# BMJ Open Trend of cerebral aneurysms over the past two centuries: need for early screening

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To cite: Burlakoti A, Kumaratilake J, Taylor J, et al. Trend of cerebral aneurysms over the past two centuries: need for early screening. BMJ Open 2024;14:e081290. doi:10.1136/ bmjopen-2023-081290

Prepublication history and additional supplemental material for this paper are available online. To view these files, please visit the journal online (https://doi.org/10.1136/ bmjopen-2023-081290).

Received 24 October 2023 Accepted 14 February 2024



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### **ABSTRACT**

Objective Cerebral aneurysms (CAs) are linked to variations in the cerebral basal arterial network (CBAN). This study aimed to find the optimal age for screening to detect brain arterial variations and predict aneurysms before rupture.

**Design** An observational, quantitative and retrospective research.

Setting The study analysed 1127 cases of CAs published from 1761 to 1938, Additionally, CT angiography images of 173-patients at the Royal Adelaide Hospital (RAH), South Australia between 2011 and 2019 were examined for the presence and the location of aneurysms in CBAN. Participants The data were collected from patients at RAH and 407 published sources, including males and females across the entire age range, up to 100 years old. Outcome measures and results Data, CAs cases, from 1761 to 1938 included (526 males, 573 females and 28 unknown sexes). The age of these patients varied from 18 months to 89 years (mean age=42, SD=18). Approximately 11.5% of the CAs occurred in patients aged <20 years. Among the 1078 aneurysms whose location was reported, 76% were located in the internal carotid (IC), middle cerebral (MC) and anterior communicating artery complex (AcomAC) regions, while the remaining 24% were in the vertebrobasilar region. Among 173 patients from RAH aged between 18 and 100 years (male=83 and female=90, mean age=60, SD=16), 94% of the CAs were found in the IC, MC and AcomAC regions. The pattern of aneurysm occurrence, as indicated by values at the 25th, 50th and 75th percentiles, along with the minimum and maximum patient ages, has remained consistent from 1761 to 2019. **Conclusion** The distribution pattern of CAs in relation to

## INTRODUCTION

Anatomical variations among components of the cerebral basal arterial network (CBAN) in addition to the trauma, infection, spontaneous dissections and collagen disorders have been linked to the formation of cerebral aneurysms (CAs) 1-3 and such variations develop during the period of embryonic life.<sup>2</sup> The period taken for the development of CAs may vary among individuals and once formed they may enlarge, compress the surrounding

sex, age and locations in the CBAN, remained steady over

the last 260 years resulting in risk of strokes early in life.

Therefore, early screening for CBAN segment variations is

advised for stroke prevention if possible.

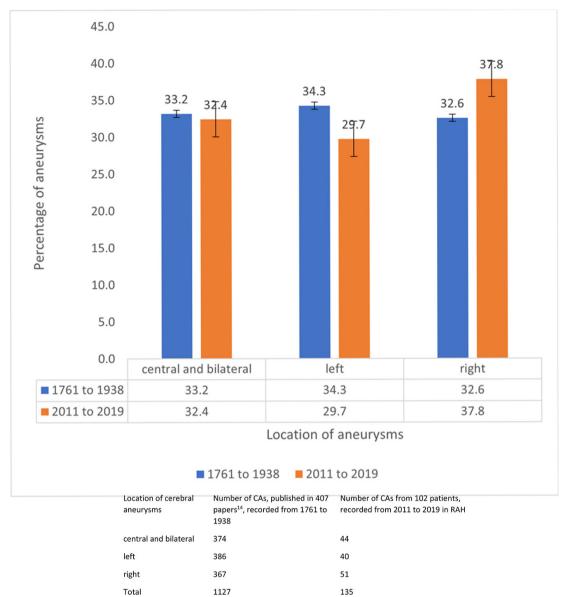
# STRENGTHS AND LIMITATIONS OF THIS STUDY

- ⇒ To our knowledge, the patterns of distribution and trends of occurrence of cerebral aneurysms have not been systematically studied over the past 260
- ⇒ Aneurysms can develop at any age in the presence of variations in cerebral basal arterial network (CBAN).
- ⇒ Early detection of variations in CBAN in infant using non-invasive Doppler ultrasound technique is recommended and continuing screening regularly as needed.
- ⇒ Reported cases from the tertiary medical centres and 407 papers published over the past 260 years may not represent the general population precisely.
- ⇒ This investigation is not a continuous study.

tissues and rupture leading to subarachnoid haemorrhage (SAH).<sup>3</sup>

CAs of all sizes have been observed to cause SAH in adults<sup>4</sup> (incidence 6–10/100 000), however, they also occur in the age group 0-20 years (incidence rate=1.4-2 per 100 **3** 000). 5-7 It is not clear that the occurrence of anatomical variation-related aneurysms is limited to any specific age. The management of complicated CAs is costly and the CAs can leave permanent disabilities or even become fatal costing millions of dollars to families and governments. 7-12 The majority of childhood SAH (ie, incidence 1.4-2 per 100000 children) are caused by the pre-existing CAs.<sup>13</sup> About 5% of the total cerebral aneurysmal cases diagnosed in the clinical setup were in the age group 0-19 years and the incidence of childhood SAH is significantly greater in the older age children. The clinical manifestation of aneurysmal cases seen later in life might be the consequence of aneurysms that developed in early childhood. Therefore, this study aims to review cases of CAs using data collected from a tertiary medical centre (Royal Adelaide Hospital (RAH), South Australia) and published sources to investigate the recent pattern of CAs and how it has changed over the past 260 years. The null hypothesis is that the advancement of





**Figure 1** Comparison of the location of cerebral aneurysms (CAs) between Royal Adelaide Hospital (RAH) sample (2011–2019) (n=135 CAs from 102 patients, orange colour) with those recorded in 407 publications (1761–1938) (n=1127 CAs, blue colour).

medical science did not lead to a reduction in the prevalence of aneurysms by age.

# MATERIALS AND METHODS Study design and setting

Two types of data were used in this study:

- 1. Type 1 data are composed of 1127 cerebral aneurysmal cases that were published in the 407 papers from 1761 to 1938, as compiled by McDonald and Korb. <sup>14</sup> These CAs were identified at autopsy and included patients of all ages (average age=41.7 years, mode age=41 years, median age=41 years, SD=17.7, age range 1.5–89 years) (online supplemental files 1 and 2).
- 2. Type 2 data were cerebral CT angiography (CTA) images obtained from 173 randomly selected patients, who visited RAH, South Australia, between January 2011 and December 2019 for a variety of cranial pathologies;

their age ranged from 18 to 100 years, males=83, females=90, mean age=60 years, median age=62 years, mode age=61 years, SD=15.72) with (n=102) or without (n=71) aneurysms (online supplemental files 2 and 3). These images were anonymised, stored in the Carestream data registry system. The consent documents taken from each patient were not provided to the researchers to ensure privacy. The research materials used in this study comprised 1229 observed cases of CAs that spread across all age groups, spanning a period of approximately 260 years.

# **Data sources and size**

Type 1 data: a range of variables (such as, the year CAs was detected, age, sex, location of the aneurysm) related to 1127 cases of CAs reported in publications from 1761 to 1938, <sup>14</sup> were transferred into an excel data file, rearranged and subjected to analysis (online supplemental

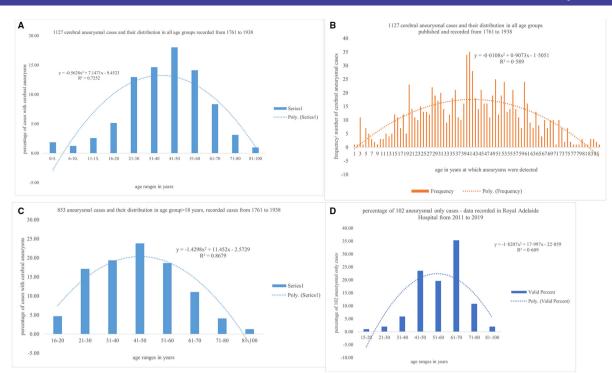


Figure 2 Figures displaying the distribution patterns of cerebral aneurysms (CAs) in different age groups recorded from 1761 to 1938 and from 2011 to 2019. A polynomial regression lines show the number and distribution of CA cases across all age groups. (A) The distribution of cerebral aneurysmal cases (n=1127) in various age groups, recorded from 1761 to 1938. 14 (B) The frequency of cerebral aneurysmal cases and their distribution (n=1127) across all age groups recorded 14 from 1761 to 1938. (C) Age-related (≥18 years) distribution of individuals affected with CAs over the past 260 years (1761–1938) (n=853), recorded 14 from 1761 to 1938, and (D) age-related (18-100 years) prevalence (%) of CAs in Royal Adelaide Hospital (RAH) sample from 2011 to 2019 (n=102). The peak prevalence occurred between 31 and 60 years (p<0.001).

file 1). Type 2 data: the cerebral CTA of 173 patients recorded from 2011 to 2019 in RAH were accessed to study the presence and absence of CAs in different locations of CBAN based on diagnoses made by clinicians. Some cases had multiple aneurysms located in the various segments of CBAN (online supplemental file 3).

The above cases of CAs were grouped into age ranges 0-5, 6-10, 11-15, 16-20, 21-30, 31-40, 41-50, 51-60, 61–70, 71–80 and >81 years and transferred into the SPSS V.25 software, for analyses (online supplemental file 1). The observation error has been tested by repeating the observation of the location of CAs in the cerebral CTA images in 20 cases, a month after the first study. There was 100% agreement of repeated observations with those of the first one. The sites of the formation of aneurysms were recorded as the left and right, internal carotid artery (ICA), middle cerebral artery (MCA), anterior cerebral artery (ACA), anterior communicating artery complex (AcomAC), posterior communicating artery, posterior cerebral artery, vertebral artery, basilar artery, posterior inferior cerebellar artery, anterior inferior cerebellar artery, superior cerebellar and pial arterial regions. In some cases, the areas of location of aneurysms seemed not to have been mentioned and those cases were tabulated under the heading of 'aneurysms located in CBAN'. Overall, the locations of nearly 1229 aneurysmal cases from both datasets were broadly divided into four categories: central and bilateral, left and right (figure 1) before being plotted in the bar charts to study the location and distribution trends of aneurysms in the arterial network over the past 260 years (figure 2). The aneurysms located in the AcomAC, and basilar arterial regions were classified as the central group of aneurysms. Additionally, in a few cases aneurysms were located simultaneously on left and right sides and those cases were grouped as 'bilateral' (online supplemental files 1 and 3 and figure 1).

### Statistical methods

Data were analysed using Excel and Statistical Package for the Social Sciences (SPSS-IBM, V.25) program (eg, descriptive and  $\chi^2$  tests). The p values <0.05 were considered as statistically significant.

Patient and public involvement
Involving patients was challenging for conducting and planning this research since researchers were allowed.

planning this research, since researchers were allowed the access only to anonymised raw data recorded in the database. As per the ethics permit, we accessed retrospective anonymised data, precluding patient involvement in research planning and execution. The shared outcome of this study will be informed to the public, families and patients who attend medical centres for various clinical visits, through a series of meetings, seminars and media releases.

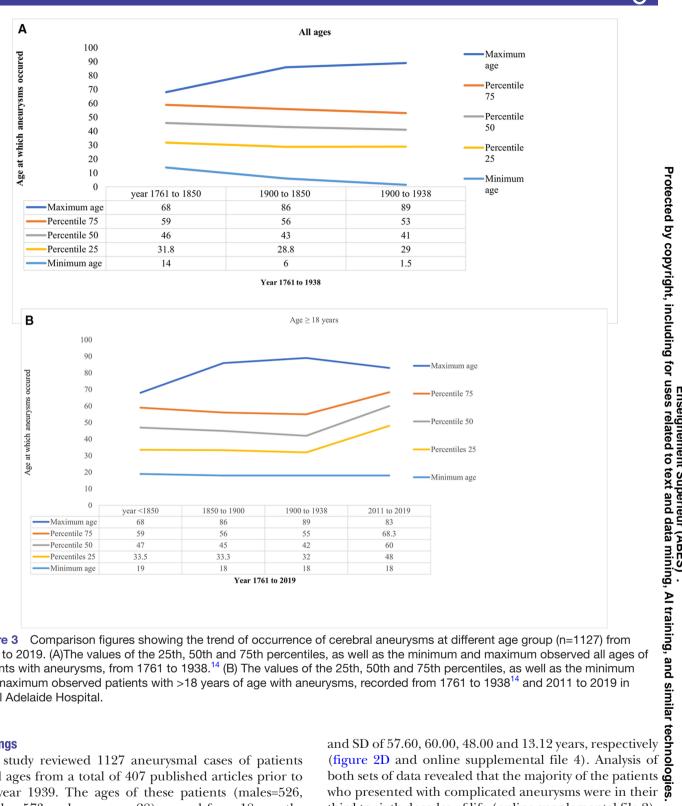


Figure 3 Comparison figures showing the trend of occurrence of cerebral aneurysms at different age group (n=1127) from 1761 to 2019. (A)The values of the 25th, 50th and 75th percentiles, as well as the minimum and maximum observed all ages of patients with aneurysms, from 1761 to 1938. 14 (B) The values of the 25th, 50th and 75th percentiles, as well as the minimum and maximum observed patients with >18 years of age with aneurysms, recorded from 1761 to 1938<sup>14</sup> and 2011 to 2019 in Royal Adelaide Hospital.

### **Findings**

This study reviewed 1127 aneurysmal cases of patients of all ages from a total of 407 published articles prior to the year 1939. The ages of these patients (males=526, females=573, unknown sex=28) ranged from 18 months to 89 years of age with an average of 41.70 years, mode of 41 years and median of 41 years (SD=17.7) (online supplemental file 2, figure 2A-C and online supplemental file 1). The second group of patients with CAs (44 males and 58 females, and n=102) from RAH (2011–2019) with the age range 18-100 years showed that the most common age for diagnosis or complication of CAs ranged from 31 to 60 years with the calculated mean, median, mode

both sets of data revealed that the majority of the patients who presented with complicated aneurysms were in their third to sixth decades of life (online supplemental file 2).

The most important aspect of the two sets of data was the wide age range of occurrence of CAs and the fact that some of the complicated aneurysmal cases appeared at an early age (figure 2A,B, online supplemental file 1). A separate analysis was conducted for 853 out of the 1127 cases of CAs recorded before 1938 (males=409, females=438, unknown sex=6), specifically focusing on the age range of 18-89 years to align the age groups with

training, and similar

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Prevalence of cerebral aneurysms in males and females: a comparison of the recent hospital-based data recorded in RAH from 2011 to 2019 with the autopsy data published before from 1761 to 1938

Sex	N=173, cases with or without cerebral aneurysms recorded in RAH from 2011 to 2019	N=1127 aneurysm cases (from 1761 to 1938) recorded in 407 publications <sup>14</sup>
Sex not defined	0	28
Female	90	573
Male	83	526
Female-to-male sex ratio	1.08	1.09

\*This study investigated the prevalence of cerebral aneurysms in both males and females, drawing a comparison between recent hospitalbased data recorded in RAH (2011-2019) and autopsy data published between 1761 and 1938. N, Number of aneurysmal cases; RAH, Royal Adelaide Hospital; RAH, Royal Adelaide Hospital.

the RAH recorded data from 2011 to 2019 (online supplemental file 2, figure 2C,D). The similarities of SD (15.45) of those 853 cases (from 1761 to 1938) and the cases that were recorded from 2011 to 2019 in RAH (13.12 years) validated the comparability of our data and the findings (online supplemental file 2). The values of the 25th, 50th and 75th percentiles, as well as the minimum and maximum observed ages of patients with aneurysms, remained relatively stable from 1761 to 1938 (figure 3A). Some of these percentile values increased slightly as life expectancy extended from 1761 to the 21st century (figure 3B). Therefore, the SD, and age distribution of adult patients with ruptured or diagnosed CAs presented in the 2011–2019 dataset were consistent with those cases reported before 1938, indicating persistence of a pattern (table 1, figures 2 and 3). Specifically, aneurysms are being frequently diagnosed in individuals aged 30-60 years, and this age range has remained relatively unchanged over the past 260 years (table 1, figures 2 and 3). Forty-nine out of 1127 cases recorded across 407 publications from 1761 to 1938 seemed not to have information about the location of aneurysms in the CBAN, however, 818 out of 1078 identified aneurysms (76%) were in the ICA, MCA and AcomAC regions and rest of them were in the vertebrobasilar region (online supplemental file 1). The location and distribution pattern of aneurysms from 102 patients recorded in RAH was consistent with 1078 cases recorded from 1761 to 1938 (online supplemental files 1-3).

In the type 2 dataset, a total of 135 aneurysms were identified in 102 individuals, with ages ranging from 18 to 83 years, across various components of CBAN (figure 2D and online supplemental file 3). Among these aneurysms, 38 (28.14%) were detected in the right MCA region, while 17 (12.6%) were in the right ICA region. In comparison, the left MCA and ICA regions had 27 (20%) and 12 (8%) aneurysms, respectively, which appeared to be lower in number compared with the right MCA and ICA regions. When considering the distribution of aneurysms based on territory, 55 out of 135 aneurysms (40.74%) in 50 patients were found in the right ICA and MCA territories, whereas 39 out of 135 aneurysms (28.88%) in 37 patients were detected in the left ICA and MCA regions (online

supplemental file 3). Out of the 102 individuals with aneurysmal cases included in the study, 33 (24.44%) had aneurysms located in the AcomAC region, accounting for 33 out of the total 135 aneurysms. An additional 5.9% of the total aneurysms (8 out of 135 aneurysms) were found in the vertebral and basilar arterial regions, as indicated in the online supplemental file 3. A majority of the CAs, 127 out of the total 135 (94% of the total), were in the MCA, ICA and AComAC regions (online supplemental file 3). Some cases had multiple aneurysms, for example, 2 cases had right ICA and MCA aneurysms, while 10 cases had left ICA and MCA aneurysms (online supplemental file 3).

There were no significant differences between male and female patients affected with CAs in all 1229 cases analysed in those two datasets ( $\chi^2$  statistic=0.83, p $\geq$ 0.36) (table 1). The sex, age of occurrence and location of CAs appear to have remained steady over the past 260 years across all age groups (table 1 and online supplemental file 2, and figure 3). The mode, mean and median age and SD of patients with ruptured or diagnosed CAs studied from 2011 to 2019 in RAH matched well with the cerebral aneurysmal cases recorded in the past considering the difference in life expectancy between the two time periods studied (1761-1938 and 2011-2019) (figure 3 and table 1).

### **DISCUSSION**

The age and locations at which CAs occur in the CBAN has not changed over past 260 years (figures 1–3, table 1) despite the life expectancy has increased over time worldwide and the progress in medicine. In the past people had shorter life span on average, and yet the CAs occurred at the same ages as they do now. 15 The life expectancy recorded at below 50 years in 1940 and even below 40 years in 1850 was way lower compared with the one recorded above 80 years of age since the year 2000 in Australia. 15 A separate analysis was done for 853 out of the 1127 CAs recorded before 1938 focusing on the age range of 18-89 years to align the age group with the currently RAH recorded data from 2011 to 2019, since there were no aneurysmal cases of children (age <18

years) in the RAH dataset. In Adelaide, there is a separate hospital for children where aneurysmal cases would have been treated, but the authors had no access to these data (online supplemental file 3). RAH is a general hospital, thus individuals aged 18 years and less are not admitted. Current study compared the cases of CAs diagnosed by CTA imaging technique (from 2011 to 2019) with those verified by surgery and autopsy, 14 since there were no cerebral angiogram facilities in early years (ie, before 1938). The cases of aneurysms are commonly diagnosed, when the patients are presented at medical centres after attacks of stroke. 16 CAs in the past seemed to be ruptured and complicated as early as 18 months of age and as late as 89 years of age with a wide range of age (online supplemental file 1). The findings suggested that the change in lifestyle or medical practice had no effect at the age/ time of formation of CAs in general population. Clinical investigation of lipid profiles in patients commenced after 1950, <sup>17</sup> and they started attributing arterial diseases and aneurysms to the hyperlipidaemia, however, the manifestation of occurrence of aneurysms by age in the past 260 years seems not to be different from the current age of occurrence. Although the lifestyle and the external influences, including medical practice, changed over more than two centuries, aneurysms still occur at approximately the same age. Therefore, aneurysms occur and rupture on their own internal circumstances and are not related to the diet, environmental and external factors.<sup>18</sup> The most likely internal factor is the severity of the variation on the segments of CBAN that adversely affects the haemodynamics resulting in the formation of aneurysms. 1 19 The condition of the arterial wall should not have changed over the last 260 years and that seems to be less significant than the variation in the components of CBAN. The segmental and communicating arteries play a crucial role in dampening the systolic pressure within the CBAN and reducing the likelihood of aneurysm formation. 1 19 The severity of arterial variation can have negative effects on the blood flow dynamics through the variant segment of the component of the CBAN. The incidence of CAs is about 3.3% in the general population and may not be diagnosed, until they get enlarged as the size of the aneurysm <3 mm in diameter can be missed.<sup>20</sup> Imaizumi et al found that the prevalence rate of CAs was 4.32% in Japan. 12 The incidence rate of CAs in childhood (age <18 years) has been reported to be 0.5%-4.6%, which is almost as common as the incidence rate observed among adults. 13 Treating cases of CAs with a diameter < 3 mm requires careful consideration, as pre-existing small aneurysms of ≤3 mm could rupture, resulting in spontaneous SAH.<sup>21</sup> The majority of CAs are detected only when they cause a stroke or other pathological effect (eg, compression of the optic tract). Individuals >18 years are no longer considered children.  $^{6\,13}$ 

Most of the symptomatic cases of CAs in the paediatric age group were observed in older children (15+ years), <sup>13</sup> and only complicated cases of CAs were generally diagnosed and reported. 22 23 If the incidence

of childhood CAs described (ranges from 0.17% to 4.6%). Si scorrected for number of years lived, it would be 18.4% of the total aneurysmal cases among adults. The adult patients included in CAs studies ideally have an age range of 18 years and above, which can include individuals up to the age of 100 years. Si Incontrast, the childhood group included in aneurysmal studies typically ranges from birth up to 18 years of age and a few studies have categorised patients who are 18 years or older under the adult group. Si When the age range, 0–18 years and 19–100 years is considered, the incidence of childhood CAs, that should the multiplied by 5 times to correct for the number of years lived, can be comparable to that in adults because the childhood period of life is much shorter of than the adulthood. Therefore, the age range of adult ygroup (≥20 years up to 100 years) included in the CAs years up to 100 years) included in the CAs years and stroke studies would be about 5 times more than the age range of children (ie, ≤18 years). The means adults have 5 times more years to develop CAs compared with children. Therefore, the incidence of childhood CAs per year is almost equivalent to adult. Si Hence, CAs could develop in early childhood in the presence of a significantly variant component of cerebral arterial anatomy, and it could take years for them to balloon before becoming symptom atic and being observed in a tertiary medical centre. The overall pattern of location and distribution of childhood CAs was similar to adult as they commonly occurred in ICA, MCA and AcomAC regions. Therefore, the development of CAs is not age related and found to be prevalent in all age ranges. Therefore, the development of CAs is not age related and found to be prevalent in all age ranges. Therefore, the development of CAs is not age related and found to be prevalent in all age ranges. Therefore, the development of CAs is a sample of cannot be advised to be 55–57 years of age in a study conducted using 1085 aneutysmal cases from 2008

for detecting brain aneurysms becomes available. The current screening recommendation is based on the congenital variations of segments of CBAN, but such variations could occur later on life in cases of pathology like atherosclerosis and could cause aneurysms. Future studies to test the association of presence of anatomical variations in CBAN in infancy and future risk of both unruptured and ruptured intracranial aneurysms in adulthood are recommended.

The estimated cost of a single stroke is approximately \$A300 000 in Australia. With a haemorrhagic stroke incidence of 10 per 100 000, the total cost amounts to \$A45 million per year in a city like Adelaide, South Australia, which has a population of 1.5 million. Regular screening for individuals with significantly variant brain arteries identified, representing 50% of the population, once every 5 years, and assuming the cost of a single CTA or magnetic resonance angiogram is about \$A100 each, the total screening cost would be \$A1.5 million per year, that means 30 times reduction in cost of strokes. Additionally, the government would receive millions of dollars in return as tax revenue from working individuals who would survive with little to no disability from potential strokes resulting from aneurysms. This study was not designed to examine the characteristics of aneurysms, but the focus was on the distribution of aneurysms in different segments of CBAN, trend of occurrence of aneurysms over the past 260 years and the comparison of CAs in all age ranges.

### Limitations

The insufficient data on the lack of personal and family history, history of smoking, lipid profile and blood pressure are limitations of this study. A larger survey and a prospective study could be conducted. A prospective study could involve using ultrasound techniques to identify variations in brain vessels among infants.

### **CONCLUSION**

Brain arterial aneurysms can develop early in the presence of variant arterial components. Screening children under 24 months using transcranial ultrasonography for variant cerebral arteries may be practical. Those with variations should undergo periodic tests for aneurysms, aiming to prevent some haemorrhagic strokes if an affordable and convenient technology for detecting brain aneurysms becomes available.

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Contributors AB is responsible for the overall content as the guarantor and accepts full responsibility for the work and the conduct of the study, has access to the data ands control the decision to publish. AB conceived the idea, collected and analysed both sets of data, took pictures, recorded videos, contributed to conceptualisation, prepared and drafted the manuscript. JK conceived the idea, contributed to the concept, aided in data interpretation, editing and the revision of the manuscript and approving the article. JT conceived the idea, contributed to the concept, aided in data interpretation, editing and the revision of the manuscript and approving the article. MH conceived the idea, masterminded and helped in statistics, data analysis and interpretation, editing and approving the article.

**Funding** The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

**Patient and public involvement** Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval The Human Ethics permit (approval number: H2014176, Research Ethics Committee, Office of Research Ethics, Compliance and Integrity, Faculty of Health Sciences, University of Adelaide) granted permission to access and use the de-identified dataset from the Carestream data registry system (Vue-RIS-version-11.0.14.35) for research. The patients gave their consent to use their clinical information for research activities.

Provenance and peer review Not commissioned; externally peer reviewed.

**Data availability statement** Data are available on reasonable request. Not applicable/uploaded as supplementary information.

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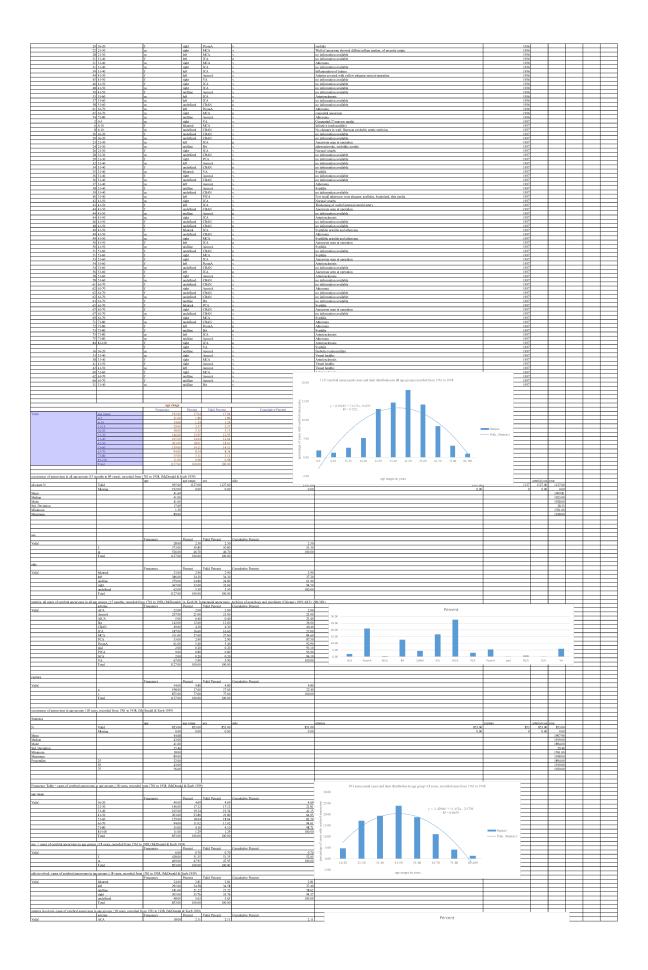
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		for early screening - An obse	rvational study		ACA-anterior cerebral artery, PC/						
	ARL Academic sterior corecular ariety. VA-veneroria steriy. UnAc-vectoria na age in years  60 51-60	sex, m-male, f-female m	side affected w bilateral	nt. FR. A posterior strener cereocus streny. Me arteries involved status of ancuryens; ruptu PcomA n	ered-y, not ruptured-n	A. Active on intertrain intertyres. Archives of neurosopy and payerinary (Caseago) 1999; 40,21; 296-348.1 arterial continues on intertraining with deposit on inner faining	year of aneuryum detected	161			
P. C.								778 179 124			
The color	20 16-20 57 51-60	f m	midline midline	AcomA y		Calcareous deposits on coats of vessels	1	125			
Company	99 51-60	f	midline midline	AcomA y AcomA y		not available no information available	1 1	25			
Column	19 16-20	m m	right left	MCA y		Annance major or sec. commissiones no information available no information available	1	26 27			
Column	39 31-40 45 41-50 35 31-40	m m	left left			no information available no information available Carotid and vertibral arteries normal		27 27 28			
Column	21 21-30 24 21-30	m	left	BA y VA y		Calcarcosa deposits on vessels  Arterior diseased, as some places cartilaginous	i	29			_
1	63 61-70	r	right left	MCA y		wall thin but firm  No bone or other appearance of disease	1	31			
A	601 51-60 42 41-50 54 51-60	m f m	right right midline	MCA y ICA y BA y		Nac with this. Jurn parastes no information available no information available					
	28 21-30	1	left left	ACA n ICA n VA n		models layer thick no information available no information available		34 34			
	68 61-70 20 16-20 90 15-60	m í	midline midline	BA n BA n		Yellow plagues on surface no information available	l l	36 37			=
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	20 16-20		undefined	PCA n		no information available Vessels thickened		H2			=
	41, 41-59 54, 51-60 20, 16-20	m r	midline midline right	BA y BA n PoomA y		no information available		i44 i44			
	58 51-60	m m	midline midline	BA y BA y BA y		no information available na	1	i46 i46			
Column	62 61-70 34 31-40	í m	right left	PomA y PomA y		no information available no information available	1	i46			
Column	52 51-60	(	1-6	ICA v		haller estados	1	148			
The color   The	33 31-40 35 31-40 39 31-40	m f	midline midline undefined	BA y BA y ICA y		no information available Other arturies healthy No calcarcous deposit in cerebral vessels		i49 i49 i49			
1	18 16-20 30 21.30	(	undefined left	PcomA y		No fatty atherografists changes in arteries	1	i49			
Column	70 61-70	í	left undefined	ICA n		no information available no information available	1	i50			
		m	left	MCA v		an information would be	1	151			=
	45 41-50 65 61-70	m m	midline undefined midline	BA n ICA n BA n		no information available no information available no information available		iS1		=	
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The content of the	56 51-60 57 51-60	m m	right right	PCA y MCA s		no information available Vessels very afteromatous	1:	i55			
Company		m m					1	155			
Company	40 31-40 52 51-60		left right	PCA m		Vessels thin but healthy Model court thickened		i56 i57			
1   2   2   2   3   3   4   4   4   4   4   4   4   4	54 51-60 56 51-60	m m	midline right	BA n		Atherona		158 158			=
Column	17 16-20 30 21-30	ſ	left left	MCA v		Embolic no information available	1:	i59			=
	35 31-40 43 41-50	í	left midline	ACA y BA y		Healthy vessels	1	i59			
	58 51-60 16 16-20 24 21-30	m f	left	MCA y ICA n BA y		Atherena no information available embolic		159 160 160			
1	47 41-50 24 21-30 14 11-15	í m	midline right	BA n MCA v		Walls of sec calcurates		61			
Column	42 41-50 48 41-50	m (	midline midline	BA y BA n		Vessels otherwise healthy no information available	1:	62 62			
1	68 61-70 80 71-80	m r	left	MCA y AcomA n			1	62			
	37 31-40	m m		BA y AICA		Healthy vessels Other vessels slightly atheromatous Athereems		64 64			
1   1   1   1   1   1   1   1   1   1	24 21-30	m m	left left midline			Soft arteries	l l	65 65			
1   1   1   1   1   1   1   1   1   1	43 41-50 46 41-50	ſ	left left	ICA y ACA s		Walls atheromatous Vessels very atheromatous	1:	65 65			
Column	51 51-60 53 51-60	f m	midline left	BA y MCA y		Arteries not diseased	1	i65			
1   1   1   1   1   1   1   1   1   1	21 21-30	f m	right left left	MCA y ACA y MCA y		no information available no information available No disease et vesels		65 65			
1   1   1   1   1   1   1   1   1   1	61 61-70 27 21-30		left left	VA y ICA y MCA v		no information available Atteries thickened no information available		l66 l67			
Section   Sect	78 71-80 13 11-15.	í m	right right	MCA y MCA y		No atheroma Rest of arteries healthy	1	167 168			
1	34 31-40 36 31-40	ſ	left midline	MCA a		Walls of ancurysmatheromatous; vessels at base not diseased	1	168			
1	40 31-40 40 31-40 50 41-50	m f	left left	MCA y MCA s		STREAM INFORMEDICES ON WEST Whose It normal Thick and creates but not materially discused	1	68 68			
March   Marc	55 51-60 56 51-60	í í	right left	PomA y MCA n MCA n			l l	68 68			
1   1   1   1   1   1   1   1   1   1	60 51-60	f	right	BA y MCA y ICA y		Vessels very afterornatous Other vessels healthy	li li	168			
1   1   1   1   1   1   1   1   1   1	19 16-20 28 21-30	m	left left	VA y MCA y		no information available	-	169			
1   1   1   1   1   1   1   1   1   1	48 41-50 48 41-50 56 51-60	f m	left right	MCA v		Abereens Several patches ofdegeneration enbasilar artery	1	169 169			
1   1   2   1   1   1   1   1   1   1	61 61-70 12 11-15. 13 11-15.	í	midline right right	BA m MCA y MCA y			li li	76			
1   1   1   1   1   1   1   1   1   1	17 16-20	m f	right left	MCA y ACA y		Embolic origin Vessels healthy	1	170 170			_
1   1   1   1   1   1   1   1   1   1	26 21-30 26 21-30	í m	left midline	MCA n		Vessel plagged by yellow fibrin (vegetations on nortic valves) Remaining vessels healthy					
Col   10	20 21-39 35 31-40 37 31-40	m m	undefined midline	PCA m BA y		no information available		170 170			
14   15   16   17   17   18   18   18   18   18   18	49 41-59 50 41-50 60 51-60	f m	midline left left	BA v PCA v VA v		no information available Slight atherorentous patches Venech diseased		170 170 170			
10   10   10   10   10   10   10   10	54 51-60	m (	midline left	AcomA y MCA y		Atherocastous Arteries at base highly Atherocastous	li li	(76 (71			
	45 41-50 61 61-70	П	midline le0	BA n		Other arteries at base atheromatous Slight atheromatous changes	1	172 172			
	20 16-20 20 16-20 20 20 20 20 20 20 20 20 20 20 20 20 20 2	í m	right undefined	PoomA y CBAN y CBAN	-	Embolic Embolic Embolic was positifin		073 073			=
	21 21-39 27 21-30 40 31-40	m m	undefined right	CBAN y MCA y				673			
11-00	24 21-30		midline undefined	BA n ICA n		W alii misk : other vessels healthy Vessels thickened no information available	1	174			
10   10   10   10   10   10   10   10	40 31-40 43 41-50	m	left right right	ICA n ICA n MCA y		Marked arteriosclerosis Walks of sac partly calcified so information sociabile.	1 1	175			
1.00	45 41-50 45 41-50 44 41-50	n n	midline left	AcomA y MCA y		Arteries slightly afteromatous Vessels normal Intrins arteriosekrosis	1	05 05		=	
10   10   10   10   10   10   10   10	56 51-60	f m	left	VA n		internal mechanicaments with thickness caronic mechanicaments no information available	1	05 05			
1.4   1.4		Ţ	midline right	AcomA n MCA y		Informe atherona of voxeds  Vessels atheronatora no information available	1	175			
1   1   1   1   1   1   1   1   1   1	34 31-40			on a		Rest of arteries free of atheroma		176			
2   2   2   2   2   2   2   2   2   2	34 31-40 34 31-40 41 41-50 50 41-50	m f	midline right	AcomA y MCA y		Androni		C/B	_	 	
1.0   1.0	34 31-40 34 31-40 41 41-50 50 41-50 63 61-70	m m f	midline right right midline right	ICA y BA y MCA n		no information available no information available	1	06 07			_
13   13   15   15   15   15   15   15	34 31-49 34 31-49 44 44-59 40 45-79 41 45-9 41 45-9 41 45-9 41 45-9 41 45-9 42 45-9 43 45-9 44 45-9 45 45-9 46 45-9 47 45-9 48 45-9	m	midline right right midline right midline left	ICA v BA y MCA n AcomA v ICA n		no information coulds!c no information voudsle! no information voudsle! no information voudsle! No other disease of voues! Voucels healthy, probably embelig Healthy words	1 1 1 1 1	06 07 07 07			
0.0   0.5   0.0	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1		midline right right midline right midline left right left right	ICA y BA y  MCA n  Accord y  ICA n  MCA y  MCA y  MCA y		ass a foresterion exceluble, as a foresterion resultable, as a foresterion resultable, See description of resultable, See de		06 07 07 07 07 07 07			
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Add   1.4   Add	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	m	midline right right right midline right keft right keft right midline right keft right right right keft right right keft right keft	ICA S BA Y MCA B X MCA S MCA		an elementar analysis.  Son deer deer analysis of the control of t		06   07   07   07   07   07   07   07			
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0   11-0   F	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1		midline right right midline right midline right midline right left right midline right left right midline midline midline midline midline midline midline midline	ECA. S.		an elementar analysis.  See description analysis.		06   07   07   07   07   07   07   07			
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15   15   25   25   25   25   25   25	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1		midline right right midline right midline right midline right midline right left right midline midline left left right midline midline midline midline midline	Li CA		an administra analolis.  See dee donne franche.  See d		076 677 777 777 777 777 777 777 777 777			
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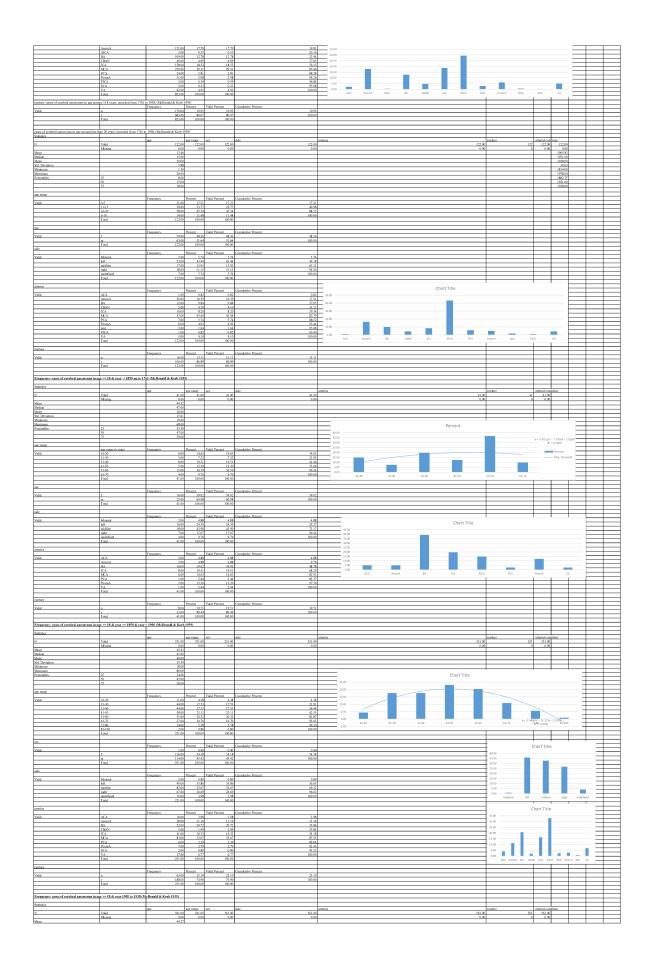
	ad to to	T F	To one	 		
1	27, 21-30 20, 16-20 30, 21-30		Fireholie (mulay-mulitia)	1886 1887		
Column		right MCA	Embolic (indocuratio)  Find of the (indocuratio)  to information available	1887	+	=
Column	36 31-40 40 31-40	midline BA right MCA	No embolism or atheroma No embolism or atheroma	1887		
	43 41-50 49 41-50		Embolic (endocarditis)	1887	=	_
The color	00 31-00	left VA	Arterioselerosis	1887	=	
Column	53 51-60	undefined VA midline BA	Arterioscierosis no information sociable	1887 1888		
Column	26 21-30 43 41-50	midline BA midline AcomA	Septic endocarditis no information available	1889 1889		
	56 51-60	midine AconA midine AconA	Arterios free of atheroma Arterioseleroses	1889	=	_
	15 II-15. 15 II-15.	nght PCA	Septse embolus endocurditis	1890	=	
1	15 11-15.	right MCA	Septic embolus endocarditis	1890	=	
1	15 11-15. 16 16-20	right MCA	Septie embolus endocarditis Septie embolus	1890		
Column			Septic embolus endocurditis Septic embolus	1890		
Column		right MCA	Scotte emission emiscarium	1890 1890	=	
	23 21-30	right MCA	Septic embolus endocarchis Sentic embolus endocarchis Sentic embolus endocarchis	1890 1890	=	_
	31 31-40 35 31-40	right MCA left MCA	Septic embolus Septic embolus endocardris	1890 1890		
March	45 41-50	Heft IMCA	Atherens	1890		
Column	50 41-50	left MCA	Atheroma Septic embolus	1890	=	_
	30 31-99 66 61-70		Anterona Atherona	1890	=	_
	35 31-40 47 41-50	midine BA	Other vessels healthy No atheroma	1892	$\vdash$	
Color   Colo	7 6-10. 10 6-10.	midline BA left VA	no information available no information available	1894 1894		
	25 21-30	midline BA	no information available	1894 1894	=	==
	26 21-30	right ICA	- information and date	1894	$\vdash$	
	28 21-30 20 21-30	midline AcomA left MCA	no information available no information available	1894		
	30 21-30 31 31-40	midline AcomA		1894	+	_
Column	32 31-40	midline BA	no information available	1894	=	==
	36 31-40 36 31-40	midline PcomA left ACA	no information available	1894	=	=
	40 31-40	midline BA	no information available no information available	1894	Ħ	$\equiv$
Column   C	41 41-50 42 41-50	right VA left ICA	no information available no information available	1894	$+ \exists i$	$\dashv$
10   10   10   10   10   10   10   10	42   41-50 42   41-50	left MCA midline BA		1894	##	$=$ $\pm$
	43 41-50	left VA	no information available	1894	##	=
Column	43 41-50	right MCA	no information available	1894 1894	Ħ	=
1	44 41.50 45 41.50	midline AcomA	no information available no information available	1894	ฮ	==
March   Marc	45 41-50 45 41-50	midline AcomA left ICA	no information available no information available	1894	$+$ $\mp$	$=$ $\top$
1	46 41-50 46 41-50	midline BA right MCA		1894 1894	##	$=$ $\pm$
	48 41-50	right ICA	no information available	1894	=	=
Column	49 41-50	left VA midline PoomA	no information available no information available	1894 1894	Ħ	
Column	50 41-50 50 41-50	LG MCA	and in Committee and Alberta	1894 1894		
1   1   1   1   1   1   1   1   1   1	51 51-60 52 51-60	right MCA	no information available no information available	1894	${}^{\sharp}$	=
	53 51-60 53 51-60	manne AcomA		1894	=	
Column   C	54 51-60	right MCA	no information available	1894	=	
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Column	56 51-60	sinks MCA	- information and date	1894		
Column	57 51-60 57 51-60	right ACA midline AcomA	no information available no information available	1894 1894	=	_
1   1   1   1   1   1   1   1   1   1	60 51-60 60 51-60		no information available no information available no information available	1894 1894	=	
A.   A.   A.   A.   A.   A.   A.   A.	60 \$1-60 61 61-70	left MCA midline BA	no information available	1894	=	_
March   Marc	61 61-70 62 61-70	right ICA midline AcomA	no information available	1894		
Column   C	63 61-70	midline AcomA	no information available no information available	1894	+	_
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Column   C	67 61-70 67 61-70	left ICA right ICA	no information available no information available	1894 1894	=	_
10   10   10   10   10   10   10   10	69 61-70 70 61-70	left ICA right ICA	no information available no information available	1894 1894		
1	72 71-80 72 71-80	right ICA	no information available	1894	+	_
1   1   1   1   1   1   1   1   1   1	73 71-80	right VA	and information and delay	1894	=	
1   1   1   1   1   1   1   1   1   1	73 71-90 73 71-90 79 71-90	midline AcomA midline BA	no information available no information available	1894 1894	=	_
1	80 71-80 81 81-100	right ACA left MCA	no information available no information available	1894 1894		
1		midline CBAN	no information available	1894		
Column	29 21-30	midline BA	no information available	1895	=	_
1.00   1.00	40 31-40	midline BA		1897	=	
1.00   1.00	50 41-50 53 51-60	left MCA left MCA	no information available no information available	1897	=	
1.00   1.00	69 61-70 28 21-30	bilateral ICA midline BA	no information available no information available	1897 1898		
March   Marc	54 51-60 70 61-70	right MCA	Extensive sclerosis with atheromatous plaques	1898	=	
1   1   1   1   1   1   1   1   1   1	68 61-70	left VA	Vessels atheromatous Embolic (endocarditis)	1900	##	=
17.0   17.0   17.0   17.0   18.0   18.4   18.5	11 11-15. 27 21-39	right pial	Embolic (molocurdita)	1901	$\blacksquare$	=
17.0   17.0   17.0   17.0   18.0   18.4   18.5	28 21-39 32 31-40	midline BA right MCA	no information available Embolic (endocaeditis)	1901	$+ \exists$	$-\mathbb{T}$
10   10   10   10   10   10   10   10	50 41-50 76 71-80		no information available	1901	##F	
10   10   10   10   10   10   10   10	29 21-30 34 31-40	undefined CBAN	normal vessels	1902	##	=
4   1.5	22 21-30	right ICA right MCA	healthy Line of atheroma at point of ancuryon	1903 1903	$\pm \mp$	=+
Main	64 61-70 27 21-30	right PcomA	Syphilis Wells of other enterior healths	1903	$+ \exists i$	$\dashv$
Column   C	40 31-40 65 61-70	right ICA right ACA	no information available Atherema	1904 1904	${}^{++}$	=
Column   C	70 (61-70 86   81-100 87 (81-100	medine BA bilateral ICA right ICA	Athereons no information available Athereons	1904 1904	##	=
Mathematical Continues   Mathematical Contin	42 41-50	midline BA right PoomA	Syphilis Marked sclerosis	1905		
A   A   A   A   A   A   A   A   A   A	50 41-50	right VA	Localized syphilitic periorieritis of ancuryon no information available	1905	<b>≠</b> ∓	$=$ $\mathbb{F}$
1		LG MCA		1906	##	=
1		bilateral VA bilateral ICA	Arterisockrouis po information available	1906	=	<b>=</b>
10   10   1   10   10   10   10   10	40 31-40	left BA left MCA	Veneta normal	1907	$oldsymbol{oldsymbol{eta}}$	
15   15   15   15   15   15   15   15	50 41-50	left ICA	no afteroma Arterios healthy	1907	##F	
10   10   10   10   10   10   10   10	55 51-60 61 61-70	bilateral MCA left ICA	no information available no information available	1907 1907	#	=
10   10   10   10   10   10   10   10	63 61-70 65 61-70	midline AcomA	no information available	1907	$\blacksquare$	=
1	68 61-70	midline BA	no information available no information available	1907 1907	$+ \exists$	$=$ $\mp$
10   10   10   10   10   10   10   10		right ICA left ICA	no information available no information available	1907	##F	
1   1   1   1   1   1   1   1   1   1	65 61-70	right MCA	Irregular, cartilaga novas thuckernang of arteries at base Athereoma Thickerning: gyobi like endosterisis obliterares	1908	##	=
10   14 de   15   15   15   15   15   15   15   1	27 21-30 44 41-50	midline BA	Other arteries permal	1910 1910	$oldsymbol{oldsymbol{arphi}}$	=+
27   1-42	57 51-60	bilateral VA left MCA		1910	igoplus	=
27   1-42	25 21-30	right AICA	Thick and hard Thickening of vessels	1910	+	=
Cl.	36 31-40 37 31-40 47 41 45	right ICA	no information available	1911	##	==
CS   S-70   f   Int   ICA   v   Internation examilier   ISI	42 41-50 42 41-50 53 53.40	nght ICA	no information available  Both cerebral arteries thickened	1911	##	=
22 (24.50) f ngh ACA y Assersynt organis 1922 1	62 61-70	left ICA midline BA	no information available Healthy vessels	1911	=	=
S113-36   f   edge   ACA   y	22 21-30	right ACA	Ancuryon congenital	1912	$oldsymbol{oldsymbol{arphi}}$	
	25 21-30 31 31-40	nubt AICA undefined CBAN	Aneuryon congenital No arteriosclerosis	1912 1912	+	=
13   13   26	32 31-40 36 31-40 31 31 44	left MCA left PICA	Ancuryon congenital Walls healthy (probably embolic) Findstale funder—files Findstale funder—files		##	=
Fig.   1,46	37 31-40 38 31-40	left MCA	Ancuryon congenial	1912	=	=
33   3.40 m right VA y Assertson congenied 992   1922   1923   1924   1924   1924   1925   19	38 31-40	right VA left MCA	Ancuryon congenital Embolic (endocarditis)	1912	$oldsymbol{oldsymbol{arphi}}$	
4) 41-50 f left ICA y Asserting congenial 1912 f left 41-50 m left MCA y Enhalter (concention) 1912 f left 150 m	42 41-59 42 41-50	left ICA left MCA	Ancuryun congenital Embolic (endocarditis)		$\pm \mathbb{I}$	=
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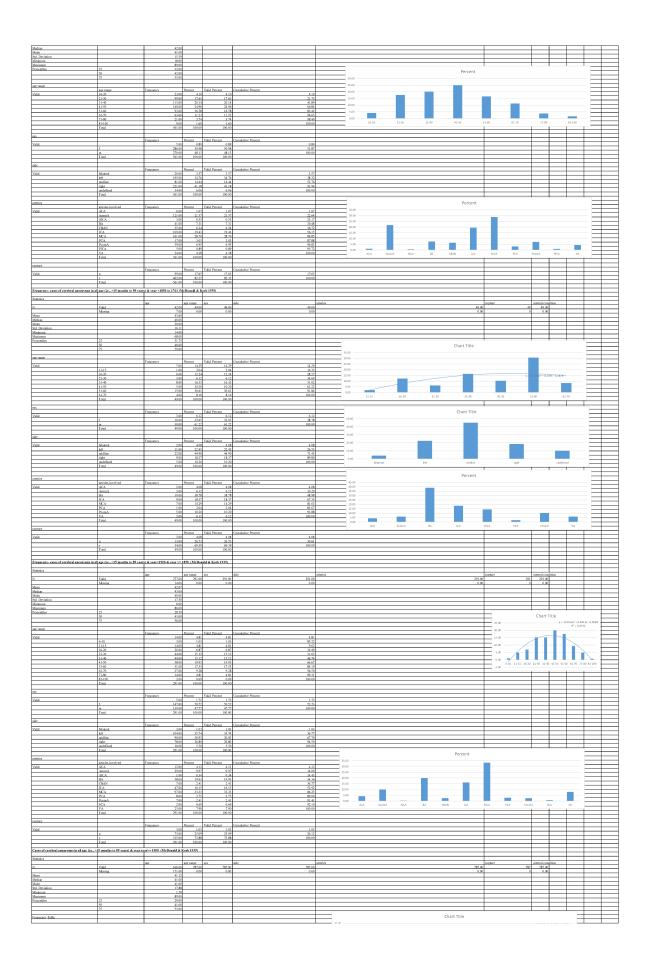
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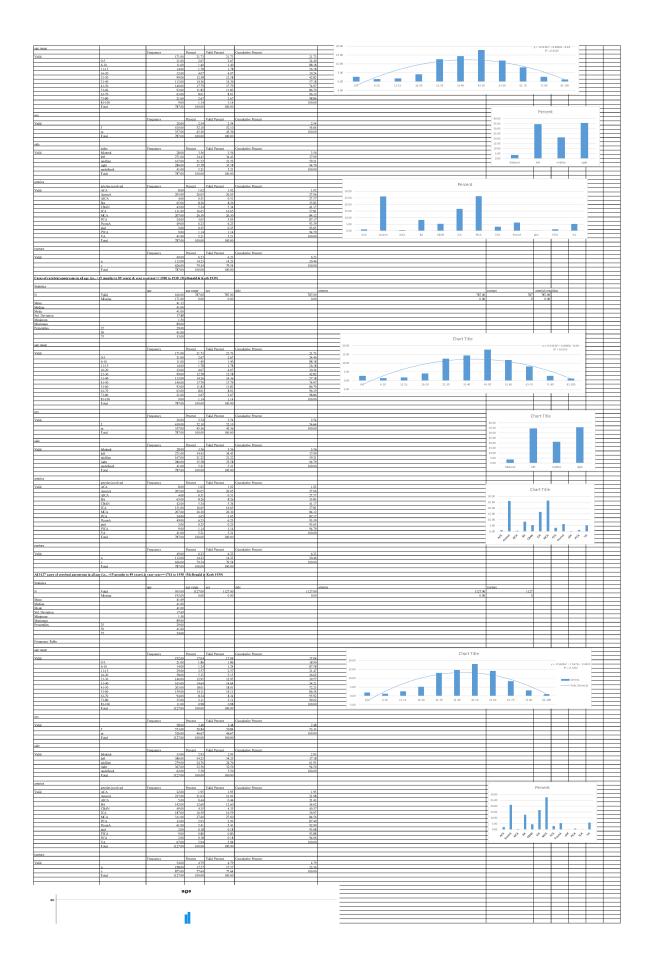
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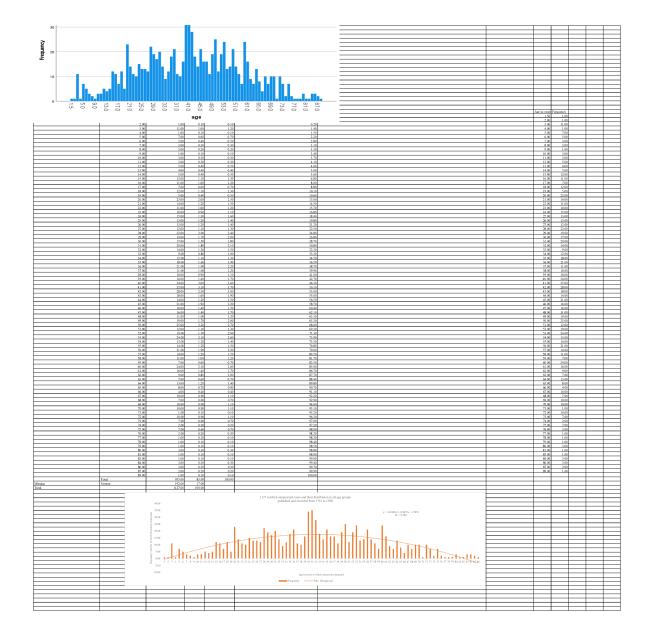
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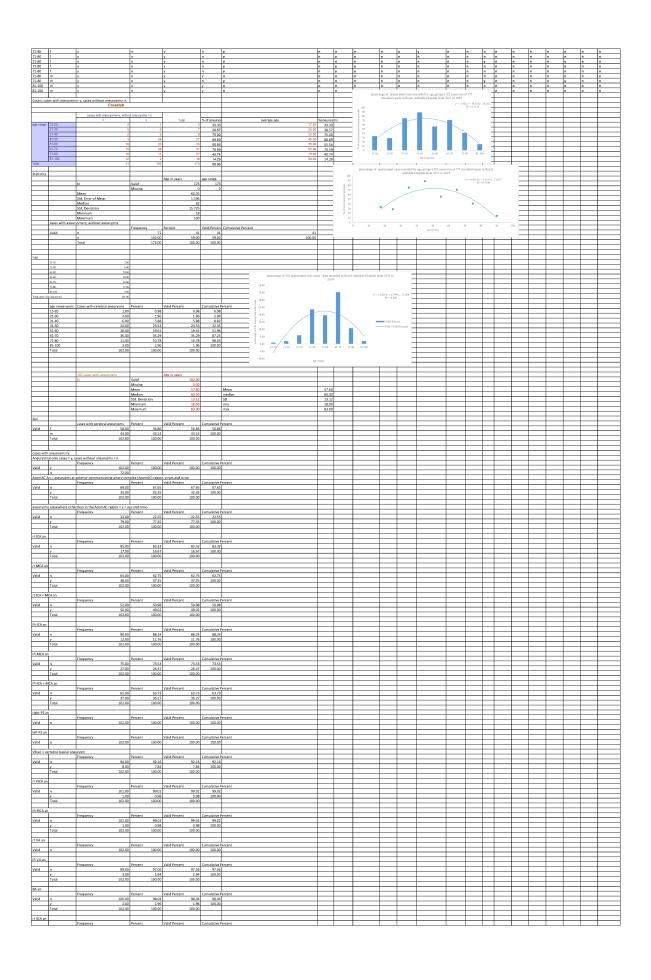
Supplementary File 2: Statistical parameters of distributions of aneurysmal cases reported from 1761-1938, <sup>14</sup> and recorded in RAH from 2011 to 2019.

1/01-1330	o, allule	102	RAH from 2 1127 patients			ns recorded	l in 407 pub	lications - 1	oublished fr	rom 1761 to
		patients	1938							
		with								
		cerebral								
		aneurysmal								
		aneurysms								
		recorded in								
		RAH from								
		2011 to								
		2019								
Statistics -		age>18,	age>18	age >=	age >=	age >=	all age,	all age,	all	all age
all cases		2011 to	years, 1761	18 &	18 &	18 &	year	year <	ages,	group, all
with		2019	to 1938	year <	year >=	year >=	<1850	1900 &	year >=	years, >400
cerebral				1850	1850 &	1900		year >=	1900	publications
aneurysms					year <			1850		
					1900					
Age in										
years										
N	Valid	102	851	41	252	560	42	278	613	935
	Missing	0	0	0	0	0	7	14	171	192
Mean age		57.6	44.7	44.3	45.6	44.3	43.6	42.6	41.2	41.7
Median		60.0	43.0	47.0	45.0	42.0	46.0	43.0	41.0	41.0
age										
Mode age		48.0	41.0	20.0	40.0	41.0	20.0	40.0	41.0	41.0
Std.		13.1	15.5	15.6	15.6	15.4	16.1	17.5	17.9	17.7
Deviation										
Minimum		18.0	18.0	19.0	18.0	18.0	14.0	6.0	1.5	1.5
age										
Maximum		83.0	89.0	68.0	86.0	89.0	68.0	86.0	89.0	89.0
age										
Percentiles	25	48.0	32.0	33.5	33.3	32.0	31.8	28.8	29.0	29.0
	50	60.0	43.0	47.0	45.0	42.0	46.0	43.0	41.0	41.0
	75	68.3	56.0	59.0	56.0	55.0	59.0	56.0	53.0	54.0
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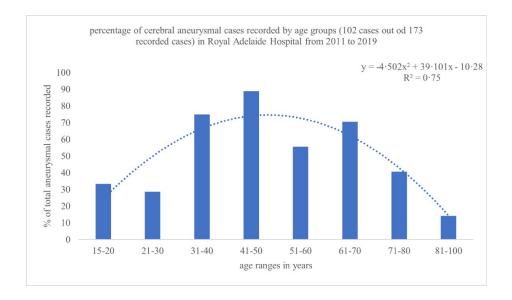
Legend: 14 = McDonald, CA & Korb, M 1939, Intracranial aneurysms, Archives of neurology and psychiatry

(Chicago), vol. 42, no. 2, pp. 298-328.

Column	Supplementa Manuscript ti	ry file 3: contains ty tle: The trend of cer	pe 2 data: The Haman Ethics permit (appreheal ancurysms over the past two cents	peoval number: H2014-1 aries: Need for early sere	76, Research Ethics Commit tening - An observational stu	tee, Office of Re	search Ethics, Compliance and Integrity, Faculty of Health Sciences, Ur	niversity of J	Adelaide) grant	ed permission to acc	ess and use t	the deidentified	data set from the Care	oviream data re	gistry system	(Vue-R	IS-version-1	1-0-14-35) for	r research				
	Abbreviation	rere Cerebral comp se: cases with cereb and C) region, voto	uted tomography angiography images of ral aneurysms—y, cases without cerebral es and nume aneurosms characters other	btained from 173 randon ancurysms—n; cases wife rebus in the Accord Co.	nly selected patients, who vis hout cerebral anearysms = y,	eases with cerel	delaide Hospital (RAH) between January 2011 and December 2019 for oral ancuryons = 1; ancuryonal only cases = y, cases withour ancuryon to othershap in the Accord Common = y = yeared name of ICA an = are	a variety of x = n; Acom	cranial pathole AC An = aneu right internal c	rgies ryens at anterior cor proted setery (et ICA)	review et M	CA un = unco	some in right middle o	erebral artery	region;								
	rt ICA + MC	A an = ancuryens in incuryen in the 2nd	n right internal carotid and middle cerebo d part of right posterior cerebral artery; le	ral arterial region; lft IC/ eft P2 an =aneurysm in t	A an = ancurysms in left inter he 2nd part of the left poster	nal carotid arter for cerebral arter	y (Iff ICA) region; Iff MCA an =aneurysms in left middle cerebral artery y; VB an = aneurysm in vertebro basilæregion; rt PICA an = aneurysm	region; lft I in the right	CA + MCA an posterior infer	-ancuryoms in left in ior cerebellar (rt PIC	A) region;	id and middle	cerebral arterial region										
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																							=aneurysm in the left superior
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	21-30	1	n n	y y	n n	n n	n n	n n	n n	n	n n	n n	n n	n	n	n	n n	n n	n n	n	n n	n n	n
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	41-50 51-60	m m	n n	y y	n n	n n	n	n n	n n	n	n n	n n	n n	n	n	n	n n	n n	n n	n	n	n n	n n
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Mathematical   Math	71-80	m m	n n	y y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n	n n	n n	n n	n n	n n	n n	n n
Mathematical   Math		m m	n n	y y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
Mathematical   Math	81-100	m m	n n	y y	n n	n n	n n	n n	n n	n n	n n	n	n n	n	n	n	n n	n n	n n	n	n n	n n	n
18	81-100	m f m	n n	y y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
Mathematical	81-100 81-100	m r	n n	y y	n n	n n	n n	n n	n n	n	n n	n n	n n	n n	n	n	n n	n n	n n	n	n n	n n	n n
Mathematical Content	81-100 81-100		n n	y y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n	n	n n	n n	n n		n n	n n	n n
	81-100 81-100	m f	n n	y y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
1.   1.   1.   1.   1.   1.   1.   1.	15-20 21-30	r m	y y	n n	y y	n n	y y	n n	n n	n	n n	n Y	n Y	n	n	n	n n	n n	n n	n	n n	n n	n n
15	31-40	m !	y y	n n	y y	n n	y y	n n	n n	n n	n n	y y	y y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
15		m r	y y	n n	y y	n n	y y	n n	n Y	n Y	y n	n Y	y y	n n	n	n	n n	n n	n n	n	n n	n n	n n
State   Stat	31-40 41-50	m r	y y	n n	y y	y n	n n	n n Y	n n	n y	n n	n n	n n	n n	n	n n	n n	n n	n n		n n	n n	n n
No.	41-50 41-50	m !	y y	n n	y y	y y	n n	n Y	n n	n Y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
Column	41-50	r m	y y	n n	y y	y n	n Y	n n	n n	n	n Y	n n	n y	n	n	n	n n	n n	n n	n	n n	n n	n
14	41-50	m f m	y y	n n	y y	n v	n Y Y	n n	n n	n n	n Y n	n Y	n Y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
No.	41-50 41-50		y y	n n	y y	n n	y y	n n	y y	Y	n n	y n	y n	n	n	n y	n Y	n n	n n	n	n	n n	n n
14   15   15   15   15   15   15   15	41-50		y y	n n	y y	n n	y y	n Y	y y	y y	n Y	y n	y y	n n	n	n n	n n	n n	n n	n	n n	n n	n n
100   100	41-50	m r	y y	n n	y y	n n	y y	n y	y V	y v	n n	0	n n	n n	n n	n	n n	n n	n n	n n	n n	0	n n
100   100	41-50	m	y y	n n	y y	y y	y y	n Y	n Y	n Y	n n	y n	y n	n n	n	n	n n	n n	n n	n	n	n n	n n
	41-50	r m	y y	n n	y y	n n	y y	n n	Y Y	y y	n n	n n	n n	n n	n n	n	n n	n n	n n		n n	n n	n n
Column   C	41-50	m r	y y	n n	y y	n v	y y	n n	n n	n n	n n	n Y	n y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
Color   Colo		r m	y y	n n	y y	n n	y y	y n	n Y	y y	y n	n n	y n	n	n	n	n n	n n	n n	n	n n	n n	n n
Color   Colo	51-60 51-60		y y	n n	y y	y n	n Y	n n	n Y	n Y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
Column   C	51-60 51-60	r m	y y	n n	y y	n y	n v	n n	n v	n v	n n	n n	n n	n n	n n	n n	0	n n	n n	n n	n n	n n	n n
Color   Colo	51-60		y y	n n	y y	n n	y y	y y	n n	y Y	n n	n n	n n	n	n n	n n	n n	n n	n n	n n	n n	n n	n n
Color   Colo	51-60 51-60	r m	y y	n n	y y	n n	y y	n n	n Y	y n y	n n	y n	y n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
Martin   M	51-60 51-60 51-60	m r	y y	n n	y y	n n	n Y	n n	n n	n n	n Y	n y	n V	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
Martin   M	51-60		y y	n n	y y	n n	y y	n Y	y n	y y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
No.		m r	y y	n n	y y	n n	y y y	n n	n y	n y	n Y	n n	y y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
No.	61-70 61-70	m r	y y	n n	y y	n n	y v	n n	y y	y y	n n	n Y	n Y	n n	n n	n Y	n n	n n	n n	n n	n n	n n	n Y
2.50   F	61-70	m f	y y	n n	y y	n y	y n	n n	y n	y n	n n	n n	n n	n n	n n	n	n n	n n	n n	n n	n n	n n	n n
130		m r	y y	n n	y y	y y	n Y Y	n n	n Y Y	n Y Y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
130	61-70	m m	y y	n n	y y	n Y	y n	n n	y n	y n	n n	n n	n n	n n	n	n	n n	n n	n n	n	n n	n n	n
\$\frac{1}{2}\frac{1}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac	61-70	m r	y y	n n	y y	y n	n y	n n	n n	n n	n n	n n	n y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
1	61-70 61-70	r m	y y	n n	y y	n v	y n	n n	n n	n n	y n	n n	n v	n n	n n	n	n n	n n	n n	n n	n n	n n	n n
130   Y	61-70	m r	y y	n n	y y	y n	y y	n n	 У У	y n	n n	n Y	n Y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
\$\frac{1}{2}\frac{1}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac{1}{2}\frac	61-70 61-70	r r	y y	n n	y y	n n	y y	n n	n n	n n	n n	y y	y y	n n	n n	n n	0	n n	n n	n n	n n	n n	n n
1	61-70	r m	y y	n n	y y	n n	y y	y n	n y	y y	n n	n Y	n Y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
\$\frac{1}{2}\triangle \text{F} & \text{V} & \text{a} &	61-70 61-70	r r m	y y	n n	y y	n n y	y n	n y n	n n	y y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
\$2.70   f   y		m 	y y	n n	y y	y y	n n	n n	n n	n	n n	n	n n	n	n n	n n	n n	n n	n n	n n	n n	n n	n n
2-20 m y n n y y y n n y y n n n n n n n n n	61-70 61-70		y y	n n	y y	n n	y y	n n	n n	n n	n n	y y	Y Y	n n	n n	n n	n n	n n	n n	n n	n n	n n	n n
2-20 m y n n y y y n n y y n n n n n n n n n	61-70 61-70	m r	y y	n n	y y	n y	n v	n n	n n	n n	n n	n n	n n	n n	n n	y n	n n	n n	n n	n n	n n	n n	n n
7.50   f   V   0   V   V   V   N   0   0   0   0   0   0   0   0   0	71-80 71-80	m r	y y	n n	y y	n n	y y	y n	n n	y n	n n	n n	n n	n n	n n	n Y	n n	n Y	n n	n n	n n	n n	n n
	/1-80 71-80	m	y y	n n	y y	n n	y Y	y y	n	Y	n Y	n	n Y	n	n	y Y	n	n n	n	n Y	n	n	n n



Valid	n	102.00	100.00	100.00	100.00									
Ift SCA an														
		Frequency	Percent	Valid Percent	Cumulative I	Percent								
Valid	n	101.00	99.02	99.02	99.02									
	y	1.00												
	Total	102.00	100.00	100.00										
age range	- patients with (n=	102) and without aneurysms (n=71												
		Frequency	Percent	Valid Percent	Cumulative	Percent								
Valid	15-20	3.00	1.70	1.70										
	21-30	7.00	4.00	4.00										
	31-40	8	4.6	4.6										
	41-50	27	15.6	15.6										
	51-60	36	20.8											
	61-70	51	29.5	29.5										
	71-80	27	15.6	15.6										
	81-100	14	8.1	8.1										
	Total	173	100	100										
Sex														
		Frequency	Percent	Valid Percent	Cumulative I	Percent								
Valid	f	90	52	52										
	m	83	48	48										
	Total	173	100	100										
	1			1	1									



Supplementary File 4: The prevalence (%) of cerebral aneurysms observed over a broad age range (18-100 years, n=173, cases with aneurysms=102, cases without aneurysm=71), with a median of 62 years, a mean of 60 years, and a standard deviation of 15.75, is shown in the chart. The peak prevalence occurs between 31-60 years (p<0.001).