

## Subarachnoid Hemorrhage as a Cause of Death in Japan

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*Summary.* Japan is more prone to subarachnoid hemorrhage than other nations, with an estimated annual death rate of 25 out of 100 000 population (in white races 20). Subarachnoid hemorrhage constitutes 6.6% of all sudden death cases of Tokyo, a far greater percentage than that of New York (4.7%) and London (4.3%). Women are more liable to subarachnoid hemorrhage, while men are more liable to intracerebral hemorrhage. Problems of causality between contingent trauma and ruptured aneurysm were discussed from medical as well as judicial point of view.

*Zusammenfassung.* Vorgelegt wird eine statistische und kasuistische Studie über Subarachnoidalblutungen in Japan. Die Mortalität betrug im Jahre 1970 25 zu 100 000 Einwohner; sie ist höher als in der Weißen (20 auf 100 000). 6,6% der plötzlichen Todesfälle in Tokio wurden verursacht durch eine Subarachnoidalblutung; in New York waren es 4,7%, in London 4,3%. Bei Frauen überwiegt die Subarachnoidalblutung, bei Männern die intracerebrale Blutung. Das Problem der Kausalität zwischen einem Trauma und der Ruptur eines Aneurysma wurde besprochen, sowohl im medizinischen als auch im juristischen Sinne.

*Key words:* Mortality — Sudden death — Causality, between trauma and rupture of aneurysm — Subarachnoid hemorrhage.

Spontaneous subarachnoid hemorrhage is a very common and therefore important cause of natural death for the Japanese. Table 1 represents some data from the vital statistics [1] on the movement of the Japanese population, which shows that 181 104 persons died because of cerebrovascular diseases in 1970, this constituting 25.4% of a total of 712 703 annual deaths, and ranks first, followed by 119 881 deaths from malignant neoplasm and 89 039 deaths caused by cardiovascular diseases. Among these 181 104 deaths from cerebrovascular diseases, there were 85 408 deaths from intracerebral hemorrhage and 58 970 deaths from cerebral thrombosis-embolism (Table 2). This statistics is based upon records of death-certificates issued by medical practitioners, and therefore may contain some inaccuracy resulting from the not always correct diagnosis for diseases made by them. According to the analytical transaction of autopsy protocols of 76 reliable pathological institutions by Katsuki *et al.* [2], 878 cases which were clinically

Table 1. Main natural causes of death in Japanese (1970)

Cerebrovascular disease	181 104	(25.4%)
Malignant neoplasm	119 881	(16.8%)
Cardio-vascular disease	89 039	(12.5%)
...		
Total annual deaths	712 703	(100.0%)

Table 2. Cerebrovascular disease as a natural cause of death in Japanese (1970)

	Male	Female	Total
Intracerebral hemorrhage	46782 48.3%	38626 45.8%	85408 47.2%
Cerebral thrombosis-embolism	30586 31.6%	28384 33.7%	58970 32.5%
Other cerebrovascular diseases	19403 20.1%	17323 20.5%	36726 20.3%
Total	96771 100.0%	84333 100.0%	181104 100.0%

Table 3. Validity of clinical diagnosis in 1744 cases of cerebrovascular disease

Anatomical diagnosis	Clinical diagnosis				
	intracerebral hemorrhage	cerebral thrombosis-embolism	subarach. hemorrhage	miscellaneous	total
Intracerebral hemorrhage	613	35	50	95	793 45.5%
Cerebral thrombosis-embolism	192	318	11	187	708 40.6%
Subarachnoid hemorrhage	73	5	138	27	243 13.9%
Total	878	358	199	309	1744 100.0%

diagnosed as intracerebral hemorrhage contained 192 cerebral thrombosis-embolism and 73 subarachnoid hemorrhage cases. Similar differences in other groups of diseases did also exist; 358 cases which were clinically diagnosed as cerebral thrombosis-embolism contained 35 intracerebral hemorrhage and 5 subarachnoid hemorrhage cases, and 199 cases, clinically diagnosed as subarachnoid hemorrhage, contained 50 intracerebral hemorrhage and 11 cerebral thrombosis-embolism cases (Table 3). From the above mentioned we can deduce an actual incidence of deaths from intracerebral hemorrhage of 793 cases or 45.5% of all cerebrovascular diseases, and that of cerebral thrombosis-embolism and that of subarachnoid hemorrhage being 708 cases or 40.6% and 243 cases or 13.9%, respectively. This corresponds with 82402 annual deaths resulting from intracerebral hemorrhage; 73529 annual deaths from cerebral thrombosis-embolism and 25173 annual deaths from subarachnoid hemorrhage (Table 4). In other words, the annual death rate per 100000 population for these 3 diseases is 82, 74 and 25 respectively. When comparing these figures with those of Kurtzke [3], who had estimated the same for the white races, the Japanese seem to be far more liable to intracerebral hemorrhage, slightly more to subarachnoid hemorrhage and less to cerebral thrombosis-embolism than Caucasians.

Table 4. Corrected incidence and mortality for types of cerebrovascular disease

Type of cerebrovascular disease	Corrected incidence		Estimated annual death rate per 100000 population	
	Japanese	(whites* for ref.)	Japanese	(whites* for ref.)
Intracerebral hemorrhage	46%	(16%)	82	(40)
Cerebral thrombosis-embolism	41%	(62%)	74	(120)
Subarachnoid hemorrhage	14%	(12%)	25	(20)
Annual total deaths	181104		181	(180)

\* Ref. [3].

Table 5. Sudden death from natural causes

	Tokyo	New York	Wien and Graz	London
Cardio-vascular	47.9%	44.9%	41.8%	73%
Cerebral	17.8%	17.9%	8.9%	12%
Respiratory	15.1%	23.1%	23.2%	8%
Digestive-urogenital	5.3%	9.7%	13.0%	1%
Miscellaneous	13.9%	4.4%	13.1%	6%
Total deaths examined	1385 (100%)	2030 (100%)	2668 (100%)	1029 (100%)

Table 6. Incidence of subarachnoid and intracerebral hemorrhage in sudden death

	Tokyo	New York	Wien and Graz	London
Subarachnoid hemorrhage	37.3%	25.7%	29.1%	29.7%
Intracerebral hemorrhage	50.6%	36.4%	56.1%	46.9%
Other cerebrovascular	12.1%	37.9%	14.8%	23.4%
Total deaths examined	247 (100%)	367 (100%)	237 (100%)	111 (100%)

Subarachnoid hemorrhage constitutes also an important cause in sudden and unexpected natural death, as this has until now been widely discussed by many authors. Table 5 shows the summary of 1385 autopsied cases of sudden natural deaths by the Tokyo Medical Examiners' Office in 1970 [4]. The results are grouped according to causes, which were found to approximate those reported previously by Helpert *et al.* [5] for New Yorkers, by Weyrich [6] for the inhabitants of Wien and Graz, and Simpson [7] for Londoners (Table 5). Out of 247 cases of cerebrovascular causes of sudden death in Tokyo, however, there were so many deaths as 92 cases from subarachnoid hemorrhage, representing 37.3% of this group or 6.6% of all sudden deaths (Table 6). This figure coincides almost with that of Weyrich, but is far greater than that of Helpert *et al.* (4.7%) and that of Simpson (4.3%) (Table 7).

Sugai [9], one of the authors, has stated, summarizing his thirteen years experience in the Tokyo Medical Examiners' Office, that, out of 679 cases of sub-

Table 7. Frequency of subarachnoid hemorrhage in sudden death

Tokyo	New York	Wien and Graz	London
6.6%	4.7%	6.2%	4.3%

Table 8. Intracerebral and subarachnoid hemorrhage as a cause of sudden death in both sexes

	Male	Female	Male + Female
Subarachnoid hemorrhage	332 (30.9%)	347 (59.3%)	679
Intracerebral hemorrhage	744 (69.1%)	238 (40.7%)	982
Total	1076 (100.0%)	585 (100.0%)	1661

Table 9. Age distribution of subarachnoid hemorrhage

Age	Male	Female	Total
19	9	2	11
20—29	20	4	24
30—39	30	25	55
40—49	100	71	171
50—59	100	106	206
60—69	58	98	156
70—79	15	36	51
80—	0	5	5
Total	332	347	679

arachnoid hemorrhage, 332 cases were men, and 347 cases were women, showing a slight preponderance in women. However, since women made up only 30% (in 1970, 4608 male and 1755 female bodies were examined) of the total number of medical examiner's case and of the natural deaths, this shows a definite evidence of the predominance of subarachnoid hemorrhage in women. If we compare two causes of sudden death, subarachnoid hemorrhage and intracerebral hemorrhage, in both sexes, it is easily understandable that men are more liable to intracerebral hemorrhage, and women, in contrary to men, are more liable to subarachnoid hemorrhage (Table 8).

Although it was up to now generally believed, that subarachnoid hemorrhage was predominantly a disease effecting young persons, there is actually a wide age distribution of this condition, ranging between 15 to 85 years, 11 in the first decade, 24 in the second, 55 in the third, 171 in the fourth, 206 in the fifth, 156 in the sixth, 51 in the seventh and 5 in the eighth (Table 9). Thus, fatal subarachnoid hemorrhage is most common in adult life and middle age. This data fairly coincides with that of Helpert *et al.* [5].

In the majority of cases, rupture of an aneurysm was that of an artery of the anterior portion of the circle of Willis. Table 10 shows the distribution of aneurysms found in our series of observation. Most of them were situated at the bifurcation of the large branches of the circle of Willis and four-fifth of them were discovered on the anterior half of the circle of Willis.

Table 10. Distribution of cerebral aneurysm

Arteries	Male	Female	Total	
Anterior cerebral	10	9	19	
Anterior communicating	100	60	160	
Internal carotid	23	51	74	
Middle cerebral	45	60	105	
Posterior communicating	4	8	12	370 (80%)
Posterior cerebral	5	1	6	
Basilar	13	33	46	
Vertebral	13	13	26	
Superior cerebellar	1	2	3	
Inferior anterior cerebellar	0	1	1	
Inferior cerebellar	3	5	8	90 (20%)
Total	217 (47.2%)	243 (52.8%)	460 (100%)	

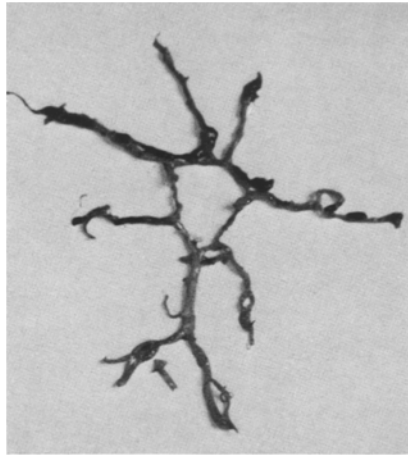


Fig. 1. Case 1. — Male 50. Stereomicroscopic appearance of a circle of Willis. Ruptured aneurysm at main bifurcation of the left vertebral artery is clearly shown (→)

Not a few cases of subarachnoid hemorrhage were sent to us for investigation, because of their unnatural mode of death; too sudden outbreak of the catastrophe and very often under the suspicion of murder. A man, aged 50 (case 1), a company chief, was found unconscious and he soon died in his office room, with some marks of physical violence on his forehead. Autopsy revealed, however, a massive subarachnoid hemorrhage arising from a ruptured aneurysm of the left vertebral artery (Fig. 1). The excoriation-bruises on his forehead was supposed to be caused by falling on the floor in an attack of unconsciousness.

The next example, a 52-year-old woman (case 2), whose history of trauma was not evident, was found unconscious and died 9 hrs later. Examination disclosed a ruptured aneurysm in the right middle cerebral artery. Fig. 2 shows the ruptured aneurysmal sac, the media of the parent artery ends abruptly at the entrance of the sac, resembling the edge of a medial defect, the intima is of irregular thickness; near the entrance, plaque-like structures simulating intimal pads or cushions, and (Fig. 3) it attenuates at the near site of the rupture, where it is accompanied by massive round cell infiltration.

Aneurysms may, during their development, embed themselves in the adjacent cerebral substance. Their subsequent rupture may give rise to an intracerebral hemorrhage, simulating



Fig. 2

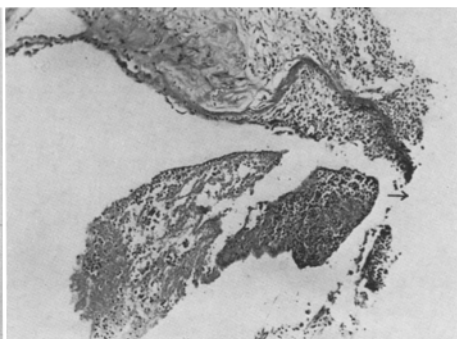


Fig. 3

Fig. 2. Case 2. — Female 52. Section through the wall of a saccular aneurysm in the right middle cerebral artery shows the abrupt cessation of the muscular layer at mouth of sac (bottom, middle), the atheromatous formation (top), and a massive round cell infiltration at the near site of the rupture (right, middle). (Hematoxylin-eosin,  $\times 40$ )

Fig. 3. Section through the site of the rupture ( $\rightarrow$ ) of the same aneurysmal sac as in Fig. 2. A massive round cell infiltration near the site of the rupture and a small thrombus in the sac (bottom half). (Hematoxylin-eosin,  $\times 100$ )

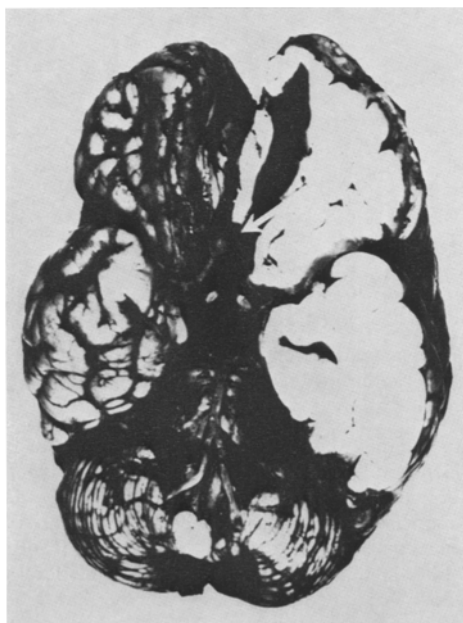


Fig. 4. Case 3. — Male 33. A lower part of the left frontal (and temporal) lobe is cut off to show a hematoma inside the lobe, which arose from the rupture of the embedded aneurysm of the anterior communicating artery ( $\rightarrow$ )

primary intracerebral hemorrhage. Examples of such cases are as follows: A taxi-driver, aged 33 (case 3), hit his occiput against the door of his own car, while his car was on a stony, uneven road. He retained consciousness, but complained, thereafter, severe headache, vertigo and

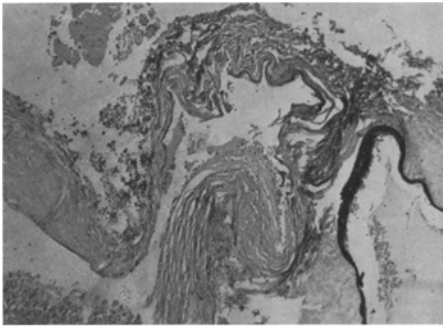


Fig. 5

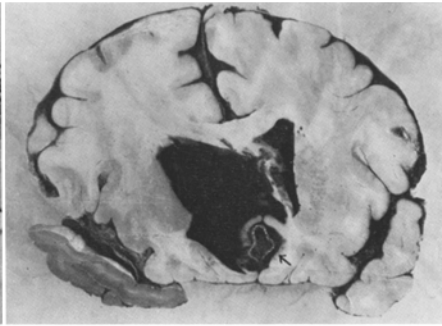


Fig. 6

Fig. 5. Section through the aneurysmal sac of case 3. The elastica lamina of the wall shows fragmentation, deep staining, and diffuse swelling. Outside the sac (to the top) a massive round cell infiltration and inside the sac (to the left bottom) a thrombus. (Elastica stain,  $\times 40$ )

Fig. 6. Case 4. — Male 64. Frontal section of the brain shows a large ruptured aneurysm which arose from the anterior communicating artery ( $\rightarrow$ ), and a large intracerebral hemorrhage in the right frontal lobe which extended into the right lateral ventricle

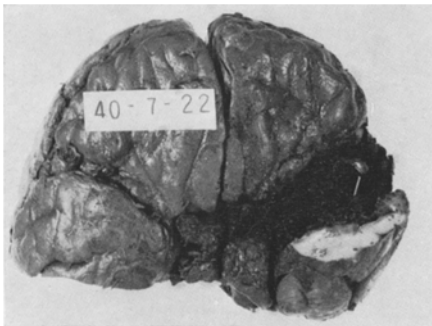


Fig. 7

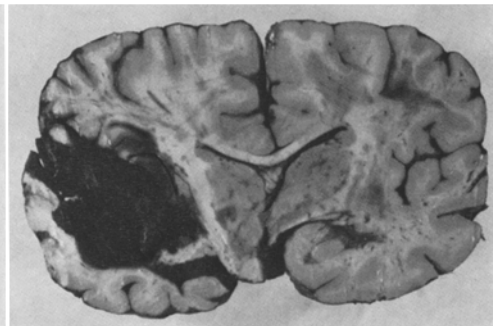


Fig. 8

Fig. 7. Case 5. — Male 48. Died 12 days after he had sustained a slight blow on his left temporal head. At autopsy, a ruptured aneurysm ( $\rightarrow$ ) from the left middle cerebral artery was found surrounded by massive clotted blood deep in the Sylvian fissure

Fig. 8. Frontal section of the brain of case 5. A large intracerebral hemorrhage arose from the rupture of the embedded aneurysm of the left middle cerebral artery

ringing of the ears. Four days later he died in hospital. Autopsy revealed a massive subarachnoid hemorrhage of the base, particularly abundant at the anterior part and a hematoma inside the left frontal lobe, which communicated with each other (Fig. 4). The bleeding originated from a rupture of a saccular aneurysm with a diameter of 5 mm, which arose from the anterior communicating artery ( $\nearrow$ ) and was then embedded in the brain substance. The fundus of the sac (Fig. 5) contained thrombus, half organised and with massive round cell infiltration outside the sac.

The following case was almost the same. Victim is a 64-year-old man (case 4), who died 10 days after having had a head injury in a traffic accident. The autopsy revealed a large intracerebral hemorrhage in the right frontal lobe, with extension into the lateral ventricle, followed by spread into subarachnoid space (Fig. 6). This was caused by a rupture of embedded aneurysm of an anterior communicating artery ( $\nearrow$ ).

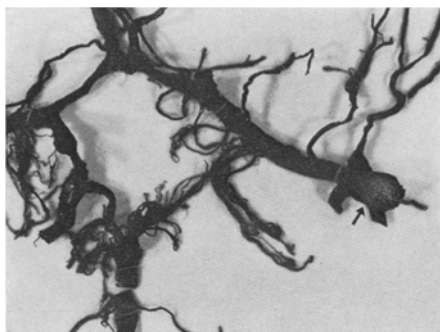


Fig. 9. Stereomicroscopic appearance of the circle of Willis in case 5. A ruptured aneurysmal sac is clearly shown (→)

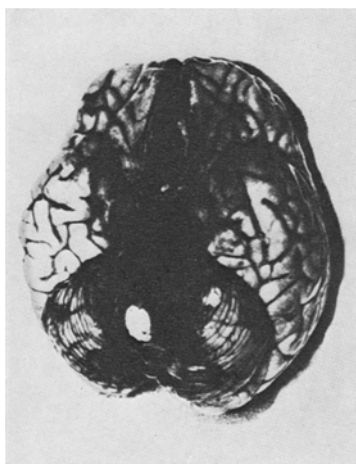


Fig. 10

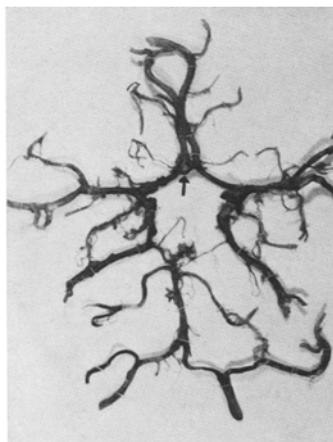


Fig. 11

Fig. 10. Case 6. — Female 39. A massive subarachnoid hemorrhage at the base of the brain of a woman who died 10 min after she had sustained a slight blow on her temporal head. A ruptured aneurysm was found in the anterior communicating artery (see Fig. 11)

Fig. 11. Stereomicroscopic appearance of the circle of Willis in case 6. Ruptured aneurysm at the anterior communicating artery (→)

The third case of embedded aneurysm deals with a 48-year-old man (case 5), who died 12 days after he had sustained a slight blow on his left temporal head by his comrade while drinking in a bar. For 10 days he felt nothing unusual, then he suddenly fell into coma with marked signs of elevated intracranial pressure. The doctor suspected a subdural hematoma and a surgical operation was performed without finding it. Twenty-four hrs after the operation he passed away. The autopsy undertaken by us disclosed the cause of death being a rupture of left middle cerebral artery in a portion far deep in the Sylvian fissure (Figs. 7—9).

Antecedent trauma is, very often, invoked as having precipitated the onset of the disease, and to make the matter worse, as having made the direct cause of the bleeding in the cranium, and an unlucky person who has by chance had an altercation with the deceased may be sent to jail on a charge of murder.

A 39-year-old woman (case 6) was fisted by her husband on the temporal head and 10 min later she was dead. Autopsy revealed an intrascapal hemorrhage on the temporal region of the head, and a massive subarachnoid hemorrhage on the base of the brain (Fig. 10). A ruptured



small aneurysm, diameter 3 mm, was found in the anterior communicating artery (Fig. 11). Upon this finding, the man was not prosecuted. The contrary to this case, the issue in the following similar case was quite adverse. The incident happened outside Tokyo. A doctor who performed the autopsy of the woman, 33 years old, found a massive subarachnoid hemorrhage at the base of the brain and ascribed her death to a blow by her husband. When the case was brought into a legal proceeding at the local criminal court, the judge ordered Ueno, one of the authors, to criticize the conclusion made by the above-mentioned doctor through investigation of the autopsy protocol and several photos taken by a policeman at the autopsy room. His conclusion was that the hemorrhage was the result of a rupture of a preexisted aneurysm located somewhere in the circle of Willis, and therefore the accused should be released. The court, however, although having admitted that the stroke was a mild one, found him guilty of his wife's death in arousing in her an emotional stress which could have precipitated the rupture of the aneurysm and sentenced the accused to 1 year imprisonment with a probation of 2 years. This case is a good example of a Japanese tradition, "Once prosecuted, difficult to be released".

### Discussion

Above described cases show how essential it is to search for aneurysm as the source of hemorrhage and in our opinion the best can be done by stereomicroscopic

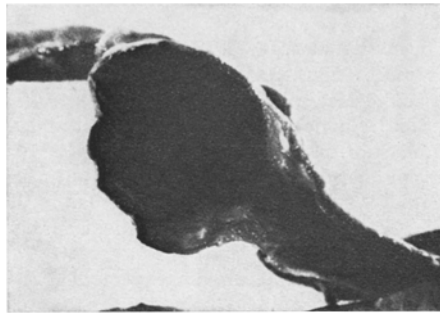


Fig. 12. Case 7. — Male 46. Magnified, stereomicroscopic view of a ruptured aneurysm with a diameter of 8 mm

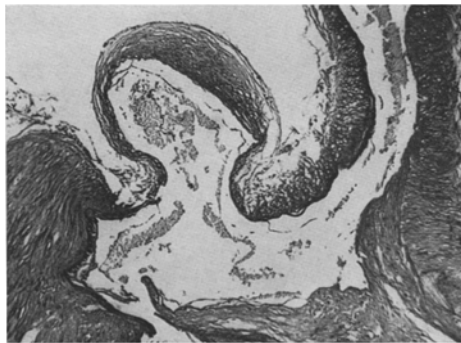


Fig. 13. Case 8. — Female 42. Longitudinal section through the bifurcation with a minute aneurysm (diameter 1.5 mm) on the distal carina between the branches of the left basilar artery. The muscular layer of one branch (left) and that of the other branch (right) end abruptly at the entrance of the sac. The intima cushion (bottom, right) is seen on the proximal carina at the angle between the right branch and the main trunk

examination of the exposed arteries of the circle of Willis on a fresh, unfixed brain after stripping the meninges and by taking away the clotted blood surrounding the artery. Fig. 12 shows one example of a stereomicroscopic photo taken at low magnification. A ruptured aneurysmal sac (diameter 8 mm) of the anterior communicating artery in a 46-year-old man (case 7) is clearly demonstrated. It can be pronounced with certainty that, if one is persevering enough to sit down at the searching desk for more than a couple of hours successive to the autopsy, he will be able to discover the source of the hemorrhage at least in seven out of ten. Sugai, one of the authors, boasts of his skill, by which he is able to discover them by this method in ten out of ten. Fig. 13 shows a minute aneurysm, diameter 1.5 mm, which was disclosed by Sugai in the left basilar artery of a 42-year-old woman (case 8), and which was the only source of the fatal subarachnoid hemorrhage. Such a small aneurysm will ordinarily be overlooked at an autopsy and a foul play may start at that point.

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