐.☐☐ sek. Høyre
5. ☐☐.☐☐ sek. Venstre

2. 360 graders vending

☐ Gjennomført: ☐ Ikke i stand ☐ missing

***INSTRUKSJON:* Snu deg rundt en hel omgang. Stans. Snu deg så rundt en hel omgang den andre veien. Det brukes ikke hjelpemidler under testen**

☐ 4 Kan snu seg sikkert 360 grader på 4 sekunder eller mindre

☐ 3 Kan snu seg sikkert 360 grader på 4 sekunder eller mindre kun en retning

☐ 2 Kan snu seg sikkert 360 grader, men trenger mer enn 4 sekunder

☐ 1 Trenger tilsyn eller muntlige ledetråder

☐ 0 Trenger støtte under vendingen

Tid vending mot høyre

☐☐.☐☐ sek.

Tid vending mot venstre

☐☐.☐☐ sek.

3.a Reise/ sette seg x 1

☐ Gjennomført: ☐ Ikke i stand ☐ Missing

3.b Reise/ sette seg x 5

☐ Gjennomført: ☐ Ikke i stand ☐ Missing

Tid 5 repetisjoner uten armbruk ☐☐.☐☐ sek.

Tid 5 repetisjoner med armbruk (hvis deltager ikke klarer uten armbruk) ☐☐.☐☐ sek.

4. 4m Gangtest

☐ Gjennomført: ☐ Ikke i stand ☐ Missing

Hvis mulig gjennomføres testen uten ganghjelpemidler

Ganghjelpemidler ved test (kryss av):

1. ☐ Uten
2. ☐ Krykke/stokk (er)
3. ☐ Rullator
4. ☐ Annet (spesifiser)_____

Tid test 1: ☐☐.☐☐ sek.

Tid test 2: ☐☐.☐☐ sek.

5. 10-meter gangtest

☐ Gjennomført: ☐ Ikke i stand ☐ Missing

	Runde-tid (sekunder og ti-deler)
Normal. hast. 1	
Normal. hast. 2	
Maks. hast. 1	
Maks. hast. 2	

Bruk av hjelpemidler

☐ Ingen
☐ Krykke/stokk (er)
☐ Rullator
☐ Annet (spesifiser)_____

6. 8-talls balansetest

☐ Gjennomført: ☐ Ikke i stand ☐ Missing

Testen er utført ☐ med sko ☐ uten sko

Total antall feiltråkk: ☐

Tid (mm:ss): ☐☐.☐☐

7. Gripestyrke

Høyre hand: ☐ Gjennomført: ☐ Ikke i stand Missing

Venstre hand: ☐ Gjennomført: ☐ Ikke i stand Missing

Dynamometer:

Transverst volargrep	Høyre hand (kg)	Venstre hand (kg)
Prøve forsøk		
2. forsøk		
3. forsøk		

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8. Nine Hole Peg Test

Antall sekunder som benyttes for å plassere alle 9 peg-er registreres (testen avbrytes etter 2 minutter).

Høyre hand: ☐ Gjennomført: ☐ Ikke i stand ☐ Missing

Venstre hand: ☐ Gjennomført: ☐ Ikke i stand ☐ Missing

	Høyre hand		Venstre hand	
	Ant. sekunder:	Ant. peg plassert:	Ant. sekunder:	Ant. peg plassert:
Forsøk 1				
Forsøk 2				