The First Three Years of the Swiss Neuropaediatric Stroke Registry (SNPSR): A Population-Based Study of Incidence, Symptoms and Risk Factors

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Original Article

Abstract

We report the results of three years of the population-based, prospective Swiss NeuroPaediatric Stroke Registry (SNPSR) of children (up to 16 years) with childhood arterial ischaemic stroke (AIS1), neonatal stroke (AIS2), or symptomatic sinus venous thrombosis (SVT). Data on risk factors (RF), presentation, diagnostic work-up, localisation, and short-term neurological outcome were collected. 80 children (54 males) have been included, 40 AIS1, 23 AIS2, and 17 SVT. The data presented will be concentrated on AIS. The presentation for AIS1 was hemiparesis in 77% and cerebellar symptoms and seizures in 20%, respectively. AIS2 presented in 83% with seizures and in 38% with abnormality of muscle tone. Two or more RF were detected in 54%, one RF in 35%. The most prominent RF for AIS1 were infections (40%), followed by cardiopathies and coagulopathies (25% each). AIS2 were frequently related to birth problems. Neurological outcomes in AIS1 and AIS2 were moderate/severe in 45% and 32%, respectively. The outcome correlated significantly with the size of infarction ($p = 0.013$) and age at stroke ($p = 0.027$). The overall mortality was 6%. Paediatric stroke is a multiple risk problem, which leads to important long-term sequelae.

Key words
Paediatric stroke · epidemiology · manifestation · risk factors · outcome

Abbreviations

SNPSR	Swiss Neuropaediatric Stroke Registry
AIS1	arterial ischaemic stroke
AIS2	neonatal stroke
SVT	sinus venous thrombosis
RF	risk factor
MRA	magnetic resonance angiography
MRI	magnetic resonance imaging
CT	computed tomography
CRP	C-reactive protein
SR	sedimentation rate
ACLA	cardiolipin antibodies
ANA	antinuclear antibodies
LA	lupus anticoagulants
MTHFR	methylene tetrahydrofolate reductase
ENT	ear, nose, and throat
MELAS	mitochondrial encephalopathy with lactic acidosis and stroke-like episodes

Introduction

With advancing knowledge on the pathophysiology and treatment options for adult stroke, research on paediatric stroke has become increasingly more important over the last decade. Recent studies suggest an incidence of 2 – 5/100 000 children/year for ischaemic stroke [5, 23], and a slightly higher incidence for neonatal ischaemic stroke of 4.7/100 000 children/year [6, 24]. For treatment decisions, a knowledge of risk factors and prognosis is important. Over the last few years there have been several

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studies on different aspects of risk factors (RF) for paediatric stroke [4,12,19,21,23,25] which confirm the fact that paediatric stroke is a multiple risk problem, differing significantly from adult stroke. The most important RF are infections and parainfectious reactions [13,29], vasculopathies [13,14,19], systemic or heart disease [23,30], and hereditary coagulopathies [4,26]. Prognosis of paediatric stroke is better compared to adult stroke. Retrospective studies, however, have shown that there is a marked morbidity, interfering with the social and professional integration of these children [2,3,9,11]. There are only limited prospective data on outcome of children after stroke [5,25].

Since January 2000, we have run a population-based stroke registry for children living in Switzerland who have suffered an ischaemic stroke or symptomatic sinus venous thrombosis. We now report our data on the incidence, manifestation, risk factors, and short-term neurological outcome of these children.

Patients and Methods

Data from the Swiss Neuropaediatric Stroke Registry (SNPSR) from 1.1.2000 until 31.12.2002

Each month a questionnaire was sent to every neuropaediatrician (n = 28) in Switzerland, and one was sent every three months to the neonatal units (n = 24). An obligatory return form was included to give initials and birth date of each child suffering an ischaemic stroke. In case of a positive answer, a questionnaire collecting data of the acute hospitalisation (as demographic data, personal and familiar risk factors, symptoms and treatment at manifestation, investigations, acute treatments) and six months later a questionnaire concerning short-term neurological outcome and treatments were sent to the responsible neuropaediatrician.

Inclusion criteria were as follows: children from birth up to 16 years who suffered, by clinical and neuroimaging criteria, of one of the following:

a) *arterial ischaemic infarction (AIS)* or b) *sinus venous thrombosis* (including preterm babies) with any transient neurological dysfunction and thrombosis of cerebral veins or venous sinuses seen on CT, MRI scan, MR venogram, and/or conventional cerebral venogram.

Subtypes of AIS included: 1) *childhood stroke (AIS1)* defined as focal neurological deficit of acute onset lasting at least 20 minutes and CT or MRI showing infarction in a localisation consistent with neurological signs and symptoms, 2) *neonatal stroke (AIS2)* (preterm babies < 37 gestational weeks excluded) defined as focal neurological symptoms or seizures, respectively, lethargy only and CT, ultrasound or MRI showing the focal ischaemic lesion. Stroke in the setting of generalised hypoxia was only included if there was a focal ischaemic lesion in an area of recognisable vascular distribution.

No obligatory investigations were required for inclusion into the study, but the following cardiac and laboratory investigations were proposed: Cardiac work-up was to include echocardiography and electrocardiography. Suggested laboratory investigations included blood count, signs for infections (CRP, SR, and serologies), signs for vasculitis (SR, platelets, quick, aPTT, ACLA, ANA, and LA), signs for metabolic disorders (lactate, ammonium, homocysteine, lipids, and amino acids/organic acids in urine), and investigations for hereditary coagulation problems (protein S, C, antithrombin III, factor V Leiden, prothrombin, plasminogen, and MTHFR). Normal values for these investigations were taken according to the local laboratory which performed the tests.

The data of the whole registry were analysed in a descriptive manner for demographics for the whole study population and, data for aetiology and RF (especially the cooccurence of several risk factors as a factor for the probable trigger of the acute event), localisation of infarction, symptoms at manifestation, therapeutic approach, as well as neurological outcome at six months for children with AIS.

RF to be were considered: 1) diseases and symptoms which were major causes for the cerebrovascular event (as moya-moya syndrome, cardiac problems, dissections) 2) and diseases and symptoms which might have contributed to the cerebrovascular event (as infections, coagulopathies).

Symptoms at manifestation were taken by history and physical findings present during the acute period from a clinical examination performed by the responsible neuropaediatrician.

Lesions of infarction were described as follows: left- or right-sided; large vascular territory (median, anterior, and posterior cerebral arteries) or small vascular territories (branches of the above vessels), localisation of the infarction (cortical, white matter, basal ganglia, thalamus, brainstem, cerebellum); single or multiple infarctions; haemorrhagic-ischaemic infarctions.

Treatments during the acute period were divided into: coagulation therapy (anticoagulation or platelet aggregation inhibition); other medications (such as antiepileptic or antibiotic treatment); and surgical procedures.

At discharge from the hospital the following data were registered: symptoms at discharge, planned rehabilitation, and prophylactic treatment against recurrence.

The neurological findings after six months were taken from a clinical examination performed by the responsible neuropaediatrician. These included: no impairment (full recovery with no signs in clinical examination); mild impairment (mild neurological findings in clinical examination without functional disturbance in daily life); moderate disturbance (moderate neurological findings in clinical examination with disturbance in daily life); severe disturbance (severe findings in clinical examination with marked disturbance in daily life and loss of certain functions or death).

Statistical analyses

Scores of the subgroups (risk factors, localisation, age groups) were tested for normal distribution by the Kolmogorov-Smirnov test. The Mann-Whitney U-test for non-parametric data was used to perform subgroup comparison. Furthermore ANOVA revealed differences of subgroup variances. Subgroups and degree

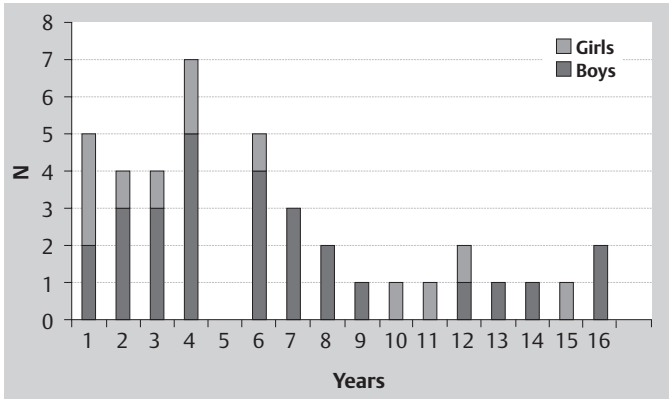


Fig. **1** Age at manifestation of arterial ischaemic stroke during childhood (n = 40).

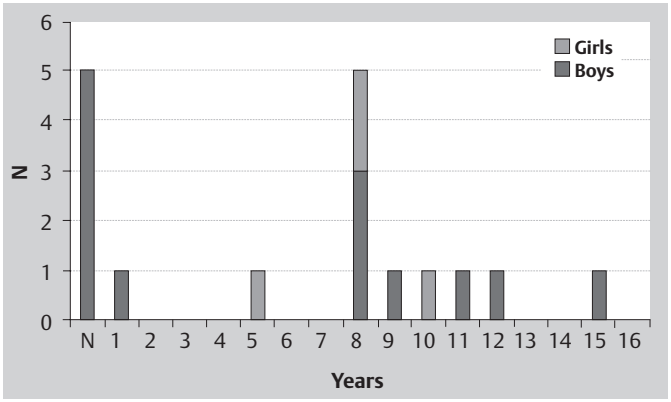


Fig. **2** Age at manifestation of sinus venous thrombosis (n = 17).

of handicap were correlated with the Pearson correlation. A p value < 0.05 was considered significant, a p value < 0.1 was considered a trend. Statistical analysis was performed using SPSS version 12.0.

The study was approved by the ethics committee from Bern, Switzerland.

Results

During these first three years, 80 children (54 boys) have been registered, accounting for an incidence for Switzerland of 2.1/100 000 children/year. Sixty-seven (67) children were of Caucasian origin.

There were 40 children with AIS1 (mean age at manifestation 5.6 years) and 23 children with neonatal infarction. In the group of SVT, 17 children were registered, five of them neonates. Figs. **1** and **2** give an overview of age at presentation of arterial ischaemic stroke and sinus venous thrombosis.

Symptoms at presentation

Symptoms at presentation for children with AIS1 are summarised in Fig. **3**. Hemisyndrome was left-sided in 16 and right-sided in 15. Headaches (in 16 children) were preceding symptoms only in 3 patients. Changes of level of consciousness were primarily fatigue and drowsiness. For children with AIS2 (not summarised in the figures) the most frequent symptoms were seizures (19 babies), followed by hypo-/hypertonia (10), apnoea (6), and irritability (5).

Localisation of infarction

Of the 40 children with AIS1, 25 suffered isolated infarctions and 13 had multiple infarctions, one of them with secondary haemorrhagic transformation. In two children with evidence of mitochondrial disorder, MRI did not reveal the localisation of infarction, in spite of marked neurological symptoms (both children with persistent hemiparesis, one of them died during the acute episode). In 27/38 cases the territory of the medial cerebral artery was affected, in six the anterior cerebral artery. 18 had involvement of the posterior circulation, in 10 of the posterior cerebral artery. Six each had a lesion within the territory of the superior or inferior cerebellar arteries. Lesions were left-sided in 19 children, right-sided in 16, and bilateral in five. A summary of affected brain structures is given in Table **1**.

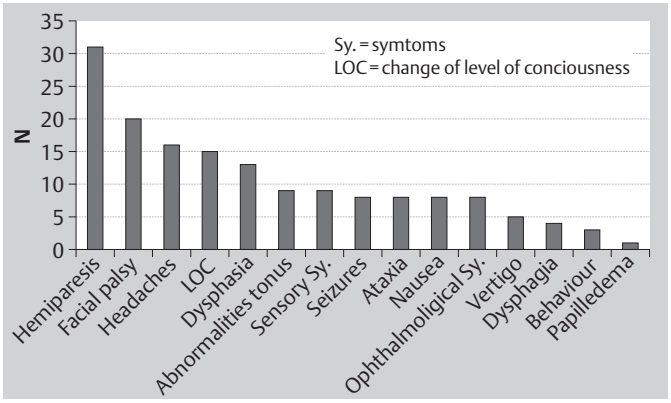


Fig. **3** Symptoms at manifestation in arterial ischaemic infarction of childhood (n = 40). Sy = symptoms; LOC = change of level of consciousness.

In children with neonatal stroke, 5/21 had multiple infarctions. Altogether, 5 children had haemorrhagic transformation. Involvement of the territory of the medial cerebral artery was present in 18/23, 16 of them on the left side. Ischaemic lesions in the vertebrobasilar territory were detected in two children. In three children, the localisation was not reported to the registry.

Risk factors

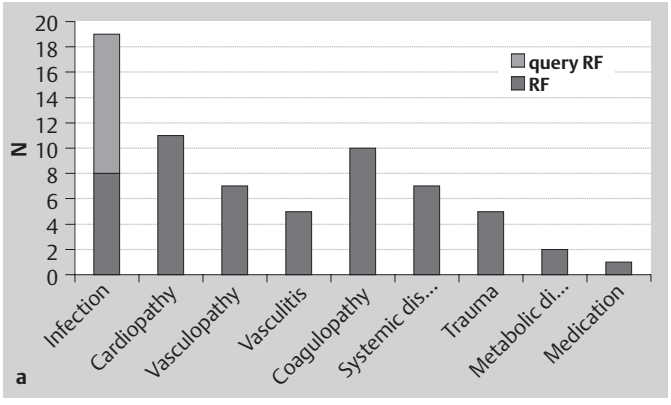
Necessary investigations were not given for inclusion into the study. Therefore investigations were initialised by the responsible neuropaediatrician according to individual indications. Some children did not undergo the complete proposed work-up. A summary of all detected risk factors is given in Fig. **4 a** and **b.**

Cardiac work-up (pathological in 11) was performed in 34/40 children with AIS1. Eight children had congenital heart malformation (once an isolated patent foramen ovale), 5 with left-to-right shunt. One of them also suffered from hyperthyroidism and was treated with antiarrhythmic agents, one underwent pulmonary lobar resection due to tuberculosis, and hereditary coagulopathies could be detected later on in three children. One

Table **1** Localisation of arterial ischaemic infarction in childhood

Main localisation			*Additional affected areas*			
	Total	*Isolated*	*BG*	*Thalamus*	*Cerebellum*	*Brainstem*
Cortex/white matter	23	10	10	3	1	1
Basal ganglia (BG)	17	3		6	1	
Thalamus	9	1	6		2	
Cerebellum	6	2	1	2		1
Brainstem	3	1			1	
White matter (isolated)	2	2				
Unknown	2*					

BG = basal ganglia. * Two children with mitochondrial disorders with lactic acidosis (see text)



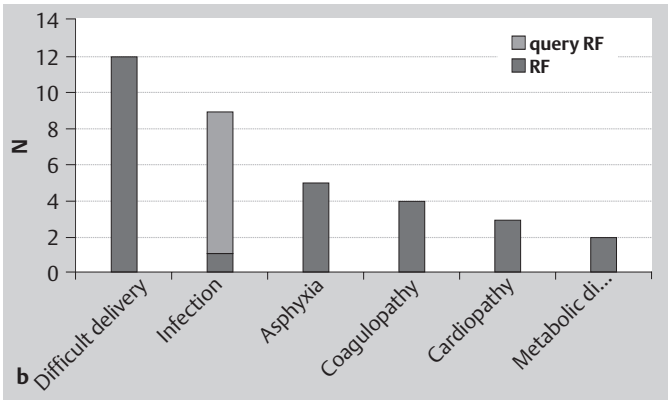


Fig. **4 a** and **b** **a** Risk factors for arterial ischaemic stroke in 40 children. **b** Risk factors for arterial ischaemic stroke in 23 neonates.

child suffered a mitral valve insufficiency without obvious malformation. There was one child with endocarditis (septic disease during treatment for leukaemia) and one child with dilated cardiomyopathy of metabolic origin (mitochondriopathy). In addition, two children with renal disease had accompanying myocardial hypertrophy. 12/23 children with AIS2 had cardiac work-up: two children showed congenital malformations and one suffered a left ventricular dysfunction after severe hypoxia.

In the group of vasculopathies (n = 7), in three children moya-moya disease could be detected by MRA and/or conventional angiography. One of these children had neurofibromatosis and was homozygous for a mutation in the MTHFR gene; one of them had trisomy 21 and suffered pharyngitis at the time of the event. Another three children had dissections of the cranial vessels; in two of them a mild head trauma preceded the acute event, one of them also suffered acute sinusitis at the time of stroke. There was one child in whom transient focal arteriopathy was shown.

In children with infections (n = 19), 11 had acute upper respiratory or ENT system infection shortly prior or at the time of the event. For eight children, the relation to stroke was more secured: for three children there was a suspicion of vasculitis during an active *Borrelia* infection, one child had a *Varicella* infection three months prior to insult, and in one child there was suspicion of vasculitis due to antiphospholipid antibody syndrome. There were two children with septic disease, and one with a complicated tuberculosis infection. Besides infection and immunological problems, further risk factors could be detected for 10/19 children: hereditary coagulopathies in five, immunosuppression due to chemotherapy and cardiac disorders in two each, and mitochondriopathy, dissection, moya-moya disease, and pneumectomy in one each. For children of group AIS2, infection was proven at time of the event for only 3/9 (sepsis, pneumonia, severe eye infection).

In children with systemic disease (n = 7), three children suffered from renal disorders and subsequent arterial hypertension, two had oncological disorders, and one each hyperthyreosis and β-thalassaemia minor. In addition, there were two children with a strong suspicion of MELAS syndrome. Both had biochemical markers for mitochondriopathy, but MELAS was not proven genetically. In the group AIS2, there were two children who had marked hyponatreamia and hypocalceamia together with hypoglycaemia.

L-Asparaginase was related to stroke in one child (with lymphoma) with AIS1.

Investigations for coagulopathies were all performed at least three months after the acute event, but were not complete in many children. In a total of 14/63 children coagulopathies could be detected, 30/63 were normal for all tested functions, and 19 had no coagulopathy work-up. In the group of AIS1, in 22 % no re-

Table **2** Summary of the investigations for coagulopathies

Coagulopathies	*Expected abnormalities***	*Abnormal results/total investigations*		
		AIS1 (n = 40)	*AIS2 (n = 23)*	*total (n = 63)*
Protein S	< 1%	2/22	0/8	2/28 (7.1%)
Protein C	0.67%	1/23	1/9	2/32 (6.25%)
*Factor V Leiden/APCR**	4%	2/25	1/10	3/44 (6.6%)
Antithrombin III	< 1%	0/23	0/9	0/32
*MTHFR**	10.4%	3/19	2/11	5/30 (16%)
Homocysteine		1/25	0/10	1/35 (2.8%)
*G 20210 A prothrombin**	1.3%	2/14	1/8	3/22 (13.6%)
Plasminogen/lipoprotein A*	4.7%	1/14	0/4	1/18 (5.5%)
ACLA		2/19	0/4	2/23 (8.6%)
ANA		1/21	0/5	1/26 (3.8%)
Lupus anticoagulans		1/18	0/4	1/22 (4.5%)
Total		16	5	21

Abbreviations: MTHFR = methylene tetrahydrofolate reductase; APCR = APC resistance; ACLA = anticardiolipin antibodies; ANA = antinuclear antibodies. * Genetic analyses. ** In healthy German population, data from [26]

sults for coagulopathy work-up were available, in the group of AIS2 this was the case in as much as 43%. Table **2** gives an overview of the detected abnormalities in relation to the children investigated.

Only one child (AIS1) was homozygous for the MTHFR mutation. In the two children with positive ACLA, there was in one an additional heterozygote MTHFR mutation and in the other protein S and C deficiency. One of the neonates with the MTHFR gene mutation had an additional protein C deficiency.

In the group of AIS1, head trauma was reported within a short time prior to the insult in five children. In one child additional risk factors were factor V Leiden heterozygous mutation and neuroborreliosis, for one child asymptomatic marked sinusitis.

In the group of AIS2, interventions during labour were registered in nine (emergency caesarean section, vacuum or forceps delivery), four of these babies suffered severe problems of adaptation (asphyxia in two, hypoglycaemia and neonatal infection in one each).

It is important to realise that 54% of all children with arterial ischaemic infarctions had more than one risk factor, which might have attributed to the acute event of stroke. In another 35%, at least one risk factor could be detected. In 11%, the aetiology of stroke remained completely uncertain.

Neurological outcome and relation to localisation, age at stroke, risk factors

Neurological outcome after 6 months is summarised in Fig. **5**. There were four children, aged 1.8 – 5.9 years, who died. One child died after recurrent stroke in the acute phase due to dissection after trauma, one child died due to renal problems, and two children died due to mitochondrial disorders.

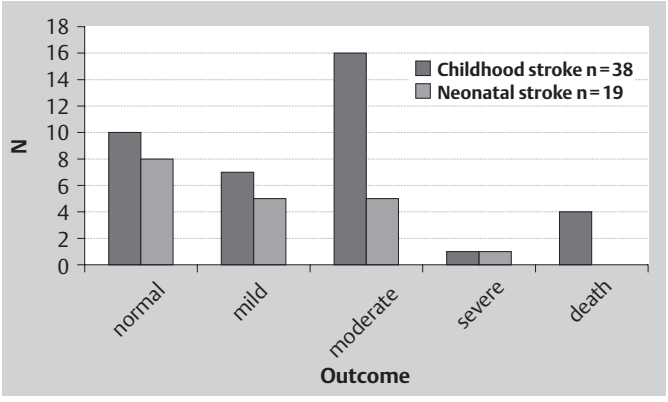


Fig. **5** Neurological outcome 6 months after arterial ischaemic stroke.

Statistical analyses revealed a significant correlation between *localisation of stroke* and *degree of handicap* ($r = 0.391$, $p = 0.018$), while ANOVA documented a significant group difference ($p = 0.047$). Children with a large extension of stroke in cortical and white matter areas with involvement of basal ganglia and thalamus showed significantly more handicaps than children with strokes in other regions. There was no correlation between other groups, especially no correlation between the group of cortical/white matter lesions and basal ganglia/thalamus lesions ($p = 0.344$) (Fig. **6**).

Age at stroke and *degree of handicap* were negatively correlated ($r = -0.335$, $p = 0.040$), meaning that the younger the child is at time of stroke the less favourable is the outcome (ANOVA, $p = 0.124$). There was a tendency ($p = 0.064$) towards worse outcome in children with early childhood stroke (0 – 4 years) compared to children aged 10 – 16 years (Fig. **7**).

There was no significant relationship between the amount of *risk factors* and *degree of handicap* after childhood stroke ($r = 0.237$,

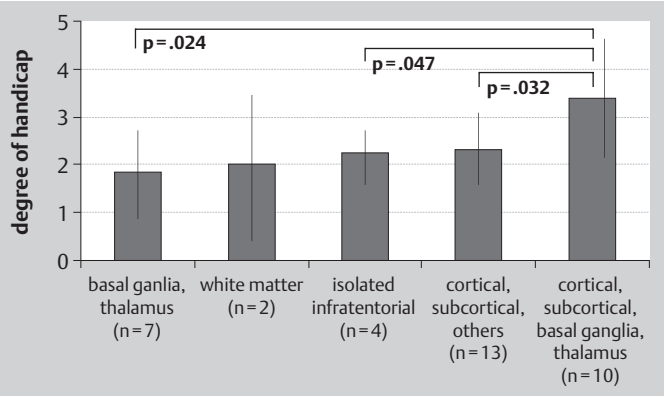


Fig. **6** Relation between localisation of infarction and degree of handicap in children with arterial ischaemic stroke (n = 36). Degree of handicap: 0 = normal; 1 = mild; 2 = moderate; 3 = severe; 4 = death.

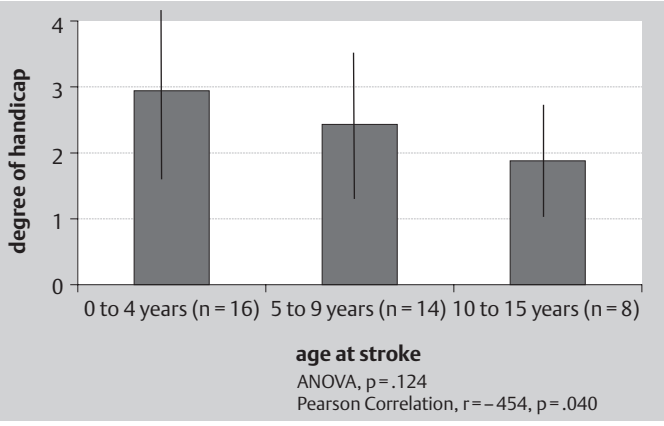


Fig. **7** Relation between age at stroke and degree of handicap in children with arterial ischaemic stroke (n = 38). Degree of handicap: 0 = normal; 1 = mild; 2 = moderate; 3 = severe; 4 = death.

p = 0.153). ANOVA did not reveal any differences concerning the amount of risk groups according to the degree of handicap (p = 0.352).

Data of outcome from children with neonatal stroke is not reported, due to the limited follow-up period.

Discussion

The strength of our study is to give population-based results. An incidence for ischaemic infarction of 2.1/100 000 children/year and the sex distribution (boys : girls = 2 : 1) are similar to other studies, at different tertiary care centres [6,10,18,23]. Most of our children have been referred to tertiary care centres. Therefore, the accordance of our data with registries based on different tertiary care centres suggests that most children with paediatric stroke in industrialised countries are referred to tertiary care centres. Stroke during childhood is very likely to be detected by symptoms and referred for further investigations, therefore we think we have registered close to all childhood strokes. However, there might be missing neonatal strokes. Symptoms might be minimal and detection of residual symptoms might be diagnosed as congenital infarction later on. In addition it was more difficult to get neonatal units to register patients correctly than neuropaediatricians.

There was a peak of frequency for AIS1 during pre-school age and a peak of frequency for SVT in early school age, a fact which was also shown in other studies [3,6,15,16,23,32]. Both peaks are likely to be related to the risk factors of infections and parainfectious reactions. The incidence of infections is known to be increased for these age groups [1,8], and therefore infectious complications are likely to be increased as well. In the study of deVeber et al [5] there was a trend for the frequency of childhood stroke and SVT to be shifted to a younger age group. A likely explanation for this effect might be the fact that Canadian children visit day care and kindergarten at a younger age and are therefore exposed to infections at a younger age. The proportion of AIS versus SVT with 79 : 21 % is similar to the observation of the group of deVeber et al [5] in a registry based on different centres. This is a further confirmation for the two studies to be representative despite slightly different designs.

Neuroimaging confirmed the clinical suspicion of infarction during the acute episode in 85 % of the children. In seven children with normal CT during the acute event, follow-up examination confirmed the suspicion within the next two days. In four neonates cranial ultrasonography was initially reported to be normal, CT and/or MRI later on confirmed the ischaemic event. These data underscore the point that emergency imaging by CT or sonography might be initially misleading. Interestingly, in two children, ischaemic lesions could not be detected in MRI, despite severe neurological dysfunction at the time. Both children had marked lactic acidosis and died later on, in both children death was related to an acute episode of lactic acidosis. This observation underlines that lesions due to mitochondrial dysfunction have a different pathophysiology than pure arterial ischaemic infarctions. This observation is also important in the initial differential diagnosis in children presenting with stroke and normal imaging.

Investigations for risk factors were performed for cardiac problems in children past the neonatal period with AIS, but in neonates there were missing results. Most concerning was the incompleteness of investigations of prothrombotic risk factors. The fact that Kirkham et al [19] and Straeter et al [34] showed that recurrence risk was related to risk factors (especially coagulopathies) enhances the importance of a full work-up. The numbers of our study group were probably still too small to detect a recurrence.

Our study confirms the results of de Veber et al [4] and Lanthier et al [23] who describe paediatric stroke to be a multiple risk factor problem. In 54 % of our children we could detect more than one risk factor and only for 11 % of children were no risk factors found. The fact that investigations were incomplete in many children emphasises this observation further. In 23 children with coagulopathies, only in three could no further risk factor be detected. This supports the suggestions of Ridker et al [28] and Lanthier et al [23] that pathological coagulopathies might in many cases be a cofactor but not the main reason for the occurrence of stroke. Prothrombotic risk factors were reported in vari-

able frequencies from 25 – 53% in several studies [4,14,17, 23,35], and for children with neonatal stroke an even higher percentage of 40 – 68% [14,25]. In our own study in 41% of all investigated children a procoagulopathy could be detected. In the study of deVeber et al [4] more boys were affected by coagulopathies than girls. However, in a normal population Revel-Vilk et al [27] did not show a preponderance of coagulopathies in males. In our study we had a relationship of affected boys : girls of 2 : 1 and a similar distribution for coagulopathies. The fact that boys outnumber girls for stroke is shown in many studies [5,23,29]. We assume that there are unknown factors increasing the risk for stroke in boys compared to girls and that coagulopathies are a cofactor, which is equally distributed for both sexes. The suggestions that other, not yet detected (genetic?) risk factors might be sex-dependent seems logical [20].

Our results concerning coagulopathies have to be taken cautiously, due to the incomplete investigations. However, we could confirm results from earlier studies [4,12,17,25,26,37] that the presence of factor V Leiden was increased (7%) compared to the normal population (4%). In addition, protein S deficiency (7%), protein C deficiency (6%), and prothrombin –G20210 A mutation (13%), were all present in a higher frequency than expected in the normal population.

The presence of anticardiolipin antibodies was lower (9%) in our patients when compared to the study of deVeber et al (33%) [4]. We only considered anticardiolipin testing to be positive if results persisted after the acute episode. In the study of deVeber et al [4] inclusion of results from the acute episode might have increased the numbers compared to ours.

Infections as a risk factor for stroke have been described in several earlier studies [13,19,23,29]. Although we did not search for specific infections in our population of children with stroke, infections were the most frequent risk factor in the subgroup of AIS1. In three children we detected an active *Borrelia* infection during the time of stroke. In the literature we could find several case reports where active *Borrelia* infection was related to the event of stroke [29,36]. In our patients we assume a transient focal arteriopathy to have been the consequence of *Borrelia* infection. In the adult literature dissection is described to be influenced by infectious/parainfectious reaction [13]. In only one child with dissection there was an asymptomatic sinusitis (detected by neuroimaging). This child had severe dissection after minor trauma, which has led to recurrent events during the acute episodes and subsequently to death. Infectious involvement of the vertebral artery might have influenced the severe course of the disease.

Ganesan et al [10] showed that systemic disease is related to a large extension of infarction, probably due to an incomplete compensatory mechanism of the cerebrovascular system. As a consequence of the large extension of infarction, children with stroke due to systemic disease had a worse prognosis. This hypothesis can be supported by our own study: in 5/6 children with stroke within a setting of systemic disease, large lesions cortically and subcortically led to moderate or severe outcome, one child died.

Perinatal problems were reported in 12/23 children with neonatal infarctions. Most of them were of minor importance and therefore it seems to us more likely to be a consequence of the infarction than actually a risk factor for infarction.

Statistical analyses have to be taken cautiously due to the small number of children and the influence of several variables, which might have confounded our results. The significance of the effect of localisation of infarction on neurological outcome ($p = 0.018$) is remarkable, but might largely have been influenced by the size of infarctions, as there was no difference between other subgroups of localisation. It seems important to enhance the observation that involvement of basal ganglia/thalamus or cortical/white matter showed no statistical significance in neurological outcome. These results could confirm the study of Ganesan et al [10]. The interpretation of influence of age at stroke can only be speculative. The effect of disturbing the process of synaptic maturation and organisation seems to outdo the expected better plasticity at this age. The point that lesions at different ages might provoke different degrees of dysfunction was also shown in one of our previous papers, where children aged between 6 – 9.5 years were most affected after cerebellar tumour compared to elder age groups [33]. It is going to be most interesting to see whether our ongoing study will reveal similar influences on neurological outcome as on neuropsychological outcome.

Conclusions

Paediatric ischaemic stroke has an incidence of 2.1/100 000 children/year. This is similar to the occurrence of paediatric brain tumours [22]. Paediatric ischaemic strokes are a multiple risk problem with infectious reactions, cardiopathies, and coagulopathies being the most frequent risk factors. Neurological outcome seems to depend on age at stroke and also size of lesion. Future research should focus on neuropsychological outcome and its variables, influence of single risk factors, and possible therapeutic approaches.

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